STUDY OF VRANA SHODHAN AND ROPAN KARMA OF NIMBADI KALKA ON CHRONIC DIABETIC ULCER

A THESIS

SUBMITTED TO THE
TILAK MAHARASHTRA VIDYAPEETH PUNE

FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

IN DRAVYAGUNA SUBJECT
UNDER THE BOARD OF AYURVED STUDIES

BY

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UNDER THE GUIDANCE OF

Dr. MRS. MEENAL LAD

DEPARTMENT OF AYURVED

January 2018

CERTIFICATE OF THE SUPERVISOR

It is certified that work entitled Study of vranashodhan and ropan karma of nimbadi kalka on chronic diabetic ulcer is an original research work done by Dr. Satish Baburao Bandgar. Under my supervision for the degree of Doctor of Philosophy (Ph.D) in Dravyaguna subject to be awarded by Tilak Maharashtra Vidyapeeth, Pune. To best of my knowledge this thesis embodies the work of candidate himself has duly been completed fulfills the requirement of the ordinance related to Ph. D. degree of the TMV up to the standard in respect of both content and language for being referred to the examiner.

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Undertaking

I Dr Satish Baburao Bandgar is the Ph. D. Scholar of the Tilak Maharashtra Vidyapeeth in Dravyaguna subject. Thesis entitled Study of vrana shodhan and ropan karma of nimbadi kalka on chronic diabetic ulcer under the supervision of Dr Mrs. Prof. Meenal Lad. Solemnly affirm that the thesis submitted by me is my own work. I have not copied it from any source. I have gone through extensive review of literature of the related published / unpublished research works and the use of such references made has been acknowledged in my thesis. The title and the content of research is original. I understand that, in case of any complaint especially plagiarism, regarding my Ph.D. research from any party, I have to go through the enquiry procedure as decided by the Vidyapeeth at any point of time. I understand that, if my Ph.D. thesis (or part of it) is found duplicate at any point of time, my research degree will be withdrawn and in such circumstances, I will be solely responsible and liable for any consequences arises thereby. I will not hold the TMV, Pune responsible and liable in any case. I have signed the above undertaking after reading carefully and knowing all the aspects there in.

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Respected Sir,

I the under signed have been enrolled under board of Ayurved studies Tilak Maharashtra Vidyapeeth Pune for Ph.D. in Dravyaguna subject.
I am submitting my thesis for Ph.D. degree titled Study of vranashodhan and ropan karma of nimbadi kalka on chronic diabetic ulcer for your further suggestion and instruction.
Thanking you,

Yours faithfully,

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Abstract

Chronic Diabetic Ulcers are one of the major health problems. Chronic Ulcers can be very well interpreted with Dushta Vrana concept in Ayurved. In Ayurveda various drugs formulations are in use for effective Chronic Ulcer management. Need is to evaluate these hypothesis and drugs with modern research methodology.

Hypothesis: Nimbadikalka formulation mentioned in Ayurved text Shrangadhar Samhita and Bhaishajya Ratanavali has both Vrana Shodhan and Ropan (i.e.Debridement and healing) properties in chronic Diabetic ulcer.

Objectives: Evidence based clinical comparison for local application of Nimbadikalka formulations, mentioned in Ayurved and modern medicine therapy for chronic Diabetic ulcer.

Research Methodology: In this study total 260 patients of Chronic Diabetic Ulcers are voluntary divided in to two equal groups. The group A, patients were treated with Nimbadi Kalka as local dressing material. In group B, conventional modern science local dressing material. In group B, conventional modern science local dressing materials like Iodine solution or Hydrogen peroxide (H_2O_2) or Eusol solution are used groups. The oral hypoglycemic agents of modern science are same for both groups. The maximum treatment period is 60 days with daily dressing. The healing assessments are done as per clinical protocol for Ulcer healing.

Results: Chi-square test P value results non- significant which indicate that both group are equally effective So, we used single proportionate test on pulled data as small frequency in major amputation ,minor amputation and deth categories patient's categories here. After that test is significant so we can conclude that both A and B treatment groups are 83.46% effective. Total 16.53% cases are non-healable with A or B treatment. The control group B is already accepted and proven treatment protocol for chronic Ulcer in the modern medicine.

Conclusion: The tropical application of Nimbadi Kalka is effective in Chronic Diabetic ulcers. Both Ayurvedic and Modern medicine are equally effective in chronic diabetic ulcer management.

Keywords: Vrana Shodhan Ropan, Nimbadikalka, Chronic Ulcers, Diabetic Ulcers.

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ABBRIVATIONS

A. H.	:	Ashtang Hriday
A.S	:	Ashtang Samgraha
B. P.	:	Charak Samhita
Ch. Su	:	Charak Sutrasthan
Ch. Ni	:	Charak Nidansthan
Ch. Chi	:	Charak chikitsastan
Ch .Ind	:	Charak Indriyasthan
Ka. Sam	:	Kashyap Samhita
M .Ni	:	Madhav Nidan
Sh .S	:	Sharangdhar Samhita
Su. Su	:	Sushruta Sutrasthan
Su.Ni.	:	Suahruta Nidansthan
Su. Chi	:	Sushruta.Chikitsasthan
Ah. Ni.	:	Ashtang Hrudya Nidansthan
Utt.	:	Uttaratantra
Y. R.	:	Yoga Ratnakar
D. M.	:	Diabetes Mellitus
A. N.	:	Ashtang Nighantu
D. N.	:	Dhanvantari Nighantu
S.N.	:	Sodhal Nighantu
M. N	:	Madhav Nidan
R. N	:	Raj Nighantu
K. N	:	Kaiydev Nighantu
B.N.	:	Bhavprakash Nighantu
N. A.	:	Nighantu Adarash
D. M.	:	Diabetes Mellitus

IDDM.	:	Insulin Dependent Diabetes mellitus
NIDDM.	:	Non Insulin Dependent Diabetes mellitus
HDL	:	High Density lipoprotein
LDL	:	Low Density lipoprotein
FFA	:	Free fatty acid
D.C.	:	Differential Count
E.S.R.	:	Erythrocyte Sedimentation rate
K.F.T.	:	Kidney Function Test
L. F.T.	:	Liver Function Test
R.S	:	Respiratory System
CVS	:	Cardiovascular System
CNS	:	Central Nervous System
PA	:	Per Abdomen
E.C.G.	:	Electro cardio gram
OGTT	:	Oral glucose tolerance test
PGDF	:	Platelet derived Growth count
B. C.	:	Before Christa
A. D.	:	Anno Domini
+	:	Same me as per index

Chapter 1. Introduction

The study to determine the global prevalence of diabetes in all age-groups all over world shows, 285 million people in the year of 2010. This figure will raise upto 438 million in 2030. The rate of urbanization among developing countries will raise upto two times till 2000 - 2030. [1]

One of the major complications of DM is foot ulceration. It is a major social, medical, and economic burden on people. However, the rate of foot ulceration and amputation differs significantly. The study from India, for the prevalence of DM shows that the chances of infection in ulcer is 6-11% and occurance of amputation was 3% in type 2 diabetic patients. In another study from India, shows the incidence of diabetic foot ulcers in the clinic population was 3.6%. The causes of ulceration among Indian people are barefoot walking, religious faith like walking on fire, use of improper footwear and lack of awareness and negligence about foot-care. This increase prevalence of foot ulceration and its complications in DM type II patients. This study also reflects increasing incidences for infection on DM patient's ulcers upto 52%.[1]

The global burden for amputation among DM foot ulcer patient is million per year. That means at every 30 seconds a limb is amputed in the world.[2]

The Indian Study shows that the prevalence of amputation in DM patient is nearly about 40,000 per every year with increasing numbers.[3]

In United states study shows, that the rough estimated cost of ulcer healing is $ 8,000 that of an infected ulcer healing is $ 17,000 and that of a major amputation is $ 45,000.In the United States, Nearly about 80,000 amputations are performed per year for diabetic foot ulcer, and the possibility of ulceration with infection in another leg in this patient is 50% within 18 months.Among these 58% causes have possibility of reamputation in 5 year from first amputation.The mortality rate for reamputed case is 20-50%. Despite of much research and development in ulcer and DM management, this ratio is static since last 30 yrs. [4]

The study for expenses in hospitalized patients of DM II with complications says the hospital stay is longer in such cases.

The hospitalized patient's expenses for ulcer (19020 INR) with any two Diabetic complications (17633 INR) spent four times more. The patients having daiabetic complication like Nephropathy (12690 INR), cardiovascular complication

(13135 INR) and retinopathy complications (13922 INR) pays three times more expenses than the patients without any complications (4493 INR).

The hospital expenses in the last two years are high and if complication of DM II it will be highest.[5]

The early intervention of diagnosis and treatment in DM Ulcer can reduce mortality rate and economical burden also[6]. The evidances also indicates that the multidisplinary and active management in foot ulceration can reduces its possibility of amputation.[7, 8].

Even though of various modern techniques and antibiotics development, chronic non-healing ulcers continue to pose a challenge to physician. The Multidrug resistance to antibiotic is also increasing.[9]

- Need of study:

As the last decade creates tremendous interest in Ayurvedic science. The demand of herbal medicine in world market is also increasing. Now a day's it is necessary to create evidences to basic principles mentioned in Ayurvedic texts, for its worldwide acceptance.To achieve above stated goal, we must reopen the Ayurvedic texts and search for appropriate medicines for a particular disease; and evaluate these hypothesis and drugs with modern research methodology.

- Selection of drug:

In Ayurveda Samhita, various drugs have been mentioned as Vranashodhak and Vranaropak which are helpful in Chronic Ulcer healing. In this study, the drugs were selected on following criteria.

- Easily available.
- Less adultery in market.
- Any person should be able to prepare and use the medicine easily.
- Non-toxic.
- Drug having both Vranashodhak and Ropak activity is selected because all chronic Ulcers are infected and Non-healing.

निम्बपत्रघृतक्षौद्रदार्वीमधुकसंयुक्तो।

तिलैश्चसहसंयुक्तोलेपःशोधनरोपणाः॥

(शा.सं.तृ.खं.११/८६) [10]

निम्बपत्रतिलादंतीत्रिवृत्सैंधवमाक्षिकं।

दृष्टव्रणप्रशमनोलेपःशोधनरोपणाः॥

(शा.सं.तृ.खं.११/८८) [11]

तिलकल्कःसलवणोद्वेहरिद्रेत्रिवृत्तघृतम्।

मधुकंनिम्बपत्राणिप्रलेपोव्रणशोधनः॥

(च.चि.२५/८५) [12]

निम्बपत्रघृतक्षौद्रदार्वीमधुकसंयुता।

वर्तिस्तिलानांकल्कोवाशोधयेद्रोपयेद्व्रणानां॥

(भै.र.४७/४५) [13]

द्रव्यमार्द्रशिलापिष्टंशुष्कंवासजलंभवेत्।

प्रक्षेपावापकल्कास्तेतन्मात्रंकर्षसंमितम्॥१॥

कल्केमधुघृतंतैलम्देयम्द्विगुणमात्रया॥

सितागुडौसमौदद्यात्द्रवाःदेयाश्चतुर्गुणाः॥२॥

(शा.सं.द्वि.ख.५/1,2) [14]

So Nimbadi Kalka formulation mentioned by Sharangdhar Samhita III /11/86 and Bhaishajya Ratnavali, 47/45 was selected. This formulation fulfilled all above stated drug selection criteria.

In this study, Non-Healing Diabetes ulcers in group-A were treated with above formulation as a kalka for external application. The observed results were compared with the treatment group B, which were treated with modern medicine drugs for local Ulcer Dressing.

Chapter 2. Aim and Objectives

1. Aim:

- To evaluate Vrana Shodhan and Ropan Karma of Nimbadikalka on Diabetic II chronic ulcer.

2. Objectives:

- To create evidence base for hypothesis mentioned in ancient text of ayurved by modern research methodology.
- A clinical comparison between ayurved treatment group and modern medicine treatment group will be assessed by approved clinical study protocol.
- To study any untoward effect of Nimbadikalka on chronic ulcer.
- To prepare easily available and effective drug therapy in chronic Diabetic II ulcer.

3. Hypothesis:

Nimbadikalka has both Vrana Shodhan and Ropan properties in healing of chronic Diabetic II ulcer.

Chapter 3. Review of Literature

3.1. Review of vrana

3.1.1 Historical Review of Vrana

Ayurveda is a science of healthy life as well as diseases. Since ages, it is serving the health needs to society. Hence Shalyatantra is evloved as a special branch of Ayurveda for surgical treatments.

Ancient surgeons have done surgeries on the various diseases.They have treated from minor conditions like warts to major conditions like non-healing ulcers, plastic surgeries and grafts.The constant research for treatments of diseases are seen from Vedic period.Many references are available such as administration of Rohini Aushadi in Kshata and Vrana, and the sheetalajaladhaara to stop the bleeding in Sadhyovrana.[15]

A] Vedic review:

Here a discriptive information about body,diseases and treatments are avilable in Rigveda and Athrvaveda. In Rigveda**,** Mantra-chikitsa, Aushadhi chikitsa and Shalya chikitsa were at its highest peak.

Ashwinikumar are "Vaidyas of god". They learnt madhuvidya i. e trasplantation surgery and pravragyavidya i.e.plastic surgery from Acharya Dadichi.They are famous plastic surgoen and organ trasnplant surgeon.[16]

Vipashyala the daughter of king Khel lost her limbs in war. However Ashwinikumar transplanted her limbs with copper /iron Metal.[17]

1.Athrvaveda:

There are many references in Atharvaveda about Apachivedhan, removal of Garbhashay and Vranchikitsa,and ligation of artery to stop bleeding.[18] In Chandogyaupanishad a reference of Madhuvidya(transplantation Surgery) is available[19].

In Bruhdaaranyak and Shathpath brahmhan reference of Acharya Dadhichi is avilable. He was famous plastic surgeon and organ transplant surgeon.So Ashwinikumar transplanted head of horse to Dadhichi and Dadhich's head to horse**.**[20] Also references of Rakshoghana, Vishalyaghana, Kriminashan, Rohan and Sandhan dravyas are mentioned.[21]

2. Agnipuran : [22]

Surgical wounds are mentioned in Agnipuran.

3. Mahabharat :[22]

At the time of Kurukshetra yuddaha, in Bhismparva and Udyoga parva and in Anushasan parva various references regarding wound healing are mentioned. It is also stated that there were so many surgeons avilable for removal of Shalya.

4. Mahavagga (Buddhist tradition):[23]

Vrana with pooya (pus) were treated with medicine such as kshaar.The external application of such medicines causes explusion of pus.

5. Koutilya Arthashastra: [24]

In Koutilya arthashastra the reference of Dushta vrana is available. Also the other references of Yantra, Shastra, Agad, Sneha, and Vastra are available.[24]

6. Jatkamala:[22]

Dushta vranas which are painful along with pus should be carefully opened and drained.The wound becomes painful when it comes in contact with salt.[25]

7.Kaadambari:[26]

Wounds are produced by constant friction.Severe injury produces disablities in the oragans. After healing the wound the scar remains for whole life.

B] Samhita kaal:

A detailed description of vrana with management is mentioned in Brihattraye and Laghuttraye.Charaka has explained 36 therapeutic measures of Vrana in Dwivraniyachikitsa adhayay in Chikitsasthan.[27] Sushrutha has mentioned 60 therapeutic measures for vrana known as Shashti–upkrama in chikitsasthan.For Vrana chikitsa, he mentioned 1St adyaya of chikitsasthana, as Dwivranachikitsa.[28]

Information of vrana is also mentioned in Bhel-samhitha, Kashyapa samhita, Gada nigraha, Chakradatta, Yogaratnakara, and Bhaishajyaratnavali and in Madhavnidan also.

3.1.2. Review of vrana

Vrana is one of the important subject mentioned in Ayurveda. It is described in detail by Sushruta. About the Vrana various references are available in Samhitas.

A) Nirukti (Derivation):

'व्रण गात्र विचूर्णने।'[29]

A word Vrana is derived from the root Vriya meaning to recover.

Vran + a, in the sense of 'Gaatra Vichurnane'[30]

Vrana is a condition, even after complete healing it leaves a scar (vranavastu) over the area. The scar remains for whole life.

व्रण गात्र विचूर्णने। व्रणयति इति व्रण:। [29]

Gaatra (body tissue or part of body) Vichurnane means rupture or destruction or discontinuation.

The discontinuity or destruction of body tissue is known as Vrana.

B) Definition of Vrana:

वृणोतियस्माद्बूढेऽपिव्रणवस्तुननश्यति|

आदेहधारणात्तस्माद्व्रणइत्युच्यतेबुधैः||४०|| [31]

Su.Su. 21/40

Vrana is a condition which even after the complete healing leaves a scar over the area, which stays as long as the person is alive.

C) Specialties of Vrana:

षण्मूलोऽष्टपरिग्राहीपञ्चलक्षणलक्षितः|

षष्ट्याविधानैर्निर्दिष्टैश्चतुर्भिःसाध्यतेव्रणः||१३४|| [32]

Su.Chi.1/134

- Six roots of vrana i.e. mulsthaan (or causes of vitiation) -Vaata, Pitta, Kapha, Rakta, Sannipataj and Agantuja
- Eight sites or vranavastu or vranaadhistan–Twak, Mamsa, Sira, Snayu, Sandhi, Asthi, Kostha, Marama.
- Five features of examination (Panchalakshan) - shape, pain, colour, odour, and discharge.
- Sixty types of treatment (Shashatividhan) - i.e. vrana treated with Shasti upkramas.

- Can be treated successfully if the four necessities, i.e. Chikitsachatushpaad are ideal.

D) Classification:[33]

Vrana is classified into two categories : Nija Vrana and Aagantuj Vrana

I) Nija or Shareeraja Vrana:[34]

Sushruta has specially mentioned Nija Vranas occurs due to Vaataja, Pittaja, Kaphaja, Raktaja and Sannipataja Dosha..

These are further classified into 15 types on the basis of vitiation of dwi-doshas and tridoshas along with Rakta.

Charaka has described Nija Vrana has 3 types i.e.due to Vaata, Pitta and Kapha.

II) Aagantuja Vrana or Sadyovrana:

It is caused by external trauma from Purusha, Pashu, Pakshi, Vyaala, Prapatana, Peedana, Prahara, Teekshnaoushadha, Agni, Kshara, Visha, Kapaala, Shringa.

Sushruta has mentioned six types as Chinna, Bhinna, Viddha, Kshata, Picchita, Ghrishta.[35]

Ashtang hridaya mentioned eight types as Ghrishta, Avakruta, Vicchinna, Pravilambita, Paatita, Viddha, Bhinna, Vidalith.[36]

Ashtanga Sangraha mentiond three types as Chinna, Viddha, Picchita.Madhava Nidana has stated same as that of Sushruta.

Sharangdhara has mentioned eight types as Avkrlupta, vilambint, Chiina, Bhinna, Vidalita, Grushta, Viddha, Nipaatit.[37]

E) Nidana of Vrana :[38]

Doshaj ,Aaharaj and viharaj

1.Vata Dosha –

Aharaj hetu: Vaataprakopak aahaaras,i.e. Ruksha,laghu,Sheet,lavana,katu,ahaara, shushkashaaka,vallura,uddhalak, etc.

Viharaj hetu :balawat vigrah,excessive panchakarmas like Vamana, Virechana, Raktamokshana,ativyaayaama and supression of Adharanneya vega vyavaya,atiadhyanan, prapatanan, langhana, jagaran

2. Pitta Dosha --

Aharaj Hetu: Pittaprakopaka aahara i.e. amla, katu, lavana, kshaara, Ushna, teekshna, laghu, vidaahi, tila taila, pinyaaka,

Viharj Hetu : krodha, shoka, bhaya, aayas i.e. sharirpida, upavaasa, maithuna.

3.Kapha Dosha–

Aharaj Hetu : Guru, madhur, snigdha, sheeta, lavana, maasha, mahaamaasha

Viharaj Hetu: avyaayaam (lack of physical exercise), aalasya, diwaswapna, (sleep during day)

F) Vrana Lakshanas:[39]

There are two types of Vrana features:

A. Samanya : Vedana (pain.)

B. Vishesha : consists of signs and symptoms caused by vitiated doshas.

I) Vataja Vrana Lakshanas:

Vrana due to vaata has shyaava or aruna varna, tanu, stabdha, kathina, ruksh, having alpa sraava and vedana baahulyata and toda bhedavat Vedana.

Table No. 1 : Vataj vrana lakshanas

Lakshanas	Sushruta[40]	Charaka[41]	M.Ni.[42]	A.H.[43]	Kashyapa[45]	A. San.[44]
Varna	Shyaava or Aruna	Shyaava	Shyaava	Shyaava, Krushna, Aruna, Bhasma kapotha or Asthi Varna	-	Shyaava, Aruna, Krushna, Bhasma of Asthi.
Vartma	Rooksha	Stabdha, Kathina	Stabdha, Kathina	---	Stambha, Kathina	-
Vedana	Todha, Bheda, Chatachata yana ,etc	Teevra Ruk, Sphurana	Maharuja	Todha, Bheda etc.	Maharuja	Sphuruna, Todha, Bheda etc.
Sraava	Sheeta, tanu, Picchila, Alpasraava	Mandhasra ava	Mandhas raava	Alpasraa va resemblin g Mastu, Maamsa, Pulakaam bu	Alpasraava	Alpa sraava resemblin g Mastu, dadhi, Kshaara, Maamsa Dhaavana, Pulakoda ka etc.

II) Pittaja Vrana Lakshanas : Vrana due to Pitta will be associated with daaha, paaka, raaga, jwara, trishna, moha and vrana has kshipra utpatti i.e sudden onset with neela, peeta varna and pootisraava.i.e. associated with pus.

Table No.2 : Pittaj Vrana lakshanas

Lakshanas	Sushruta[46]	Charaka[47]	M.Ni[48]	A.H.[49]	Kashyapa[50]	A.San.[51]
Varna	Peeta, Neelabha	-----	---	Neela, Kapila,Pingala,Peeta,	----	Peeta, Neela Krushna, Pingala, Haritha,
Utpatti	Kshipra,i.e. .sudden onset	----	---	Kshipra	---	Kshipra
Anya lakshanas	Daaha, Paaka, Raaga, with Peeta Pidaka	Trushna, Moha, Jwara, Sveda,	Trushna, Moha, Jwara, Kleda, Daaha	Raaga, Paaka, pain resembling Vrana caused by Kshaara	Jwara, Daaha, Moha, Trushna	Daaha, Raaga, Paaka, Jwara, Dhoomayana
Sraava	Sraava resembling Kimshuka flower, Ushna.	Pootisraava	Pootisraava	Sraava is warm, large in quantity. resembling Kimshuka Taila or solution of Bhasma	Pootisraava	Sraava large in quantity. resembling Gomootra, solution of Bhasma, Kimshuka or Mrudveeka or Taila

III) Kaphaja Vrana Lakshanas:

Vrana due to Kapha will have paandu or shweta varna associated with ugra kandu, manda vedana, shukla, sheeta, pichhila and ghana Sraava, stabdh sirasnayujalvat.

Table No. 3: Kaphaj Vrana lakshanas

Lakshanas	Sushruta[52]	Charaka[53]	M.Ni.[54]	A.H.[55]	Ka. Sam[56]	A.S.[57]
Varna	Paandu	Paandu	Paandu	Paandu	Paandu	Paandu
Vartma	Sthoola, covered with StabdhaSiraSnaayujaala, Katina	Snigdha, Guru, Bahupicch a	Bahupi ccha, Guru, Snigdha	Sthoola, Katina, covered with SiraSnaay uJaala	Sthaimi thya, Maardh ava	Snigdha, Kathina, Sthoola
Anya lakshanas	Mandavedana, severe Kandu, feeling of heaviness	Manda, vedana, Chirakaari	Mandav edana, Sthaimi thya	Alparuja, Kandu	Manda, vedana, Chiraka ari	Mandha vedana, Kandu, Swaapa, Sthaimithya
Sraava	Shukla, Sheeta, Saandra	Alpasamkl eda	AlpaSa mkleda, Chirapa aki	Large qty. of Sveta Ghana sraava	Atisraa va	Sraava resembling Navaneeta, Tila pishta, Naarikeloda ka

IV) Raktaja Vrana Lakshanas:

This Vrana will have features similar to that of Pittaja Vrana; it has Pravaala (Rakta) Varna, Raktasraava covered with network of Krishna sphota, smells like Turanga or Vaajisthaana.

Table No. 4: RaktajaVrana lakshana's

Lakshanas	**Sushruta**[58]	**M. Ni.**[59]	**A.H.**[60]	**A.Sam.**[61]
Varna	Pravaala Dhala Nichaya	Raktha	Pravaala (Raktha)	Pravaala (Raktha)
Vartma	Covered with network of Krushnasphota, Pidaka	---	----	Covered with Krushnasphota, Pidaka
Anyalakshanas	Smells like Turangasthaana, Vedanaayuktha, DhoomayanaSheela and having features of Pitta	-----	Smells like VaajiSthaana, has other features of Pitta	Smells like VaajiSthaana, has other features of Pitta
Sraava	RakthaSraava	RakthaSraava	SaraktapooyaSraava	SaraktapooyaSraava

V) Dvidoshaja Vrana lakshanas: [62]

Sushruta has explained the lakshanas depending upon combination of doshas while Vagbhata and Madhavakara have the stated same.

Table No. 5: Dvidoshaj Vrana Lakshanas

Lakshanas	**V-P**	**V-K**	**P-K**	**V-R**	**P-R**	**K-R**
Aakruti	-	-	-	-	Ghritamanda	-
Gandha	-	-	-	-	Meenadhavantoya	-
Varna	Aruna, peeta	-	-	Rakta, Aruna	-	Rakta
Vedana	Toda, Daha, Dhoomayana	Toda, Kandu	Daaha, Ushna	Toda, supta	-	Kandu
Sraava	Peeta, Aruna	Sheeta, Picchila, Alpa	Peeta, Paandu	Rakta, aruna	Ushna, Krishna	Rakta, Paandu
Anya lakshanas	-	Rooksha, Guru, Daruna	Guru	Ruksha, Tanu	Mridu, Visarpa	Guru, Picchila, Snigdha

VI) Tridoshaj or Sannipataj Vrana lakshanas: [63]

Table No. 6: Tridoshoj or Sannipataj Vrana lakshanas

Lakshanas	V-P-R	V-K-R	P-K-R	V-P-K	V-P-K-R
Vedana	Spuran, Toda, Daha, Dhoomayan	Kandu, Sphuran, Chumchumayan	Daha, Kandu	has vedana of three types	Nirdahan, Nirmathan, Spuran, Toda, Daha, Kandu
Varna	-	-	-	has varna of three types	-
Sraava	Peeta, Tanu, Rakta	Paandu, Ghana, Rakta	Paandu, Ghana, Rakta	has sraava of three types	Nana varna
Anya lakshanas	-	-	Paaka, Raga	-	Paaka, Raga

G) DushtaVrana: [64] **(Infected wound)**

Dushta means getting vitiated by doshas. If vrana has bad smell (foul odour), has abnormal colour with profuse discharge, severe pain intensity and takes long period to heal it is known as Dushta Vrana. The features of Dushta Vrana vary as per present dosha predominanance

Lakshanas of DushtaVrana: [64]

Lakshanas depending upon the discharge, consistency, shape and chronicity according to various samhitas is as follows;

Table No.7: Dustha Vrana Lakshanas

Sushruta[65]	**Charka**[66]	**M.Ni.**[67]	**A.H.**[68]	**A.S.**[69]
Atisamvrutha or Ativivrutha, Ati-katina or Mrudu, Utsanna or Avasanna. Atisheeta or Usna, having one of the colours Rakta, Krushna Peeta, Shukla etc, Bhairava (fearful features), filled with Pootipooya, Mamsa, Sira, Snaayu etc. Moving in oblique track (Unmargi) having Amanoghna Darshana, Amoghnagandha, atyartVedanayukta, associated with Daaha, Paaka, Raaga, Kandu, Shopha, Pidaka etc. Discharging excessivelyDushtaShonitha,Dheergakalaanubandha	Mentioned 12 characteristic features indicating the advanced stage of morbidity of Vrana. Swetatva, Avasanna Vartmatva, Atisthoola-Vartmatva, Atipinjaratva, Neelatva, Syaavatva, Atipidakatva, Rakta-Krushnatva Atipootitva, Ropyatva Kumbhi-Mukhatva, Vranas with Pootigandha, Vivarna, Bahusraava, Maharuja	Discharges Pooti Sraava, or Dushta Ashruk, has Utsangi (sinuses) inside, Chirastitha, emits Pooti Gandha & doesn't possess any features of Shuddha Vrana	Samvrutta or Vivrutta, Katina or Mrudu Atiutsanna or Avasanna, Atiushna or Atisheeta, Raktatwa or Paanduta, Discharges Pooti Pooya covered with Pooti Maamsa, Sira, Snaayu, associated with Atiruk, Daaha, Swayathu, Kandu & other complications, Dheerga Kaalanubandha	Atisamvrutha or Ativivrutha, Atimrudu or Katina, Atiutsaadha or Avasaadha, Atisheeta or UsnaRakta, Krushna, or Paanduta, covered with Pooti Maamsa, Sira, Snaayu, etc. Discharges Pooti Pooya, Daaha, Paaka, Kandu, Svayathu, Vedana Pitaka, etc. appearing as Upadravas, Dheerga Kaalanubandha

H) ShuddhaVrana: [70] (Clean wound)

The Vrana which is not vitiated by doshas, not invaded by tridoshas, having Shyaava Oshta, developed Sama Pidaka and is not associated with Vedana and Sraava, is said to be ShuddhaVrana.

Vrana which resembles Jihwatalaabha,(i.e. edges and floor) mrudu, snigdha, slakhsna; and there is no vedana and sraava also it is good looking i.e. suvyvastith, is said to be Shuddha. Shuddha Vrana consist all these features. Sushruta and Vagbhatta have mentioned the almost similar features

.

Table No. 8: ShuddhaVrana Lakshanas

Sushruta[71]	**Charka[72]**	**M.Ni[73]**	**A.H[74]**	**A. S.[75]**
Not vitiated by tridoshas having Shyaava Oshta resembles Jihvatalaabha which is Mrudu, Snigdha, free from Vedana, Sraava, good looking, and has developed Samapidika.	no Atirakta, nati-Paandu, nati Shyaava, na ati Ruk, nati Utsanna nati Utsangi.	Resembles Jihva talaabha is Atimrudu, Slakshna, Snigdha, Suvyavasthitha, AlpaVedana, and Nirasraava.	Resembles vrana Jihvatalabha in redcolor, Mrudu, Slakshna with ShyaavaOshta, Samapidika, having UnnataMadhya, not acommpanied with any Upadravas.	Not vitiated by Doshas, resembles Jihva in colour, and is Slakshna having Shyaava, Oshta centre being elevated or even not having Vedana, Sraava

I) RuhyamaanaVrana: [76] (Healing wound)

Lakshanas:

Vrana posseses Kapota Varna, it is free from Kleda and has Sthira Pitika is said to be Ruhyamaana Vrana.Vrana area is coverd by small outgrowths (granulation tissues). Similar type of information is given by Vagbhata and in Madhavanidaana also.

J) Samyak Roodha Vrana:[77] (Healed Wound)

Lakshanas:

Vrana healed without eruptions (Granthi), without hardness, no pain (Vedana) and swelling, colour of the site should be similar to that of surruounding skin and the site should be flat such Vrana is known as Samyak Roodh Vrana.

K) Representation of classification of Vrana due to various reasons :

Vrana	Kaarana ----	Nija, Aagantuja.
	Avastha - ---	Dushta, Shuddha, Ruhyamaana, Roodha.
	Aakruthi ---	Aayatha, Chathurasra, Vrutta, Triputaka.
	Saadhyasaadhyatha---	Sukhasaadhya, Kruchrasaadhya, Yaapya, Asaadhya.

L) Samprapti: [78]

Doshas being aggrivated by their respective causative factors and gets lodged in any of Vrana Sthaanas.So, vranotapatti occurs.Vaata, Pitta, Kapha being aggrivated by their respective causative factors and give rise to NijaVrana.

M) Examination of Vrana: [79]

Examination of Vrana and patient, suffering from Vranalakshanas are to be carried out in 3 different ways. They are Darshana, Sparshana and Prashna.

i) Darshana:

By Darshana Pareeksha age of patient, site of Vrana, Aakruthi, Varna, condition of Vrana, Vranasraava, number of vrana, etc. can be elicited.

ii) Sparshana:

It helps in deciding the hardness or softness of Vrana, increase or decrease of local temperature, tenderness, bleeding, etc.

iii) Prashna:

By Prashna Pareeksha the cause of Vrana, type of Vedana, Agni, Bala, Saatmya etc. are to be examined. Sushruta mentioned Shadvidha Pareeksha for the diagnosis. Darshana and Sparshana should be done by Panchaindriya Pareeksha. Shushruta mentioned about Vrana examination that every surgeon examines the mulsthan of Vrana, also vrana adhisthan i. e. Panchlakshan and parigrahee i.e. vrana site. Then surgeon can decide about Shasthi upakram and with the help of chikitsa chatushpada the treatment of ulcer becomes easier.[28]

Table No.9: Examination Method of Vrana

Sr. No.	Exam Method	Points to be examined
1	Darshan	Age, Colour, body parts, Sense organ etc
2	Sparshana	Hot and cold, soft and hard, etc
3	Prashana	Cause of disease (hetu), Pains, satmya, strength of Agni

N) Vrana Vastu or Vrana Adhistan: [80]

There are eight sites of vrana known as vranavastu. They are twak, mamasa, sira, snayu, asthi, sandhi, kostha and marma. Charaka has mentioned meda instead of sandhi and antarashrya for kostha.

Madhavakara (Madhavanidana) explained Samaanya and Vishesha lakshanas in case of injury to Maamsa, Sira, Snaayu, Sandhi, Asthi, and Marma.

Table No.10: Vrana vastu and its lakshanas according to Madhavkara [81]

Vranasthaana	Lakshanas
Injury to Maamsa, Sira, Snaayu, Sandhi, Asthi	**Saamanya Lakshanas**: - Bhrama, Pralaapa, Patana, Strasthangata,Vicheshtana, Pramoha, Glani, Ushnata, Moorcha, Teevraruja, dischrge of Rakta resembling Mamsodaka, loss of functions of Indriyas etc.
	Vishesha Lakshanas
Injury to Sira	Profuse discharge like Indragopa.
Injury to Snaayu	Decrease in height, drooping, loss of Pain, Vrana takes long time to heal
Injury to Sandhi	Increase in Shopha, severe pain, loss of strength, total loss of function etc
Injury to Asthi	Severe pain continuously throughout the Day and night, no relief in any posture.
Injury to Maamsa Marma	Pallor, loss of tactile sensation.

O) Vranakruti or Shape of Vrana: [82]

Vrana resembles following shapes which are curable:

- Ayata (Elongated)
- Chaturastra (square or rectangular)
- Vritta (Circular)
- Triputak (Triangular)

According to Vaghbhat the shape of vrana is depends on the shape of shalya. Shape of agantuja Vranas are Aayatha, Chaturasra, Trayastra, Mandalina, Ardhachandraakaara, Vishaala and swastika, Kutila etc.Some resembling Sharaavanirmana madhyascha, others with elevation in the centre. Aagantuja Vranas have numerous shapes, except the main four shapes others are difficult to cure.

P) Vrana Varna (colour of the Vrana): [83]

The colour of the vrana site, gives an indication of the involved dosha as well as the prognosis.

Table No.11: Vrana Varna as per involvement of Doshas.

Dosha	Colour of Vrana
Vaata	Bhasma (ash), Kapota, Asthi, Parusha, Aruna (reddish black), Krushna.
Pitta & Rakta	Neela, Peeta, Haritha, Shyaava, Krushna, Rakta, Kapila (brownish), Pingala (faint brown).
Kapha	Sweta, Paandu (dull yellowish), Snigdha (appears unctuous).
Sannipaataja	As tridoshas are mixed, the site acquires various colours.i.e. Sarva Varna.

Q) Vrana Gandha: [83, 84]

Vrana gandh is one of the important examinations of vrana. According to Sushruta vataj vrana has Katu gandh, Pittaj has Tikshna, Kaphaj has Visra, Rakaj has Lohagandh and Sannipatik vrana has Vyamishra gandh. Vyamishra i.e associated with all types of smells. He also mentioned that smell similar to laja, Atasi tail and Til taila (oil) are also prakrut smelles of vrana. Other smells are known as vikrut gandh. According to Charaka, Vrana has eight types of Gandha like Ghruta, Tail, and vasa i.e serum of Flesh, Puya, Rakta, Shavya, Amla and Pootik.

R) Vrana Vedana: [85]

Vrana vedana are related with the doshas involvement.

i) Vrana vedana due to vata dosha:

Vrana vedana due to vaat dosha like Toda, Bheda, Tadana, Chedan, Ayamana, Manthana, Vikshepan, Chimchimayan, Nirdahana, Avabhanjana, Sphotan, Vidarana,Utpatana, Kampan. Due to vata dosh pain comes on and off. There is no consistency with respect to site, cause etc.

ii) Vrana vedana due to pitta dosha:

Oosha, Chosa, Paridaha, Dumayan, the area of vrana appears like burning amber, pain like Khsaara has been applied to the Vrana. These are the types of vedana due to pitta dosha.

iii) Vrana vedana due to Kapha dosha:

Kandu, Gaurava, Suptatva, Alpa vedana, Stambha, Saithya are the types of pain due to kapha dosha.

iv)Vrana vedana due to Rakta dosha: Similar to pitta dosha.

v) Vrana vedana due to Sannipataj vrana:

Vrana vedana due to involvement of tridosha and hence all types of vedana occurred.

S) Vrana Sraava: [86]

i) Vrana sraava due to dosha

Sushrut and Vaghbhat have mentioned the list of discharges based on vrana adhisthan and involvement of doshas.

Table No.12: Vrana sraava according to doshas

Dosha	**Vrana Sraava**
Vaat	Parusha, Shyaava, Dadhimastu, Kshaarodak, Maamsa- dhaavana, Pulakodaka
Pitta	Gomeda, Gomootra, Shanka, Kashaayodaka, Maadhveeka Taila
Kapha	Navaneeta, Kaseesa, Majja, Naarikelodaka, Varaahavasa
Rakta	like lakshanas as pitta but associated with more rakta srava
Sannipataj	Priyanguphala, Naarikelodaka, Kaanjeeka etc.

Features of sraava mentioned by Vagbhata are similar to Sushruta. Charaka has explained 14 types of sraava. They are Lasseka, Jala, Pooya, Asruk, Haridra, Aruna, pinjar, Kashaaya, Neela, Harita Snigdha, Ruksh, Sita, and Asita.

ii)Vrana Sraava according to Sthaana:[86]

Table No.13: Vrana Sraava according to Sthaana or Vrana Adhisthan

Sthaana	**Sraava**
Twak	Salilaprakasha, Peetaavabaasa.
Maamsa	Sarpiprakasha, Sheeta, Picchila.
Sira	Rakta Atipravruthi, Pooya comes out after Paaka.
Snaayu	Snigdha, Ghana, Singhanaka pratima, Sarakta.
Asthi	Discharge mixed with Rakta, Majja.
Sandhi	Picchila, Saphenarudhira.
Kostha	Discharges mixed with Asruk, Mootra, Pureesha, Pooya, and Udaka.

T) Saadhyaasadhyatha:

i) Sukh saadhya Vrana: [87]

- Vrana arising in tarun vaya (it heals because of pratyagra dhaatus),
- Dhruda (Body having Sthira Bhau, Mamsa, Sira, Snayus etc.)
- Pranvannta.
- Satwawanta (Do not suffer from Vedana caused by Darun Kriya)
- Vrana arising in pratham dhatu i.e. Twak, Mamsa Adhistan.
- Vrana akrutis like Ayata, Chaturasra, Triputak, and Vrutha.
- Patient should be Atmvanta.
- Vrana treated by Kushal Vaidyas.
- Vrana sites are sphik, paayu, Prajanan (genitila), Lalata,Gandha, Oshta, Prushta, karna Phalakosha, Udara.
- Vrana of recent origin and free from upadrava

ii) Kruchasadhya Vrana: [88]

- Vrana in patient having old age,
- Patient is Krush, Alppraana, and Bheeru etc.
- Vranas having vikrut aakruti.
- Vranas situated in sthaan like Danth, Nassa, Apanga, Srotra, Naabhi, Jathara, Sevani, Nitamb, Parshwa, Kukshi, Vrana associated with people suffering from Kustha, Visha, Shosha, Madhumeha.
- Vrana treated by quack
- Vrana which emits phena, pooya, anil
- Bhagandhara with an internal opening
- Vrana formed over Kati and Asthi
- Patient who is anaatmavatitha

iii) Yappya Vrana: [89]

- Vrana manifested in the following conditions can be treated but not completely cured Avapattika, Niruddhaprakasa(Phimosis), Sannirudh Guda, Sannirudh Jathara, when krimi infest vrana, Granthi, Krimi in abdomen, Madhumeha,sikatameha,Vaatkundalika, asthila(BPH), Dantsharkara, upakush, Kanthashalulak, Visarapa, etc.

iv). Asaadya Vrana: [90]

- Vrana with an elevated floor of excessive granulation tissue (mamsa pindavat) with excessive discharge. Containing pooya inside associated withvedana, Discharge of vasa, meda, majja, mastulunga. Koshtastha vrana having discharge of peeta or asita varna, mootra, pureesha etc. and those having discharge of pooya and rakta.
- Vrana having ostha like Ashwa apaana, protruded like goshringa,
- Those discharging dushta rudhira, Tanu, Sheeta, Picchila sraava, elevated in centre, some are Santoolavata contains Snaayu, Jala, having Durdarshana.
- Vrana at Upanakha,over marma, over bones of leg (Janghasthi), Blind external fistula in Ano, Vrana over Median Raphae,
- Vrana secondary to diseases of bones of pelvic region.
- Vrana situated in all ground materials (Sarvtogath) with anumukh and mamsa budbuda
- Vrana situated in sira and Kantha from which air escapes making of sound
- Discharge of blood with pus in a heena mamsa person,
- Associated with upadravas like Arochaka, Avipaak, Kaasa, Shwas
- Bhinna vrana in Shira, Kapal followed by appearance of Mastulung.
- Features of all the three vitiated doshas or with Kassa and Shwas are incurable
- Vrana discharging Vasa,Majja, and Mastulung are curable if casued by aagntuj karanas, otherwise it is incurable due to vitiation of three doshas.
- Vranas with pulkodaka sraava from pakwashaya, Ksharoodaka type of sravva from raktashya, kalaaya type of sraava from amashya and trik sandhi Pradesh
- Vrana situated in deeper dhatu (uttarotar Dhatu)
- If it is not cured in a particular time sadhya vrana becomes Yaapya, Yappya vrana becomes assadhya, and finally Assadhay may kills person.
- Vrana manifested with fatal signs i.e. vrana with Arishta lakshanas.

U) Vrana Chikitsa: [91]

Charak has mentioned 36 upkramas for the treatment of vrana where Sushruta has mentioned 60 upkramas. Sharangdhar has mentioned Saptakramas like pralepa, pradeha, pachan, darana, vrana shodhan, ropan. Charka has explained Samanya and Vishesh Chikitsa for Vrana.[92]

I) Samanya Chikitsa:

Vranitasya should be given Shodhan, therapies through Vamana or Virechana.

II) Vishesh Chikitsa:

A) Vaataja vrana chikitsa[93]

Persons suffering from Vaataja Vrana should be treated with complete Snehapana, Swedana, Upanaha, Pradeha, and Parisheka which are of unctuous nature.

B) Pittaj Vrana Chikitsa[94]

Persons suffering from pittaj vrana should be treated with virechana, pradeha, parisheka, sarpipana, prepared by sheetala, madhura, and tikta dravyas.

C) Kaphaj Vrana chikitsa [95]

Person suffering from Kaphaja Vrana should be treated with Pradeha, Parishechana, with drugs which are Kashaaya, Katu, Rooksha, Ushna and Laghu, Paachana etc.

D) Saptaupakrama and Shashti Upakramas [96]

They are mentioned in treatment of Vrana Shopha.

They are Vimlapan, Avasechan, Upanaha, Paatan, Shodhan, Ropana, Vaikritaapaham and these are elaborately explained by Sushruta in the Shashti upakrama.

Table No.14: Shashti Upakramas of Vrana mentioned for vrana by Sushruta and other Acharyas[97]

Upakrama	Sushruta[97]	Charak [98]	Kashyap[99]	A.S & A.Hr [100]
Apatarpana	+	-	-	-
Aalepa	+	-	Pralepa	Pradeha
Parisheka	+	-	+	+
Abhyanga	+	-	-	+
Swedana	+	-	-	+
Vimlapana	+	-	-	+
Upanaha	+	-	+	-
Pachana	+	-	-	+
Visravan	+	-	+	+
Snehana	+	-	+	-
Vamana	+	-	-	+
Virechana	+	-	-	+

Chedana	+	+	-	-
Bhedana	+	Patana	-	-
Darana	+	-	-	+
Lekhana	+	+	-	-
Eshana	+	+	-	-
Aharana	+	-	-	-
Vyadhana	+	+	-	-
Sravana	+	-	-	-
Sivana	+	+	-	-
Sandhana	+	+	-	-
Pidana	+	+ Avapidana	-	+
Shonit sthapana	+	-	-	-
Nirvapana	+	+	-	+
Utkarika	+	-	-	-
Kashaya	+	+	-	-
Varti	+	-	-	+
Kalka	+	-	+	-
Sarpi	+	+	-	Ropan ghrit
Taila	+	+	-	Ropan taila
Rasakriya	+	-	-	+
Avachoornan	+	+	-	Choorna
Vrana shodhana	+	kathinakara, mardavakara	-	+
Utsaadana	+	+	-	+
Avasaadana	+	+	-	+
Mrudukarana	+	maradavakarma, Aalepana.	-	+
Daranakarma	+	Kaathinyakara aalepa.	-	+
Ksharkarma	+	+ Daha	-	+
Agnikarma	+	+ Daha	-	+
Krishnakarma	+	Varnya	Savarnikaran	Savarnikaran

Pandukarma	+	Varnya	Savarnikaran	Savarnikaran
Pratisarana	+	-	-	-
Romasanjanan	+	+Lomarohana	-	+
Lomapaharana	+	-	-	-
Basti	+	-	-	-
Uttarbasti	+	-	-	-
Bandha	+	+	+	-
Patradana	+	Patrachedana, (bahya abhyantar)	-	-
Krimighna	+	-	-	-
Brimhana	+	-	-	-
Vishaghna	+	-	-	-
Shirovirechana	+	-	-	-
Nasya	+	-	-	-
Kavala dharana	+	-	-	-
Dhoom	+	-	-	-
Madhu-Sarpi	+	-	-	-
Yantra	+	-	-	-
Aaharana	+	Bhojya	-	-
Rakshavidhana	+	-	-	-
Shophaghna	-	+	-	-
Shamana	-	+	+	-
Chadana	-	+	-	-
Shodhanalepa	-	+	-	+
Ropanalepa	--	+	-	+
Ropana	-	+	+	-
Utklinnaprakshalana	-	-	+	Prakshalan
Shodhana	-	-	+	-
Prachhena	-	+	-	-

Among the 36 upakramas mentioned by Charaka, Shophaghna i.e. the treatment of Vranashopha; involves Raktavsechan, Langhana, Snehan, Pralepa, Pradeha, Upanah etc. These karmas are considered consizely 11 Upakramas mentioned by Sushruta for Vranashopa.

Also, Charaka has discussed about Shastra karmas. He also mentioned Eshankarma separately & Aharankarma has been covered under six surgical measures. Charaka has describes vranopkrama as Shodhanakashaya, Taila, Ghrita, Ropanakashaya, Utsadan, Avasadana, Aalepana etc.

Sushruta has mentioned vranopkrama as Shodhana, Ropana & Vaikrutapaham etc.

Shodhan and Ropan Defination

In this study Shodhan and ropan concept are more important for treating chronic ulcers.As the all chronic ulcers are infected, so they need vran shodhan and ropan both to heal completely.

A.Defination of Shodhan:

I) शोधनम्

स्वयमेव विदिर्ण शस्त्रेण भेदितं वा व्रणं यानि द्रव्याणि शोधयन्ति, तानि शोधनाणि इत्युच्यन्ते |

शोधन--व्रणशुध्दिकर:। (च.सू.२५/८५)

शोधनं पुन:ष्टविधं ------कषाय, वर्ति ,कल्क, घृतं, तैल, रसक्रिया, चूर्ण,धूपन भेदेन। शोधनद्रव्य विस्तरस्तु सूश्रूते सूत्रस्थाने ३७ तमे अध्याये चिकित्सास्थाने प्रथमे च द्रष्टव्य:।

Shodhan can be concluded as cleaning of ulcer i.e. removel of pus and dead tissue.

B. Defination of Ropan

II) रोपणं

रोप्य्-- रोपणीय: यथाव्रणा:

'बहिशुध्दा:' इवाभान्ति रोप्यस्ते संप्रकिर्तिता:। (च.सू.२५/२५)

यथा कषायो रस:॥(च.सू. २६/४३,६)

यथा स मधुकास्तिला रोपणा:॥(अं.स,सू.३९)

यथा रोपणं मधु:।(अ.ह्रु.सू.५\५२)

रोपणं कषाय, वर्ति ,कल्क, घृतं , तैल,रसक्रिया, चूर्णभेदेन सप्तविधं भवति। रोपणद्रव्य विस्तरस्तु सूश्रूते सूत्रस्थाने ३७ तमे अध्याये चिकित्सास्थाने प्रथमेअध्याये च द्रष्टव्य:||

व्रणं रोपणं तत्सप्तविधं--कषाय, वर्ति ,कल्क, घृतं , तैल,रसक्रिया, चूर्णभेदेन।(सु.सू.३७,२२- २८)

रोपणकल्क व्रणरोपणार्थपयुज्यमान: कल्क(सु.सू.३७,२४) ,

कल्कद्रव्याणि ---समंग:सोम:,सरला, सोमवल्क: चन्दन:| काकोल्यादिगणा:च।(सु.सू.३७,२४)

शुध्दं व्रणं यानि द्रव्याणि रोपयन्ति,तानि रोपणानि इत्युच्यन्ते। रोपणं कषाय, वर्ति ,कल्क, घत, तैल,रसक्रिया, चूर्णभेदेन सप्तविधं भवति।रोपणद्रव्य विस्तरस्तु सूश्रूते सूत्रस्थाने ३७ तमे अध्याये चिकित्सास्थाने प्रथमेअध्याये च द्रष्टव्य: |

It can be concluded as after cleaning of ulcer it's healing by normal tissue growth and closure of ulcer by healthy skin.

Thus these 60 procedures (shashti upkramas) can treat Vrana effectively.

V) Pathyaapathya: [101]

Pathya:

Vranita should consume Jeerna Shaali, Odhana which is made warm unctuous and taken with Jaangala Maamsa. Soup prepared from Tanduliyaka, Jeevanti, Vaartaaka, Patola, Kaaravellaka, Daadima, Aamalaka etc.Vranit should avoid day sleep, excercise.

Apathya:

Vranit must avoid Navadhaanya, Maasha, Tila, Kalaaya, Kulattha, Nishpaava, Hareeta Shaaka, Katu, Amla, Lavana Rasa substances, Guda, Sushka Shaaka, eatables made from Pishta, Ajaa, Anoopa, Maamsa, Sheeta Udaka, Krushara, Paayasa, Dadhi, milk etc.Vranit person should avoid Vaata, Aatapa, Raja, Dhooma, Atibhojana, Bhaya, Shoka, Krodha, Raatri Jaagarana, Vishamaashana, Vyayaama, Upavaasa, Chankramana etc.

W) Upadravas:

These are mainly classified as Vranasya Upadravas, Vranitasya Upadravas[102]

- **Vranasya Upadravas**: -are five related with abnormality in Aakruthi, Vedana, Gandha, Sraava, Varna.
- **Vranitasya Upadravas**: -Jwara, Atisaara, Moorcha, Hikka, Chhardi, Arochaka, Shwaasa, Kaasa, Avipaaka, Trushna.

Charaka mentioned the 16 types of Upadravas they are: Visarpa, Pakshaaghaata, Sirasthambha, Apataanaka, Moha, Unmaada, Vrana Ruk, Jwara, Trushna, Hanugraha, Kaasa, Chhardi, Atisaara, Hikka, Shwaasa and Vepathu.[103]

X) Vrana as upadrava:

Vrana itself is an upadrva in diseases such as Prameha, Visarpa, Vaata rakta, shotha, Kushta, Arshas(bahya).

Y) Vrana Arishtha lakshans (Fatal signs of Vrana): [104]

Sushruta has described Vrana-arishta lakshnae (fatal signs) as follows;

i) Gandhavaikrut: - (fatal sign based on odour):

Vrana patient emits odour resembling that of Madya (alcohol), Honey, Ghee, Jasmine, Lotus, Sandal etc. It is one of important fatal sign.

ii) Sparsh vaikrut (fatal sign based on touch):

When patient complaints severe heat and burning sensation at vrana site but vrana itself having sheetsparsh (Cold). It is also one of the important fatal sign.

iii) Aakruti vaikrut: (fatal sign based on shape.)

Vrana having irregular shapes viz; flag, elephant, horse, etc.; also, Patient feels that Vrana site covered by a fine powder like substances, but actually nothing is at Vrana site; Then Vaidya can conclude about patient's death.

iv). Shabdvaikrut: (Fatal sign based on sound)

Vrana produces various sounds viz Ghurgurayan and there is discharge of air or gas from Vrana.All these are indications of death.

3.2 Review of Prameha and Madhumeha

Diabetic foot ulcer is a major complication of Diabetis Mellitus. It becomes one of the medical, social and economic problems worldwide.India being termed as capital of DM. Ayush department of India also declared theme 'Mission Madhumeha through Ayurved'for year 2016- 2017.

3.2.1. Vedic Period (4000 B.C.-1500B.C.)

The ancient references of 'Prameha' are found in Vedas of India.

The word Prameha literally means 'to flow'. It is derived from the Sanskrit root 'Mih-Sechane.'

The two terms 'Aasarava'and 'Prameha' are used in Atharvveda and in Kaushiksutra, respectively.Sayan and Kesavabhatta, the well-known commentator of Vedas describes 'Asrava 'as 'Mutratisara.'

Whitney (1962) and Griffith interpreted this as flux and morbid flow respectively. William Cullen (1712-1794) added the word 'mellitus' to Diabetes, like the Prameha-Madhumeha concept of the ancient ayurvedic classics.[105]

3.2.2. Samhita Period (2000B.C.to 800A.D.):

I) Charak Samhita:

Charak explained the etiology, symptmatology, especially samprapti, types, complications and treatment of Prameha. He explained Prameha in Charak Nidansthan.4 and chikitsa in Charak Chikitsastan 6. In Ch.Su17, he described the samprapti of margavrodhajanya madhumeha and Sapta pramehapidaka.

In Charak Indriyasthan 9, he mentioned that the madhumeha is one of the 'ashtau maharog' which are difficult to cure.[106]

II) Sushruta Samhita:

Sushrut explained nidan panchak of Prameha and Pramehajanya saptapidaka in Nidan sthan.6. He elaborately describes the treatment in Chikitsasthana.He has explained three different Adhayay.They are Sushrut Chikitsasthana11Prameha Chikitsitadhaya, Sushrut Chikitsasthana12,Pramehapidaka Chikitsit and Sushrut Chikitsasthana13 Madhumehachikitsa adhyaya.[107]

He used "Kshoudrameha" synonym to Madhumeha.

III) Ashtang Hridaya: [108]

Vagbhat describes Prameha in Ni .10 and in chi.12; He has explained the two types of Madhumaeha i.e. Dhatukshayajanya and Avrutpathjanya.

IV) Harita Samhita:[108]

He has explained Prameha is a papajanya vyadhi and mentioned that there are13 types of Prameha with names like Puyameha, Ghrutmeha etc. (HA. II, Sth1/9)

V) Bhel Samhita: [108]

He has mentioned two types Narasya -swakrutam Prameha and Prakruti-prabhavam prameha.

VI) Kashyapa Samhita: [108]

He has discussed the symptoms of Pramehi Child in Vedanadahaya and explained eight pidaka in Dwivraniyachikitsaadhyaya.

3.2.3) Medieval Period (800 A.D. to 900A.D.):[108]

I) Madhavnidan:

He repeated collective description of Madhumeha narrated by Charaka, Sushruta and Vaghabhatta.

II) Gayadas:

In 'Nayyachandrika' he has narrated 'Avilmutrata' is because of presence of dushya. (Ny.C.Su.Ni.6/6)

III) Dalhan:

In his 'Nibandha Samgraha' tika he has mentioned that female cann't suffer from Prameha.

IV) Sharangdhara Samhita:

Sharahngdhara mentioned that there are 20 types of Prameha in poorvakhandha 7ThChapter (59-62).

V) Bhavaprakasha:

He described Prameha and Madhumeha along with some new herbomineral preparations.

VI) Yogratnakar:

Prameha and Madhumeha with its treatment are also explained by Yogratnakar.

3.2.4 Prameha

Madhumeha is a disease mainly occurs due to the metabolic disturbances, sedentary lifestyle and sometimes due to genetic predisposition.To understand the complete Prameha with its all details; we must study the Hetu, Samprapti, Poorvarupa, Roopa.

1). Nirukti (Etymology):[105]

The word 'Prameha' composed of two sub words i.e. Pra (Upasarg-Prefix) and 'Meha'.

The word meha is derived from the root 'Mih Sechane' meaning to profuse (watering) and the Pra-means excessive quantity and frequency. Therefore, the Prameha is said to be 'Prabhuta Mutrata' and 'Avil Mutrata.'

प्रकृष्टो मेह: यस्मिन रोगे स: प्रमेह:।

The word Madumeha consists of two words i.e Madhu and Meha.

The 'Madhu' word is derived from the root "Manyante Visheshen Janayati Jana Yasmin." The root "Manjane" is applied by Dha Adesha and it shows the similarity of Urine in taste, colour and appearance etc.

In Sanskrit literature madhu word is used for Pushparasa, Jalam, Makarand, Kshir and for madhura rasa, etc.

Thus, one can conclude that if a disease in which excretion of urine is like madhu (honey) in its colour, taste, smell and consistency then it is known as Madhumeha.

Sushruta has used the term 'Kshoudrameha' for Madumeha[109]and suggested that Prameha is not treated in a time then it is converted into Madhumeha.[110]

2). Paribhasha (Defination):

'Prakarshena prabhutam prachuram varam varammeheti mutratyagam karoti iti Prameha' - Ma.Ni.33/1

प्रकर्षेण प्रभूतं प्रचूरं वरं वरं मेहेति मुत्रत्यागं करोति इति प्रमेह:।

मा.नि ३३/१

Prameha is a syndrome which includes all those clinical conditions which are characterized by increased quantity of urine associated with or without the increased frequency of micturation. Polyuria and turbidity of the urine are two essential presenting features of the disease.

3). Synonyms:

Followings are some of the synonyms mentioned in Samhitas:

A. Ojomeha:

Ojomeha is a subtype of Vataj Prameha.Since Vata affects the Oja, so patient passes the Kashay Urine.

B. Kshoudrameha:

This synonym is used by Sushruta. This type of Pramehi patient passes urine like madhu. (Honey)

C. Paushpameha:

Pushpmeha word is described in Anjan Nidana asPushparasa resembles with madhu.

4) Classification of the Prameha:

In Brihattrayi, the Prameha is classified based on dosha dominance and the classification as follows.

A.Classification of Prameha

I Classification based on Dosha bheda

II. Classification based on Sadyasadytwa

B. Classification of Madhumeha

I). Classification based on Dosha bhed:

Twenty types of Prameha have been described by different authors of ayurvedic Samhitas. Among these, ten are of Kaphaja type, six are of Pittaja type and four belongs to Vataja type.

Table No.15: Classification of Prameha based on Dosh:[111]

Sr. No.	Type	Charaka	Sushruta	Vagbhatta	Madhava
Kaphaja Meha					
	Udakameha	+	+	+	+
	Ikshubalikarasmeha	+	+	Ikshumeha	Ikshumeha
	Sandrameha	+	+	+	+
	Sandraprasadmeha	+	Surameha	Surameha	Surameha
	Shuklameha	+	Pishtameh	Pishtameha	Pishtameha
	Shitameha	+	Lavanameha	+	+
	Siktameha	+	+	+	+
	Shanairmeha	+	+	+	+
	Alalmeha	+	Phenameha	Lalameha	Lalameha
	Shukrameha	+	+	+	+
Pittaja Meha					
	Ksharameha	+	+	+	+
	Kalameha	+	Amlameha	+	+
	Nil meha	+	+	+	+
	Lohitmeha	+	Shinitameha	Raktameha	Raktameha
	Manjishthameha	+	+	+	+
	Haridrameha	+	+	+	+
Vataja Meha					
	Vasasmeha	+	+	+	+
	Majjameha	+	Sarpimeha	+	+
	Hastimeha	+	+	+	+
	Madhumeha	+	Kshaudrameha	+	+

II) Classification based on Sadyasadytwa:

Prognosis is an invitable part of Chikitsa and the success of treatment depends on an unbiased prognosis. Therefore, we must study the classification depends on Sadyasadyatva.

Table No.16: Classification according to Sadyasadyatwa.

Sadhya	Yapya	Asadhya
Kaphaja	Pittaja	Vataja
Sthula(Obese)	Usually not much obese	Krisha(Asthene)
Apthyanimittaja(Acquired)	Acquired	Sahaja(heriditary)
Early Stage	Acute Stage	Advanced Stage
Without complication	With complication	with complication

III) Classification based on hetubheda:

1) Sahaj and2) Apathynimittaj

IV) Classification based on samprapti:

1).Avaranjanya

2) Dhatukshayjanya

1) Santarpanjanya

2) Apatarpanjany

V) Classification based on dehaprakruti bheda:

1) Sthula 2) Krush

Let us consider one by one.

VI) Classification based on etiological factor:

A) Sahaj :(congenital)

Sahaj Prameha occurs due to Beejadosh.Charak mentioned that the Sahaj Prameha is a Kulaj Vikar and is Asadhay**.**[112]Matru pitru beeja dosha are responsible for Sahaj Prameha.

B) Apathyanimittaja:(acquired)

The word Apathya suggests its etiology. Apathya hetusevan and ati aahar causes to Apathynimittaja Madhumeha. This type is again classified according to etiology and Samprapati.

IV. Classification based on Samprapti:

1. **Avaranajanya and Dhatukshayjanya:**

In Avaranajanya Madhumeha[113], kaphavardhaka hetusevan leads to vata avarana, due to vruda vata, oja ksharana occurs and udakavaha, medovaha srotas get affected. Patient passes madhur and kasharaja mutra. In Dhatukhashyajanya Samprapti [114], due to vatavardhak hetu, vataprakopa occurs and dhatukshay takes place.Dhatukshay

and ojakshaya occurs and mutravaha, udakvaha srotas affected. This is the dhatukshayjanya Prameha.

2. Santarpanjanya and Apatarpanjanya

Santarpanjanya i.e.due to excessive nutritional diet; Kaph vitiates and kaph gets aggrevated. The more intake of such diet vitiates kapha, pitta, mamsa and meda. It causes Prameha by forming avarana of vata.

On the contarary, in Apatarpanjanya Prameha dhatu phoshan is insufficient and vata gets aggravated it leads to Apatarpanjanya Prameha.

V. Classification based on Dehaprakruti bheda: [115]

1. Sthula

2. Krush (asthenic)

The sthula and krush classification is akin to obese and non-obese division.

4) Nidana of Prameha:

Let us consider one by one. According to Charak, the disease is mainly due to tridosha. However, Sushrut mentioned that it is due to Sahaj and Apathynimittaj.[116]

1) Sahaj Prameha

Matru and Pitru beeja dosh are the main reason of Sahaj Prameha. If garbha gets affected from Matru and Pitru beeja dosh then by birth patient suffers from Prameha. In conclusion, Prameha is hereditary disease.

Charaka also explained that Sahaj Madhumeha is a kulaj vikar, due to veekrut beeja (sperm and ovum). Here the patient is prone to disease because of above factor.[117]

Charak stated that an excessive intake of madhura rasa by parents may get affected the kaphadhatu and it leads to veekruti (deformity) in sperm and ovum, thus during pregnancy mother and foetus gets affected by Prameha.

2) Apathynimittaj

a) General etiological factors of Prameha[118]

Asyasukha i.e. sedentary lifestyle

Sawpansukha.

Excessive intake of curd or preparations from curd

Excessive intake of udaka, Aanoop mamsa i.e. meat of domestic aquatic land animals.

Payamsi i.e. excessive intake of milk and milk preparations.

Navannapanam i.e. new grains and drinks

Guda vaikrutam i.e. various preparation of sugar and jagary.

Other substance which aggravates kapha may cause Prameha.

Vagbhata said that due to ahitakar aahar and vihar, meda, mutra and kapha are vitiated; this is the main causes of Prameha.[119]

Sushruta mentioned that sheeta, snigdha, madhur, medya (fatty) and drava (liquid) type of food are the responsible for Prameha[120].

B) Kaphaj Prameha Nidana[121]

The following factors which aggrivates the kapha i.e. kaphaprakopak.

Intake of tila, pisthtanna, paaysa (a type of milk preparation), krishara, vilepi and preparations of sugarcane.

Intake of milk, fresh wine, curd preparations, sheet aahar

Reduced physical activity.

Sedentary life style.

Restoring regimens which can increase mere kapha, fat and urine.

C) Pittaj Prameha Nidana[122]

Intake of ushna, kshaar, amla, lavana, ktu dravya (i.e. Spicy food)

Ajeerna and vishaam aahar sevan,

Exposure to excessive heat, sun, emotions like anger and physical exertion.

Shushruta stated that vata, kapha, shonita and meda are involved in samprati of Pittaja Prameha[122]

D) Vataja Prameha Nidana[123,124]

Nidan of vataj prameha is as follows,

Excessive intake of katu, tikta, ruksha, sheeta veerya aahar,

Excessive physical exercise,

Excessive treatment of panchakarmas viz; vaman, virechan, aashthapan and shirovirechan.

Suppression of natural urgs like mala, mutra, upavas, ratri-jagran, excessive blood loss, ati shok, udvega (worry/sorrow) and irregular posture of body are the main causes of vataj Prameha.

Stress is also found to be common cause in all types of Prameha.

Charak said that vataj prameha, due to its own properties becomes Asadhya.

Vasameha, Majjameha, Hastimeha and Madhumehaare the four types of vataj prameha .[125]

5) Poorva roopa:

Bahumutrata is one of the important poorvaroop of prameha. It predicts the nature of disease.It is clearly stated in Sushruta samhita, Poorvaroopa transformed into roopa in association with Bahumutrata[116]. Madhumeha is classified under the Doshaj type of Prameha;So poorvaroopa of Prameha can be taken as poorvroopa of Madhumeha.

Table No. 17: Poorvroopa of Prameha[126-129]

	Cha	Su.	A.H.	A.S.	M.Ni
Kesheshu Jatilibhava	+	+	-	+	-
Asya Madhurya	+	-	+	+	+
Karapadadaha	+	+	+	+	+
Karapada Suptata	+	-	-	-	-
Mukha Talu Kantha Shosha	+	-	+	+	-
Pipasa	+	+	-	+	+
Alasya	+	-	-	+	-
Kaye Malam	+	-	-	+	-
Kaya Chhidreshu Upadeha	+	-	-	+	-
Paridaha Angeshu	+	-	-	-	-
Suptata Angeshu	+	--	-	+	-
Shatpada Pipilika Mutrabhisaranam	+	-	+	+	-
Mutre cha Mutradosham	+	-	-	-	-
Visra sharer Gandha	+	+	+	+	-
Sarvakala Nidra	+	-	-	+	-
Sarvakala Tandra	+	+	-	+	-
Snigdha Gatrata	-	+	-	+	-
Pichhila & Guru Gatrata	-	+	-	-	-
Madhur Mutrata	-	+	-	-	-
Shukla Mutrata	-	+	-	+	-
Sada	-	+	-	+	-
Shwasa	-	+	-	+	-
Keshanakhativriddhi	+	+	+	-	-
Sheeta Priyata	+	-	+	+	-
Hridaya Netra Jihwa Shravanopdeha	-	-	+	-	-
Sweda	+	-	+	+	-
Dehe Chikkanata	-	-	-	-	+

6) Roopaavastha of Prameha: [130]

In Shusruta samhita it is stated that person should be diagnosed as Pramehi when poorvroopas are assocated with the Bahoomutrata.[130]

Gyadasa while describing specific disease state that poorvaroopa get converted into roopa i.e. vyadhi prabhava.

1) Specific Roopa of Madhumeha

Urine Characteristics:

a) Prabhuta Mootrata: -

This is the cardinal sign of Prameha described by all Acharyas.Urine of Madhumehi is kashaya, madhura, pandu varna and ruksha quality.[131]

As per Sushruta, [132] the urine of Madhumehi resembles to that of honey and same description is stated by Vagbhata.[133]

b) Avila Mutrata (Turbidity): -

Due to dushya, dosha and mutra patient passes turbid urine.

C) Picchila Mutrata: -

Patient passes the urine picchila and madhura.

2) Associated Signs and Symptoms:

Sushruta has described two types of Prameha along with their manifestations as follows:

i) Sahaja Pramehi (Krisha-Asthenic)

- Ruksha (Dry body)
- Alpashi (Eat less amount of food)
- Bhrish Pipasa (Excessive thirst)
- Parisarpansheelata (Restless, always desires to wander)

ii) Apathyanimittaja (Sthula-Obese)

- Bahuashi (consumes excess amount of food)
- Snigdha (Unctuous body texture)
- Shayyasanswapnasheela (prefer sedentary life style)

D) Kashyapa has described the symptoms as follows**: -**

Akasmata mutra Nirgaman—Child excretes urine suddenly.

Akranta mutra—Flies get attracted towards urine.

Shweta and Ghanamutrata. —Child passes urine turbid and Shweta.

Kashyapa has also narrated symptoms like Gaurava (Heaviness of the body), Baddhata (tightness) and Jadata (Steadiness, laziness).

7. Samprapti

Samhita describes three different sampraptis, based on Dosha dominanace.

A). Samprapti of Kaphaja Prameha: [134]

Charak mentioned in the NidanSthan and Chikitsasthana about the etiological factors which causes to kaph-prakopa.He also mentioned 'Bahudrava sleshma' is main causative factor.[135]

In Kaphaj Prameha, vikrita kleda is increased. The body tries to move it out. Some part of its get converted into Swed, because 'swedasya kledavidruti.' So it produces 'Chikkanata dehe.' Remaining kleda is taken to basti; therefore, prabhoota mootrata is seen.Due to vikrit Kleda various abnormalities seen in urine. Thus, due to dusit dosha-dyshyas, dhatu-shaithilya,, excessive production of kapha, meda and mutra is seen and 'Bahoodravmootra'symptom is found.[136]

Sushruta narrated dushyas in each doshaj type of Prameha. He stated In Kaphaj Prameha Kapha vitiates along with vata, pitta and meda. [137]

B) Samprapti of Pittaj Prameha: [138]

Due to hetu-sevan pitta dosha gets provoked. TheUshna Guna of Pitta,Vilyana of dravansha from dhatus takes place.This pitta along with increased kleda vitiates dhatus like meda, mamsa, Prameha occures.

Shushruta mentioned that in Pittaj Prameha, Shonit also vitiated with vata and kapha.[139]

C) Vataj Prameha: [140]

The samprapti of Vataj prameha as per Charaka, kapha, pitta and kleda are Ksheena, Vivrudha Vata with Ashayapakarshagati pulls kleda and other dusyas like majja,oja rasa,meda,to Basti.In relation to Basti,kleda is increased but this is kshayatmaka Samprapti.Thus, the dushyas like vasa, majja, lasika and oja vitiated one by one and are brought to Mootraashay and eliminated in mootra form.[140]

Shushruta mentioned that samprati of vataj Prameha occurs due to pitta, meda, kapha, vasa and majja.[141]

As per Vagbhat, first kaphaj prameha is produced, then as the sampraprti progresses, the same turn to Pittaj and ultimately Vataja

As we go to kaphajmeha to vatic the gati of dosha-dushya sammurcchana can be explained as,

Table No.18: Relation between Dosha, Duysh and Madhumeha

Dosha prakopa	Dushya	Meha produced
Kapha	Rasa, mamsa, meda	Kaphaja
Pitta	Rakta	Pittaja
Vata	Remaining Pradhana Dhatus	Vataja

Shusrut has mentioned that if Prameha is not treated in a particular time then it is converted into Madhumeha. So, let us consider about Madhumeha.

8. Madhumeha: -

Madhumeha is very well explained by Charaka when Prameha not treated in particular time it gets converted into Madhumeha. Madhumeha is a vyadhi (disease).[142] Vagbhatta explained two types of Madhumeha according to samprati.[143]

A) Dhatukshayajanya madhumeha

B) Avranjanya Madhumeha

1. Vataj Madhumeha: [144]

Charaka described detailed samprati of Vataj Prameha. Due to hetusevan vata provokes and vitiated vata with the saarbhoot dhatu like vasa, majja, oja and lasika, carries towards mootrashya resulting into Vataj Prameha.

The ruksha guna of vata carried out Ksyatamaka samprapti.So, the dusyas like mamsa, meda and oja are carried out towordas mootrashaya by asyapakarsha gati and bahumootrata takes place. This condition is termed as madhumeha.

2. Madhumeha due to Shuddha Vata: [145]

Due to kapha and pitta kshya (loss) and with the kshya of vasa, majja, lasika and oja, vata dosha gets aggrevated and draws oja towards basti leading to madhumeha.

A) Dhatukshayajanya Madhumeha: [146]

Dhatukshaya mainly occurs due to excessive santarpan and apatrapan also. Due to apatarpan or excess santarpan, dhatupariposhankrama gets disturbed and thus the vata gets provoked.

The kshaya of vital dhatus vasa, majja, lasika and oja leads to vataprakopa. Vata makes ksharana of dhatus. Dhatus are also having Shiathilya.So, through mutravaha srotas mutra, lasika, vasa are moved out and when Kapha and Pitta gets depleted the vata provocation occurs and it leads to kshay of vasadi dhatus.[147]

B) Avaranjanya Madhumeha: -

Vagbhata mentioned this type of madhumeha but not explained.[148]

However, Charak explained it very well. This is the unique contribution of Charaka. Due to hetusevan like guru, snigdh, amla, new grains, excessive exercise and excessive panchkarma measure Kapha and Pitta gets provoked and vitiates meda and mamsa. They in turn cause obstruction of normal pathway of vata. Thus, obstructed vata aggrivates by making avaran with dusyas and carries the vitiated doshas towards basti resulting into Madumeha.[148]

1. Kalprabhavaja Madhumeha: [149]

Sushrut explained all types of Prameha, if not treated in particular time, it converted into Madhumeha. This is the later stage of disease. It is asadhya.

3.2.9. Samprapti Ghataka's of Madhumeha: -

1. Dosha:

Kapha : Bahu +Abaddha in avaranjanya madhumeha
Kshin in dhatukshayajanya madhumeha

Pitta : Vriddha-in avaranjanya madhumeha
Kshina in dhatukshayajanya madhumeha

Vata: Avritta- in avaranjanya madhumeha
Vriddha-in dhatukshayajanya madhumeha

2. Dhatus: [150-157]

Rasa, Rakta, Mamsa, Meda, Majja, Vasa, Lasika, Oja, Shukra, Ambu, Sweda

3. Srotas: Medovaha, Mutravaha, Udakavaha

4. Srotodushti: Atipravritti, Sanga

5. Prakopa: Sarva sharira

6. Prasara: Rasayani

7. Sthanasamshraya.: Mutravaha Srotas

8. Agni: Dhatwagnimandya

9. Ama: Dhatugata (Aparipakwa Dhatu)

10. Udbahva: Amashaya

11. Swabhava: Chirkari

1. Dosh involvement:

1. Kapha dosh: -

Charaka described as 'Kaphakrutch sarvum'. It indicates the importance of this dosha. Kapha is produced from Rasa; therefore Rasa and Meda are necessarily to be considered. Provakation of kledaka kapha results into sharirshaithilya; and results into atinidra, tandra, aalasya etc.

2. Pitta dosh: -

Due to dushit pitta and its drava guna rakta, lasika, sweda and rasa these dusyas get affected. Thus swedavridhi, visragandha, paridaha, pipasa and sosha these symptoms of pittadosha vridhi appeared.

3. Vatadosha: -

This is the predominated dosha in Madhumeha samprapti. Vata aggravates due to its own etiological factors or because of avarna caused by kapha, pitta and meda.

The provocated vata affects the dusya like vasa, majja, and oja, lasika.All these vital constituents are moving out. Thus, all the dhatus becomes durbale. This leads to poor vyadhikshamatava. Thus, the symptom manifests are karshya, daourbalya, angasuptata and parisarpansheela.

In all prameha samprapties, Vyana acts as the collector of kleda and Apana as excreter.

2. Dhatu involvement: -

Dosha, dushya and nidan are the three responsible factors for the manitestation of every disease.

Charaka enumerated a special group known as dushaya visesha.He mentioned dusyvisesha in chikitsasthan also. Dusyvisehas are Rasa, Rakta, mamsa, Meda, Majja, Shukra, kleda, vasa, lasika and oja.[151].

Sushruta explained the dushyas along with basic type and mentioned that meda is a comman dushya in each type.[152]

All the ten dushyas along with sweda are mentioned by Vagbhatta.

2. Dhatu and Dushya involvement:

Due to combination of these dushayas 20 types of Prameha occured

I) Rasadhatu: -

As kapha gets vitiated rasa also vitiated. Vagbhata also mentioned that 'Rasoapi shleshmavat'[153]So as shusrut said that vitiation of rasadhatu results into Staulya and Karshya[154]

So Rasdhatu plays important role in the disease. Vitiated rasa produces symptoms like aganimandya, alasya, glani, karshya, klaibya and aganimandya in madhumeha.

II) Raktadhatu: -

It gets vitiated causing complications like Pidaka, Vidradhi, Alaji.

III) Mamsadhatu: -

It is involved in kaphaj Prameha and avaranjanya madhumeha. Due to vitiation of mamsa dhatu, it loses its normal consistency, develops shaithilya and provides 'Avakash' in the body. It turns into putimamsa pidaka.[155]

IV) Medadhatu: -

It is the important dushya in all types of Prameha, both quantitatively and qualitatively. Due to vitiation of doshas 'meda evam upchiyate na tathetar dhatava.' i.e. further dhatu develop gradually. The Dhatus prior to it also develop Kshaya. Meda itself is also having vikrit vriddhi but there is prakrut medakshaya. i.e all the dhatues becomes Ksheena and shithil. Vikrit Meda can produce abnormality in Sira and produces Siradaurbalya.Here bahuabddameda is Dushyavishesha.

Arundatta commented that, it is important to occur meda-kshaya, which produces. Ayushorhasa, javoparadha, krucharvyavyata, general debility, daurgandhya, Swedabadha, excessive appetite and thrust.

V) Majjadhatu: -

If Majja dhatu vitiated, Vatakshaya occurs. Vitiatied majja produces symptoms like netrang gaurava, angagaurava in Madhumeha. Due to Vataprakop with majja excessive mutra secretes leading to majjameha.

VI) Shukradhatu: -

Prameha is a kulaja vikara and occurs because of beeja dosha. Shukra possesses an important role in Sahaj Prameha. Apana and vyan are the main causative factors for shukradosha and Prameha. Due to vata aggrivation shukrakshaya occurs, and this vata causes to shukrameha also.

VII) Oja: -

Oja is the main dushya of madhumeha. Due to vataprakopa and avaran of oja it carries towards basti and excretes through urine.

Due to ojakshya symtoms like gurugatrata, nidra, tandra, daurbalya are produced Ojakshaya produces rukshata[156],so one can easily understand manifestations of Krushapramehi or Sahaj pramehi.

VIII) Kleda: -

Kleda is an important dushya in Prameha. The literary meaning of kleda is wetness, moisture. Kleda establishes abdhatwa in the dhatus. Increased kleda increases bahudravshleshma and when dosha and dushya gets vitiated with this bahuabddha kleda, it gets converted into mutra and results in an increased frequency of mutra.

Thus, due to vrudha kleda prabhutmutrata, swedavriddhi, saithilya, daurgandhya and avilamutrata are seen.

ix) Vasa: -

Vasa is upadhatu of mamsa and is main dushya is vataj prameha. So vasameha is a type of Vataj Prameha.

x) Lasika:-

Lasika is one type of bodyfluid. Dushti of lasika described in Hastimeha

Xii) Sweda:-

This dushya is separately mentioned by Vagbhatta, and is mainly related with kleda and meda.Sweda aggrivation results in symptoms like swedavruddhi, daurgandhya, picchilagatrata and snigdhagatrata etc.

Only, Shushrut mentioned that in madhumeha sweda becomes sweet in nature.[157]

3. Srotas: -

In Madhumeha along with mutravahasrotas, there is a reference of sroto dushti only related to Mutravaha srotas but as per symptoms, we can easily understand involvement of medovaha, mamsavaha, swedavaha and udakavaha srotas too

9. Sadhyasadhytwa:

Sadyasadyta depends upon Hetu, Nidan, Rupa and Mootrapariksha

i)Sadhya

Kaphaj Prameha is sadhya[158] as dosha and Meda also possesses same properties and dushay are of same gunas.

ii) Kruchhasadhya -

Pittaj prameha and Vataj prameha are Kruchhasadhya. Madhumeha occurs due to avaranjanya and is difficult to treat i.e. kruchsadhya[159]

iii) Yapyatwa:-

Pittaj meha are said to be yapya.[160]

iv) Asadhytwa:-

Vataj prameha is incurable because there is involvement of vital dhatus like majja, oja, and as dosha and Dushays are of Vruddha guna.[161]

Charaka mentioned madhumeha because of bejjadosha is incrurable i. e Asadhy.[162]. Sushruta mentioned madhumeha in association with prameh- pidaka is asadhya.[163] Dhatukshayajanya vataja madhumeha is Asadhaya.

10. Upadrava:

Charak has described the general complications where as Sushrut and Vagbhata have described it according to the dosha predominance.

1. General Updrava: -

Trushna, Atisara, Jwara, daha, daurbalya, Arochak, Apachana Pootimamsa, Pidaka, Alaji and vidradhi. These upadravas takes place due to long term anubandha of Prameha disease .[164]

2.Doshaj updravas: -

Sushrut mentioned twenty upadravas as per Dosha dominanace. These upadavas are as mentioned by Shushrut and Vagbhatta.

A) Kaphaja meha Upadravas :[165]

Makishikosarpanam,Aalsya,Mamsopachya,Pratishyaya,Shaithilya,Arochaka, Avipaka, kaphaprasek, Chhardi, Nidra, Kaasa and Shwasa.

B) Pittaj meha Upadravas:[166]

Vrushanayoavadaranam,Bastibheda,Medratoda,Hridshula,Amlika,Jwara,Atisara, Arochaka,Paridhumayanam,Daha,Murrchha,Pipasa,Nidranasha,Pandurog, Pittavinmutranetratva and Vibheda.

C) Vataj meha Upadravas :[167]

Hridgraha, Laulya, Anidra, Stambha, Kampa, Shula, Buddha purishatvaand shosha, kasa, Shvasa.Prameha and Sthaulya are closely related, therefore the complications of Sthaulya also can be observed in Prameha.

11. Prameha Pidaka-

Brihatttrayi have described Prameha pidaka is a major complication of Prameha.Charaka mentioned 'saptapidaka' as upadrava of madhumeha; [168] where as Sushruta and Vagbhata mentioned ten pidaks, [169] and Kashayap mentioned astha pidaka.[170]

These pidaka may develop without Prameha in the person having medodushti only. Sushruta mentioned that madhumeha along with pidakais asadhya. He told that these pidaka occurs due to tridosha and vitiated meda and mamsa.

These pidaka are as follows:

Table No.19: PRAMEHA-PIDAKA

Pidaka	Charaka	Sushruta	Vagbhatta
Sharavika	+	+	+
Kacchhapika	+	+	+
Jalini	+	+	+
Sarshapi	+	+	+
Alaji	+	+	+
Vinata	+	+	+
Vidradhi	+	+	+
Putrini	-	+	+
Masurika	-	+	+
Vidarika	-	+	+

- प्रमेहिणां या पिडका मयोक्ता रोगाधिकारे पृथगेव सम्।
  ता: शल्यविध्द्धि: कुशलैचिकित्स्या: शस्त्रेण संशोधनरोपणैश्च्॥
  च. चि.६/५८
- शराविकाद्या पिटीका: शोफ़वत समुपाचरेत।
  अपक्का व्रणवत्पक्वा:| तासां प्राग्रुपं एव च।
  क्षिरि व्रुक्षांम्बु पानाय बस्तमुत्रं च शस्यते।
  तीक्ष्णं च शोधनं प्रायो दुर्विरेच्य हि मेहिन::। अ. ह्रु.चि. १२ /८

Charak suggests that these pidakas are treated by sansodhan and ropan aushadhaies, by a good surgeon.If these pidakas are not treated in particular time, can be it converted into vrana; and these pidakas requires surgical intervention.

12. Differential Diagnosis:

To differentiate Prameha, from other diseases, Charaka has explained the presence or absence of poorvaroopas; if colour of urine is haridra or rudira, and it is associated with poorvaroopas of prameha, then we can diagnose prameha. But if poorvaroopa of prameha are absent, it is only rakta and pitta vriddhi.[171]

If patient passes urine as like honey, madhur, Sapiccha, think about diifferntial diagnosis in two ways, one if symptoms are due to dosha-dusya kshya, then it is

considerd as upadrava of Vataj Prameh; and if the symptoms are due to Santarpanjanya hetu then it is considerd as upadrava of Kaphaj Prameha.[172]

Charaka mentioned that, if Medhumeha is present, it is difficult to distinguish from kaphaja mehas.

13. Arishta Lakshana (Fatal signs):

Charaka has mentioned arishta laksanas related to Prameha i.e. the indications of ensured death. He said that if the flies are attracted towords patient after bath patient will die due to Prameha.He also said that if he daily drinks various kinds of oil and ghees in his dream also; he may die due to Prameha.[173]

In Indriyastana, Charak stated that if the madhumehi patient losses his balmamsa; then the Vyadhi becomes Achikitsaya i.e. difficult to cure. Thus, Madhumeha is one of the "Ashtau maharog."[174,175]

3.3. Review of Ulcer and Chronic ulcer

1. History[176]

The science of wound healing has an exciting journey over the ages. Since the caveman, man has been tending to his wounds. Wound care evolved from magical incantations, potions, and ointments, to a systematic text of wound care and surgery from Sushruta, Hippocrates and Celsus.

The Egyptians were masters in applying and arranging bandages, and they recognized the cardinal signs of infection and inflammation.Egyptian drug therapy can be regarded as having evolved from a system rooted in magic and empirical observation. Pressure sores have been found on 5,000-year-old mummies in Egypt. The ancient Egyptians used honey as a wound treatment.

The 1650 B.C. Edwin Smith Surgical Papyrus, a copy of a much older document, describes at least 48 different types of wounds. A later document (Ebers Papyrus, 1550 B.C.) relates the use of concoctions containing honey (antibacterial properties), lint (absorbent properties), and grease (barrier) for treating wounds. These same properties are still considered essential in contemporary daily wound management. Yet another very early account of wound healing dating back to about 2000 B.C. suggests that the Sumerians employed two modes of treatment: a spiritual method consisting of incantations and a physical method of applying poultice-like materials to the wound.

Hippocrates, a Greek physician and surgeon, 460-377 B.C., known as the father of medicine, used vinegar to irrigate open wounds and wrapped dressings around wounds to prevent further injury. He washed ulcers with wine and after having softened them by oil, he dressed them with fig leaves. Galen, a notable Roman surgeon, was first to recognize that pus from wounds inflicted by the gladiators preceded wound healing.

Plinio used mineral remedies as lead and silver, Galen used spice ointments. These advances achieved in wound care and surgery for healing wounds by Hippocrates and Celsus were lost after the fall of the Roman Empire.In Europe, the middle ages were a regression of wound care back to potions and charms. It was not until the time when

large armies started using muskets and cannons, that surgical wound care emerged again.

The 19th century brought significant advances in wound treatment. Joseph Lister, a Professor of Surgery in London, recognized that antisepsis could prevent infection. Lister placed carbolic acid into open fractures to sterilize the wound and prevent sepsis. Changes were also made to sterilize the surroundings of a wounded patient. Hand washing prior to care along with sterilization of instruments as well as wearing of gowns, masks and gloves began in 1880s.

The scale of wound infections was most evident in times of war. During the American Civil War, infected wounds accounted for some 17,000 deaths. World War I brought new types of wounds from advanced weaponry and contamination from the trenches. A Belgian military surgeon, Depage, introduced wound debridement and delayed wound closure and would use microbiological assessment to determine if wound was safe for closure.

The use of antibiotics usheredin a new era in wound care. Penicillin was first used clinically in 1940. However, the use of antibiotics did not end wound infections as resistant bacteria and new surgical interventions has risen. The primary method for wound management is prevention. The use of systemic antibiotics and surgical drainage and excision of damaged tissue are primary methods of wound management in present day.

2. Definition of Ulcer: [177,178]

A lesion on the surface of the skin or mucous surface caused by superficial loss of tissue usually with inflammation.

An open sore or lesion of skin or mucous membrane accompanied by sloughing of inflamed necrotic tissue

An injury especially one in which in skin or another external surface is torn pierced, cut or otherwise broken, it is the discontinuity or break of the surface.Though both ulcer and wound have some similar and dissimilar features, they mean the same– disruption in epitheliail surface.A wound with superficial loss of tissue from trauma is not primarily an ulcer, but may become ulcerated if infection occurs.

Chronic ulcers: Chronic ulcers are the wounds that fail to heal; in general, they have a fibrotic margin and a bed of granulation tissue which may include areas of slough (necrotic tissue).

3. Physiological and Pathological consideration of wound healing: [189]

Wound healing is a dynamic, interactive process involving soluble mediators, blood cells, extracellular matrix and parenchymal cells. Wound healing has three phases' inflammation, tissue formation, and tissue remodeling that overlap in time.

A) **Inflammation:**

Tissue injury causes the disruption of blood vessels and extravasation of blood constituents. The blood clot reestablishes hemostasis and provides a provisional extracellular matrix for cell migration. Platelets not only facilitate the formation of a hemostatic plug but also secrete several mediators of wound healing, such as platelet-derived growth factor, that attract and activate macrophages and fibroblasts. However, in the absence of hemorrhage, platelets are not esential to wound healing. Numerous vasoactive mediators and chemotactic factors are generated by the coagulation and activated-complement pathways and by injured or activated parenchymal cells.These substances recruit inflammatory leukocytes to the site of injury. Infiltrating neutrophils cleanse the wounded area of foreign particles and bacteria and are then extruded with the eshar or phagocytosed by macrophages.

In response to specific chemo attractants, such as fragments of extracellular-matrix protein, transforming growth factor β, and monocyte chemo attractant protein 1, monocytes also infiltrate the wound site and become activated macrophages that release growth factors such as platelet-derived growth factor and vascular endothelial growth factor, which initiate the formation of granulation tissue. Macrophages bind to specific proteins of the extracellular matrix by their integrin receptors, an action that stimulates phagocytosis of microorganisms and fragments of extracellular matrix by the macrophages.

Adherence to the extracellular matrix also stimulates monocytes to undergo metamorphosis into inflammatory or reparative macrophages. Adherence induces monocytes and macrophages to express colony- stimulating factor 1, a cytokine necessary for the survival of monocytes and macrophages; tumor necrosis factor a, a

potent inflammatory cytokine; and platelet-derived growth factor, a potent chemo attractant and mitogen for fibroblasts. Other important cytokines expressed by monocytes and macrophages are transforming growth factorα, interleukin-1, transforming growth factor β, and insulin like growth factor I. The monocyte- and macrophage-derived growth factors are almost certainly necessary for the initiation and propagation of new tissue formation in wounds; because macrophage depleted animals have defective wound repair. Thus macrophages appear to have a pivotal role in the transition between inflammation and repair.

B) Epithelialization:

Epithelialization of wounds begins within hours after injury. Epidermal cells from skin appendages such as hair follicles quickly remove clotted blood and damaged stroma from the wound space. At the same time, the cells undergo marked phenotypic alteration that includes retraction of intracellular tonofilaments dissolution of most inter-cellular desmosomes, which provide physical connections between the cells; and formation of peripheral cytoplasmic actin filaments, which allow cell movement. Furthermore, epidermal and dermal cells no longer adhere to one another, because of the dissolution of hemidesmosomal links between the epidermis and the basement membrane, which allows the lateral movement of epidermal cells. The expression of integrin receptors on epidermal cells allows them to interact with a variety of extracellular-matrix proteins (e.g., fibronectin and vitronectin) that are interspersed with stromal type I collagen at the margin of the wound and interwoven with the fibrin clot in the wound space.The migrating epidermal cells dissect the wound,separating desiccated eschar from viable tissue. The path of dissection appears to be determined by the array of integrins that the migrating epidermal cells express on their cell membranes.

The degradation of the extracellular matrix, which is required if the epidermal cells are to migrate between the collagenous dermis and the fibrin eschar, depends on the production of collagenase by epidermal cells, as well as the activation of plasmin by plasminogen activator produced by the epidermal cells.Plasminogen activator also activates collagenase (matrixmetalloproteinase1) and therefore facilitates the degradation of collagen and extracellular-matrix proteins.

One to two days after injury, epidermal cells at the wound margin begin to proliferate behind the actively migrating cells. The stimuli for the migration and proliferation of epidermal cells during reepithelialization have not been determined, but several possibilities exist. The absence of neighbour cells at the margin of the wound (the "free edge" effect) may signal both migration and proliferation of epidermal cells.Local release of growth factors and increased expression of growth-factor receptors may also stimulate these processes.Leading contenders include epidermal growth factor,transforming growth factor a, and keratinocyte growth factor. As reepeithelialization ensures, basement-membrane proteins reappear in a very ordered sequence from the margin of the wound inward, in a zipper like fashion. Epidermal cells revert to their normal phenotype, once again firmly attaching to the reestablished basement membrane and underlying dermis.

C) **Formation of Granulation Tissue:**

New stroma, often called granulation tissue, begins to invade the wound space approximately four days after injury.Numerous new capillaries endow the new stroma with its granular appearance.Macrophages, fibroblasts, and blood vessels move into the wound space at the same time. The macrophages provide a continuing source of growth factors necessary to stimulate fibroplasia and angiogenesis; the fibroblasts produce the new extracellular matrix necessary to support cell in growth; and blood vessels carry oxygen and nutrients necessary to sustain cell metabolism. Growth factors, especially platelet-derived growth factor 4 and transforming growth factor β 1, in concert with the extracellular-matrix molecules, presumably stimulate fibroblasts of the tissue around the wound to proliferate, express appropriate integrin receptors, and migrate into the wound space. Indeed, platelet-derived growth factor accelerates the healing of chronic pressure sores and diabetic ulcers, and basic fibroblast growth factor has been used with some success to treat chronic pressure sores.

The structural molecules of newly formed extracellular matrix, termed the provisional matrix, contribute to the formation of granulation tissue by providing a scaffold or conduit for cell migration. These molecules include fibrin, fibronectin, and hyaluronic acid. In fact, the appearance of fibronectin and the appropriate integrin receptors that bind fibronectin, fibrin,or both on fibroblasts appears to be

the rate-limiting step in the formation of granulation tissue.The fibroblasts are responsible for the synthesis, deposition, and remodeling of the extracellular matrix. Conversely, the extracellular matrix can have a positive or negative effect on the ability of fibroblasto synthesize, deposit, remodel, and generally interact with the extracellular matrix. Cell movement into a blood clot of cross-linked fibrin or into tightly woven extracellular matrix may require an active proteolytic system that can cleave a path for cell migration.A variety of fibroblast derived enzymes, in addition to serum-derived plasmin,are potential candidates for this task, including plasminogen activator, collagenases, gelatinase A, and stromelysin. After migrating into wounds,fibroblasts commence the synthesis of extracellular matrix.The provisional extracellular matrix is gradually replaced with a collagenous matrix, perhaps as a result of the action of transforming growth factor β1. Once an abundant collagen matrix has been deposited in the wound, the fibroblasts stop producing collagen, and the fibroblast-rich granulation tissue is replaced by a relatively acellular scar.Cells in the wound undergo apoptosis 35 triggered by unknown signals. Dysregulation of these processes occurs in fibrotic disorders such as keloid formation, morphea, and scleroderma.

D) Neovascularization:

The formation of new blood vessels is necessary to sustain the newly formed granulation tissue. Angiogenesis is a complex process that relies on extracellular matrix in the wound bed as well as migration and mitogenic stimulation of endothelial cells.

The induction of angiogenesis was initially attributed to acidic or basic fibroblast growth factor. Subsequently, many other molecules have also been found to have angiogenic activity, including vascular endothelial growth factor, transforming growth factor β, angiogenin, angiotropin, angiopoietin 1, and thrombospondin, to name but a few.Low oxygen tension and elevated lactic acid may also stimulate angiogenesis. Many of the molecules mentioned above appear to induce angiogenesis by stimulating the production of basic fibroblast growth factor and vascular endothelial growth factor by macrophages and endothelial cells. Activated epidermal cells of the wound secrete large quantities of vascular endothelial cell growth factor. Basic fibroblast growth factor may set the stage for angiogenesis during the first three

days of wound repair, whereas vascular endothelial-cell growth factor is critical for angiogenesis during the formation of granulation tissue on days 4 through 7.

In addition to angiogenesis factors, appropriate extracellular matrix and endothelial receptors for the provisional matrix are necessary for angiogenesis. Proliferating microvascular endothelial cells adjacent to and within wounds transiently deposit increased amounts of fibronectin within the vessel wall. Since angiogenesis appears to require the expression of functional fibronectin receptors by endothelial cells, the perivascular fibronectin may act as a conduit for the movement of endothelial cells into the wound. Protease expression and activity are also necessary for angiogenesis.

The series of events leading to angiogenesis may be as follows. Injury causes destruction of tissue and hypoxia. Angiogenesis factors such as acidic and basic fibroblast growth factor are immediately released from macrophages after cell disruption, and the production of vascular endothelial-cell growth factor by epidermal cells is stimulated by hypoxia. Proteolytic enzymes released into the connective tissue degrade extracellular-matrix proteins. Fragments of these proteins recruit peripheral-blood monocytes to the site of injury, where they become activated macrophages and release angiogenesis factors. Certain macrophage angiogenesis factors, such as basic fibroblast growth factor, stimulate endothelial cells to release plasminogen activator and procollagenase. Plasminogen activator converts plasminogen to plasmin and procollagenase to active collagenase, and in concert these two proteases digest basement membranes. The fragmentation of the basement membrane allows endothelial cells stimulated by angiogenesis factors to migrate and form new blood vessels at the injured site. Once the wound is filled with new granulation tissue, angiogenesis ceases and many of the new blood vessels disintegrate because of apoptosis 46. This programmed cell death probably is regulated by a variety of matrix molecules, such as thrombospondins 1 and 2, and antiangiogenesis factors, such as angiostatin, endostatin, and angiopoietin 2.

4) Wound Contraction and Extracellular-Matrix Reorganization:

Wound contraction involves a complex and superbly orchestrated interaction of cells, extracellular matrix, and cytokines. During the second week of healing, fibroblasts assume a myofibroblast phenotype characterized by large bundles of actin-

containing microfilaments disposed along the cytoplasmic face of the plasma membrane of the cells and by cell–cell and cell–matrix linkages. The appearance of the myofibroblasts corresponds to the commencement of connective-tissue compaction and the contraction of the wound.

The contraction probably requires stimulation by transforming growth factor β1 or β 2 and platelet-derived growth factor, attachment of fibroblasts to the collagen matrix through integrin receptors, and cross-links between individual bundles of collagen. Collagen remodeling during the transition from granulation tissue to scar is dependent on continued synthesis and catabolism of collagen at a low rate. The degradation of collagen in the wound is controlled by several proteolytic enzymes termed matrix metalloproteinases, which are secreted by macrophages, epidermal cells, and endothelial cells, as well as fibroblasts17. The various phases of wound repair rely on distinct combinations of matrix metalloproteinases and tissue inhibitors of metalloproteinases.

5) Tensile strength:

Wounds gain only about 20 percent of their final strength in the first three weeks, during which time fibrillar collagen has accumulated relatively rapidly and has been remodeled by contraction of the wound. There after the rate at which wounds gain tensile strength is slow, reflecting a much slower rate of accumulation of collagen and, more important, collagen remodeling with the formation of larger collagen bundles and an increase in the number of intermolecular cross-links. Nevertheless, wounds never attain the same breaking strength (the tension at which skin breaks) as uninjured skin. At maximal strength, a scar is only 70 percent as strong as normal skin.

6. Clinical signs of infection:

Although a detailed discussion of the many conditions associated with abnormal ulcer healing is beyond the scope of this review, several examples will illustrate the multifactorial nature of these conditions. Diabetic ulcers are an excellent example of how multiple physiologic and biochemical defects can lead to impaired healing. They usually occur in patients who are unable to sense and relieve cutaneous pressure because of neuropathy. Ischemia secondary to vascular disease impedes

healing by reducing the supply of oxygen and other nutrients. Diabetic ulcers are also prone to infection because of impaired granulocytic function and chemotaxis. Other abnormalities associated with diabetic ulcers include prolonged inflammation, impaired neovascularization, decreased synthesis of collagen, increased levels of proteinases, and defective macrophage function. Keloids and hypertrophic scars that are characterized by excess accumulation of collagen within the wound are examples of fibroproliferative disorders. In these conditions, abnormalities in cell migration and proliferation, inflammation, synthesis and secretion of extracellular-matrix proteins and cytokines, and remodeling of the wound matrix have all been described. Increased activity of fibrogenic cytokines (e.g., transforming growth factor β1, insulin-like growth factor 1, and interleukin-1) and exaggerated responses to these cytokines have also been noted. In addition, abnormal epidermal–mesenchymal interactions and mutations in regulatory genes have recently been proposed to help explain abnormal healing.

7. Clinical experience with growth factors:

The overall clinical experience with growth factors and other mediators to accelerate wound healing has been discouraging. This is not surprising, considering that wound repair is the result of a complex set of interactions among soluble cytokines, formed blood elements, extracellular matrix, and cells. It is possible that combinations of various growth factors given at precisely timed intervals would be more effective in promoting healing. Indeed, synergistic effects on wound repair have been demonstrated for several growth-factor combinations65. Among these factors, only recombinant platelet-derived growth factor has been approved by the Food and Drug Administration (FDA) for the treatment of wounds.

8. Insights from fetal wound healing:

Fetal wounds reepithelialize rapidly. Unlike adult epidermal cells, which resurface the wound by “crawling” across it, embryonic epidermal cells are pulled forward by the contraction of actin fibers that draw the wound edges together as the opening of a purse is closed by a purse string. Fetal wounds also heal without scarring. One reason for this may be the small amount of transforming growth factor β1, a scarpromoting cytokine, in fetal skin. The addition of transforming growth factor β1 to fetal wounds results in scarring. Furthermore, fetal skin is rich in

metalloprotein that may promote scar less healing. Scarring is reduced in adult rats given neutralizing antibodies to transforming growth factors β1 and β2 and those given transforming growth factor β3, which down-regulates the other transforming growth factor β isoforms. This result supports the central role of transforming growth factor β1 in scar formation.

9. Cytokines important in ulcer healing:

Cytokine- major source- target cells and major effects:

1) Epidermal growth factor- family- Epidermal and mesenchymal regeneration
2) Epidermal growth factor- Platelets- Pleiotropic-cell motility and proliferation
3) Transforming growth factor α- Macrophages, epidermal cells -Pleiotropic-cell motility and proliferation Heparin-binding epidermal growth factor- Macrophages Pleiotropic-cell- motility and proliferation
4) Fibroblast growth factor-family- Wound vascularization
5) Basic fibroblast growth factor-Macrophages-endothelial cells Angiogenesis and fibroblast proliferation
6) Acidic fibroblast growth factor -Macrophages, endothelial cells- Angiogenesis and fibroblast proliferation
7) Keratinocyte growth factor- Fibroblasts Epidermal-cell motility and proliferation
8) Transforming growth factor β-family -Transforming growth factors β 1 andβ2- Platelets, macrophages - Fibrosis and increased tensile strength, Epidermal-cell motility, chemotaxis of macrophages and fibroblasts, extracellular-matrix synthesis and remodeling.
9) Transforming growth factor β 3-Macrophages - Antiscarring effects.
10) Other Platelet derived growth factor- Platelets- macrophages, epidermal cells Fibroblast proliferation and chemo attraction, macrophage chemo attraction and activation.
11) Vascular endothelial growth factor- Epidermal cells, macrophages- Angiogenesis and increased vascular permeability.
12) Tumor necrosis factor α-Neutrophils- Pleiotropic expression of growth factors.
13) Interleukin-1- Neutrophils- Pleiotropic expression of growth factors.
14) Insulin-like growth factor I- Fibroblasts, epidermal cells- Re-epithelialization and granulation-tissue formation.

15) Colony stimulating factor 1 -Multiple cells- Macrophage activation and granulation tissue formation.

10. Classification of ulcers: [180]

A. Non-specific ulcer

B. Specific ulcers

C. Malignant ulcers

A. Non-specific ulcer:

1**) Traumatic ulcer**: Any trauma to the skin. Bony proneness are more prone for the ulcers. Trauma may be physical, chemical, mechanical. They are mostly irregular in shape, painful.

2**) Infective**: The primary ulcers are infected by various microorganisms. These are small or multiple, with discharge.

3) **Arterial ulcer/ Ischemic ulcer**: These are due poor peripheral circulation. This may see in various arterial diseases like emboli, atherosclerosis, Diabetes, vasculitis, etc. These ulcers mostly seen on the anterior and outer aspects of the leg, dorsum of the foot, on the toes or the heel. Pain are the main symptom with punched out edge.

4) Venous ulcers: These ulcers are due to hypertension in vein mostly seen in varicose vein, Thrombosis and Phlebitis.

Venous ulcers are most common on inner side just above medial malleolus of leg. The shape is usually ovoid, single in number with irregular, thin blue margin, pale granulation tissue, Pigmentation and eczema is seen in the vicinity of ulcer. These ulcers are usually shallow. Pain is minimal mostly in initial period.

5) Neurogenic ulcers: Occurs due to, impairment of the nutrition of tissues, inadequate blood supply and neurological deficit or repeated trauma to the insensitive part of the body. These ulcers mostly seen on heel when patient is non-ambulatory. Edge is punched out, base is slightly indurated and floor is covered with slough. Surrounding skin has no sensation and these are painless. E.g. Bed sore, perforating ulcers.

6) Martorells ulcer: Mostly seen in hypertensive/ atherosclerotic peoples.

7) Cryopathic ulcer: These result from cold trauma.

8) Tropical ulcer: Characteristic feature of this ulcer is callousness towards healing. Edge is slightly raised and exudes copious serosanguineous discharge. Pain is an important symptom. In some cases, it destroys surrounding tissue and spreads widely.

9) Diabetic ulcer: The small injury to the diabetic patients' tissue may cause chronic infection and ulcer formation. Ulceration is worsening by ischemia due to diabetic atherosclerosis, infection or peripheral neuritis. The sensation is reduced in chronic cases. Toes and feet are normally affected.

10) Miscellaneous ulcers: Ulceration mostly seen in anemia, leukemia, polycythemia, systemic sclerosis, Rheumatoid arthritis, ulcerative colitis, poliomyelitis, arteriovenousfistula, various collagen disorders, chronic lymph edema, and cortisone ulcers etc.

(B) Specific ulcers: These are seen in T.B, syphilis, soft chancres, leprosy, Actinomycosis and Meleny's ulcer

1) Tuberculous ulcer: Seen in bursting of cold abscess; may form tuberculosis lymphnode, TB of bone or joint.It is oval generally with irregular crescent border, often multiple in number.

2) Syphilitic ulcer: Ulcers due to syphilis are seen in all 3 stages. Single, painless, indurated base, oval to round and deep saucer shape.

3) Soft chancre: Mostly seen on genital part. These are often painful, gradually become pustular and ulcerate to form soft sores.These are multiple, soft, rounded painful, and readily bleed and edges are undermined.

4) Actinomycosis: This condition causes multiple ulcers. At first area becomes indurated, nodules appear, which soften and later ulcerates in various places. Surrounding skin often looks bluish in colour. Discharges yellow colour sulphur granules.

5) Leprosy ulcer: Mainly seen in the extremities of foot and hand. It is due to peripheral neuropathy and pressure induced parenchyma

6) Meleny's ulcer: These are in post-operative wounds commonly seen over abdomen, thorax, etc., it is very painful with signs of toxemia. It has undermined edges, foul smelling, granulation tissue with seropurulent discharge.

C) Malignant Ulcer : Rodent ulcer/Basal cell carcinoma :

It is locally invasive carcinoma of basal layer of epidermis. It is of low grade malignancy. It is commonly seen on the face, above line from corner of mouth to ear, innercanthus of eye, nose on and around nasolabial fold, has risen and pearly white

beded edge, irregular in shape and floor covered with coat of dried serum, epithelial cells.

i) Epithelioma (squamous cell carcinoma): This occurs commonly, in the dorsum of hands, in the face, limbs, lips, vulva, penis etc. It has normal temperature and usually not tender, oval or circular in shape with raised and everted edge, indurated base and floor is covered by necrotic tumor, serum & blood.

ii) Marjolins ulcer: It is the name given to a squamous cell carcinoma which arises in a chronic benign ulcer or scar. It is slow growing malignant lesion, painless and edge is not always raised and everted.

11. Classification of wound: [181,182]

Wounds can be classified into 5 types

1). Incised wounds- Caused by sharp objects, edges of the wound are sharp. Tends to gape and bleed freely.

2). Lacerated wounds- Causedby bluntobjects, edges of the wound are jagged. Causes minimal bleeding because of crushing.

3). Penetrating wounds- (variation of punctured wound) - Stab injuries of abdomen are notorious, depth is more.

4). Crushed or contused wounds- Caused by blunt trauma.

5). Abrasion- Caused by scraping away of superficial skin layer and is very painful.

12. Causes of ulcer: [183]

1) Vascular: venous, arterial, lymphatic, vasculitis

2) Neuropathic: diabetes, spina bifida, leprosy

3) Metabolic: diabetes, gout

4) Connective tissue disease: Rheumatoid arthritis, Scleroderma, Systemic lupus erythematosus, Pyoderma gangrenosum (often reflection of systemic disorder)

5) Haematological disease: red blood cell disorders; sickle cell disease, white blood cell disorders: Leukaemia, plateletdisorders: Thrombocytosis

6) Dysproteinaemias: cryoglobulinaemia, amyloidosis

7) Immunodeficiency: HIV, immunosuppressive therapy

8) Neoplastic: basal cell carcinoma, squamous cell carcinoma, metastatic disease

9) Infectious: bacterial, fungal, viral**.**

10) Traumatic: pressure ulcer, radiation damage.

11) Iatrogenic: drugs induced.

13. Factors Influencing Ulcer Healing:

A) Local factors: [183]

- Inadequate blood supply
- Increased skin tension
- Poor surgical apposition
- Wound dehiscence
- Poor venous drainage
- Presence of foreign body and foreign body reactions
- Continued presence of microorganisms
- Infection
- Excess local mobility, such as over a joint

B) Systemic factors: [183]

- Advancing age and general immobility
- Obesity
- Smoking, Alcohol, Tobaco chiwing
- Malnutrition
- Deficiency of vitamins and trace elements
- Systemic malignancy and terminal illness
- Chemotherapy and radiotherapy
- Immunosuppressant drugs,
- corticosteroids, anticoagulants
- Connective tissue disorders
- Impaired macrophage activity
- (Malacoplakia)

14. Forms of Ulcer Healing: [184]

I) Healing By 1st Intension:

In simple and non-infective ulcer of healthy person healing by minimum scar formation.

II) Healing By 2nd Intension:

In marked tissue destruction ulcer can heal without edges approximation by any surgical procedure.

III) Healing By 3rd Intension:

Frist wound can heal naturally, when the ulcer is free of infection then it is surgically closed.

15. Abnormal wound healing: [183]

- Sinus and Fistula formation
- Malignancy
- Osteomyelitis
- Contractures and deformity in surrounding joints
- Systemic amyloidosis
- Heterotopic calcification
- Colonization by multiple drug
- Resistant pathogens, leading to antibiotic resistance
- Anemia
- Septicemia
- Excess scar and Keloid formation due to excess deposition of extracellular matrix at ulcer site.
- Deficient Scar Formation: found I ulcer in which the granulation tissue is inadequate
- Dehiscence of ulcer Herniation of wound: Seen due to increased pressure within ulcer. Mostly seen in abdominal ulcer.
- Ulceration: Due to inadequate blood supply and perfusion as in Varicose ulcer, Atherosclerosis, Neuropathic ulcers

16. Laboratory investigations before treating a wound: [183]

- **Hemoglobin**-Anemia may delay healing
- **White cell count**- Infection
- **Platelet count**- Thrombocytopenia
- **Erythrocyte sedimentation rate (E.S.R.) and C-reactive protein** -Non-specific markers of infection and inflammation; useful in diagnosis and monitoring treatment of infectious or inflammatory ulceration.
- **Urea and creatinine**–High urea impairs wound healing. Renal function important when using antibiotics.
- **Albumin Protein loss**- delays healing

- **Glucose, hemoglobin A1C**- Diabetes mellitus
- **Markers of autoimmune disease-**Such as rheumatoid factor, antinuclearantibodies, anticardiolipin antibodies, lupus anticoagulant Indicative of rheumatoid disease, SLE and other connective tissue disorders. Cryoglobulins, cryofibrinogens, prothrombin time, partial thromboplastin time -Haematological diseases.
- **Deficiency or defect of:** Antithrombin III, protein C, proteinS, factor V LeidenVascularThrombosis.
- **Haemoglobinopathy screen Sickle cell anemia, Thalassemia**
- **HIV status** -Kaposi's sarcoma
- **Serum protein electrophoresis**-Bence-Jones proteins Myeloma
- **Urine analysis** - Protein loss, Urinary tract infection, connective tissue disease
- **Kidney function test:** BUN, serum Creatinine, Serum Electrolytes
- **Liver function test:** Liver cirrhosis, Jaundice, Liver parenchymal disease.
- **Wound swab:** culture and sensitivity of infective organism.

17. Non-healing Ulcers: [185-188]

A wound or ulcers which does not reduce in area (simple length x width) by at least 50% in 4 weeks, has greater than 90% likelihood of non-healing at 12 weeks, should an ulcer progressing too slowly, it is appropriate to alter the modality to restart healing.

Non-healing Ulcers: have traditionally been defined as those that fail to progress through an orderly sequence of repair in a timely fashion. Such wounds are sometimes thought of as being caused by neglect, incompetence, misdiagnosis, or inappropriate treatment strategies. However, some wounds are resistant to all efforts of treatment aimed at healing, and alternative end points should be considered; measures aimed at improving the quality of life will be paraamount in these instances.

A chronic ulcer is defined as a wound that does not heal within an expected time frame (i.e. 6 weeks) despite optimal correction of any underlying pathological processes interfering with the body's normal process of wound healing. Most of these wounds fall into three types: Venous ulcers; Pressure ulcers; and Diabetic ulcers

18. Clinical features of Non-Healing Ulcer: [189]

- Absence of healthy granulation tissue
- Presence of necrotic and unhealthy tissue in the wound bed
- Excess exudate and slough
- Lack of adequate blood supply
- Failure of re-epithelialisation
- Cyclical or persistant pain
- Recurrent breakdown of wound
- Clinical or subclinical infection

19. Prevalence of Chronic Ulcers: [190-194]

The study from India shows that etiology of chronic ulcers included systemic conditions such as diabetes, atherosclerosis, tuberculosis, and leprosy. Other major causes included venous ulcers, pressure ulcers, vasculitis, and trauma. The study report stated that inappropriate treatment of acute traumatic wounds was the most common cause of the chronic ulcer.[190]Chinese study shows that the principle etiology (67%) of ulceration is trauma or traumatic wounds compounded by infection. Diabetic ulcers, venous ulcers, and pressure ulcers accounted for 4.9%, 6.5%, and 9.2%, respectively. Most of these wounds were seen in farmers and other agricultural workers.[191-192]

It has been reported that ulcers related to venous insufficiency constitute 70%, arterial disease 10%, and ulcers of mixed etiology 15% of leg ulcer presentations.[193]The remaining 5% of leg ulcers result from less common pathophysiological causes, and this latter group comprise considerable challenges in diagnosis, assessment, and management.[194]

One of major cause of non-healing ulcers is Diabetes. For the treatment of chronic Diabetic ulcer, one should know the pathophysiological understanding very well.

20. Pathophysiology of Diabetic Ulcer: [195]

The major contributing factors in Pathophysiology are Prolonged Hyperglycemia, Neuropathy, Ischemia, and Infection.

1 .Diabetic Neuropathy:

The changes ofNeuropathyin Diabetes are developing in Sensory, Motor and Autonomic nervous system. The prolonged Hyperglycemia increases enzyme action of Aldose Reductase and Sorbitol Dehydrogenase. These enzymes convert intracellular glucose to Sorbitol and Fructose. The accumulation of these sugar molecules in nerve result in decrease synthesis of Myonositol in nerve this enzyme is essential in normal nerve conduction signals. The enzymatic and metabolic changes in Diabetes get accumulate in peripheral nerve which reduces sensations.

In sensory neuropathy, there is loss of sensations of sole and peripheral skin.This result in repeated trauma. In Motor Neuropathy, the muscles of feet are affected more. This result in reduce control on pedal muscles. The unequal distribution of force while walking result in pressure point in feet. The pressure point skin is thickened and results in formation of Callus on feet. InAutonomicNeuropathy, there is loss of control on peripheral blood vessels. This result in Ischemic changes in local circulation. The Autonomic Neuropathy also results in dryness of skin and hair fall. The ultimate effect of this result in fissuring of skin. The thick callus formation in feet acts as a foreign body for feet. The pressure created by callus causes brushing and extravasation of blood and serum from micro capillary. The dry skin and fissuring skin creates readymade culture media for bacteria result in ulcer formation.

2) Vasculopathy:

The chronic Diabetes affects small as well as large blood vessels.

A) Microangiopathy: The occlusion of Arterioles and Capillaries result in patchy ischemic changes which result in gangrene formation. The acid Schiff positive causes thickening of small vessels. Also, the muscular control of vessels is losses due to Autonomic Neuropathy.

B) Macroangoipathy: Formation of Atheroma plaques in vessel wall is predisposing for thrombus formation.

C) Monckeberg's sclerosis: The thickening of blood vessel due to calcification of muscular coat of arteries.

D) Intimal fibrosis: This type of thickening of arterial vessel is due to normal aging phenomenon.

3) Infection:

In chronic Diabetes patient, the prolonged Hyperglycemic media is available at fissuring or crack site. The defense mechanism in chronic cases is also low. This form the nedus place for bacteria to grow. The underlying abces and cellulitis also causes poor glycemic control of body. This creates viscous cycle of hyperglycemia and infection. Infectionare due to surface organisms like Staphylococcus, Streptococcus.Other organism are aerobic gram positive and gram negative like Ecoli, Klebsella, and Proteusexcetra.

21. Ulcer Assessment guidelines: [196]

1. History: The chronicity of Diabetes and poor control of Diabetes result in complication of Diabetes like Neuropathy, Vasculopathy, Nephropathy, Retinopathyetc.The history of Hypertension, habits like Alcohol, tobacco, Smoking, Obesity is also important in Vasculopathy and defense mechanism. Family history is also important in disease prognosis. Occupation history is important for pressure distribution on ulcer and Glycemic control.

2. Occupation: Pressure phenomenon, work culture-Venous ulcer in long standing people, Neuropathy in drivers etc.

3. Ulcer examination guidelines:

A. General examination:

- The Nutrition, Gait, Psychological, Edema, Anemia, pigmentation
- Systemic examination: CVS, RS, PA, CNS
- Feet Examination: Shape, Size, Deformity, Thickening, Callosity

B. Ulcer examination:

- Shape-Oval, Circular, Irregular (Arterial, Venous, Diabetic, Pressure, Other)
- Size-Length, Depth, Breadth of Fistula.
- Skin-Healthy, Dry, Ischemic, Pigmentation, Maceration, Nails.
- Location-Medial malleolus- Venous ulcer,
- Lateral malleolus-Arterial ulcer,

- Plantar surface- Diabetic ulcer,
- Sacrum-Pressure ulcer.
- Edges: Slopping-Venous, Punched-Arterial, Rolled-Basel cell Carcinoma, Everted-Squmaus cell Carcinoma, Purple-Vasculitis, Undermining-Tuberculosis, Syphilis ulcer.
- Bed-Necrotic, Slough, Black
- Secretion: Serous, Pus, Hemosangio
- Granulation: Pink Colour-Healthy Granulation,
- Red Ischemic-Unhealthy infected,
- Black-Necrotic tissue, absent,
- Over granulation-Non-healing tendency
- Odour: Mild, Moderate, Sever, Foul
- Vascular Assessment:

  1) Pallor
  2) Ischemic Changes
  3) Peripheral pulse
  4) Capillary refilling time.
- Neurological Assessment:

  1. Sensory Assessment: Pain, Touch with 10 gm. Monofilament.
  2. Motor Assessment: A) leg deformity- Claw, Charcot
     B) Ulcer pressure points
     C) Tendon reflux
     D) Muscle power.

3.Autonomic Assessment-Dryskin, hairloss, hyperpigmentation, local temperature.

- Infection: Signs of Inflammation, Fever, pain, redness, edema etc. pus discharge
- Osteomyelitis: X-ray examination.

TABLE. No. 20: Table of ulcers signs and symptoms with their differential diagnosis[197]

Signs↓ Type→	Arterial	Venous	Diabetic	Other
Site	Lateral malleolus	Medial malleolus	Planter area	Variable
Size	Small	Small-Large	Small-Large	Variable
Skin	Pale	Variable	Necrotic	Variable
Bed	Pale	Variable	Necrotic	Variable
Shape	Round	Irregular	Round	Round
Edge	Smooth	Irregular	Smooth	Variable
Oder	No	?	++	Few
Pain	+	?	?	+

22. Diabetic Ulcer Grades:[198-199]

Diabetic Ulcer Grades Classification According to Wagner's& Armstrong University of Texas:

Table No.21: Diabetic ulcer grades classification

Grades	0	1	2	3	4	5
A)	Callus or Scar	Superficial Infection	Wound up to Tendon/Capsule	Penetrating to Bone /Joint	Necrotic foot	Entire foot Necrosis
B)	Ischemia	Ischemia	Ischemia	Ischemia	Ischemia	Ischemia
C)	Infection	Infection	Infection	Infection	Infection	Infection
D)	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection

23. Management of DiabeticUlcers: [200]

The aim of any ulcer treatment is to assure quick healing with durability form of healing and with minimal scar formation.

A) General medical and surgical treatment aspects:

- Careful inspection
- Vascular assessment
- Neurological assessment
- Describesite, size, shape, edges, surrounding skin, pain, discharge, ulcerbed, and granulation tissue.
- Routine investigation and special investigations.
- Internal Antibiotics, Analgesics, Medical management of Diabetes, and associated disease.
- Multivitamins, proper Nutrition
- Rest with offloading techniques, use of walker.
- Surgical debridement as per requirement, restore artery supply, vascular reconstruction, drainage, Amputation as per requirement.
- Daily dressing, regular assessment
- Proper footwear.

B) Local management: [201,202]

1. Drainage: The proper drainage of pus, deberies, and gases from hidden pockets by probing and debridement techniques is called drainage.

2. **Debridement**: The process of removal of dead necrotic tissue or foreign material from and around a wound to expose healthy tissue is called debridement.

A. Types of debridement are: [201,202]

1. Surgical debridement: done in wounds with a large amount of necrotic tissue.

2. Mechanical debridement: this technique has been used for decades in wound care. By allowing a dressing to proceed from moist to wet, and manually removing the dressing, is a form of non-selective debridement. It is done in wounds with moderate amount of necrotic debris, hydro therapy, saline irrigation and saline moistened gauze comes under this.

3. Debriment Devices: [203-205]

a) Vacuum assisted closure (VAC): Intermittent negative pressure at 125mm of Hg at ulcer surface promotes the healing process. By improving blood supply, growth factor, nutrients flow.

b) Radiant heat bandages: The heat improves local perfusion, subcutaneous oxygen tension, which enhances healing process.

c) Tropical hyperbaric oxygen therapy: By achieving atmospheric pressure of 1.02 to 1.03 atm at ulcer site stimulates collagen formation, fibroblast growth. Hyper baric oxygen is lethal to anaerobes. The hyperbaric oxygen is administered by using sealed polythene bag over ulcer area. The 100% oxygen is delivered at ulcer area with pressure of 20 to 30 mm of Hg.

d) Whirlpool hydrotherapy: Water under high pressure is used to debride ulcer area.

e) Autolytic: It uses the body's own enzymes and moisture to rehydrate, soften and liquefy the slough. It is achieved with hydro colloids, hydrogels, and transparent films.

4) Chemical debridement: These are chemical desloughing agents.

A) Hydrogen peroxide (H_2O_2): it releases nascent oxygen, allows aerobic bacteria to grow they eat away the slough. Usually it is mixed with acriflavine. Acriflavine is a mild antiseptic and irritant it promotes the growth of granulation tissue.

B) Eusol: Edinbergh University Solution. It is a sodium hypochlorite (NaOCl). It acts by releasing nascent chlorine, which combines with slough to form water soluble complexes. In dilute concentrations it kills fibroblasts, neutrophils and endothelial cells in tissue culture.

Eusol delays the appearance of Hydroxyproline (the amino acid marker of wound collagen content) and prolongs the acute inflammatory response. It has no role in the treatment of open wounds that are clean and healing well with no signs of invasive infection.

C) Povidone iodine: Strong bactericidal for gram positive and negative organisms (it has a broad spectrum of activity but its anti-bacterial effect is reduced by contact with pus or exudate). It should not be used in patients who are sensitive to iodine.

D) Chlorhexidine: It is the topical antiseptic which is effective against a wide range of gram positive and negative organisms and some fungi.

5) Biological: maggot debridement therapy is an old remedy that has been revised and proven to be invaluable in cleansing non-healing wounds. This simple procedure involves the placement using restrictive dressing of live disinfected maggots into non-healing wounds to promote for cleansing of necrotic tissue and initiation of the healing process.

6) Topical agents: [206]

A wide variety of topical wound cleaning agents being available and bacteriostatic agents being promoted for local wound application. Some of them are:

Povidine iodine 2.5%-It is Bactericidal

Chlorhexidine solution: Bactericidal

Bactracin: Antibacterial

Hydrogen peroxide (H2O2)

Dankin's solution (Chlorazene) 0.25%Bacteriocidal

Sucralfate: Its antimicrobial activity is by its macrophage activity. It prevents the release of cytokines from damaged skin cells there by exerting anti-inflammatory and smoothening effect.

- PDGF: Helps to rapidly heal chronic non-healing Ulcers. PDGF derived from patients own blood. (Platelet derived growth factor i.e.PGDF)
- Collagen dressings: The Collagen is obtained from bovine extract. Available I the form of gel granules and incorporated with alginate dressing. Collagen provides additional proteins for healing.
- Hyaluronic acid: Enhances the structural organization of extracellular matrix which increases meiotic activity.
- Oxandrolone: This is anabolic steroid, it is anticatabolic and protein sparing properties which enhances protein synthesis.
- Silver Arglaes: Powerful antibacterial agent like Silver Nitrate, Silver Oxide, Silver Chloride.
- Growth factor: Use of Becaptermin, Vecombinant platelet derived growth factor.
- Human skin: By tissue culture technique as a derma graft.

C) Dressing: [206]

Dressing: Covering the ulcer area with various materials

Feeling dead spaces with medicated gauze to avoid anaerobic organism growth.

Appling the moisture and medicated solution and packing the Ulcer.

Providing proper cushioning and covering ulcer area.

Appling bandaging and sticking for wrapping.

Appling cast or splint to protect and support the ulcer.

Dressing materials:[206]

1) Regular dressing: Cotton and cotton fibers gauze.
2) Conventional dressing: The regular dressing material allows evaporating moisture and entry of microorganisms. Also, these materials have tendency to bind ulcer tissue which causes pain and damage. This lead to develop composite dressing materials like Tule grass fibers, these materials incorporated with Vaseline, antibacterial agent.
3) Synthetic dressing: Films these are polymer sheet coated adhesive on one side. Used in superficial wound.
4) Foam and sprays: These dressing sheets of foamed solution of polymer such as polyvinyl alcohol. These materials have advantage of thermal insulation and thus maintain moisture in ulcer surface. Further they are gas permeable, nonadherent, light and comfortable.

3.4 Review of Diabetes Mellitus:

1. Definition:

Diabetes Mellitus is a clinical syndrome, due to impaired metabolism and characterized by chronic hyperglycemia. There is a greater or lesser impairment in the metabolism of carbohydrates, lipids and proteins.[207] Hyperglycemia is due to deficiency in insulin secretion, or decreased glucose utilization and increased glucose production.[208] The term diabetes was originally introduced to describe the clinical symptom associated with high glucose levels.

2. Prevalence

The prevalence of Diabetes is continued increasing dramatically.It is undoubtedly one of the most challenging health problems in the 21stCentury. According the latest 2016 data from WHO, globally nearly about 422 million adults are living with Diabetes.[209]The global prevalence of diabetes has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population.[210]The International Diabetic Federation estimates in 2013 that 381 million peoples having Diabetes.[211]

The number is projected to almost double by 2030.[212] Diabetes occurs throught the world, but it is most common (type 2) in more developed countries. The increase in diabetes in developing countries is due to the trend of urbanization and life style changes, increasing sedentary lifestyle, less physically demanding work and the global nutrition transition.

The number of people with type 2DM is increasing in every country. Almost half of deaths attributable to high blood glucose occur before the age of 70 years.[213]WHO reports that diabetes will be the 7th leading cause of death in 2030.[214]

India have 69.2 million people living with Diabetes (8.7%) as per the data.[215]India is projected to be capital to109 million individuals with diabetes by 2035.[216]According to International diabetic Fedration, India had more diabetic than any other country in the World. 80% of people with DM living in low and middle-income countries. Presently as many as 50%of people with diabetes are undiagnosed. So, there is need to detect Diabetes early in its course. Considering the importance of the disease we must review some important features of the Diabetes Mellitus.

3. Types: [217]

There are three main types of diabetes mellitus

1) Type 1 Diabetes Mellitus (T1DM): Insulin dependent Diabetes Mellitus (IDDM) or "Juvenile diabetes"
2) Type 2 Diabetes Mellitus (T2DM):Non-insulin dependent Diabetes mellitus (NIDDM) or "Adult"Onset of diabetes is primarily due to lifestyle factors and genetics.
3) Gestational Diabetes (GD): Resembles type 2 diabetes, but occurs during pregnancy and may improve or disappear after delivery.

4. Classification of diabetes mellitus.[218]

IDDM and NIDDM these types were proposed by WHO in 1980 and 1985 have disappeared.

The new classification system identifies four types diabetes mellitus; type1, type 2, other specific and gestational. The etiological classification is as below.

Table no. 22: Etiological Classification of Diabetes Mellitus. Adapted from WHO

I. Typ1 Diabetes mellitus	3. Nicotinicacid
A.Autoimmune	4. Glucocorticoids
B.Idiopathic	5. Thyroid hormones
II.Type2Diabetesmellitus	6. Diazoxide
Ranges from relative insulin deficiency to	7. β-adrenergic agonists
disorders	8. Tiazides
of insulin secretion and insulin resistance	9. Dilantin
III. Other specific types of diabetes mellitus	10. α interferon
A. Genetic defects in β-cell function	ii. Infections
1. Chromosome 12, HNF-1α (MODY 3)	1. Congenital rubeola
2. Chromosome 7, glycosidase (MODY 2)	2. Cytomegalovirus
3. Chromosome 20, HNF-4α (MODY 1)	iii. Infrequent forms of autoimmune
4.Mitochondrial DNA	diabetes
5. Monogenic diabetes	1. Stiff-man syndrome)
B. Genetic defects in insulin action	2. Antibodies against insulin
1. Type A insulin resistance	receptors
2. Leprechaunism	iv. Other syndromes occasionally

3.Rabson-Mendenhallsyndrome 4.Lipotrophicdiabetes C.Disease of the exocrine pancreas 1.Pancreatitis 2.Pancreatectomy/trauma 3.Neoplasia 4.Cysticfibrosis 5.Hemochromatosis 6.Fibrocalcificpancreatopathy D.Endocrinopathies 1.Acromegaly 2.Cushingsyndrome 3.Glucagonoma 4.Pheochromocytoma 5.Hyperthyroidism 6.Somatostatinoma 7.Aldosteronoma i. Pharmacologically or chemically induced 1.Vacor 2. Pentamidine	associated with diabetes 1. Down syndrome 2. Klinefelter syndrome 3. Turner syndrome 4. Wolfram syndrome 5. Friedreich ataxia 6. Huntington's chorea 7. Lawrence-Moon-Biedel syndrome 8. Myotonic dystrophy 9. Porphyria 10. Prader-Willi syndrome **IV. Gestational diabetes mellitus** Occurs in mostly in women during gestation.

5. Clinical Features of Diabetes Mellitus: [219]

Most of the symptoms are similar in both types of diabetes but develop more rapidly in type1 diabetes and more typical. These are

- 3P's; polyuria (increased urination)
- Polydipsia (excessive thirst)
- Polyphagia(excessive appetite)
- Burning/tingling/pricking sensation in the hand or feet.
- Fatigue, feeling of tiredness, lethargy
- Blurred vision
- Pruritis vulvae, Balanitis (genital Candidiasis)
- Unexplained weight loss
- Nocturia

6. Criteria for diagnosis.[220]

The 1997 ADA recommendations for diagnosis of DM focus on (FPG) Fasting Plasma Glucose, while WHO focuses on (OGTT) Oral Glucose Tolerance Test. Here, we are considering about FPG.

i) Random Blood Sugar:

Should be less than 200 mg/dl

Random is defined as any time of day without regard to time since last meal.

ii) Fasting Blood Sugar:

Should be less than 126 mg/dl.

Fasting is defined as no caloric intake per at least 8 hrs.

iii) PPBS (2 hrs.) (Post parandial blood sugar):

Should be less than 140 mg/dl

IN a patient with characteristic sign and symptoms of diabetes, a fasting venous plasma glucose >126 mg/dl, or a random venous plasma glucose >200 mg% confirmed on repeat testing. Diagnostic –ve test for diabetes does not mean that the person will never get DM.

7. Investigations:[221]

- Blood glucose Tests:
- Fasting blood sugar (FBS)
- Post Parndial blood sugar (PPBS)
- Random blood sugar
- Oral glucose Tolerance tests(OGTT)
- Hb A1C (glycosylated haemoglobin)
- Urine tests-glucose, albumin. Ketones, etc.
- Insulin tests
- S. Insulin
- Insulin sensitivity test

Other complimentary tests

- Glycated serum protein (GSP)
- S. Fructosamine
- C. Peptide--It is a simple, cost effective, non-invasive method of assessment of beta cell capacity.
- Blood Urea

- Lipid profile
- E.C.G.
- HbA1C—It is a form of haemoglobin that is measured primarily to identify the three-month average plasma glucose concentration. A normal non – diabetic HbA1C is 3.5-5.5%. For non-diabetics, the usual reading is 4-5.9%. For people with diabetes, an HbA1C level of 6.5% is considered good control.

8. Etiology and Pathogenesis of DM

In both types of DM, environmental factor and genetic susceptibility is important.

A) DM I

Type 1 DM (IDDM) is a T-Cell mediated autoimmune disease.[222] It involves destruction of the insulin- secreting β cells in the pancreatic islets and this process takes many years. Features of diabetes do not become evident until a majority of βcells (70-90%) are destroyed.[223] This type is a result of complete or near total insulin deficiency.

When 70-90% β cells have been destructed, Hyperglycemia, with associated classical syndrome is occurs.

I) Pathology: [224,225.]

Approximately 85% of patients have circulating islet cell antibodies, and the majorities also have detectable anti-insulin antibodies before receiving insulin therapy. Most islet cell antibodies are directed against glutamic acid decarboxylase (GAD) within pancreatic β cells.[224]

Due to autoimmune destruction of pancreatic β-cells, deficiency of insulin secretion occurs. It results into metabolic changes related with T1DM. Due to reduced insulin secretion, the function of pancreatic α-cells is becoming abnormal and excessive secretion of glucagon occurs. Normally, glucagon secretion reduced by hyperglycemia but in type 1 DM, glucagon secretion is not suppressed by hyperglycemia.[225] Elevated glucagon levels aggravates metabolic defects due to insulin deficiency. Insulin deficiency is the basic defect in T1DM, but a defect in the administration of insulin is also present. Deficiency in insulin leads to uncontrolled lipolysis and elevated levels of free fatty acids in the plasma. It suppresses glucose metabolism in peripheral tissues as skeletal muscle.[225] This reduces glucose utilization. Also, insulin deficiency in T1DM causes impaired glucose, lipid and protein metabolism.

DM 1 is associated with other autoimmune disorders. Such as thyroid disease, Addison's disease, pernicious anemia and vitiligo also.[226]

ii) Genetic Predispositon: [227]

Genetic factors affect about one –third of susceptibility of type 1 DM.

HLA (Human –Lympocytes Antigen); DR3 or DR4 are associated with increased susceptibility to Type, candidate gene and genome-wide association have implicated other genes in type 1 diabetes, e.g. CD25, IL2RA. The genes associated type's diabetes overlap with autoimmune disorders.

iii) Environmental Factors

Environmental factors have an important role in promoting clinical expressions of the disease. Due to reduced exposure to micro organism in early childhood suppress maturation of immune system and increases susceptibility to autoimmune disease.

iv) Viral:

Viral infections in the pancreas affect function of β cells e.g. mumps, coxackie B4, rubella, etc.[228]Stress may precipitate type 1DM by stimulating counter – regulatory hormones.[229]Various nitrosamines, coffee and (BSA) Bovine serum albumin i.e. (a constitute of cow's milk) have been proposed as dibetogentic.[230]Thus, due to all these factors and due to β cells destruction adequate insulin secretion inhabits and normal glucose level cannot be maintained for long time.Eventually, all type1diabetic patients will require insulin therapy to maintain Normoglycemia.

B) DM II

i)Pathology [231]

It is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production and abnormal fat metabolism.

Type 2 is more complex condition than type 1 DM, because there is a combination of two main pathological defects one is reduced insulin secretion through insulin resistance to the actions of insulin in liver and muscles together. Second is there is reduced insulin secretion due to impaired pancreatic β Cell functions. It causes insulin deficiency.[232]

a) Insulin Resistance:

Type 2 DM, often associated with other disorders, particularly central obesity, hypertension and dyslipidaemia (i.e. elevated level of LDL Cholesterol and decreased level of HDL cholesterol.) [233]

Primary cause of insulin resistance is unclear but. Intra –abdominal 'central' adipose tissue releases large quantities of FFAs. These FFAs is known cause of peripheral insulin resistance and hepatic insulin resistance. Elevated plasma FFAS levels impairs insulin stimulated glucose uptake into muscles. Also, adipose tissue releases several hormones like adipokines which influence sensitivity to insulin in other tissues.[234]

b) Pancreatic β Cell failure:[235]

In type 2 DM, FFAS and elevated plasma glucose creates toxic effects on pancreatic β Cell, hence insulin secretion reduced.

Due to reduction in β Cell nos. ∝Cell mass is unchanged and glucagon secretion is increased; which may contribute to the hyperglycemia.

ii) Genetic Predisposition:[236]

In monozygotic twin's concordance rates for type 2 diabetes is 100%

iii) Environmental factors

DM II is associated with overeating especially combined with obesity. It is also common in the middle –aged and elderly people. Thus, this is the most common form of Diabetes mellitus. It is highly associated with a family history of diabetes, older age, obesity and lack of exercise and sedentary life style.[237]

C) Other type:

MODY[238] (maturity onset diabetes of the young):

It is defined as hyperglycemia diagnosed before the age of twenty –five years and treatable five years without insulin, in cases where islet cell antibodies (ICA) are negative.

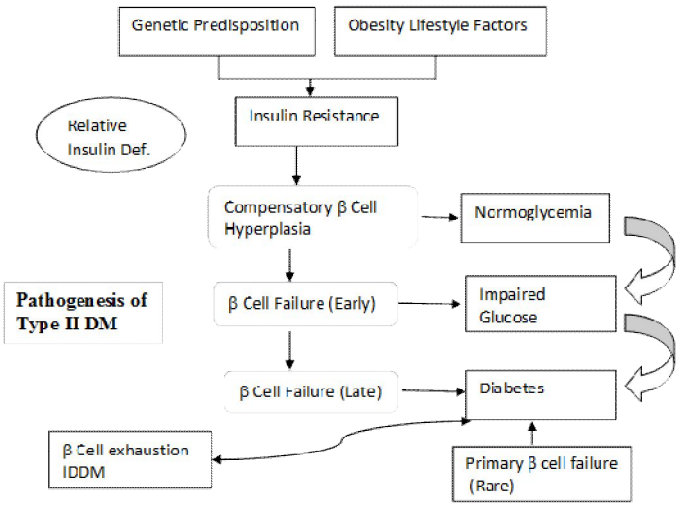

Fig. No. 1 Flow chart of Pathology of Diabetes Mellitus II

9. Complications of Diabetes Mellitus: [239]

Complications of Diabetes mellitus fall into two major divisions i.e. Acute Complications & Chronic Complications. The complications resulting from the disease are associated with the damage or failure of various organs such as the eyes, kidneys & nerves.

Acute Complications:

- Hypoglycemia
- Diabetic Ketoacidosis (DKA)
- Non Ketoic hyperosmolar state

Chronic Complications:

- Macrovascular Complications:
- Coronary artery disease.
- Peripheral Vascular disease.
- Cerebro vascular disease.
- Microvascualar Complications:
- Diabetic Eye disease
- Retinopathy (non-proliferative/proliferative)
- Macular edema
- Glaucoma
- Cataracts
- Diabetic Neuropathy
- Poly neuropathy /mono neuropathy
- Autonomic neuropathy.
- Diabetic Nephropathy
- Other
- Gastro intestinal [gastroparesis, diarrhoea]
- Genito urinary [uropathy /sexual dysfunction]
- Dermatologic infections.
- Diabetic foot.

10. Treatment

The goals of therapy for type 1 or type 2 DM are

- Eliminate symptoms related to hyperglycemia.

- Reduce or eliminate long term microvascular complications of DM.
- Allow the patient to achieve as normal life style as possible.So,the management can be planned as under
- Education of patient about DM, nutrition and exercise.
- Monitoring the level of Glycemic control.
- Assessment of glycemic control
- Oral hypoglycemic agents.
- Insulin

1. Education of Patient: [240]

Diabetes self management education is an important part of treatment. A variety of strategies and techniques should be used to provide adequate education and problem management, let us consider.

- **Nutrition**[241]

Medical Nutrition Therapy (MNT) is an integral component of DM.

Nutritional recommendation for Dm

- **Carbohydrate:**

Patient, especially type1 DM has been advised to maintain a regular intake of carbohydrates through the day. But by using insulin analogues or (CSII) continuous subcutaneous insulin injections, allowed greater flexiblity in the timing and choice of carbohydrate intake.

People with type 2 diabetes, limitations of retimed carbohydrate and restriction of total caloric intake is important.

- **Fat:** The intake of total fat should be restricted to less than 35% of energy intake.
- **Diabetic foods and sweeteners:** Low –calorie and sugar–free drinks are useful for patients with diabetes.
- **Weight Management:**[242]

Patients having DMII are overweight or obese, and many anti-diabetic drugs including insulin encourage weight – gain. So, weight loss reduction can be achieved by reduction in energy intake and an increase in energy consumption through physical activity.

1. **Exercise:**

   Diabetic patient should advise to regular physical activity for approximately 30 minutes daily, as this improves insulin sensitivity and lipid profile and lowers blood pressure.Programs for the treatment & prevention of Diabetes mellitus. It reduces the risk of cardiovascular disease, hyperlipidemia, hypertension & obesity.

2. **Self Monitoring of Blood Glucose (SMBG)** [243]

   It permits the well informed and motivated patient to make appropriate adjustments in treatment (i.e. particularly insulin dose). BG monitoring is most useful in patients having risk of hypoglycemia, during undercurrent illness and prescription of corticosteroids and during changes in therapy.

3. Assessment of Glycemic Control :[244]

Oral drugs are used to lower blood glucose level by achieving following goals.
Drugs that primarily stimulate insulin secretion.
Drugs that alter insulin action.
Drugs that principally affect absorption of glucose.
Glycated HB or HbA1C should be measured in all individuals with DM. Postprandial and nocturnal hyperglycemia may not be detected by SMBG of fasting pp but will be reflected in HbA1C.

4. Insulin Administration:

Insulin is obtained from pork pancreas or is made chemically identical to human insulin by recombinant DNA technology or chemical modification of pork insulin.[245]

Type of insulin depending on action.[246]

Rapid – acting - (Insulin analogues, lispro, aspart, glulisine)
Short Acting – Soluble
Intermediate – Isophone (NPH) Lente
Long acting – (Bovine alternate)

- **Insulin dose regimen**[247]

  The choice of regimen depends on the patient's degree of glycemic control, the severity of underlying insulin deficiency, the patient's lifestyle.

  Twice daily administration of a short-acting and intermediate acting insulin given in combination before breakfast and the evening meal, is the simplest

regimen and still used commonly. Hypoglycemia, weight gain, peripheral edema, insulin antibodies these are side effects of insulin therapy.

The prevalence of diabetes is high at the population level. So, there is an economical, psychological, social burden on our healthcare system and the individuals living with the disease.The impact of diabetes is reaches in a wide state, it is essential to each countryfor implementation of preventive and curative measures.Lifestyle modification will undoubtedly play a key role in the ultimate solution to the problem of diabetes.

3.5 Review of Drug

I. Daruharidra (Berberis aristata)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1. Vedakalin review:

The World's first ever literature is said to be Veda. Vedic era is from 6000 B.C.to 600 B.C. The Vedic literature is very vast.It contains the knowledge about various sciences. It is dispersed in Samhita, Brahman, Upanishad and Vedanga, Samhita have many branches and each has its own characteristics.[248]

Daruharidra is mentioned in Vedic literature with various names as follows-

1. Dharuharidra: In Keshav Paddhatti 3/10 Kwatha of Daruharidra and Haridra is used for Avasinchanarth in the management of Khalitya.[249]

2. Putudaru-Putadru-as a Devdara ccording to Mujumdar. According to Acharya P.V. Sharma Daruharidra which is used as a Samindha in yagya.In ShathpathBramanyak it is mentioned as aromatic and inflammable. Siddha Ghruta for Anjjan and Abhyangarth.[250]

2. SamhitakalinReview: [251-53]

I. Charak Samhita: [4]

- It is included in Aragwadhadi Varga. (Ch.Su.3/10)
- It is constituent of Pittaj Pramehahar Churna. (Ch. Vi.6/32)
- It is constituent of Rakttapittanashakyog Churna.
- It is constituent of Dahashamakchurna in Madatyayachikitsa. (Ch.Chi 4/73)
- It is constituent of Svarnakar lepa in Vrana Chikitsa. (Ch.Chi 25/116)
- It is constituent of Dahashamak lepa in Aragvadhadi Adhyaya. (Ch.Su.3/26)
- It is constituent of Mustadichurna in Kushtha Chikitsa. (Ch.Chi 7/65)
- It is constituent of Vatpittajanya Shothahar Churna.
- It is constituent of Shothahartaila in Shotha Chikitsa. (Ch.Chi 12/25)
- It is constituent of Anjan used inVisha Chikitsa. (Ch.Chi 23/69)
- It is constituent of Amruta Ghruta in Visha Chikitsa. (Ch.Chi 24/5)
- It is constituent of Phalatrikadi Kwatha in Prameha Chikitsa. (Ch.Chi 6/40)
- It is constituent of Kushthanashak Lepa in Kushatha Chikitsa. (Ch.Chi7 /84)
- It is constituent of DarviGhruta Kushatha Chikitsa. (Ch.Chi 7/135)
- It is constituent of VranaShodhak Kashaya in Vrana Chikitsa. (Ch.Chi 25/84)

- It is constituent of VranaRopak Taila in Vrana Chikitsa. (Ch.Chi 25/93)It is constituent of Visarpa Avchurnan in Visarpa Vrana Chikitsa. (Ch.Chi 20/94)

II. Sushrut Samhita: [252]

- It is constituent of Siddha Ghruta in Kushtharog Chikitsa. (Su.Chi.9/31)
- It is constituent of Lepa in Kshudraroga Chikitsa.
- It is constituent of Swedanarth Lepa in Keetakalpaadhya Chikitsa. (Su.Chi.20/6)
- It is constituent of Shodhan kalka siddha Taila. (Su.Su.37/16)
- It is constituent of Lepa in Kshudra roga Chikitsa. (Su.Chi.9/35)
- It is constituent of Abandhya Vrana Rasakriya Dwivraniya roga Chikitsa.
- It is constituent of Siddhataila in SadyaVrana roga Chikitsa. (Su.Chi.1/73)
- It is constituent of NadiVrana Shodhanarth kalka in Bhagandarroga Chikitsa. (Su.Chi.2/75)
- It is constituent of Vajraktaila in Nadirog Chikitsa.
- Lepa in Kshudraroga Chikitsa (Su.Chi.9/55)
- It is constituent of siddha taila in MahaKushtaroga Chikitsa. (Su.Chi.10/15)
- It is constituent of Vajraktaila in MahaKushtaroga Chikitsa. (Su.Chi.10/16)
- It is constituent of Lavanmeha sevanarth dravya. (Su.Chi.11/9)
- It is constituent of Vajraktaila in MahaKushtharoga Chikitsa. (Su.Chi.9/57)
- It is constituent of Ropantaila in SadyoVranaroga Chikitsa. (Su.Chi.2/75)
- It is constituent of Siddha Ghruta Jwararoga Chikitsa (Su.Ut.39/227)

III) Ashtanga Hriday: [253]

- It is constituent of Avachurnan in VisarpaRogaChikitsa. (A.Hr.Chi.19/50)
- It is constituent of Chaurna in Shotharoga Chikitsa.
- It is constituent of Kashya in Shotharoga Chikitsa. (A.Hr.Chi.17/2, 32)
- It is constituent of Churna in KushtharogaChikitsa. (A.Hr.Chi.19/50)
- It is constituent of BalaTaila inVatarogaChikitsa.
- It is constituent of Kashya in BalarogSarvadoshhara Churna. (A.Hr.Chi. 21/75)
- It is constituent of Bhutrao Ghruta in Bhootpratishedroga Chikitsa. (A.Hr.Ut. 5/19)
- It is constituent of Siddha kalka pan in Granthi Arbud Nadi Vrana roga Chikitsa. (A.Hr.Ut.30/32)

- It is constituent of Lepa in Ajgallika Vrana in Kshudraroga Chikitsa. (A.Hr.Ut.32/2)
- It is constituent of Churna in Kaphajmeharoga Chikitsa. (A.Hr. Chi121/6, 7)
- It is constituent of Kashya in Shotharoga Chikitsa. (A.Hr. Chi 17/32)
- It is constituent of KashayaChurna inKushtharoga Chikitsa. (A.Hr. Chi19/37)
- It is constituent of KawalChurna in Mukharoga Chikitsa. (A.Hr.Ut.22/56)
- It is constituent of Siddha Ghruta in Vranaroga Chikitsa. (A.Hr.Ut.25/67)

IV.NighantuKaal:[254]

- It is mentioned in Haritkyadi Varga of Ashtanga Nighantu.
- It is mentioned in GuduchyadiVarga of Dhanvantari Nighantu.
- It is mentioned in GuduchyadiVarga of Shodhal Nighantu.
- It is mentioned in AbhayadiVarga of Madanpal Nighantu.
- It is mentioned in AushadhiVarga of Raj Nighantu.
- It is mentioned in Mishraprakaran Haritkyadi Varga of Bhavprkash Nighantu.
- It is mentioned in Daruharidradi Varga of Nighantu Adarsha.

3) Morphology of Daruharidra: [255]

Daruharidra consists of dried stem of Berberis aristata DC. (Fam. Berberidaceae); an erect,spinous,deciduous shrub,usually1.8-3.6 m in height found inH imalayanranges at an elevation of 1000-3000 m, and in the Nilgiri hills in South India.

a) Macroscopic structure:

Drug available in spices of variable length and thicken.Bark is about 0.4 - 0.8 cm thick, pale yellowish-brown, soft, closely and rather deeply furrowed, rough and brittle.

Xylem portion is yellow, more or less hard, and radiate with xylem rays, pith mostly absent, when present it appears small. Yellowish-brown when dried, fracture short in bark region, splintery in xylem; taste, bitter.

b) Microscopic structure:

Stem-Shows rhytidoma with cork consisting of 3-45 rectangular and squares, yellow coloured, thin-walled cells, arranged radially, sieve elements irregular in shape. Thin walled, a few cells containing yellowish-brown contents. Phloem fibers arranged in Tangential rows, consisting of 1-4 cells. Each fiber short thick-walled,

spindle-shaped, lignified having wide lumen; half inner portion of rhytidoma traversed by secondary phloem rays. Phloem rays run obliquely consisting of radially elongated parenchymatous cells. Almost all phloem ray cells having single prismatic crystals of calcium oxalate, a few cells of rhytidoma also contain prismatic crystals of calcium oxalate. Stone cells also found cattered in phloem ray cells in groups, rarely single, mostly elongated. A few rounded, arranged radially, some of which contain a single prism of calcium oxalate crystals. Secondary phloem is a broad zone, consisting of sieve elements and phloem fibers, traversed by multi seriate phloem rays. Sieve elements arranged in tangential bands and tangentially compressed cells alternating with single to five rows of phloem fibers, phloem fibers short, lignified, thick-walled having pointed ends. Secondary xylem consisting of xylem vessels, tracheid's, xylem fibers and traversed by multi seriate xylem rays; xylem vessels numerous,small to medium sized , distributed throughout xylem region in groups or in singles, groups of vessels usually arranged radially; isolated vessels cylindrical with rounded or projected at one or both ends with spiral thickening ; xylem fibers numerous, lignified,large,thickwalled with wide lumen,and pointed tips; xylem rays quite distinct, straight, multiseriate, consisting of radially arranged rectangular cells, each ray 30-53 cells high, 8-12 cells wide, a few ray cells containing brown content.

C) Powder:

Colour-Yellow ; shows mostly fragments of cork cells, sieve elements, yellow colored phloem fibers entire or in pieces, stone cells in singles or in groups, numerous prismatic crystals of calcium oxalate, xylem vessels having spiral thickening, thick-walled, lignified xylem fibers and ray cells.

D) Identity, Purity And Strength : [255]

Foreign matter not more than 2 per cent, Appendix 2.2.2.

Total Ash not more than 14 per cent, Appendix 2.2.3.

Acid-insoluble ash not more than 5 per cent, Appendix 2.2.4.

Alcohol-soluble extractive not less than 6 per cent, Appendix 2.2.6.

Water-soluble extractive not less than 8 per cent, Appendix 2.2.7.

E) Part in use: Bark, fruit, root, stem, wood.

F) Chemical composition: [256]

It contains various chemical components. Berberaine, Oxyberberaine, Berbamine, Aromoline, Karachine, Palmatie, Oxycanthine, Taxilamiene these are the main chemical compounds.

G) Kalpas of Daruharidra: [255]

BhringarajTaila, Ashwagandharishta, Khadiradi Gulika, Khadirarishta, JatyadiTaila, Triphaladi Ghruta.

H) Properties and Action: [255]

1. **Rasa**: Tikta
2. **Vipaka**: Katu
3. **Virya:**Ushna
4. **Guna:**Rukshya,Ushna
5. **Karma:**Urdhwajatrugataroghara,Varnya,Vranashotha,Twakdoshhar, kandughnana, Pramehahar, Vishagnahar.

I) Therapeutic uses: [255]

Kandu, Kushtharoga, Medoroga, Mukharoga, Vrana, Shotha, Atisar, Urustambha, Kapharoga, Mukha, Karnaroga, Netraroga, Meha, Vishamjwara, Rakttapitta

J) Dose –Kwath 5-10 ml, Churna 1-3 gm

4) Classification:

1) Classification in Samhita: [256]

Table No. 23: Classification of Daruharidra according to Samhita.

CharakaSamhita	**SushrutaSamhita**	**AshtangaHridaya**
Lekhaniya Gana	Haridradi Gana	ShoroveerechaniyaGana
Arshoghana Gana	Mustadi Gana	HaridradiVarga
Kandughana Gana	Lakshadi Gana	MustadiVarga
Apatarpaniya Varga	Ropan Gana	Nasya Dravyasangraha
Nabhipakhar Varga	-	-

2) Classification According to Nighantu: [254]

Table No.24: Classification of Daruharidra according to Nighantu.

Nighantu	**Varga**
AshtangaNighantu	Haridradi Gana
DhanvantariNighantu	GuduchyadiVarga
Shodhal Nigh antu	GuduchyadiVarga
Madanpal Nighantu	AbhayadiVarga
Raj Nighantu	PipalyadiVarga
KaiyadevNighantu	AushadhiVarga
BhavprakashNighantu	MishraprakaranHaritkyadiVarga
Nighantu Adarsha	DaruharidradiVarga

3) Taxonomical Classification: [257]

Table No 25: Taxonomical Classification of Daruharidra

Kingdom	Plantae
Unranked	Angiosperms
Unranked	Eudicots
Order	Ranunculales
Family	Berberidaceae
Genus	Berberis
Species	B. aristata

L) Paryayi Nama and Nirukati of Daruharidra: [254, 258,259]

- **Daruharidra**: Yellow colour bark and flowers.
- **Katankarer**i: Spinous leaves.
- **Kantakini**: Spinous plant.
- **Kaleyak**: It removes doshas.
- **Kusumbhala**: It gives yellow colour dye.
- **Krumihara**: It has anti helmentic and antibiotic activity.
- **Darvi**: Its bark is important part.
- **Pachampacha**: Improves digestion and liver function.
- **Parjanya:** Its fruitning season is rainy season.
- **Parjani:** This prevents from diseases.
- **Pitadaru, Pitadru**: Its bark is in yellow colour.
- **Vishodhani:** Its action is as body purifier.

M) Parayayi Nama of Daruharidra: [254]

Table No. 26: Parayayi Nama of Daruharidra according to Nighantu

ParyayiNama	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N
Darvi	+	+	+	+	+	+	+
Darunisha		+		Ratridaru	+	+	
Nisha					sthirnisha	+	
Katankateri	+	+		+		+	+
Katankati				+			
Kantaki							
Parjanya	+	+		+	+	+	+
Parjanita							+
Pita					+	+	+
Darupita					+		
Pittachanda			+				
Daruharidra				+	+	+	+
Haridru							+
Haridra			+				
Pitadru				+	+		+
Pitadaru				+		+	+
Pachampacha	+	+		+	+		+
Kasdtarajni		+					
Kaleyak		+					+
Pitahav		+					
Pitakam							+
Kamvati					+		
Kanchani			+				
Hemvarna		+					
Hemkanta		+	+				
Suvarnavarna				+			
Kusumbhak		+					
Kusumbhala			+				
Karkakkini			+		+		
Kamini					+		

N) Vernacular names: [255]

- **Sanskrit: Katamkateri**, Darvi
- **Bengali: Daruharidra**
- **English:** Indian Berberry
- **Guajarati**: Daruharidra, Daruhuladur
- **Hindi:** Daruhaldi, Darhald

- **Kashmiri:** Kannadarishana, Maradarishina, Daruhaladi
- **Malayalam :** Maramannal, Maramanjal
- **Marathi :** Daruhalad
- **Oriya:** Daruharidra, Daruhalidi
- **Punjabi:** Sumalu
- **Tamil :** Gangeti, Varatiumanjal
- **Telugu** : Manupasupu
- **Urdu:** Darhald

O) GunakarmatmakaVivechana: [254]

Table No.27: Gunapanchak of Druharidra according to Nighantu

Properties	A.N	D.N	S.N.	M.N.	R.N.	K.N.	B.N.
Guna	-	Ruksha					Ruksha
Rasa	-	Tikta		Tikata	Tikata		Katu
	-			Katu	Katu		Tiktta
Virya	-	Uashna					Ushana
Vipaka	-						

Table No. 28: Dosha, karma and Rogaghanata of Daruharidra:[254]

Activity	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N.
Dosha karma							Pittanuta,Kaphaanuta
Pramukha Karma		Urdhwajatruroghara, Rujahar,Vishodhini,	Vishodhini,				Varnya,Twakadosh-hhara
Rog-ghnata		Vrana,Meha,Kandu,Krumi,Pinas, Aruchi	Krumihar, Hemakanta	Netrya, karnya rogjeeta, AsyaRogajeeta, Meha, Kushta, Visha, Vrana	Vrana,Meha, Kandu,Veesarpa,Twakdosha,Visha,Karna,Akshiroga	Karnarog Netrarog	Netraroga,Mukharoga,Meha,Pandu,Vrana,Shothha, Astra

P) Shloka references of Various Nighantu for Paryayinam, Gunapanchak, Dosh Karma and Rogghanta of Daruharidra :[253]

1 .अष्टाङ्गनिघण्टु–२०. हरिद्रादिगण

हरिद्रादिगणवक्ष्यैगौरीश्यामाचनिर्विषा|

निशाक्षपाचरात्रिश्चवरालोमशमूलिका||१५२||

स्वर्णवर्णाहरिद्रातुनिशाह्वारजनीतथा|

दार्वीकटङ्कटेरीचपर्जन्याचपचम्पचा||१५३||

2.धन्वन्तरिनिघण्टु–१. गुडूच्यादिवर्गदारुहरिद्रा

विशोधनीकृमिहरापीनसारुचिनाशिनी||६०||

अन्यादारुहरिद्राचपीतद्रु:पीतचन्दनम्||६१||

निर्दिष्टाकाष्ठरजनीसाचकालेयकंस्मृतम्

कालीयकंदारुनिशादार्वीपीताह्वपीतकम्||६२||

कटङ्कटेरीपर्जन्यापीतदारुपचम्पचा|

हेमवर्णवतीपीताहेमकान्ताकुसुम्भका||६३||

तिक्तादारुहरिद्रास्याद्रूक्षोष्णाव्रणमेहजित्

कर्णनेत्रमुखोद्भूतांरुजंकण्डूंचनाशयेत्||६४||

3. शोढलनिघण्टु-नामसङ्ग्रह (प्रथमभाग) - १. गुडूच्यादिवर्गदारुहरिद्रा

दाव्र्यांतुदारुपूर्वासाहरिद्रापीतचन्दनम्

काञ्चनीकर्कटकिनीतच्चकालेयकंस्मृतम्||१३५||

विशोधनीकृमिहराहेमक्रान्ताकुसुम्भला|

द्रविडश्रीर्जयन्तीचपीतद्रु:पीतकंमतम्||१३६||

4.मदनपालनिघण्टु - १. अभयादिवर्गदारुहरिद्रा

दार्वीदारुहरिद्राऽन्यापीतदारुचपञ्चधा|

कटङ्कटेरीपित्तद्रु:स्वर्णवर्णाकटङ्कटा|

दार्वीतद्वद्विशेषातुनेत्रकर्णास्यरोगजित्॥२३०॥

5. राजनिघण्टु -६. पिप्पल्यादिवर्गदारुहरिद्रा

अन्यादारुहरिद्राचदार्वीपीतद्रु पीतिका।

कालेयकंपीतदारुस्थिररागाचकामिनी॥२००॥

कटङ्कटेरीपर्जन्यापीतादारुनिशास्मृता।

कालीयकंकामवतीदारुपीतापचम्पचा।

स्यात्कर्कटकिनीज्ञेयाप्रोक्तासप्तदशाह्वया॥२०१॥

तिक्तादारुहरिद्रातुकटूष्णाव्रणमेहनुत्।

कण्डूविसर्पत्वग्दोषविषकर्णाक्षिदोषनुत्॥२०२॥

6. कैयदेवनिघण्टु - १. ओषधिवर्ग**दारुहरिद्रा**

कटङ्कटेरी पर्जन्या दार्वी दारुनिशा निशा ।

पीता दारुहरिद्रा स्यात् पीतद्रुः पीतचन्दनम् ॥१११६॥

पचम्पचा हेमकान्तिः पीतदारुः कटङ्कटी ।

तद्वद् दार्वी विशेषेण कर्णनेत्रास्यरोगजित् ॥१११७ ॥

7.भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - २. हरीतक्यादिवर्गदारुहरिद्रा

दार्वीदारुहरिद्राचपर्जन्यापर्जनीतिच।

कटङ्कटेरीपीताचभवेत्सैवपचम्पचा॥१७५॥

सैवकालीयकःप्रोक्तस्तथाकालेयकोऽपिच।

पीतद्रुश्चहरिद्रुश्चपीतदारुचपीतकम्॥१७६॥

II) Nimba (Azadirecta indica)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1) Vedic review of Nimba: [260]

In Vedic litareture Nimba is discribed.

The Samindha of Nimba is not allowed in Yagya.It is used for Dantadhavan.[261]

JeminiGruhayasutra: 1/1, KoutubGruhyasutra: 2/6/9,GobhilGhruhya sutra: 1/5/15 BodhayanPitrumethusutra:3/4,AtharvashirshyaPrishistya:21/3/2,Vishnu Dharmasutra: 61/14[262]

Pichumanda is describe in PainiyaGanapath: 3/10/24 [263]

2) Samhitakalin Review:[264-66]

I. CharakSamhita:[264]

- As a constituent of KhadiradiGhruta in KushthaRog Chikitsa. (Ch.Chi.7/152)
- As a constituent of MahatikkataGhruta in Krumi Chikitsa.(Ch.Chi.7/153)
- As a constituent of AragvadhadiAdhya for TwakRogChikitsa.(Ch.Su.3/38)
- As a constituent of VamakDravya In Apamargatanduliya Chapter. (Ch.Su.2/7)
- As a constituent of Shakavarga in RakttapittaRogChikitsa. (Ch.Chi.4/38)
- As a constituent of Churna in PittajPramehaRogChikitsa. (Ch.Chi.6/30)
- As a constituent of MustadiChurna in KushthaRogChikitsa.(Ch.Chi.7/65)
- As a constituent of Churna for Bath Kushtha RogChikitsa.(Ch.Chi.7/129)
- As a constituent of Churna in VranaDhavan of Kushtha rog Chikitsa.(Ch.Chi.7/157)

II. SushruraSamhita:[265]

- As a constituent of DhoopanDravya in VranitaupasanaChikitsa.(Su.Chi.19/28)
- As a constituent of Siddha Ghruta in KushthaRogaChikitsa. (Su.Chi.9/46)
- As a constituent of SiddhaGhruta in VidradhiChikitsa. (Su.Chi.16/17)
- As a constituent of Siddha Ghruta in JwaraChikitsa. (Su.Ut.39/226)
- As a constituent of DhoopanDravya in VranitopasanaChikitsa. (Su.Su.19/28)
- As a constituent of Mahavajarak Tail in KushtaRogaChikitsa. (Su.Chi. 9/58)
- As a constituent of DhoopanDravya in Agrohopkranik Adhyaya Chikitsa. (Su.Su5/18)

- As a constituent of ShodhanTaila in MishrakAdhyaya. (Su.Su. 37/17)
- As a constituent of Churana for Snan, Lepa in KushtaChikitsa. (Su.Chi. 9/14)
- As a constituent of Kwatha in Kushta and Krumi in Kushtha Chikitsa. (Su.Chi 9/51)
- As a constituent of Kwatha in Prameha Chikitsa. (Su.Chi. 11/8,9)
- As a constituent of Siddha Tail in Granthi Apachi Arbuda Chikitsa. (Su.Chi. 18/47)
- As a constituent of Lepa in Kshudra Roga Chikitsa.(Su.Chi. 20/21)

III. AshtangaHriday: [266]

- As a constituent of Rasayan Churna in Rajyakshma Chikitsa.(A.Hr.Chi.5/28)
- As a constituent of ShodhanadiGana for Vaman. (A.Hr.Su.15/1)
- As a constituent of ShodhanadiVarga for Pitta shamanarth.(A.Hr.Su.15/6)
- As a constituent of Churna in Sannipatic Jwara Chikitsa.(A.Hr.Chi.1/65)
- As a constituent of Churna in PittajPrameha Chikitsa.(A.Hr.Chi.12/8)
- As a constituent of Ghruta in Vidradi, Gulma Chikitsa.(A.Hr.Chi.13/11)
- As a constituent of Kashayam in VranashodhanChikitsa.(A.Hr.Chi.13/35)
- As a constituent of Mahatikttak Ghruta in Kshtharoga Chikitsa .(A.Hr.Chi.19/9)
- As a constituent of DhoopanDravya in Vranadhoopanarth. (A.Hr.Su.29/26)
- As a constituent of Kwatha in DushtaVarana Dhavan in Vrana Chikitsa. (A.Hr.Ut.25/42)
- As a constituent of Churana in Shodhan and Ropan in Vrana Chikitsa. (A.Hr.Ut.25/43,55)

IV. Nighantu Kala: [267]

- It is mentioned in Guduchyadi Varga of Ashtanga Nighantu.
- It is mentioned in Guduchyadi Varga of Dhanvantari Nighantu.
- It is mentioned in Guduchyadi Varga of Shodhal Nighantu.
- It is mentioned in Abhayadi Vargaof Madanpal Nighantu.
- It is mentioned in Prabharadi Varga of Raj Nighantu.
- It is mentioned in Mishraprakaran Guduchyadi Varga of Bhavprakash Nighantu.
- It is mentioned in Nimbadi Varga of Nighantu Adarsha.

3. Morphology of Nimb: [268]

N**imba (Leaf):** Azadirachta indica A. Juss Syn. Melia

Azadirachta Linn. (Fam. Meliaceae); a moderate sized to fairly large evergreen tree. Attaining a height of 12-15 m with stout trunk and spreading branches, occurring through out the contry up to an elevation of 900 m.

Description:[268]

a) Macroscopic:

Leaves-Compound, alternate, rachis 15-25 cm long, 0.1 cm thick; leaflets with oblique base, opposite, exstipulate, lanceolate, acute, serrate, 7-8.5 cm long and 1.0-1.7cm wide,

Colour - slightly yellowish-green;

Odour - indistinct

Taste - bitter

b) Microscopic:

Leaf-Midrib -leaflet through midrib shows a biconvex outline; epidermis on either side covered externally with thick cuticle; below epidermis 4-5 layered collenchyma present;stele composed of one crescent-shaped vascular bundle towards lower and two to three smaller bundle towards upper surface ; rest of tissues composed of thin-walled, parenchymatous cells having secretory cells and rosette crystals of calcium oxalate; phloem surrounded by non-lignified fibre strand; crystals also present in phloem region.

Lamina - shows dorsiventral structure; epidermis on either surface, composed of thinwalled, tangentially elongated cells, covered externally with thick cuticle; anomocytic stomata present on lower surface only; palisade single layered; spongy parenchyma composed of 5-6 layered, thin-walled cells, traversed by a number of veins; rosette crystals of calcium oxalate present in a few cells; palisade ratio 3.0-4.5; stomatal index13.0-14.5 on lower surface and 8.0-11.5 on upper surface.

Powder - Green; shows vessels, fibres, rosette crystals of calcium oxalate, fragments of spongy and palisade parenchyma.

C) Identity, Purity and Strength :[268]

Foreign matter not more than 2 per cent, Appendix 2.2.2.

Total Ash not more than 10 per cent, Appendix 2.2.3.

Acid-insoluble ash not more than 1 per cent, Appendix 2.2.4.

Alcohol-soluble extractive not less than 13 per cent, Appendix 2.2.6.

Water-soluble extractive not less than 19 per cent, Appendix 2.2.7.

D) Chemical constituents: [269]

About 100 chemical constituents mostly Triterpenoids of protolimonoids, Limonoids few non Triterpenoids constituents are Azadirictin Azadiractoldeacetyl Azadiractol and Sterols.

E) Part used: [268]

Bark, leaf, flower, fruits, oil.

F) Properties and action: [268]

1) Rasa: Tikta, Kshya,

2) Guna: Ruksha, Ushna, Laghu

3) Virya: Sheeta

4) Vipaka: Katu

5) Karma:[268]

Grahi, Vatal, Pittashamak, Kaphashamak,Vranaropak, Vranashodhak, Putihar, Daahaprashaman, Kandughna, Kushtahar, Rakttashodhak, Shothagna, Aampachk

G) Important formulations: [268]

Kashishadi Taila, Jatyadi Ghruta, Arogyavardhini vati, PanchagunaTaila.

H) Therapeutic uses: [268]

Jwara, Krumiroga, Kushtha, Netraroga, Prameha, Vrana, Amavat, Visharoga

I) Dose:[268] 1-3 g. of the drug in powder form.

10-20 ml of the drug for decoction.

4. Classification:

A) Classification According to Samhita:[270]

Table No.29: Classification of Nimba according to Samhita

CharakaSamhita	SushrutaSamhita	AshtangaHridaya
Kandughana	Aragvadhadi, Pramehaghna	Vaman varga
Vamak	Guduchyadi, Dantashodhan	Guduchyadi
Langanarth	Lakshadi	Aragvadhadi
	Urdhwabhagh har	Tiktakvarga
	Rakshoghana	PittashamakVarga
	Shodhan	

B) Classification According to Nighantu: [267]

Table No.30: Classification of Nimba according to Nighantu

Nighantu	Varga
AshtangNighantu	Guduchyadi/Aragwadadhi Gana
Dhanvantari Nighantu	Guduchyadi Varga
Shodhal Nighantu	Guduchyadi Varga
Madanpal Nighantu	Abhayadi Varga
Raj Nighantu	Prabhadradi Varga
Kaiydev Nighantu	Aushadhi Varga
Bhavprakash Nighantu	Guduchyadi Varga
NidgantuAdarsh	Nimbadi Varga

C) Taxonomical Classification: [272]

Table No.31: Taxonomical Classification of Nimba

Kingdom	Plantae
Unranked	Angiosperms
Unranked	Edicots
Unranked	Rosids
Order	Spindales
Family	Meliaceae
Genus	Azadirachta
Species	A.Indica

D) Parayayi Nama of Nimba According to Nighantu[272]

Table No. 32: Parayayi Nama of Nimba According to Nighantu

Paryayi Nama	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N
Nimba	+	+	+	+	+	+	+
Arishta	+	+	+	+	+	+	+
Pichumanda	+	+	+	+	+	+	+
Shukpriya	+		+			+	
Dhanya	+						
Kustumbaru	+						
Dhanik	+						
Dhanyak	+						
Niyamano		+	+	+		+	
Neta		+	+	+	+	+	
Sutikttak		+	+	+	Vartittak	+	Tikttak
Sarvatobhadra		+	+	+		+	
Prabhadra		+		+	+	+	
Paribhadrak		+	+	+	+	+	+
Subhadra			+			+	
Yavneshta			+			+	
Chardano			+		+	+	
Hingunirryas			+		+		+
Peetsaro			+	+			
Ravipriya			+				
Kushtaha				+			
Devdatta				+			
Ravi				+			
Suryako				+			
Kakphala					+		
Koreshto					+		
Dhamano					+		
Pavaeshta					+		
Veshirnaparni					+		

J)Paryayinama Nirukati:[268]

- **Arishta**: It eradicates many diseases.
- **Chardan**: It induces vomiting.
- **Hinguniryas**: Plant exudates secretion like Hingu.
- **Kakphala**: Fruits are eaten by crows.
- **Khrumighna**: It is anthelminthic.
- **Malak**: Useful for health in many ways.
- **Nimba**: Useful for health in many ways.
- **Niyamak**: Useful for health in many ways.
- **Paribhadrak**: Useful for health in many ways.
- **Pichumanda**: Cures skin diseases.
- **Puyari**: It cures pus formation.
- **Shukapriya**: Parrots gather on this tree.
- **Sutiktak**: It is one of best better drug.
- **Vartwachya**: Bark is used as medicine
- **Sarvatobhadra**: It is good in all the ways.
- **Neta**: It is the first in medicine.

K) Vernacular names:[268]

- **Sanskrit**: Arishtak, Pichumarda
- **Assamese**:Mahanim
- **Bengali**: Nim, Nimgach
- **English**: Margosa Tree
- **Gujrati** : Limba, Limbado, Limado, Kohumba
- **Hindi** : Nim, Nimba
- **Kannada**: Nimba, Bevu, Oilevevu, Kahibevu, Bevinama
- **Malayalam**:Veppu, Aryaveppu, Nimbam,Veppa
- **Marathi** : Balantanimba, Limba, Bakayan, Nim, Kadunimb
- **Oriya**: Nimba
- **Punjabi**: Nimba, Bakan, Nim
- **Tamil**: Vemmu, Veppu, Arulundi, Veppan
- **Telugu**:Vemu, Vep

L) Guna karma of Nimb:

Table No. 33. : Gunapanchak of Nimb according to Nighantu: [267]

Properties		A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N.
Guna	Laghu		+					
Rasa	Tiktta		+			+	+	
Virya	Sheeta		+		+	+	+	+
Vipaka	Katu				+		+	+

M) Dosha, karma and Rogaghanatta of Nimba:

Table No.34: Dosha, karma and Rogaghanata of Nimba according to Nighantu:[267]

Nighantu Karma	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N.
Dosha karma	Pittanuta Kaphaan uta	Pittanuta Kaphaan uta, Astrasho dhan	Pittanu ta Kapha anuta	Vatkar Pittaghana Kaphakar	Pittanuta Kaphaan-uta	Pittanuta Kaphaan-uta Vatkar	Pittanuta Kaphaan -uta Vatkar
Pramukh a Karma	Vishodha n,	Apakwa vranapa chayetV ranapak washodh ayet	Vranaj eet	Bhedan Kushtahar Vrnajeet	Shothaghn a	Grahi Hrudya Krumihar Netroroghar Agnikrut Hrudya	Grahi Bhedan
Rog-ghnata	Chardi Kushta Vish Jwara Kandu Prameha Dushtavr ana	KushtaK andu Vrana Taila-Vatraktt a Ashmari ghna	Chardi Meha Hrudr-og Krumi Tail-Raksh-oghna Keshy a Palitya -har	Chardi Kushta Hrullas Meha Vrana	Chardi Visha Krumi Dushtavr-ana Hrudrog Vidah	Shwas Aruchi Kushta Jwara Meha	Shwas Aruchi Kushta Jwara Meha Chardi Krumi Vrana Ahrud-ya

N) Shloka references of Various Nighantu for Paryayi Nama, Gunapanchak, Dosha Karma and Rogghanta of Nimb: [267]

1. अष्टाङ्गनिघण्टु–८. गुडूच्यादिगणगुडूच्यादिगण

गुडूचीपद्मकारिष्टधानकारक्तचन्दनम्|

पित्तश्लेष्मज्वरच्छर्दिदाहतृष्णाध्नमग्निकृत्||६६||

निम्बोऽरिष्टोगुडूच्यादौपिचुमन्दःशुकप्रियः|

धान्याकुस्तुम्बुरुःधान्यंधनिकाधान्यकंतथा||६७||

2.कैयदेवनिघण्टु–४. द्रववर्ग-करञ्ज, निम्बतैल

करञ्जनिम्बजेसोष्णेव्रणशोधनरोपणे ||३२२||

नात्युष्णंनिम्बजंतिक्तंकृमिकुष्ठकफप्रणुत् |

अभ्यङ्गान्नावनात्क्षीरभोजिनःपलितापहम् ||३२३||

१. ओषधिवर्गनिम्ब

निम्बोनियमनोऽरिष्टःपिचुमन्दःसुतिक्तकः|

सुभद्रःसर्वतोभद्रःप्रभद्रःपारिभद्रकः||८७८||

कृमिघ्नश्छर्दनोनेतायवनेष्टःशुकप्रियः|

निम्बस्तिक्तःकटुःपाकेलघुःशीतोऽग्निवातकृत् ||८७९||

ग्राह्यहृद्योजयेत्पित्तकफमेहज्वरकृमीन् |

कुष्ठकासारुचिश्वासहृल्लासश्वयथुव्रणान्||८८०||

ग्राहिप्रवालंनिम्बस्यरक्तपित्तकफकृमीन् |

कुष्ठघ्नंवातजननंनेत्ररोगान्विनाशयेत् ||८८१||

तद्वत्पत्राणिनिम्बस्यव्रणघ्नानिविशेषतः |

शलाकानिम्बपत्रस्यकासश्वासविनशिनी ||८८२||

कृमिघ्नातुवरिष्ठास्यात्कुष्ठज्वरविनाशिनी |

चक्षुष्यंनिम्बपुष्पञ्चकृमिपित्तविषप्रणुत् ॥८८३॥

वातलंकटुपाकंस्यात्सर्वारोचकनाशनम् ।

फलंतिक्तंरसेपाकेकटुकंभेदनंलघु ॥८८४॥

अरूक्षमुष्णंकुष्ठघ्नंगुल्मार्शःकृमिमेहनुत् ।

निम्बस्यपक्वंमधुरंसतिक्तंस्निग्धंफलंशोणितपित्तरोगे ।

कफेप्रशस्तंनयनामयघ्नं,क्षतक्षयघ्नंगुरुपिच्छलञ्च ॥८८५॥

निम्बबीजस्यमज्जाचकृमिकुष्ठविशोधनः ।८८६।

3.धन्वन्तरिनिघण्टु - १. गुडूच्यादिवर्गनिम्ब

निम्बोनियमनोनेतापिचुमन्दःसुतिक्तकः।

अरिष्टःसर्वतोभद्रःप्रभद्रःपारिभद्रकः॥३१॥

निम्बस्तिक्तरसःशीतोलघुःश्लेष्मास्रपित्तनुत्।

कुष्ठकण्डूव्रणान्हन्तिलेपाहारादिशीतलः ॥३२॥

अपक्वंपाचयेच्छ्लोफंव्रणंपक्वंविशोधयेत् ॥३३॥

4.धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्गनिम्बतैल

नात्युष्णंनिम्बजंतैलंकृमिकुष्ठकफापहम्।

वातरक्तप्रशमनंमदात्प्रश्मीरुजापहम् ॥१३१॥

5.भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - ४. गुडूच्यादिवर्ग-निम्ब

निम्बःस्यात्पिचुमर्दश्चपिचुमन्दश्चतिक्तकः ।

अरिष्टःपारिभद्रश्चहिङ्गुनिर्यासइत्यपि ॥८१॥

निम्बःशीतोलघुर्ग्राहीकटुपाकोऽग्निवातनुत् ।

अह्रद्यःश्रमतड्कासज्वरारुचिकृमिप्रणुत् ॥८२॥

व्रणपित्तकफच्छर्दिकुष्ठहृल्लासमेहनुत् ।

निम्बपत्रंस्मृतंनेत्र्यंकृमिपित्तविषप्रणुत् ॥८३॥

वातलंकटुपाकञ्चसर्वारोचककुष्ठनुत्|

निम्बफलंरसेतिक्तंपाकेतुकटुभेदनम् |

स्निग्धंलघूष्णंकुष्ठघ्नंगुल्मार्शःकृमिमेहनुत् ||८४||

6.भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - ४. गुडूच्यादिवर्ग-पारिभद्र

पारिभद्रोनिम्बतरुर्मन्दारःपारिजातकः|

पारिभद्रोऽनिलश्लेष्मशोथमेदःकृमिप्रणुत् |

तत्पत्रंपित्तरोगघ्नंकर्णव्याधिविनाशनम् ||८७||

7. मदनपालनिघण्टु –१. अभयादिवर्ग-निम्ब

निम्बोनियमनोनेताऽरिष्टःस्यात्पारिभद्रकः |

सुतिक्तःसर्वतोभद्रःपिचुमन्दःप्रभद्रकः|

कुष्ठहादेवदत्तश्चरविसन्निभसूर्यकौ||१३७||

निम्बःशीतोलघुर्ग्राहीकटुपाकोऽग्निवातकृत् ||१३८||

व्रणपित्तकफच्छर्दिकुष्ठहृल्लासमेहनुत् |

निम्बपत्रंस्मृतंनेत्र्यंकृमिपित्तविषप्रणुत्||१३९||

तत्फलंभेदनंस्निग्धमुष्णंकुष्ठहरंलघु |

अपक्वंपाचयेन्निम्बःपक्वंचपरिशोषयेत् ||१४०||

8. राजनिघण्टु – ९. प्रभद्रादिवर्ग-प्रभद्र

अथनिगदितःप्रभद्रःपिचुमन्दःपारिभद्रकोनिम्बः|

काकफलःकीरेष्टोनेताऽरिष्टश्चसर्वतोभद्रः||७||

धमनोविशीर्णपर्णीपवनेष्टःपीतसारकःशीतः|

वरतिक्तोऽरिष्टफलोज्येष्ठामालकश्चहिङ्गुनिर्यासः||८||

छर्दनश्चाग्निधमनोज्ञेयानाम्नान्तुविंशतिः||९||

प्रभद्रकःप्रभवति शीततिक्तकःकफव्रणक्रिमिवमिशोफशान्तये|

बलासभिद्वहुविषपित्तदोषजिद्विशेषतोहृदयविदाहशान्तिकृत्॥१०॥

9. राजनिघण्टु - १५. क्षीरादिवर्ग-निम्बतैल

निम्बतैलंतुनात्युष्णंक्रिमिकुष्ठकफापहम्॥११७॥

10. शोढलनिघण्टु

नामसङ्ग्रह (प्रथमभाग) - १. गुडूच्यादिवर्ग-निम्ब

निम्बोनियमनोनेतापिचुमन्दःसुतिक्तकः|

अरिष्टःसर्वतोभद्रःसुभद्रःपारिभद्रकः॥११८|

शुकप्रियश्चीर्णपर्णोयवनेष्टोवरत्वचः|

छर्दनोहिङ्गुनिर्यासःपीतसारोरविप्रियः॥११९॥

III) Yashtimadhu (Glycyrrhiza glabra Linn)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1.Vedic review of Yashtimadhu:[273]

In Vedic literature Yashtimadhu is described as,

Madudha: According to KaushikSutra Jesthamadha, Shonakiya Atharvaved Samhita 6/101/3 Sayanbashya MadhuVruksha or Yashtimadhu. According to Mujumdar, Honey Plant. [274]

Klintak: Gobhil Sutram1/10/10[275]

Jeshtimadhu:VruschikdanshLepanarth,Streevashikaranpayog.KeshavPadhatti32/5,35/21,38/17[276]

Madhuk: Atharvaparishista as a Samindha Dravy, for speech improvement ShonakiyaAtharvved Samhita1/34/1 in marriage Manibandhanarth Shonakiya Atharvved Samhita 1/34[276]

Madhuyashthika: Discribed in MoolavidhiPrakaran Praskar Ghruhya Sutra 1/21[277]

Mdhula: Accordig to SayanBhashya itis describe asVishanashini, Mashakjambhini. Sarpavisha Maitrayani Samhita 4/9/1[278]

2. Samhitakalin Review: [279-281]

I. Charak Samhita: [279]

- As a constituent of Vamakdravya in Apamargatanduliya chapter. (Ch.Su.2/7)
- As a constituent of Vatahar Lepa in Argvadhdi chapter. (Ch.Su.3/21)
- As a Constitutuentof Sandhaniya, jeevaniya, KanthyaGana in Shadaveerechaniyashatashritiya chapter.(Ch.Su.4/9)
- As a constituent of Pratham Bramhya rasayan. (Ch.Chi.1/49)
- As a constituent of Jwaranashak kashaya in Jwarachikitsa. (Ch.Chi.3/19)
- As a constituent of Abhangya Taila in Kushtha chikitsa. (Ch.Chi.7/133)
- As a constituent of Jivantyadi Ghruta in Jwarachikitsa. (Ch.Chi.3/250)
- As a constituent of Haridradi Ghruta in Pandurogchikitsa. (Ch.Chi.16/53)
- As a constituent of Lepan Dravya in Visarpa chikitsa. (Ch.Chi.21/74)
- As a constituent of Lepan Dravya in Vranarog chikitsa. (Ch.Chi.21/74)
- As a constituent of Lepan Dravya for Shoolashaman in Vranachikitsa. (Ch.Chi.25/47)
- As a constituent of Lepan for Vranashodhan in Vranachikitsa. (Ch.Chi.25/48)

II. Sushrut Samhita: [280]

- As a constituent of Lepan for Pittaj Arbud in. (Su.Chi.18/346)
- As a constituent of Lepa for Khardhaha in Kharapakvidhi Adhaya. (Su.Su.11/21)
- As a constituent of lepa for Rakttastravahar in Shonitvraniya Chapter. (Su.Su.14/36)
- As a constituent of Lepan for Asamyak Vrana in Karnavedhan Chikitsa. (Su.Su.16/6)
- As a Constituent of Kashay for Pittajanyashool in Mishrakadhaya. (Su.Su.37/4)
- As a constituent of jeevniya, Bruhaniya, Vrushya, kakolyadi Gana in Dravyasangrahaniya Chapter.(Su.Su.38/35)
- As a constituent of Sarivadi Gana for Pitta, Daha,Trushna Shaman, in Dravyasangrahaniya Chapter .(Su.Su.38/39)
- As a constituent of Anjanadi Gana for pitta and Dahashaman in Dravya Sangrahniya chapter (Su.Su.38/41)
- As a constituent of Lepa for Vranapandu karma in Vrana chapter. (Su.Chi.1/96)
- As a constituent of Lepa for Vranapratisaraniya Vrana chapter. (Su.Chi.1/99)
- As a constituent of Kwath for Kaphaj vatarakta chapter. (Su.Chi.5/10)
- As a constituent of Siddhaghruta for dusita vrana in Vidharadhi Chapter. (Su.Chi.16/17)
- As a constituent of Hita dravya in VishaChikitsa. (Su.Chi.8/131, 32)

III. AshtangHriday:[281]

- As a constituent of JeevaniyaGana in Shodhanadi gana chapter.(A.Hr.Su.15/8)
- As a constituent of Nyogrodhadi Gana in Shodhanadigan chapter (A.Hr.Su.15/41)
- As a constituent of VranaLepa in Shastrakarmavidhi chapter.(A.Hr.Su.29/55)
- As a constituent of VranaLepa in Ksharagnikarma chapter.(A.Hr.Su.30/34)
- As a constituent of Siddhaghruta in Rajyakshma chikitsa chapter. (A.Hr.Chi.5/16)

- As a constituent of Kashya in VidradhiVrudhi chikitsa chapter. (A.Hr.Chi.13/11)
- As a constituent of Lepa for daha inVatarakta chikitsa chapter. (A.Hr.Chi.22/28)
- As a constituent of Siddha taila for Ropan in Sadyovrana chapter. (A.Hr.Ut.26/55)
- As a constituent of Kashya in Shotharoga chikitsa chapter. (A.Hr.Chi.17 31/)
- As a constituent of Vrana lepa in Vrana chikitsa chapter.(A.Hr.Chi.1/96)

IV. Nighantu Kaal:[282]

- It is mentioned in Sarivadi/Vachadi Varga of Ashtanga Nighantu.
- It is mentioned in Guduchyadi Varga of Dhanvantari Nighantu.
- It is mentioned in Guduchyadi Varga of Shodhal Nighantu.
- It is mentioned in AushadhiVarga of Kaiyadev Nighantu.
- It is mentioned in Abhayadi Varga of Madanpal Nighantu.
- It is mentioned in Pippalyadi Varga of Raj Nighantu.
- It is mentioned in Mishrakprakaran Haritkyadi Varga of Bhavprakash Nighantu.
- It is mentioned in Palashadi Varga of Nighantu Adarsha.

3. Morphology of Yashtimadhu: [283]

Yashtimadhu consists of dried, unpeeled, stolon and root of Glycyrrhizaglabra Linn, (Fam. Leguminosae), a tall perennial herb, upto 2 m high found cultivated in Europe, Persia, and Afghanistan and too little extent in some parts of India.

1. Description: [283]

a) Macroscopic:

Stolon consists of yellowish brown or dark brown outer layer, externally longitudinally wrinkled, with occasional small buds and encircling scale leaves, smoothed transversely, cut surface shows a cambium ring about one-third of radius from outer surface and a small central pith, root similar without a pith, fracture, coarsely fibrous in bark and splintery in wood, Odour, faint and characteristic taste is sweetish.

b) Microscopic:

Stolon- transverse section of stolon shows cork of 10-20 or more layers of tabular cells, outer layers with reddish-brown amorphous contents, inner 3 or 4 rows having thicker,colourless walls,secondary cortex usually of 1-3 layers of radially arranged

parenchymatous cells containing isolated prisms of calcium oxalate, secondary phloem abroad band,cells of inner part cellulosic and outer lignified, radially arranged groups of about 10-50 fibres, surrounded by a sheath of parenchyma cells, each usually containing prism of calcium oxalate about 10-3 μ long, cambium form tissue of 3 or more layers of cells,secondary xylem distinctly radiate with medullary rays, 3-5cells wide, vessels 168 about 80-200μ in diameter with thick, yellow, pitted, reticulately thick end walls, groups of lignified fibres with crystal sheaths similar to those of phloem, xylem parenchyma of two kinds, those between the vessels having thick pitted walls with out inter-cellular spaces, the remaining with thin walls, pith of parenchymatous cells in longitudinal rows, with inter-cellular spaces.Root-transverse section of root shows structure closely resembling that of stolon except that no medulla is present, xylem tetra ,usually four principal medullary rays at right angles to each other unpeeled, drug cork shows phelloderm and sometimes without secondary phloem all arenchyrnatous tissues containing abundant, simple,ovalor rounded starch grains,2-20μin length.

2 Identity, purity and strength: [283]

Total Ash not more than 10 per cent, Appendix 2.2.3.

Acid-insolubl ash not more than 2.5 per cent, Appendix 2.2.4.

Alcohol-soluble extractive not less than 10 per cent, Appendix 2.2.6.

Water-soluble extractive not less than 20 per cent, Appendix 2.2.7.

3. Constituents: [284]

Glycyrrhizine, prenylatedbiaurone, licoagrone, 7-acetoxy-2methyl-isoflavone, asrtagalin, liquiritigenin, Glycyrrhizin, glycyrrhizic acid, glycyrrhetinic acid, asparagine, Sugars, resin and starch.

4. Properties and action: [283]

1) **Rasa**: Madhura
2) **Guna**: Guru, Snigdha
3) **Virya**: Sheet
4) **Vipaka**: Madhur

5)Karma: Baly, Dahaprashamanan, Keshya, Shothhar, Medhya, Trushnanigrahan,Vatanuloman, Mrudurechan, Shonitsthapan, Kaphanissaran, Kanthya, Mutral, Shukravardhan, Varnya, Jeevaniya, Rasayan, Chakshushya, Sandhaniya

5. Important formulations:[283]

Eladigulika, Yashtimadhutailaa, Kalyanleha.

6. Therapeutic uses: [283]

Vranashodhan, Ropan, Vaatvikar.Amavata, Vatarakta, Shirorog, Trushna, Vaman, Vibhandha, Amlapitta, Shwas, Kas, Hikka, Yakashma, Mutrakruccha, Vajikar.

7. Dose: [283] 2-4 gm of the drug in powder form.

8. Classification According to Samhita: [285]

Table No.35: Classification of Yashtimadhu according to Samhita

Charaka Samhita	**Sushruta Samhita**	**Ashtanga Hridaya**
Jeevaniya	Haridradi Gana	Ambashtadi Varga
Sandhaniya	Bruhatyadigana	Niruhan Varga
Varnya	KakolyadiGana	PittasanshamanGana
Kanthya	Sarivadi Gana	JeevaniyaGana
Kandughna	AnjanadiGana	SarivadiGana
Snehopag	Ambashthdai Gana	Patoladi Varga
Vamanopag	Shodhan Gana	Haridradi Varga
Asthapanopag	Sthanyajanan Varga	Nyogrodhadi Varga
Angamarda Prashaman	GarbhasthapanVarga	Madhur Skanda
Shoonitsthapangan	Keshyaranjan Varga	VamakGana
Garbhasthapan	Medhya Rasayan	Niruhaupyogi Varga
		RaktasthambhakVarga

9. Classification According to Nighantu: [282]

Table No.36: Classification of Yashtimadhu according to Nighantu

Nighantu	**Varga**
Ashtang Nighantu	Sarivadigana,Vachadigana
Dhanvantari Nighantu	Guduchyadivarga
Shodhal Nighantu	Guduchyadivarga
Madanpal Nighantu	Abhayadivarga
Raj Nighantu	Pipalyadivarga
Kaiydev Nighantu	Aushadivarga
Bhavprakash Nighantu	Hritakyadi Varga
Nighantu Adarsha	Palashadi Varga

10. Taxonomical Classification: [286]

Table No.37: Taxonomical Classification of Yashtimadhu

Kingdom	Plantae
Unranked	Angiosperms
Unranked	Edicots
Unranked	Rosids
Order	Fabales
Family	Leguminoceae / Faboideae
Genus	Glycyrrhiza
Species	Glabra

11. Vernacular Names:[283]

- **Assamese**:Jesthimadhu, Yeshtmadhu
- **Bengali** : Yashtimadhu
- **English** : Liquorice root
- **Gujrati** : Jethimadha, Jethimard, Jethimadh
- **Hindi** : Mulethi, Mulathi, Muleti, Jethimadhu, Jethimadh
- **Kannada** : Jestamadu, Madhuka, Jyeshtamadhu, Atimadhura
- **Kashmiri** : Multhi
- **Malayalam** : Irattimadhuram
- **Marathi** : Jesthamadh
- **Oriya** : Jatimadhu,Jastimadhu
- **Punjabi :** Jethimadh, Mulathi
- **Tamil** : Athimadhuram
- **Telugu** : Atimadhuramu
- **Urdu :** Mulethi, Asl-us-sus

12. Parayayi Nama of Yashtimadhu according to Nighantu:[282]

Table No.38: Parayayi Nama of Yashtimadhu according to Nighantu

Nighantu → / **ParyayiNama ↓**	**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N**	**N.A.**
Yashtimadhuk	+	+	+	+	+	+	+	+
Yashti		+	+		+	+	+	+
Mdhuk	+	Madhuyastya	+	+		+	+	+
Klintak	+		+		+	+	+	+
Mdhuparni					Madhu-valli	+		

13. Paryayi Nama:[287]

निरुक्ति

1. Madhuk: Sweet as like honey.

2. **Yashtimadhu**: It is available in sweet wooden form

3. **Kleetak**: It diminishes impotency.

14. Gunakarmatmaka Vivechana: [282]

Rasapanchaka i.e.Guna, Rasa, Vipaka, Virya, and Karma.;with these characteristics, the drug can work. These are described in all Nighantus.

Table No.39: Gunapanchak of Yashtimadhu: [282]

Properties		**A.N**	**D.N**	**S.N**	**M.N.**	**R.N.**	**K.N.**	**B.N.**	**N.A.**
Guna	Sheet		+	+	+		+	+	+
	Guru				+		+	+	+
	Snigdha						+	+	+
Rasa	Madhur		+	+	+	+	+	+	+
	Tiktta					+			+
Virya	Sheet		+	+	+	+	+	+	+
Vipaka	Madhur								+

Table No.40: Dosha, karma and Rog-ghnata of Yashtimadhu according to Nighantu:[282]

Guna karma	A.N.	D.N.	S.N	M.N.	R.N.	K.N.	B.N.
Dosha karma	Pittaghna	Pittaghna		Pitta-jeet	Pitt-aghna	Pitta-ghna Vata-ghnaRaktt-ghna	Pitta-ghna Vata-ghna Rakttaghna
	Astrajeet						
Pramukha Karma	Stanyad-oshhara	Vrushya, Shoshhara	Sho-shv nashi	Bal-ya	Chaksus-hya	Chakshu, Bala, Varna-kruta, Shukral	Chakshu, Bala, Varna-kruta, hukral
Rog-ghnata	Trushna			Truta	Trushna	Trushna	Trushna
						Glani	Glani
	Shosha	Shosha			Shosha	Khata	Kshya
	Daha						
	Jwara						
	Vamak	Chardi		Cha-rdi		Chardi	Chardi
	Atisaraghana						
	Meda						
	Adhyavathar						
		Vishaha				Vishaha	Vishaha
					Hru-droga		
					Vrana	Vrana	Vranashotha
						Shotha	
						Keshya	Keshya
						Swarya	Swarya

15. Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha Karma and Rogghanta of Yashtimadhu: [282]

1.अष्टाङ्गनिघण्टु –

1) २.सारिवादिगण

सारिवोशीरकाश्मर्यमधूकशिशिरद्वयम्|

यष्टीपरुषकंहन्तिदाहपित्तास्रतृट्ज्वरान्||२३||

सारिवादिगणंवक्ष्येपुराप्रोक्तातुसारिवा|

वीरण्यामव(भय)लामज्जकोशीरममृणालकम्||२४||

वीरंवीरणमूलंचबहुमूलंरणप्रिया|

सारिवादिगण

यष्टीमधुकयष्ट्याह्वामधुकंक्लीतकाह्वयम्|

परुषकोमृदु फलोरोषजोधन्वनच्छद||२९||

1.अष्टाङ्गनिघण्टु - १९.वचादिगण

वचाजलददेवाह्वनागरातिविषामयाः|

हरिद्राद्वययष्ट्याह्वकलशीकुटजोद्भवाः||१४९||

वचाहरिद्रादिगणावामातीसारनाशनौ|

मेदःकफाढ्यपवनस्तन्यदोषनिबर्हणौ||१५०||

वचादोप्राग्वचाप्रोक्तामुस्तातुजलदाह्वया|

गाङ्गेयीकुरुविन्दाचदेवाह्वाभद्रमुस्तकम्||१५१||

2. धन्वन्तरिनिघण्टु - १. गुडूच्यादिवर्ग

मधुयष्टी

मधुयष्टीचयष्टीचयष्टीमधुमधुस्रवा|

यष्टीकंमधुकंचैवयष्ट्याह्वंमधुयष्टिका||१५९||

मधुयष्टीस्वादु रसाशीतपित्तविनाशिनी|

वृष्याशोषक्षयहराविषच्छर्दिविनाशनी||१६०||

3. शोढलनिघण्टु

(प्रथमभाग) - १.गुडूच्यादिवर्गमधुयष्टी

मधुयष्ट्यांचयष्टीचयष्टीमधुमधुस्रवा|

मधुकंक्लीतिकासाचक्लीतनंचमधूलिका||१९६||

मधुपर्णीरसासौम्याविरसाशोषनाशिनी|१९७|

4.मदनपालनिघण्टु – १. अभयादिवर्गमधुयष्टी

मधुयष्टीक्लीतनकंयष्टीमधुमधूलिका|

यष्ट्याह्वंमधुकंयष्टिमधूकंजलजामधुः|

मधुयष्टीगुरुःशीताबल्यातृट्छर्दिपित्तजित्||८७||

5. राजनिघण्टु –६. पिप्पल्यादिवर्गयष्टीमधु

यष्टीमधुर्मधुयष्टीमधुवल्लीमधुस्रवा|

मधुकंमधुकायष्टीयष्ट्याह्वंवसुसम्मितम्||१४४||

मधुरंयष्टिमधुकंकिञ्चित्तिक्तंचशीतलम्|

चक्षुष्यंपित्तहृद्रु च्यंशोषतृष्णाव्रणापहम्|१४५||

6.कैयदेवनिघण्टु - १.ओषधिवर्गयष्टीमधु

यष्टीमधुकंमधुकंमधुयष्टीमधुश्रवा||१०१||

यष्टीमधुक्लीतनकंयष्ट्याह्वंक्लीतनंमधु|

यष्टीकमपरांभोजामधुपर्णीमधुलिका||१०२||

यष्टीहिमागुरुःस्वादु श्चक्षुष्याबलवर्णकृत्

सुस्निग्धाशुक्रलाकेश्यास्वर्यापित्तानिलास्रजित्||१०३||

व्रणशोफविषच्छर्दितृष्णाग्लानिक्षतापहा|१०४|

१.ओषधिवर्गयष्टीमधु

यष्टीमधुकंमधुकंमधुयष्टीमधुस्रवा||११२८||

यष्टीमधुक्लीतनकंयष्ट्याह्वंक्लीतकंमधु|

मधुयष्ट्यपराम्भोजामधुपर्णीमधूलिका||११२९||

मधुकंमधुरंवृष्यंवर्ण्यंस्वर्यंहिमंगुरु

सुस्निग्धंबृंहणंकेश्यंवातपित्तकफापहम्||११३०||

सद्यःक्षतास्रतृट्छर्दिक्षयशोफव्रणान्हरेत्|११३१|

7. भावप्रकाश-Aपूर्वखण्ड-मिश्रप्रकरण - २. हरीतक्यादिवर्गयष्टीमधु

यष्टीमधुतथायष्टीमधुकंक्लीतकंतथा|

अन्यत्क्लीतनकंतत्तुभवेत्तोयेमधूलिका||१२८||

यष्टीहिमागुरुःस्वाद्वीचक्षुष्याबलवर्णकृत्|

सुस्निग्धाशुकलाकेश्यास्वर्यापित्तानिलास्रजित्|

घ्राणशोथविषच्छर्दितृष्णाग्लानिक्षयापहा||१२९||

IV. Til (Sesamum indicum)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1. **Vedic review:**[288]

Jaetil: Mostly it is wild variety of Tila.TheYavagu of this Tila is mentioned.[289]

Tila is mentioned in Atharvaveda at 8-8-3, 5-3-10, 4-7-3, 2-8-3, 6-140-2. [290]

Til is not mentioned in Rugveda, but mentioned in other Samhita with Masha, and Shishir Dhanya. As Gramya and Aranya types. Tail is extracted from it. Tilapishti is used as a fuel, as a dietry item, Keshya.[291]

2. Samhitakalin review: [292-94]

I. CharakSamhita: [292]

- As a constituent of Haradradi Lepa dravya in Aaragvadhadi chapter. (Ch.Su.3/14)
- As a constituent of Sthavarsnheha yoni adrvya in Snehaadhya chapter. (Ch.Su.13/10)
- As a Rakttadushti hetu dravya in Vidhishonit chapter. (Ch.Su.24/6)
- As a Shukravardhak dravya in Annapanvidhi chapter. (Ch.Su.27/270)
- As a Prameha hetu dravya in Prameha nidan chapter. (Ch.Ni.4/5)
- As a constituent of lepa in Raktaj Arsha chikitsa chapter. (Ch.Chi.14/220)
- As a constituent of Upnahdravya in Dwivraniya chapter. (Ch.Chi.25/51)
- As a constituent of VatajVrana Lepa dravya in Dwivraniya chapter. (Ch.Chi.7/74)
- As a constituent of Daha in Dwivraniya chapter. (Ch.Chi.7/78)
- As a constituent of Lepaniya dravya in Vatavyadhi chapter. (Ch.Chi. 28/114)

II. SushrutSamhita: [293]

- As a constituent of Vranaropakdravya in Dwivraniya chapter. (Su.Chi.1/65)
- As a constituent of Vranashodhakdravya in Dwivraniya chapter.(Su.Chi.1/69)
- As a constituent of Kalka in PittajVrana in Dwivraniya chapter. (Su.Chi.2/93)
- As a constituent of Kalka in Kaphaj Vrana in Dwivraniya chapter. (Su.Chi.2/94)
- As a constituent of Siddha Tail in Bhagnna chikitsa chapter.(Su.Chi.3/55)

- As a constituent of Sarvakushtanashak lepa Dravya in Kushtarog chikitsa chapter. (Su.Chi.9/10)
- As a constituent of Lepa in Vidradhi chikitsa chapter.(Su.Chi.16/13)
- As a constituent of Taila in VranaShodhan in Granthi, Apachi chikitsa chapter.(Su.Chi.18/7)

III. AshtangaHriday:[294]

- The Guna of Tila is mentioned in Annaswaroopvidnyaniya chapter. (A.Hr.Su.6/23)
- As a constituent of Madyamkshar Dravya in Ksharagnikarma chapter. (A.Hr.Su.30/12)
- As a constituent of Vataj vidradhi lepa Dravya in Vidradhi-Vrudhi chikitsa chapter. (A.Hr.Chi.13/4)
- As a constituent of Pittaj vidradhi lepa Dravya in VidradhiVrudhi chikitsa chapter. (A.Hr.Chi.13/5)
- As a Constituent of Kaphaj vidradhi lepa Dravya in Vidradhi Vrudhi chapter.(A.Hr.Chi.13/6)
- As a Constituent of lepa Dravya for shool in Vataraktta chikitsa chapter(A.Hr.Chi.22/30)
- As a constituent of lepa Dravya for Upanah in Vranachikitsa chapter. (A.Hr.Ut.25/35)
- As a constituent of lepa Dravya for Vranashodhan in Vranachikitsa chapter. (A.Hr.Chi.25/47)
- As a constituent of lepa Dravya for Shodhan ropan in Vranachikitsa chapter. (A.Hr.Chi.25/54)

A) Nighantu Kaal:[295]

- It is mentioned in SuvarnadiVarga of Dhanvantari Nighantu.
- It is mentioned in Taila Varga of Shodhal Nighantu.
- It is mentioned in Paniyadi andDhanyak Varga of Madanpal Nighantu.
- It is mentioned in Kariradi, Ksheeradi, Shalyadi Varga of Raj Nighantu.
- It is mentioned in Dhanyavarga, TailaVarga of Bhavprajkash Nighantu.
- It is mentioned in TilaVarga of Nighantu Adarsha.

3. Morphology of Til

Description:[296]

a) Macroscopic:

Seed white, brown, grey or black, flattened, ovate in shape, smooth or reticulate,2.5 to 3 mm long and 1.5 mm broad, one side slightly concave with faint marginal line and an equally faint central line; taste- pleasant and oily.

b) Microscopic:

Testa of seed shows single layered palisade-like, thin-walled, yellowish colouredcells, and the rest of the testa composed of collapsed cells; endosperm 3 layered, rarely 2 layered,consisting of cellulosic polygonal cells of parenchyma containing fixed oils and small aleuronegrains ; cotyledons two, externally covered with thin cuticle; single layered epidermal cell, followed by a single row of palisade- like cells; rest of the tissues consist of polygonal, parenchyma cells containing fixed oil and aleurone grains.

Powder - Blackish coloured; shows palisade-like cells in surface view, parenchymacells, aleurone grains and oil globules.

c) Identity, purity and strength: [296]

Foreign matter not more than 2 per cent, Appendix 2.2.2.

Total Ash not more than 9 per cent, Appendix 2.2.3.

Acid-insoluble ash not more than 1.5 per cent, Appendix 2.2.4.

Alcohol-soluble extractive not less than 20 per cent, Appendix 2.2.6.

Water-soluble extractive not less than 4 per cent, Appendix 2.2.7.

Fixed Oil not less than 35 per cent, Appendix 2.2.8

T.L.C.-T.L.C. of alcoholic extract on Silica gel 'G' plate using Toluene: Ethylacetate (9 : 1) shows under UV (366 nm) three fluorescent zones at Rf. 0.57, 0.64 (both light blue) and 0.72 (blue). On exposure to Iodine vapour five spots appear at Rf. 0.08, 0.57, 0.64, 0.72and 0.94 (all yellow). On spraying with Vanillin-Sulphuric acid reagent and heating the plate for ten minutes at 110^{o} C seven spots appear at Rf. 0.08, 0.57, 0.64, 0.72 (allviolet), 0.76, 0.84 (both light violet) and 0.94 (violet).

D) Constituents:[297]

Natural lipid, glycolipids, phospholipid, arginine, cysteine, histidine, isoleucine, isoleucine, leucine, lysine, methionine, phenyalanine, threonine, tryptophan, tyrosine, valine, α- and β-globulin, ascorbic acid, pyridoxine, riboflavin, sesamol, thiamine, galactose, glucose, lychone.

E. Properties and action:[296]

1. Rasa: Madhura, Katu, Tikta, Kashya

2. Guna: Vyavayi, Guru, Snigdha, Sukshma

3. Virya: Ushna

4. Vipaka: Madhura

5. Karma: Balya, Keshya, Pittala, Rasayana, Sangrahi, Vataghna, Varnya, Vishaghna, Snehana, Swarya, Snehopaga, Kushtahara, Mutrabandhaka, Medhavardhaka, Agnivardhaka, Avasadakara, Keshya, Karnapalivardhak, Kaphakopaka, VranaSamsadhaka, Vranapachaka, and Dahanashak, Bhagnaprakopak, Vajikara, AgnibalaVardhaka.

F. Important formulations: [296]

Narasimha Churna, Jatiphaladya Chura,Haridradi Lepa, Tiladi Upanaha, Tiladi Yoga, Priyaladi Yoga, Mustadi Upanaha, Sunthyadi Churna, PathyadiGulika, Hingvadi Yoga, Bhallatakadi Modaka.

G.Therapeutic uses: [296]

Ashmari, Akshiroga, Atisara, Amasula, Galaganda, Gulma, Hikka, Krumi, Kshaya, Kasa, Kushta, Peenasa, Pradara, Pravahika,Raktatisara, Visarpa, Udvarta, Yonishula, Udara,Raktapitta,Vatarakta, NadiVrana¸Vrana, Svitra, Granthi, Khalitya, Palitya, Dantaroga,Atidgdha .

H. Dose : [296] Powder 5-10 gm/day.

5.Classification :

A) Classification According to Samhita: [298]

Table No.41: Classification of Tila according to Samhita

CharakaSamhita	**SushrutaSamhita**	**AshtangaHridaya**
SthavarSneha	Pachak gana	Snehavarga
Agryasangraha	Shodhak Gana	GundushDravya
Vatavijay Sneha	Dantashodhan Dravya	DhupanDravya
	Kesharajan Dravya	DravPadarth
	Vajikaranarth	

B) Classification according to Nighantu: [295]

Table No.42: Classification of Tila according to Nighantu

Nighantu	**Varga**
Dhanvantari Nighantu	Suvarnadi Varga
Shodhal Nighantu	Taila Varga
Madanpal Nighantu	Paniyadi, DhanyaVarga
Raj Nighantu	Karveeradi,Ksheeradi,ShlyadiVarga
Kaiydev Nighantu	Drava,Dhanya,TailaVarga
Bhavprakash Nighantu	Drava, ,TailaVarga
Nighantu Adarsha	TilaVarga

C) Taxonomical Classification: [299]

Table No.43.: Taxonomical Classification of Tila

Kingdom	Plantae
Unranked	Angiosperms
Unranked	Edicots
Unranked	Asterids
Order	Lamiales
Family	Pedaliaceae
Genus	Sesamum
Species	Indicum

6. Vernacular names:[296]

- **Sanskrit** : Tila
- **Assamese** : Simmasim
- **Bengali** : Tilagachh
- **English** : Sesame, Gingelly-oil Seeds
- **Gujrati** :Tall
- **Hindi** : Tila, Teel, Tili
- **Kannada** : Accheellu, Ellu
- **Malayalam** : Ellu
- **Marathi** : Tila
- **Oriya** : Til
- **Punjabi** : Til
- **Tamil** : Ellu
- **Telugu** : Nuvvulu
- **Urdu** : Kunjad

Table No.44: Parayayi Nama of Tila according to Nighantu:[295]

Nighantu→ ParyayiNama ↓	**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N**	**N.A.**
Tila	-	Tila		+	+			+
Homadhyana	-	Homadhyana			+			+
Pavitra	-	Pavitra			+			+
Pitrutarpan	-	Pitrutrapan			+			
Papaghna	-	Papaghna			+			
Pootdhanya	-	Pootdhanya			+			
Jartil	-	Jartil		+	+			+
Vanodbhava	-	Vanodbhava		Vajat	+			+
Vajat	-			Tilodbhava				
Tilodbhava	-			Tailapushpa				
Tailapushpa					Taruni			

7. निरुक्ति:[299]

Til: It is Snigdha. It gives Snigdhata to all over body.

8. Gunakarmatmaka Vivechana:[295]

Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma; with these properties the drug can work. These are described in all Nighantus.

Table No.45: Gunapanchak of Tila

Properties		**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N.**	**N.A.**
Rasa	Madhur			+	+	+			
	Katu		+	+	+		+	+	
	Tiktta		+		+			+	
	Kashya		+	+	+		+	+	
							+		
Guna	Guru		+	+	+	+	+		
	Snigdha		+	+	+	+	+		
	Ushna		+	+	+	+	+		
	Vavayeee		+	+			+		
	Vishada		+		+		+		
	Grahi		+						
	Tikshna				+	+	+		
Vipaka	Madhur		+					+	+
Virya	Ushna		+	+	+	+	+	+	+

Table No.46: Dosha, karma, and Rogghanata of Til: [295]

Gunakarma	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N.
Dosha karma		Kaphapittaanuta	+	Vatakaphahar	Kaphapittaanuta	+	+
						Rakttapittakar	
Pramukha Karma		Balya Grahi Vrana Varnya Shodhak Dipan Stayna	+ + + + +	+ + + + 	+ + + + +	+ + + Tarpan	 +
Roga		Vatroga	+				
		Kesharoga	+				
		Vrana	+	+	+	+	+
		Twacharog	+				
		Bhagna					
		Kushta	+	+	+	+	+
		Urdhawajatruroga	+	+	+	+	+
		Krumi					
		Danta					
		Kandu	+		+	+	+

8. Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha, Karma and Rogghanta of Til:[295]

1.धन्वन्तरिनिघण्टु - ६. सुवर्णादिवर्गतिल

तिलस्तुहोमधान्यंस्यात्पवित्रःपितृतर्पणी|

पापघ्नःपूतधान्यश्चजर्तिलस्तुवनोद्भवः||११५||

तिलोरसेकटुस्तिक्तोमधुरस्तुवरोगुरुः|

विपाकेकटुकःस्वादुःस्निग्धोष्णःकफपित्तनुत्|११६||

बल्यःकेश्योहिमस्पर्शस्त्वच्यःस्तन्योव्रणेहितः|

दन्त्योऽल्पमूत्रकृद्ग्राहीवातघ्नोऽग्निमृतिप्रदः||११७||

तिलतैल (तैलविशेष)

स्नानाभ्यङ्गावगाहेषुतिलतैलंविशिष्यते|

तद्वद्धस्तिष्वपानेषुनस्यकर्णाक्षिपूरणे||१२०||

अन्नपानविधौवाऽपिप्रयोज्यंवातशान्तये|

छिन्नभिन्नयुतल्पिष्ठमथितक्षतपातिते||१२१||

भग्नेस्फुटितविद्धाग्निदग्धविश्लिष्टदारिते|

भयाभिहितनिर्भुग्नेमृगव्यालादिभक्षिते||१२२||

तैलयोगश्चसंस्कारात्सर्वरोगापहोमतः|१२३|

3. शोढलनिघण्टु: गुणसङ्ग्रह (द्वितीयभाग) - १४. तैलवर्ग

तिलतैलंकषायानुरसंबल्यंचपित्तकृत्|

दीपनंवातविध्वंसिस्वादुतीक्ष्णंव्यवायिच||७६४||

त्वद्रोषकृदचक्षुष्यंसूक्ष्मोष्णंकफकृन्नच|

कृशानांबृंहणायालंस्थूलानांकर्शनायच||७६५||

कृमिघ्नंबद्धविण्मूत्रंसंस्कारात्सर्वरोगजित्|

छिन्नभिन्नच्युतोत्पिष्टमथितोत्कृत्तचर्मणि॥७६६॥

भिन्नस्फुटितबद्धाग्निदग्धविश्लिष्टदारिते।

तथोपहतनिर्भुग्नेमृगव्यालादिभक्षिते॥७६७॥

सेकाभ्यङ्गावगाहेषुतिलतैलंविशिष्यते।

तद्वद्वस्तिषुपानेषुनस्येकर्णाक्षिपूरणे॥७६८॥

अनुपानविधौचापिप्रयोज्यंवातशान्तये।

त्वच्यंकेश्यंतदभ्यङ्गेकपालेन्द्रियतर्पणम्॥७६९॥

तैलप्रयोगादजरानिर्विकाराजितश्रमाः।

4.मदनपालनिघण्टु–८. पानीयादिवर्ग-तैल

तैलमुष्णंगुरुस्थैर्यंबलवर्णकरंसरम्।

वृष्यंविकाशिविशदंमधुरंरसपाकयोः॥१३६॥

सोष्णंकषायनुरसंतिक्तंश्लेष्मानिलापहम्।

विपाकेमधुरंतीक्ष्णंबृंहणंरक्तपित्तजित्॥१३७॥

श्लेष्मलंकटुविण्मूत्रत्वग्गर्भाशयशोधनम्।

दीपनंमतिदंकेश्यंव्यायामव्रणमेहनुत्॥१३८॥

श्रोत्रयोनिशिरःशूलनेत्ररोगविनाशनम्।

मथितच्युतविच्छिन्नभग्नव्यालविषादिषु।

क्षतेऽग्निदग्धेतत्पथ्यंपानाभ्यङ्गादिभिःसदा॥१३९॥

घृतमब्दात्परंपक्वंहीनवीर्यंप्रजायते।

तैलंपक्वमपक्वंवाचिरस्थायिगुणाधिकम्॥१४०॥

मदनपालनिघण्टु–१०. धान्यवर्ग-तिल

तिलपुष्पस्तैलफलस्तिलपिञ्जोपरःसितः।

जातिलोवनजातःस्यादाढकीतुवरीमता॥४७॥

तिलःकषायो मधुरस्तिक्तकःकटुकोरसे |

तिलोग्राही गुरुःस्वादु ःस्निग्धोऽस्रकफपित्तलः॥४८॥

बल्यःकेश्योहिमस्पर्शस्त्वच्योव्रणहितःपरम्|

वन्योऽल्पमूत्रकृद्धातुनाशनोऽग्निमतिप्रदः॥४९॥

कृष्णःश्रेष्ठतमस्तेषुशुक्रलोमध्यमःसितः|

अन्येहीनतराःप्रोक्तास्तज्ज्ञैरक्तादयस्तिलाः॥५०॥

5. राजनिघण्टु–१०. करवीरादिवर्ग-तिलक

तिलकोविशेषकःस्यान्मुखमण्डनकश्चपुण्ड्रकःपुण्ड्रः|

स्थिरपुष्पःछिन्नरुहोदग्धरुहोरेचकश्चमृतजीवी ॥४२॥

तरुणीकटाक्षकामोवासन्तःसुन्दरोऽभीष्टः |

भालविभूषणसञ्ज्ञोविज्ञेयःपञ्चदशनामा॥४३॥

तिलकोमधुरःस्निग्धोवातपित्तकफापहः|

बलपुष्टिकरोह्यग्नेलघुर्मेदोविवर्धनः॥४४॥

तिलकत्वक्कषायोष्णापुंस्त्वघ्नीदन्तदोषनुत्|

क्रिमिशोफव्रणान्हन्तिरक्तदोषविनाशनी ॥४५॥

राजनिघण्टु - १५. क्षीरादिवर्ग-तिलतैल

तिलतैलमलङ्करोतिकेशंमधुरंतिक्तकषायमुष्णतीक्ष्णम्|

बलकृत्कफवातजन्तुखर्जूव्रणकण्डूतिहरंचकान्तिदायि॥१०९॥

राजनिघण्टु - १६. शाल्यादिवर्ग-तिल

तिलस्तुहोमधान्यंस्यात्पवित्रःपितृतर्पणः|

पापघ्नःपूतधान्यश्चजटिलस्तुवनोद्भवः॥१११॥

स्निग्धोवर्णबलाग्निवृद्धिजननस्तन्यानिलघ्नोगुरुः

सोष्णःपित्तकरोऽल्पमूत्रकरणःकेश्योऽतिपथ्योव्रणे|

सङ्ग्राहीमधुरःकषायसहितस्तिक्तोविपाकेकटुः

कृष्णःपथ्यतमःसितोऽल्पगुणदःक्षीणास्तथान्येतिलाः॥११२॥

6. कैयदेवनिघण्टु - ४. द्रववर्गतैलवर्ग

तैलंस्नेहोत्तमंस्नेहमुख्यंचतिलसम्भवम्।

अभ्यञ्जनंचुप्पडनंम्रक्षणंचप्रकीर्तितम्॥२९६॥

एरण्डफलसम्भूतमेरण्डकमितिस्मृतम्

उमातैलंचक्षौमंस्यात्कटुतैलंचसार्षपम्॥२९७॥

कुसुम्भतैलंकौसुम्भंआरुष्करमरुष्कजम्।

तुवरोत्थंतौवरकंनिम्बतैलंचनिम्बजम्॥२९८॥

कैयदेवनिघण्टु - ४. द्रववर्ग-तैलसामान्यगुण

तैलंस्वयोनिवत्तत्रतिलतैलंवरंगुरु।

कषायानुरसंतिक्तंमधुरंरसपाकतः ॥२९९॥

विकाशिविशदंसूक्ष्ममुष्णंसंस्पर्शवीर्ययोः।

मेदोविलेखनंकेश्यंतर्पणंरक्तपित्तकृत् ॥३००॥

निहन्तिकेवलंवातंकफयुक्तंचदीपनम् ।

व्रणजन्तुप्रमेहघ्नंव्यवायिकफकृन्नच॥३०१॥

मेधामांसबलस्थैर्यवर्णमार्दवशुक्रकृत्

बद्धमूत्रपुरीषञ्चगर्भाशयविशोधनम् ॥३०२॥

योनिकर्णशिरःशूलशमनंलघुताकरम्

त्वग्दोषजिच्चचक्षुष्यमभ्यङ्गेभोजनेऽन्यथा ॥३०३॥

श्लक्ष्णंपुरीषंबध्नातिस्खलितंतुप्रवर्तयेत्।

रूक्षादिकृद्धपवनस्रोतःसङ्कोचतोयदि।

रसोऽसम्यक्क्वहन्काश्र्यंकुर्याद्रक्ताद्यवर्धयन् ॥३०४॥

तेषुप्रविष्टंरसतःसौम्यस्निग्धत्वमार्दवैः|

तैलंक्षमंरसंनेतुंकृशानांतेनबृंहणम् ॥३०५॥

व्यवायिसूक्ष्मतीक्ष्णोष्णसरत्वैर्मेदसःक्षयम् |

शनैःप्रकुरुतेतैलंतेनस्यात्स्थौल्यनाशनम्॥३०६॥

अवृष्येतैललवणेजातूकर्ण्योऽब्रवीदिदम्

योनिकर्णशिरःशूलंनाशयेल्लघुताकरम् ॥३०७॥

योनेर्गर्भाशयस्यापिशोधनंक्लमनाशनम् |

छिन्नभिन्नोत्पिष्टविद्धपतितच्युतपीडिते॥३०८॥

लकुटाद्यैरभिहतेविश्लिष्टेस्फुटितेक्षते|

दग्धेव्यालदिदष्टेचसन्धिमुक्तादिकेहितम्॥३०९॥

सेकाभ्यङ्गावगाहेषुपाननावनवस्तिषु |

संस्कारेचान्नपानानांप्रयोज्यमिदमेवहि॥३१०॥

तत्तुसंस्कारसंयोगविशेषात्सर्वरोगजित्

अब्दादूर्ध्वंघृतंपक्वंहीनवीर्यंचकेवलम्

तैलंविपर्ययंविद्यात्पक्वेचापक्वएववा ॥३११॥

कैयदेवनिघण्टु - ३. धान्यवर्ग-तिल

तिलस्तैलफलःपूतःस्नेहपूरफलोऽपरः|

तिलपिञ्जस्तिलपेजोवनजोऽन्यस्तुजर्तिलः॥८०॥

तिलःकषायोमधुरस्तिक्तकःकटुकोरसः|

विपाकेकटुकःस्वादुःसुस्निग्धोबलकृत्गुरु॥८१॥

केश्योव्रणहितस्त्वच्योहिमस्पर्शोऽनिलापहः|

दन्त्योऽल्पमूत्रोमेधाग्निकफपित्तविवर्धनः ॥८२॥

तिलेषुशुक्रलःकृष्णःप्रधानोमध्यमःसितः|

अन्योऽरुणादयोज्ञेयागुणैर्न्यूनतरास्तिलाः॥८३॥

7.भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - ९. धान्यवर्ग-तिल

तिलःकृष्णःसितोरक्तःसवर्ण्योऽल्पतिलःस्मृतः।

तिलोरसेकटुस्तिक्तोमधुरस्तुवरोगुरुः।

विपाकेकटुकःस्वादुःस्निग्धोष्णःकफपित्तनुत्॥५३॥

बल्यःकेश्योहिमस्पर्शस्त्वच्यःस्तन्योव्रणेहितः।

दन्त्योऽल्पमूत्रकृद्ग्राहीवातघ्नोऽग्निमतिप्रदः॥५४॥

कृष्णःश्रेष्ठतमस्तेषुशुक्लोमध्यमःसितः।

अन्येहीनतरःप्रोक्तास्तज्ज्ञैरक्तादयस्तिलाः॥५५॥

भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - २०. तैलवर्ग-तैल

तिलादिस्निग्धवस्तूनांस्नेहस्तैलमुदाहृतम्।

तत्तुवातहरंसर्वविशेषातिलसम्भवम् ॥१॥

भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - २०. तैलवर्ग-तिलतैल

तिलतैलंगुरुस्थैर्यबलवर्णकरंसरम् ।

वृष्यंविकाशिविशदंमधुरंरसपाकयोः ॥२॥

सूक्ष्मंकषायानुरसंतिक्तंवातकफापहम्।

वीर्येणोष्णंहिमंस्पर्शेबृंहणंरक्तपित्तकृत्॥३॥

लेखनंबद्धविण्मूत्रंगर्भाशयविशोधनम्।

दीपनंबुद्धिदंमेध्यंव्यवायिव्रणमेहनुत्॥४॥

श्रोत्रयोनिशिरःशूलनाशनंलघुताकरम्।

त्वच्यंकेश्यंचचक्षुष्यमभ्यङ्गेभोजनेऽन्यथा॥५॥

छिन्नभिन्नच्युतोत्पिष्टमथितक्षतपिच्चिते।

भग्नस्फुटितविद्धाग्निदग्धविश्लिष्टदारिते॥६॥

तथाऽभिहतनिर्भुग्नमृगव्याघ्रादिविक्षते|

वस्तौपानेऽन्नसंस्कारेनस्येकर्णाक्षिपूरणे|

सेकाभ्यङ्गावगाहेषुतिलतैलंप्रशस्यते||७||

रूक्षादिदुष्टःपवनःस्रोतःसङ्कोचयेद्यदा|

रसोऽसम्यग्वहन्काश्र्यंकुर्याद्रक्तान्यवर्धयन्||८||

तेषुप्रवेष्टुंसरत्वसौक्ष्म्यस्निग्धत्वमार्दवैः|

तैलंक्षमंरसंनेतुंकृशानांतेनबृंहणम्||९||

व्यवायिसूक्ष्मतीक्ष्णोष्णसरत्वैर्मेदसःक्षयम् |

शनैःप्रकुरुतेतैलंतेनलेखनमीरितम्||१०||

द्रुतंपुरीषंबध्नातिस्खलितंतत्प्रवर्तयेत्|

ग्राहकंसारकञ्चापितेनतैलमुदीरितम्||११||

भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - २०. तैलवर्ग-घृत, तैलपरिभाषा

घृतमब्दात्परंपक्वंहीनवीर्यंप्रजायते|

तैलंपक्वमपक्वंवाचिरस्थायिगुणाधिकम्||१२||

V. Madhu/ Honey (Apisindica Mellifera)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern science.

1. Vedic review: [300,303]

- As a ahariya DravyaAtharaveda. [300,303]
- As a Pranij Dravya Atharvaveda 9-1-1 to24 [301,303]
- As a Madhu, Saragh, SapptamadhurPadarth Superior in categeory. Atharva Veda: 9-1-22, 23, 19, 17, 16, 4, 3, 2, 1 [300,303]

2. Samhita kalin review: [304-6]

I . Charaksamhita:[304]

- It is used for Mamsa and Shonitvardhan in Kshataksheena chapter. (Ch Chi 11/30)
- The Properties of Madhu are mentioned in Annapaanvidhi chapter (Ch Su 27/243-246)
- It is used for the healing of open wound (Ch Chi 25/65)
- It is costituents of Nimbadi kalka for shodhana and ropana of Vrana. (Ch Chi 25/83-85)
- It is mentioned in Twakvishuddhikar yoga in Vranachikitsa (Ch Chi 26/114)

II. Sushrut Samhita : [305]

- The Properties of Madhu are mentioned in Dravadravyavidhi chapter. (Su. Su 45/132)
- It is use during operative process of Baddhagudodar and Chidrodar. (Su chi 15/17)

III .Ashtanga Hriday: [306]

- It is mentioned in Twakvishuddhikar yoga in Vranachikitsa. (A.U.25/58)
- It is mentioned in kalka dravya forVranalepa in Vranachikitsa. (A.U.25/55)
- The Properties of madhu are mentioned in Dravdravyavidnyaneeyam chapter (AH .Su 5/52-53)
- As a constituetion of Gandusha dravya for ropana in Mukharoga. (AH Su 22/7)

IV.Nighantu Kaal: [307]

- It is mentioned in SuvarnadiVargaofDhanvantari Nighantu.
- It is mentioned in Namasanghrah, Parishishtya of Shodhal Nighantu.
- It is mentioned in Aushadhi Varga of Kaiyadev Nighantu.

- It is mentioned in Ikshukadi Varga of Madanpal Nighantu.
- It is mentioned in Paniyadi Varga of Raj Nighantu.
- It is mentioned in Madhu Varga of Bhavprakash Nighantu.

5. Constituents: [308]

Honey has approximately 40% fructose, 30% glucose, 5% sucrose and 20% water. It also contains several amino acids, antioxidants, vitamins, minerals, glucose oxidase, which produces hydrogen peroxide, and gluconicacid, which gives the honey an acidic pH of 3.2-4.5

6. Properties and action: [307]

- **Rasa**: Madhur,Kashya
- **Vipaka**: Madhur
- **Virya** : Sheet
- **Guna** : Rukshya,Laghu,Grahi
- **Karma**:Lekhan,Vrushya,Pittavatghna,Stanya,Chakshushya, Mukharoghar,Varnya,Vranaropak,Krumighna,Atisaraghna,Shwasaghna, Kasahar, Kanthya.

7. Important formulations: [309]

Chavanprash, Agastiprash, Nimbadikalka, Haritkyadiavleha, Mustadi yapanBasti.

8. Therapeutic uses: [307]

Mukharog, Netrarog, Shwas, Kas, Kshya, Atisar,Vrana, Kushta, Varnya,Bastidravya, Strotoshodhan.

9. Dose: [309] 5-7 gm/day orally

10. Classification: [310]

A. Classification According to Samhita: [310]

Table No.47: Classification of Madhu according to Samhita:

Charaka Samhita	Sushruta Samhita	Ashtanga Hridaya
Vamanopag Dravya	Lehaniya Gana	NiruhanVarga
Aahariya Dravya	Kumar Rasayan	Madhurskanda
Agryasangraha	Annapansangraha,Dravdravya	Dravapadarth
		Mehaghanavarga
		Gandush Dravya

B.Classification According to Nighantu:[307]

Table No.48: Classification of Madhu according to Nighantu

Nighantu	**Varga**
Dhanvanri Nighantu	Suvarnadi
Shodhal Nighantu	Parishishtya,Namasangraha
Madanpal Nighantu	Ikshukadi
Raj Nighantu	Paniyadi
Kaiyadev Nighantu	Aushadhi
Bhavprakash Nighantu	Madhu

C. Taxonomical Classification:[311]

Table No.49: Taxonomical Classification of Madhu

Kingdom	Amimalia
Phylum	Arthropoda
Class	Insecta
Order	Hymenoptera
Family	Apidae
Genus	Apis
Species	Mellifera

11. Vernacular names: [312]

- **Sanskrit:** Madhu
- **Bengali**: Madhu
- **English** : Honey
- **Gujrati** : Madh
- **Hindi** : Shahad
- **Kannada**:Jenutupa
- **Malayalam**:Taen
- **Marathi**: Madha
- **Tamil**: Taene
- **Telugu**:Taene
- **Urdu**:Shahad

12. Parayayi Nama of Madhu according to Nighantu:[307]

Table No.50: Parayayi Nama of Madhu according to Nighantu:

Nighantu → / ParyayiNama ↓	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N	N.A.
Madhu		+	+	+	+	+	+	+
Pushparasa		+	+	+	+		+	+
Pushpaasav		+	+		+	+		+
Makarand			+	+		+	+	+
Pushparas		+	+		+	+	+	
Saragh		+	+	+	+	+		+
Pavitra					+			

13. Madhu Nirukti:[313]

- Madhu (honey) is derived from the verb 'dham' (to blow) reversed.
- Madhu (honey) means Soma is derived from (the root) mad (toenxhilarate) and is compared with soma (because the analogy of exhileration). This other meaning of Madhu (wine) is derived from the same root also.

14. GunakarmatmakaVivechana: [307]

Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma.with these properties the drug can work. These are described in all Nighantus.

Table No.51: Gunapanchak of Madhu[307]

Properties		A.N	D.N	S.N	M.N.	R.N.	K.N.	B.N.
Guna	Ruksh		+	+	+	+	+	+
	Laghu		+	+				
	Grahi		+	+	+	+	+	+
	Vishad		+		+		+	+
Rasa	Madhur		+		+	+	+	+
	Kashaya		+			+	+	+
Vipaka	Madhur		+					
Virya	Sheet		+					

Table No.52: Dosha, Karma, Rogghnata of Madhu:[307]

Gunakarma	A.N.	D.N.	S.N.	M.N.	R.N.	K.N.	B.N.
Dosha karma	Pittaghna	Pittaghna		Pitta-jeet	Pitt-aghna	Pitta-ghna Vata-ghna	Pitta-ghna Vata-ghna
	Astrajeet					Ratt-aghna	Ratt-aghna
Pramukha Karma	Stanyad-oshhara	Vrushya Shoshhara	Sho-shvinashi	Balya	Chaksus-hya	Chakshu, Bala,Varna-kruta, Shukral	Chakshu, Bala,Varna-kruta, Shukral
Rog-ghnata	Trushna			Truta	Trushna	Trushna	Trushna
						Glani	Glani
	Shosha	Shosha			Shosha	Khata	Kshya
	Daha						
	Jwara						
	Vamak	Chardi		Cha-rdi		Chardi	Chardi
	Atisaraghana						
	Meda						
	Adhyavathar						
		Vishaha				Vishaha	Vishaha
					Hru-droga		
					Vrana	Vrana	Vranashotha
						Shotha	
						Keshya	Keshya
						Swarya	Swarya

6.Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha,Karma and Rogghanta of Madhu :[307]

6 .कैयदेवनिघण्टु - १. ओषधिवर्गमधु

पुष्पासवःपुष्परसोमक्षिकाविट्चसारघम्|

माक्षिकंपौत्तिकंक्षौद्रंभ्रामरंमधुजातयः||१७३||

माक्षिकंतैलसङ्काशंपौत्तिकंघृतवर्णकम्

क्षौद्रंतुकपिलंविद्यात्भ्रामरंशशिवर्णकम्||१७४||

2. सामान्यमधुगुण

मधुस्वादु हिमंरूक्षंकषायानुरसंलघु

दीपनंग्राहिचक्षुष्यंस्वर्यंवर्ण्यंविलेपनम्||१७५||

सौकुमार्यकरंवृष्यंहृद्यंस्रोतोविशोधनम्

सूक्ष्मंमेधाकरंछेदिव्रणशोधनरोपणम् ||१७६||

विशदंरोचनंह्लादिप्रसादजननंजयेत्|

मेदःपित्तकफश्वासहिध्ममेहवमिक्षयान् ||१७७||

दोषत्रयातिसारास्रतृष्णादाहविषकृमीन्|

कुष्ठार्शोरक्तपित्तघ्नंयोगवाहिचवातलम् ||१७८||

वातलंवातकोपेऽपिवर्षासुमधुशस्यते|१७९|

2.धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्ग मधु

मधुक्षौद्रंतुमाक्षीकंमाक्षिकंकुसुमासवम्|

पुष्पासवंसारघंचतच्चपुष्परसंस्मृतम्||२२७||

माक्षिकंभ्रामरंक्षौद्रंपौत्तिकंछात्रकंतथा|

आर्घ्यमौद्दालकंदालमित्यष्टौमधुजातयः||२२८||

माक्षिकंतैलवर्णंस्यात्क्षौद्रंतुकपिलंभवेत्|

पौत्तिकंघृतवर्णंतुश्वेतंभ्रामरमुच्यते||२२९||

आपीतवर्णंछात्राख्यंपिङ्गलंचाघ्र्यनामकम्|

औद्दालंस्वर्णसदृशंदालंचपाटलंस्मृतम्|२३०||

7. सामान्यमधुगुण

कषायानुरसंरूक्षंशीतलंमधुरंमधु|

दीपनंलेखनंबल्यंव्रणरोपणमुत्तमम्||२३१||

सन्धानंलघुचक्षुष्यंस्वर्यंहृद्यंत्रिदोषनुत्|

छर्दिहिक्काविषश्वासकासशोषातिसारजित्||२३२||

रक्तपित्तहरंग्राहिकृमितृण्मोहहृत्परम्|२३३|

8. धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्गभ्रामरमधु (विशिष्टमधुगुण)

पैच्छिल्यात्स्वादु रूपत्वाद्भ्रामरंगुरुसञ्ज्ञितम्

भ्रामरंकुरुतेजाड्यमत्यन्तंमधुरंचतत् ||२३३||

धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्गक्षौद्रमधु

क्षौद्रंविशेषतोज्ञेयंशीतलंलघुलेखनम्|२३४|

धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्गमाक्षिकमधु

तस्माल्लघुतरंरूक्षंमाक्षिकंप्रवरंस्मृतम्||२३४||

उष्णैर्विरुध्यतेसर्वविषान्वयतयालधु|

उष्णार्तरुक्षरुष्णौर्वातन्निहन्तितथाविषम्||२३५||

तत्सौकुमार्याच्चतथैवसेव्यंवनौषधीनांरससम्भवाच्च|

उष्णैर्विरुध्येतविशेषतस्तुतथाऽऽन्तरिक्षेणजलेनवाऽपि||२३६||

4.मदनपालनिघण्टु–९. इक्षुकादिवर्गमधु

मधुपुष्पासवःपुष्परसोमाक्षिकमीरितम्|

माक्षिकंपौत्तिकंक्षौद्रंभ्रामरंमधुविस्तरात्||२३||

माक्षिकंतैलसङ्काशंपौत्तिकंघृतसन्निभम्

क्षौद्रंकपिलवर्णंस्याद्भ्रामरंस्फटिकोपमम्॥२४॥

मधुशीतंलघुस्वादु रूक्षंग्राहिविलेखनम्

चक्षुष्यंदीपनंस्वर्यंव्रणशोधनरोपणम्॥२५॥

वर्ण्यंमेधाकरंवृष्यंविशदंरोचनंजयेत्

कुष्ठार्शःकासपित्तासृक्कफमेहक्लमक्रिमीन्॥२६॥

मदतृष्णावमिश्वासहिक्कातीसारहृद्ग्रहान्

दाहक्षतक्षयास्रंतुयोगवाह्यल्पवातलम्॥२७॥

माक्षिकंमधुषुश्रेष्ठंनेत्रामयहरंलघु

पौत्तिकंमधुरूक्षोष्णंपित्तदाहास्रवातकृत्॥२८॥

क्षौद्रंमाक्षिकमप्येवंविशेषान्मेहनाशनम्

भ्रामरंरक्तपित्तघ्नंमूत्रजाड्यकरंगुरु॥२९॥

नवीनंमध्वभिष्यन्दिस्निग्धंश्लेष्महरंसरम्

5. राजनिघण्टु–१४. पानीयादिवर्ग-मधु

मधुक्षौद्रंचमाक्षीकंमाक्षिकंकुसुमासवम्

पुष्पासवंपवित्रञ्चपित्र्यंपुष्परसाह्वयम्॥११४॥

माक्षिकंभ्रामरंक्षौद्रंपौत्तिकंछात्रकंतथा

आर्घ्यमौद्दालकंदालमित्यष्टौमधुजातयः॥११५॥

नानापुष्परसाहाराःकपिलावनमक्षिकाः

याःस्थूलास्ताभिरुत्पन्नंमधुमाक्षिकमुच्यते ॥११६॥

येस्निग्धाञ्जनगोलाभाःपुष्पासवपरायणाः

भ्रमरैर्जनितंतैस्तुभ्रामरंमधुभण्यते॥११७॥

पिङ्गलामक्षिकाःसूक्ष्माःक्षुद्राइतिहिविश्रुताः

ताभिरुत्पादितंयत्तुतत्क्षौद्रंमधुकथ्यते॥११८॥

अन्नजामक्षिकाःपिङ्गाःपुत्तिकाइतिकीर्तिताः|

साधारण्यामधुहितंतत्तुल्यामधुशर्करा||१३३||

उष्णैःसहोष्णकालेवास्वयमुष्णमथापिवा|

आमंमधुमनुष्याणांविषवत्तापदायकम्||१३४||

कीटकादियुतमम्लदूषितंयच्चपर्युषितकंमधुस्वत¦

कण्टकोटरगतञ्चमेचकंतच्चगेहजनितञ्चदोषकृत्||१३५||

दण्डैनिहत्ययदुपात्तमापास्तदंशंतादृग्विधंमधुरसायनयोगयोग्यम्||१३६||

हिक्कागुदाङ्कुरत्रिशोफकफव्रणादिदोषापहंभवतिदोषदमन्यथाचेत्

3.शोढलनिघण्टु-नामसङ्ग्रह (प्रथमभाग) - १०. परिशिष्टमधु

क्षौद्रंमधुमाक्षिकंचमाक्षीकंसारघंचतत्|

पुष्पासवःपुष्परसोमकरन्दरसस्तथा||६||

गुणसङ्ग्रह (द्वितीयभाग) - १५. मधुवर्ग

मधुवर्ग

मधुस्वरहितंमेध्यंहृद्यंलेखनदीपनम्|

चक्षुष्यंछर्दितृट्श्लेष्मविषहिध्मास्रपित्तनुत्||७९५||

कुष्ठमेहकृमिच्छर्दिश्वासकासातिसारजित्|

व्रणशोधनसन्धानरोपणंवातकोपनम्||७९६||

मधुरंकटुकंपाकेतत्तुल्यामधुशर्करा|

VI. Ghruta

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern science.

1. Vedic review: [314]

As a Aahariya Dravya.

2. Samhitakalin review: [315-17]

I.Charak Samhita:[315]

- It is mentioned in vranashodhan lepa in Dwivraneeya chapter (Ch Chi 25/83-85)
- It is used for Mamsa and shonitvardhan in Kshataksheena chapter (Ch Chi 11/30)
- It is mentioned for Ghritapaan in vaatpittaj jwara(Ch Chi 3/164)
- The properties of Ghrita are mentioned in Annapaanvidhi chapter (Ch Su 27/231-233)
- Indication of ghritapaan in various condition are metioned in snehaadhyay (Ch Su 13/41-43)
- SanskarAnuvartan quality of Ghrita is mentioned snehaadhyay (Ch Su 13/13)
- Properties of Ghrita as Dahashaman is mentioned in snehaadhyay (Ch Su 13/41-14)

II.Sushrut Samhita:[316]

- The Properties of Ghrita are mentioned in Dravdravyavidnyaneeyam chapter (Su Su 45/96)
- The Properties of Cow Ghrita are mentioned in Dravdravyavidnyaneeyam chapter (Su Su 45/97)
- It is mentioned for snehapaan in different types of sdyovrana (Su Chi 2/23-25)
- It is mentioned for lepa, Parishek for dahashaman in sadyovrana (Su Chi 2/27)
- It is mentioned in Shashti Upakrama of Vranachikitsa (Su Chi 1/8)
- It is constituents of Shodhan ghrita for Pittaj dhah in deep wound (Su Chi 1/56)
- It is mentioned in nirwapan lepa chikitsa in shashtiupakrama of vrana (Su Chi 1/49)
- It is mentioned for the nirwapan of sadyovranachikitsa (Su chi 1/130)
- It is mentioned for Ropan karma in all types of Vrana (Su chi 1/79)

- It is used during operative process of Baddhagudodar and Chidrodar (Su chi 15/17)
- It is mentioned in Twakvishuddhikar yoga in Vranachikitsa (A.U25/58)
- It is mentioned in kalkadravya for Vranalepan in Vranachikitsa(A.U25/55)
- It is constituents of Yashtimadhu Ghrita for Vedanasthapana in sadyovranachikitsa (AH U 26/6)
- It is constituents of Pittashamaklepa in sdyovranachikitsa (AH u 26/8)
- The Properties of Ghrita are mentioned in Dravdravyavidnanyaneeyam chapter (AH Su 5/37-39)
- The Properties of Puran Ghrita are mentioned in Dravdravyavidnyaneeyam chapter (AH Su 5/40)
- It is mentioned for ghrita sevana in Bheshaj ksheena sharir. (AH Su 5/30)

III. NighantuKaal: [318]

- It is mentioned in ViprakirnaPrakaran of AshtangaNighantu.
- It is mentioned in SuvarnadiVarga of DhanvantariNighantu.
- It is mentioned in Gunasagharah GhrutaVarga of Shodhal Nighantu.
- It is mentioned in PaniyadiVarga of MadanpalNighantu.
- It is mentioned in Drava ghrutaVarga of Kaiyadev Nighantu.
- It is mentioned in Mishraprakaran GhrutaVarga of Bhavprakash Nighantu.

3. Constituents: [319]

Gross Composition of Ghee. Cow ghee Buffalo Ghee Fat 99.0—99.5% 99.0—99.5% Saturated fat 46% cis-monoene 29% trans-monoene 7% Diene 13% Polyene 5% ¹riglycerides (triacylglycerols) SSS 42% 49% SSU 42% 39% SUU 14% 11% UUU 2% 1% Diglycerides (diacylglycerols) 4% Monoglycerides (monoacylglycerols) 1% ºnsaponifiable matter Cholesterol 300 mg Lanosterol 9 kg 100 g~1 Lutein 4 kg 100 g~1 Squalene 60 kg 100 g~1 Vitamin A 9 kg 100 g~1 Vitamin E 28 kg 100 g~1 Ubiquinones 6 kg 100 g~1 S"saturated, U"unsaturated. Table adopted from the works reviewed by Achaya (1997).

4. Properties and action: [318]

1) **Rasa** : Madhur

2) **Guna** : Snigdha,Sheet,Guru

3) **Virya** : Sheeet

4) **Vipaka** : Madhur

5) **Karma**: Balya, Rasayan, Vayasthapan, Jeevaniya,Bruhaniya, Indriyauttam, Vatpittaghana, Kantiojakar, Vranaropak.

6. Important formulations:[320]

Chavanprash, Kalyanakghruta, Tiktakghruta, Panchatikakghrutagugul.

7. Therapeutic uses: [315]

Jirnajwara, Yakshma, Rakttapitta, Unmmad, Apasmar, Netraroga, Vajeekaran, Shwas, Kas, Mutrakruchha.

8. Classification According to Samhita: [321]

Table No.53: Classification of Ghruta according to Samhita

Charaka Samhita	Sushruta Samhita	Ashtanga Hridaya
Bruhan dravya	Rakshoghna Gan	DravapadarthVarga
Snehan dravya	Bruhaniya Gan	
Agryasangharah	SarvapathyaVarga	
Pittashamangan	KumarrasayanVarga	
	Agryasanghrahar	
	VajeeekaranVarga	

9. Classification According to Nighantu: [318]

Table No.54:.Classification of Ghruta according to Nighantu

Nighantu	Varga
Ashtang Nighantu	Viprakirna Prakaran
Dhanvantari Nighantu	SuvarnadiVarga
Shodhal Nighantu	Gunasangraha GhrutVarga
Madanpal Nighantu	Paniyadi Varga
Raj Nighantu	Ksheera Varga
Kaiydev Nighantu	DravavargaGhruta Varga
Bhavprakash Nighantu	GhrutaVarga
NighantuAdarsha	

10. Vernacular names:[322]

- **Sanskrit** : Ghrut
- **Bengali** :Ghee
- **English** :Clearified Butter
- **Gujrati** :Ghee

- **Hindi** :Ghee
- **Kannada** :Tuppa
- **Malayalam** :Neyyuh
- **Marathi** :Toop
- **Punjabi** :Ghyo
- **Tamil :** Ney
- **Telugu** :Neyyi
- **Urdu :**Ghee

11. Parayayi Nama of Ghruta according to Nighantu: [318]

Table No.55: Parayayi Nama of Ghruta according to Nighantu

Nighantu → ParyayiNama↓	**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N**	**N.A.**
Ghrut	+	+	+	+	+		+	+
Ajjya	+	+	+	+	+		+	+
Havi	+	+	+	+	+		+	+
Sarpi	+	+	+	+	+	+	+	+
Navneetam	+	+			+	+		
Ghrutalya	+							
Jeevaneeya		+				+		
Pavitra		+			+	+		
Amruta		+		+	+	+		+
Snehottam			+					
Tejasam					+			
Abhidhara						+		

12. GunakarmatmakaVivechana:[318]

Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma.with these properties the drug can work. These are described in all Nighantus.

Table No.56: Gunapanchak of Ghruta

Properties		**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N.**	**N.A.**
Guna	Snigdh			+	+	+		+	+
	Guru			+	+			+	+
	Sheet			+	+	+	+	+	+
Rasa	Madhur		+	+	+	+	+	+	+
Virya	Sheet		+	+	+	+	+	+	+

13.Dosha, karma, Rogghnata of Ghruta

Table No.57: Dosha, karma, Rogghnata of Ghruta:[318]

Nighantu	**A.N.**	**D.N.**	**S.N.**	**M.N.**	**R.N.**	**K.N.**	**B.N.**
Dosha karma		Vattapittaghana,	Vattapittaghana,	Vattapittaghana,	Vattapittaghana,	vatpitthar	Vatpitthar,kap-hakar
Pramukha Karma		Vrushya, Jeevaneeya, Vayastapan ,Balya, Rasayan,Dhee,Dhruti, Medha, Smruti	Snehan Brhun, Vrushya,Rasayan, Dhee, Dhruti, Smruti ,Medha	Vrushya, Vayastapan, Rasayan, Medha,	Snehan Brhun, Vrushya,Rasayan, Dhee,Dhruti, Smruti ,Medha	Snehan, Brhun, Vrushya, Rasayan, Dhee,Dhruti, MedhaSmruti	Snehan, Brhun, Vrushya,Rasayan, Dhee,Dhruti, Smruti ,Medha
Rog-ghnata		KshatkseenaVisha, Chakshurog Vrana,	Vrana,	Udavarta, unamadaJwaraShoola ,Anaha, Vrana, rana,	Vrana,	Chakshus-ya,Rakttapitta,Vrana,vataroga,Khataksheen,Agnimandya, Ajirna, udavarta, Ykshma.	Udavarta, unamadaJwara,Shoola ,Anaha, Vrana,

13. Shloka references of Various Nighantu for Paryayinama, Gunapanchak, Dosha, of Ghrut.

Karma and Rogghanta:[318]

1.अष्टाङ्गनिघण्टु - २७.विप्रकीर्णप्रकरण

घृतमाज्यंहविःसर्पिःनवनीतंघृतालय॥३२५॥

2.धन्वन्तरिनिघण्टु - ६.सुवर्णादिवर्गघृत

घृतमाज्यंहविःसर्पिःपवित्रंनवनीतजम्

अमृतंचाभिधारश्चजीवनीयंप्रकीर्तितम्॥१४०॥

सहस्रवीर्यंविधिवद्धृतंकर्मसहस्रकृत्१४१।

शस्तंधीस्मृतिमेधाग्निबलायुःशुक्रचक्षुषाम्

बालवृद्धप्रजाकान्तिसौकुमार्यस्थिरार्थिनाम्॥१४१॥

क्षतक्षीणपरीसर्पशस्त्राग्निग्लपितात्मनाम्

विपाकेमधुरंशीतंवातपित्तविषापहम्॥१४२॥

चक्षुष्यंबल्यमग्र्यञ्चगव्यंसर्पिर्गुणोत्तरम्॥१४३॥

3.शोढलनिघण्टुगुणसङ्ग्रह (द्वितीयभाग) - १३.घृतवर्ग

घृतंहितंधीस्मृत्यग्निबलायुःशुक्रचक्षुषाम्

बालवृद्धप्रजाकान्तिसौकुमार्यस्वरार्थिनाम्॥७४८॥

क्षतक्षीणपरीसर्पशस्त्राग्निग्लपितात्मनाम्

वातपित्तविषोन्मादशोषालक्ष्मीज्वरापहम्॥७४९॥

स्नेहानामुत्तमंशीतंवयसःस्थापनंपरम्

सहस्रवीर्यंविधिवत्तच्चकर्मसहस्रकृत्॥७५०॥

पुराणंजयतिव्याधीन्शिरःकर्णाक्षियोनिजान्

मदापस्मारमूर्छायांव्रणशोधनरोपणम्॥७५१॥

उग्रगन्धंपुराणंस्याद्दशवर्षस्थितंचयत्

लाक्षारसनिभंशीतंतद्धिसर्वग्रहापहम्॥७५२॥

अपस्मारग्रहोन्मादवतांशस्तंविशेषतः।

पूर्वोक्तांश्चाधिकान्कुर्याद्गुणांस्तदमृतोपमम्॥७५३॥

तद्वच्चघृतमण्डोऽपिरूक्षस्तीक्ष्णस्तनुश्चसः।

गव्यंघृतंचचक्षुष्यंवातपित्तकफापहम्॥७५४॥

विपाकेमधुरंशीतंबल्यमग्र्यंगुणोत्तमम्।

आजंघृतंदीपनीयंचक्षुष्यंबलवर्धनम्॥७५५॥

कासेश्वासेक्षयेचापिपथ्यंपाकेचतत्लघु।

औष्ट्रंकटुःघृतंपाकेकृमिशोफोदरापहम्॥७५६॥

दीपनंकफवातघ्नंकुष्ठगुल्मोदरापहम्।

आविकंलघुपाकंचनातिपित्तकरंघृतम्॥७५७॥

कफेऽनिलेयोनिदोषेशोफेकम्पेचतद्धितम्।

माहिषंसर्पिःशीतंचवातपित्तविनाशनम्॥७५८॥

मधुरंरक्तपित्तघ्नंगुरुपाकिकफावहम्।

सर्वमैकशफंसर्पिःकषायंकफनाशनम्॥७५९॥

बद्धमूत्रपुरीषंचवीर्योष्णंलघुपाकिच।

स्त्रीणांसर्पिःप्रधानंतुचक्षुष्यममृतोपमम्॥७६०॥

वृद्धिंकरोतिदेहाग्न्योर्लघुपाकंविषापहम्

कारेणवंघृतंहन्तिकफकुष्ठविषक्रिमीन्॥७६१॥

कषायंबद्धविण्मूत्रंतिक्तमग्निकरंलघु।

उष्ट्रीणांचापिनारीणांगर्दभीनांपयांसिच॥७६२॥

घृतकार्येषुयोज्यानिघृतंतेषांनविद्यते।

उक्तानिपूर्वशास्त्रेषुपूर्वाचार्यैःक्रमेणतु॥७६३॥

मदनपालनिघण्टु–८.पानीयादिवर्ग-घृत

घृतमाज्यंहविःसर्पिराघारममृताह्वयम्

घृतंरसायनंस्वादु चक्षुष्यंगुरुदीपनम्॥१२७॥

शीतवीर्यंविषालक्ष्मीवातपित्तानिलापहम्

अत्यभिष्यन्दिकान्त्योजस्तेजोलावण्यबुद्धिकृत्॥१२८॥

उदावर्तज्वरोन्मादशूलानाहव्रणाञ्जयेत्

स्निग्धंकफप्रदंरूक्षंक्षयवीसर्परक्तजित्॥१२९॥

स्वर्यंक्षतहरंप्रायःशस्यतेबालवृद्धयोः

घृतंक्षीरभवंग्राहिशीतलंनेत्ररोगजित्॥१३०॥

निहन्तिपित्तदाहास्रमदमूर्छाभ्रमानिलान्

पुराणङ्कटुकंपाकेसर्पिर्दोषत्रयापहम्॥१३१॥

श्रोत्रनेत्रशिरःशूलकुष्ठापस्मारशोथजित्

योनिरोगज्वरश्वासकुष्ठार्शोगुल्मपीनसान्॥१३२॥

निहन्तिदीपनंबस्तिनस्यपूर्तिषुशस्यते

घृतमण्डोऽपिघृतवद्गुणैस्तीक्ष्णोलघुःसर॥१३३॥

दशवर्षात्परंसर्पिःकौम्भमित्यभिधीयते

रक्षोघ्नंलघुतस्मात्तुगुणैःश्रेष्ठंमहाघृतम्॥१३४॥

घृतस्यगुणदोषौतुक्षीरतुल्यौसमादिशेत्

सर्वेषुगुणकृद्द्रव्यमाविकंनिन्दितंपुनः॥१३५॥

5.राजनिघण्टु - १५.क्षीरादिवर्गघृत

घृतमाज्यंहविःसर्पिःपवित्रंनवनीतजम्

अमृतंचाभिधारश्चहोम्यमायुश्चतैजसम्॥४॥

5.राजनिघण्टु - १५.क्षीरादिवर्ग-गोधृत

धीकान्तिस्मृतिदायकंबलकरंमेधाप्रदंपुष्टिकृत्वातश्लेष्महरंश्रमोपशमनंपित्तापहंहृद्यदम्वह्नेर्वृद्धिक

रंविपाकमधुरंवृष्यंवपुःस्थैर्यदंगव्यंहव्यतमंघृतंबहुगुणंभोग्यंभवेद्भाग्यत॥७७॥

6. कैयदेवनिघण्टु - ४.द्रववर्ग–घृतवर्ग

अभिधारंजीवनीयंपवित्रंनवनीतजम्॥२६३॥अमृताह्वयमाधारंसर्पिराज्यंघृतंहवि॥२६४।

४.द्रववर्गसामान्यघृत

घृतंस्नेहोत्तमंशीतंमधुरंरसपाकयोः॥२६४॥

जीवनंबृंहणंवृष्यंवर्ण्यमिन्द्रियभावनम्

व्रण्यंस्वर्यंमृदु श्लक्ष्णंवयसःस्थापनंपरम्॥२६५॥

अल्पाभिष्यन्दिचक्षुष्यंश्लेष्मलंगुरुदीपनम।

बुद्धिमेधाप्रजाकान्तिस्मृत्योजोबलपुष्टिदम्॥२६६॥

सौकुमार्यकरंतेजोमेदोलावण्यवर्धनम्

वातपित्तहरंरक्षःपाप्मालक्ष्मीविषापहम्॥२६७॥

स्नेहनंरक्तपित्तघ्नंप्रशस्तंबालवृद्धयोः।

शूलाजीर्णज्वरोन्मादोदावर्तानाहयक्ष्मनुत्॥२६८॥

क्षतक्षीणपरीसर्पशस्त्राग्निग्लपितात्मनाम्

निरामेषूत्तमंसर्पिःस्नेहानांप्रागुदाहृतम्॥२६९॥

योग्यसंस्कारकद्रव्ययोगवाहितयाघृतम्

असङ्ख्याःभजतेशक्तीःसंस्कारस्यानुवर्तनात्॥२७०॥

गव्यघृत-

गव्यंसर्पिःस्वादुपाकेवातपित्तकफापहम्

वृष्येष्वग्र्यंपरंबल्यंघृतश्रेष्ठंत्रिदोषजित् ॥२७१।

7.भावप्रकाश

पूर्वखण्ड-मिश्रप्रकरण - १८.घृतवर्ग

घृतमाज्यंहविःसर्पिःकथ्यन्तेतद्गुणाअथ

घृतंरसायनंस्वादु चक्षुष्यंवह्निदीपनम्

शीतवीर्यंविषालक्ष्मीपापपित्तानिलापहम्॥१॥

अल्पाभिष्यन्दिकान्त्योजस्तेजोलावण्यबुद्धिकृत्

स्वरस्मृतिकरंमेध्यमायुष्यंबलकृद्गुरु॥२॥

उदावर्तज्वरोन्मादशूलानाहव्रणान्हरेत्

स्निग्धंकफकरंरक्षोरक्तक्षयविसर्पनुत्॥३॥

भावप्रकाश-पूर्वखण्ड-मिश्रप्रकरण - १८. घृतवर्ग-गोघृत

गव्यंघृतंविशेषेणचक्षुष्यंवृष्यमग्निकृत्

स्वादु पाककरंशीतंवातपित्तकफापहम्॥४॥

मेधालावण्यकान्त्योजस्तेजोवृद्धिकरंपरम्

अलक्ष्मीपापरक्षोघ्नंवयसःस्थापकंगुरु॥५॥

बल्यंपवित्रमायुष्यंसुमङ्गल्यंरसायनम्

सुगन्धंरोचनंचारुसर्वाज्येषुगुणाधिकम्॥६॥

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Chapter 4. Materials and Methodology

Drug formulation mentioned in Sharangdhar Samhita III /11/86 was selected for this study, it contains:[324]

Table No.58: Drugs used for Nimbadi Kalka

Drugs:	**Latin Name:**	**Family**	**Part used:**
Nimba	Azadiricta indica (Linn.)	Meliaceae	Patrachurna (Leaves) Powder
Ghrut (Ghee)	-	-	Cow Ghee
Kshodra (Honey)	Apisindica Mellifera	Apiadae	Honey
Darvi (Daruharidra)	Barberis aristata (DC.)	Berberidaceae	Kashta and Mula (Steam & Roots) Powder
Madhuka (Yashtimadhu)	Glycerrhiza glabra (Linn.)	Leguminosae.	Mula (Roots) Powder
Tila (Sesame Seed)	Sesamum indicum (Linn)	Pediliaceae	Beeja (Seed) Powder

The proper and potent drugs mentioned above are collected personally to insure about its quality. They are authentified from Agharkar research institute Pune Maharashtra. ---Annexure-I

1. Method of drug preparation:

Nimbadi Kalka is prepared according to reference of Sharangdhar Samhita Di.Kh.5/1-2 and Bhaishajya Ratnavali 47/45 [325]

2. Apparatus: Mortar and pestle, spoon, Bowels, Dressing tray.

All the above-mentioned drugs are weighed properly on weight balance. In mortar with the help of pestle a homogenous paste is prepared. The finished product is taken out in sterile bowels and used for patients dressing purpose.

The minimum standardization of Nimbadi Kalka is done according to Ayurvedic Pharmacopeia of India Part-II, Vol-I, 2007 from Indian Drug Research Laboratory Pune Maharashtra. ---Annexure-II

3.Sample size:

Total 260 patients of chronic Non-Healing Diabetes ulcer are selected by simple randomized method irrespective of Gender,Cast,and Occupation etc.These 260 patients are divided in two equal groups on patient's willingness.Group A-130 patients have received Ayurved treatment for ulcer care. Group B-130 patients have received Allopathic treatment for ulcer care.

4.Clinical study design:

Randomized Experimental trial with two arm

5 Dose: Local application as a paste on ulcer area up to skin level. Once in a day for maximum 60 days..

6.Period of treatment:

Maximum sixty days in hospitalized patients

7. Place of work:

MIMSR Medical College and YCR Hospital, Ambajogai Road Latur, District: Latur, State: Maharashtra 413512India.

8. Inclusion criteria for patients:[326,327]

- Age below 75 yrs
- Gender-Both
- Non healing diabetic ulcer for > 5-6 week
- All Diabetes Type –II patients
- Heamodynamicaly stable
- All diagnosed patients were hospitalized
- Patient's reciving modern medicine (oral hypoglycemic agent) for Diabetes II
- I and II Grade of ulcers: ---Annexure IV

8. Exclusion criteria: [326,327]

Known cases of following disease condition

- Inadequate blood supply
- Poor glycemic control
- Non-adherence with treatment plan
- End-stage renal disease
- Transplant recipients
- Differing individual goals

- Malnutrition
- Connective tissue disorders
- Systemic conditions such as sickle cell disease
- Osteomyelitis
- Immobility
- Heart disease
- Dementia
- Cancer
- AIDS and
- Advancing age. (age above 75 years)

9. Consent:

Written informed consent of each pt. in his own language is taken. According to ICMR-7 Points guideline: ----------Annexure III

10. Permition of Ethics committee:

Is taken from concerned authority: --------Annexure IV

11. Case paper: ----------Annexure V

12. Dressing method:

1) Ayurved treatment group: (Group-A)

Ulcer cleaning and debridement was done regularly as per ulcer need with the help of normal saline water, sterile cotton, forceps, scoop and blade. Ulcer was dried with sterile cotton gauze. After that Nimbadi kalka was apply on ulcer area up to the superficial skin and dry bandage was applied on it. Patients were advised for rest and offloading techniques were taught to patients.

2) Treatment group :(Group-B)

Ulcer cleaning and debridement was done regularly as per ulcer need with the help of normal saline water, H_2O_2, Eusol solution, Betadine solution, Forceps, blade sterile cotton, and scoop. Ulcer was dried with sterile gauze and Ointment Betadine was applied to all ulcer area. After that a dry bandage was applied on it. Patients were advised for rest and offloading techniques were taught to patients.

Regular internal treatments of diabetes for both groups of patients were followed as per Allopathic physician's opinion.

13. Observation and Assessment during treatment period:[326,327]

i) History:

The chronicity of Diabetes and poor control of Diabetes result in complication of Diabetes like Neuropathy, Vasculopathy and Nephropathy, Retinopathy etc. The history of Hypertension, Habits like Alcohol, Tobacco, Smoking, and Obesity is also important in Vasculopathy and defense mechanism. Family history is also important in disease prognosis. Occupation history is important for pressure distribution on ulcer and Glycemic control.

ii) Occupation:

Pressure phenomenon, work culture-Venous ulcer in long standing people, Neuropathy in drivers etc.

iii) General examination:

The Nutrition, Gait, Psychological, Edema, Anemia, Pigmentation

iv) Systemic examination: CVS, RS, PA, CNS

Feet Examination: Shape, Size, Deformity, Thickening, Callosity

V) Ulcer examination:

a) Shape- Oval, Circular, Irregular (Arterial, Venous, Diabetic, Pressure, Other)

b) Size - Length, Depth, Breadth, Fistula.

c) Skin-Healthy, Dry, Ischemic, Pigmentation, Maceration, Nails.

d) Location- Medial malleolus- venous ulcer,
Lateral malleolus-Arterial ulcer.
Planter surface- Diabetic ulcer,
Sacrum - Pressure ulcer.

f) Edges: Slopping-Venous,
Punched-Arterial,
Rolled-Basel cell Carcinoma,
Everted- Squamous Cell Carcinoma,
Purple-Vasculitis,
Undermining -Tuberculosis, Syphilis ulcer.

g) Bed- Necrotic, Slough, Black

h) Secretion: Serous, Pus, Hemosangio

I) Granulation: Pink Colour-Healthy Granulation,

Red Ischemic-Unhealthy infected,

Black -Necrotic tissue, absent,

Over granulation-Non-healing tendency

j) Odour: Mild, Moderate, Sever, Foul,

k) Post healing pigmentation.

l) Surrounding Skin colour: Pink, Ischemic, Red, Black, and Exzematic.

VI) Vascular Assessment:

1) Pallor 2) Ischemic Changes 3) Peripheral pulse 4) Capillary refilling time.

VII) Neurological Assessment:

1) Sensory Assessment: Pain, Touch with 10 gm. Monofilament.

2) Motor Assessment: A) leg deformity- Claw, Charcot B) Ulcer pressure points C) Tendon reflux D) Muscle power.

IX) Assessment-Dry skin, hair loss, hyper pigmentation, local temperature

X) Infection: Sigs of Inflammation-- Fever, pain, redness, edema etc. pus discharge

Osteomyelitis: X-ray examination.

XI) Systemic examination: CVS, RS, CNS, PA was done daily.

14. Variables for Ulcer healing Assessment Criteria:[327]

1. Ulcer Size Sq. cm: Ulcer size in square center meter

1) 1-20 sq. cm= 1 grade,
2) 21-40 sq. cm= 2 grade,
3) 41-60 sq. cm= 3 grade,
4) >61 sq. cm= 4 grade

2. Ulcer shape: O=Oval shape, C=Circular, I=Irregular

3. Ulcer Bed: Ulcer bed colour

1) Pink colour =1 Grade,
2) Red Ischemic =2 Grade,
3) Black colour= 3 Grade,
4) Yellow/Green= 4 Grade

4. Granulation tissue: Granulation colour

1) Pink colour= 1 Grade,
2) Red Ischemic = 2 Grade,
3) Absent= 3 grade,

4) Over granulation= 4 Grade

5. Secretion:

1) No secretion= 1Grade,
2) Serus secretion= 2 Grade,
3) Hemosangionus=3 Grade,
4) Pus secretion=4 Grade

6. Pain: On Pains Scale

1) 0-1= 1 grade (No pain),
2) 2-4= 2 Grade (Mild pain),
3) 5-7= 3 Grade pain (Moderate pain),
4) 8-10= 4 Grade pain (Sever pain).

7. Smell:

1) No smell = 1 Grade,
2) Mild smell =2 Grade,
3) Intolerable smell=3 Grade,
4) Foul smell= 4 Grade

Daily observations of patients from both groups for Local as well as systemic observation are noted and mention in case paper.

15. Laboratory investigations before treating a wound:

Hemoglobin, White cell count, Platelet count, ESR, C- reactive protein, KFT, LFT, Glucose fasting and post prandial, HbA1C, Urine analysis, Wound swab culture and sensitivity of infective organism. All above observations are mentioned in case paper.

16. Ulcer healing outcome consideration:

Reduction in Ulcer area approximately 20-40% after 2-4 week is a good sign of healing. The healing outcome assessment is done at interval of 20 days, 40 days, and 60 days.

Clinical healing outcome was considered as:

- Healing with complete Epithelization.
- Partial healing without Epithelization but wound contracture
- Minor Amputation
- Major Amputation
- Death of patient

All observations noted were presented in information tables.

17. Statistical method:

The Variables were compared by using Chi-square test, paired T-test, Wlicox test, and proportionate test. P value of <0.05 was considered as significant. For this open controlled design with two arm study.

Figure No: 2 Drugs constituents of Nimbadi Kalka

Daruharidra root

Daruharidra branch

Nimba patra

Yashtimadhu plant

Yashtimadhustem

Figure No:3 Drugs constituents of Nimbadi Kalka

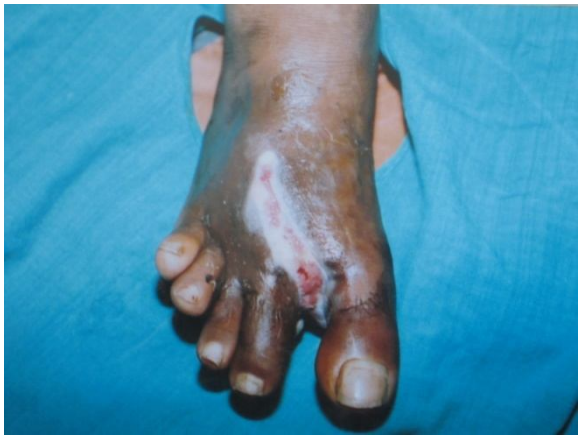
Tila Plant and seed

Ghee

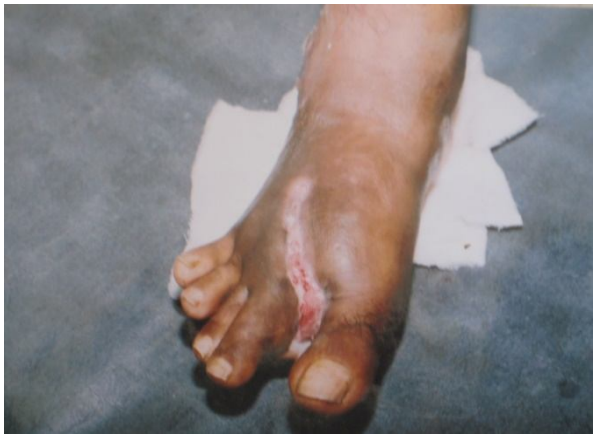
Honey

Nimbadi kalka

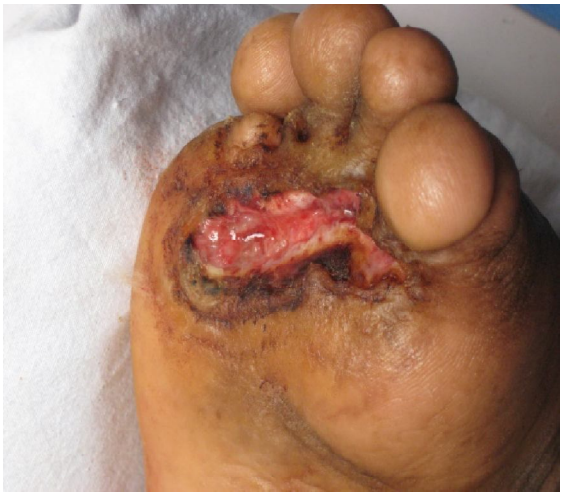
Figure No: 4 Patients Ulcer and its healing outcome



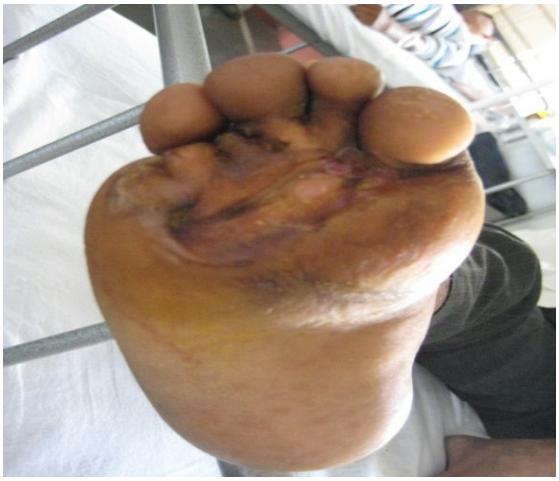
Foot Ulcer on 8th day treatment



Foot Ulcer on 38th day of treatment

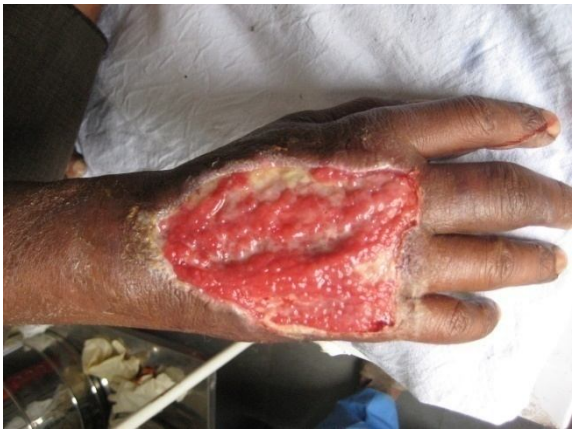


On 16th Day

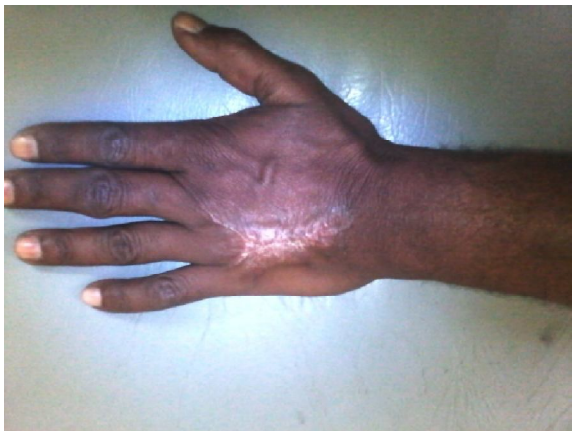


On 36th Day

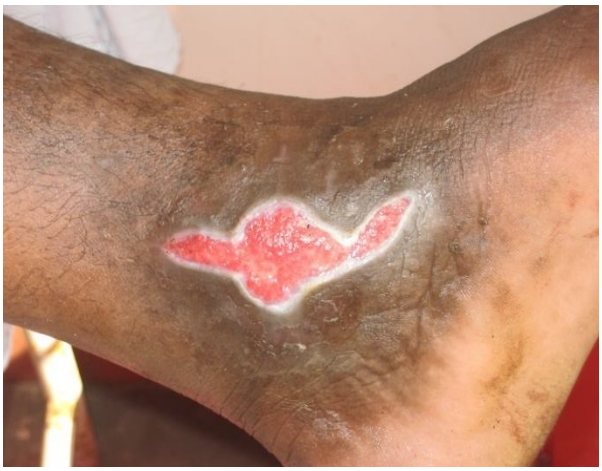
Figure No: 5 Patients Ulcer and its healing



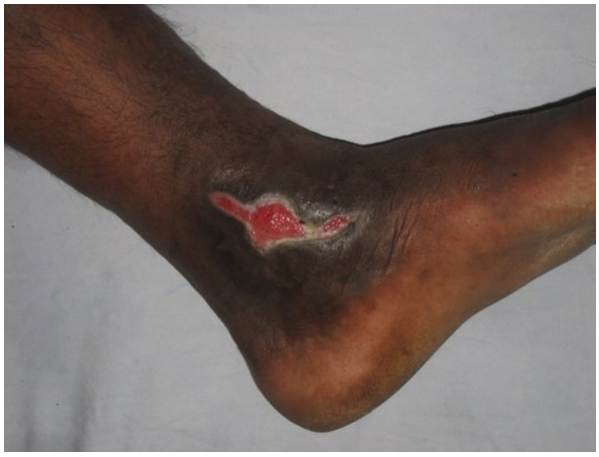
After 19th day of treatment



After 21st days of treatment

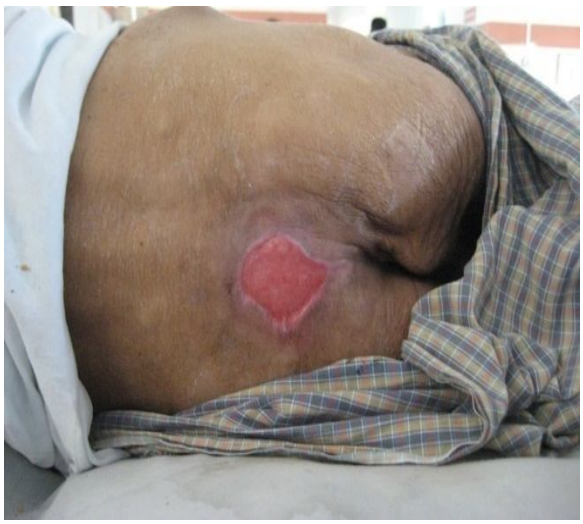


After 13th day of treatment

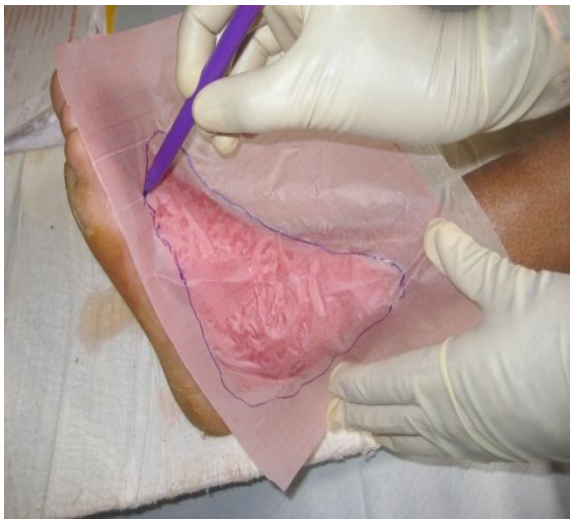


After 31st days of treatmen

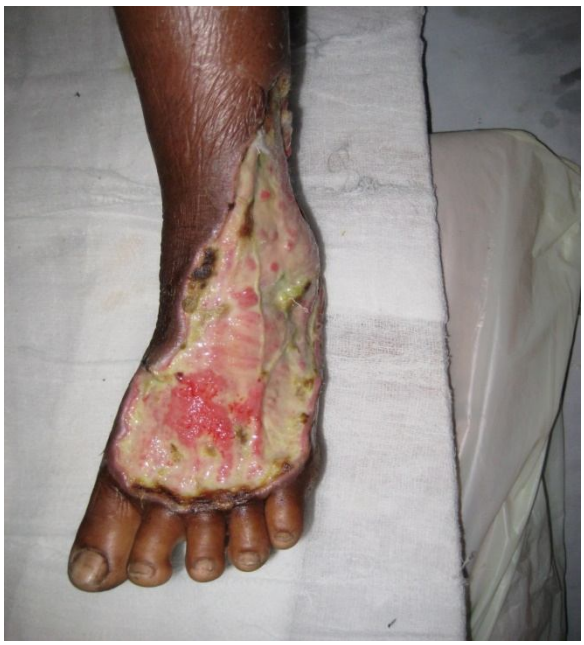
Figure No: 5 Patients Ulcer and its healing



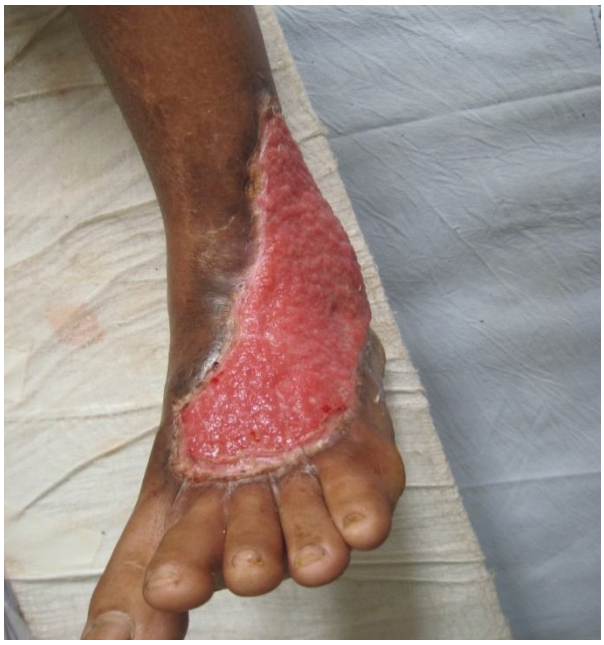
Bed Sore after 49th day



Measurement of tracing technique



1st day of treatment



48th day of treatment

Chapter 5. Observations and Results

Total 260 patients are selected in this study. They are divided in two equal groups 130 each; Group A and group B. Group A receives Ayurved treatment and Group B receiving Allopathic/Modern-Medicine treatment. The Master-chart description is given below.

Master-chart Variable description and data coding:

1. Sr, No. Serial Number

2. Group: A=Ayurvedic treatment group, B=Allopathic treatment group

3. Occupation: W= Worker, H = Housewife, S=Sedentary work

4. Gender: M=Male, F= Female

5. Age: Age in years

6. Chronicity: Chronicity of ulcer in weeks

7. Hop. stay: Hospital stay in days

8. Part involved: Ulcer part involved

U=Upper limb, L= Lower limb, G = Gluteal area

9. Initial Ulcer grade:

Initial ulcer grade on 1st day According to its classification.

1= Ulcer grade 1 2=Ulcer grade 2

10. BSL F: Blood sugar level fasting mg/dl

11. BSL PP: Blood sugar level post prandial mg/dl

12. Ulcer size

i. **Day 1 S Z:** Ulcer size on 1st day in square centimeter

ii. **Day 20 S Z:** Ulcer size on 20th day in square centimeter

iii. **Day40 S Z:** Ulcer size on 40 th day in square centimeter

iv. **Day 60 S Z:** Ulcer size on 60 th day in square centimetre

Grading of size: 1-20 sq. cm= 1 grade,
21-40 sq. cm= 2 grade,
41-60 sq. cm= 3 gra de,
>61 sq. cm= 4 grade

13. Shape:

i. **Day 1 SP:** Ulcer shape on 1 st day

ii. **Day 20 SP:** Ulcer shape on 20 th day

iii. **Day 40 SP:** Ulcer shape on 40th day

iv. **Day60SP:** Ulcer shape on 60th day

Grading of shape O=Oval shape, C=Circular, I=Irregular

14. Ulcer Bed:

i. **DAY1BD:** Day 1st ulcer bed

ii. **DAY20BD:** Day 20th ulcer bed

iii. **DAY40BD:** Day 40th ulcer bed

iv. **DAY60BD:** Day 60th ulcer bed

Grading of Ulcer bed colour

i. Pink colour=1 Grade

ii. Red Ischemic =2 Grade

iii. Black colour = 3 Grade

iv. Yellow/Green= 4 Grade

15. Granulation:

i. **Day1GR:** Granulation on 1st day

ii. **Day20GR:** Granulation on 20th day

iii. **Day40GR:** Granulation on 40th day

iv. **Day60GR:** Granulation on 60th day

Grading of granulation tissue colour:

i. Pink colour = 1 Grade

ii. RedIschemic =2 Grade

iii. Absent = 3 grade

iv. Over granulation = 4 Grade

16. Secretion:

i. **Day1SEC:** Secretion on 1st day

ii. **Day20SEC:** Secretion on 20th day

iii. **Day40SEC:** Secretion on 40th day

iv. **Day60SEC:** Secretion on 60th day

Grading of secretion.

i. No secretion = 1 Grade

ii. Serus secretion = 2 Grade

iii. Hemosangionus = 3 Grade

iv. Pus secretion = 4 Grade

17. Pain:

i. **DAY 1 PN**: Day 1st pain

ii. **DAY 20 PN**: Day 20th pain

iii. **DAY 40 PN**: Day 40th pain

iv. **DAY 60 PN**: Day 60th pain

Grading of pain on pain scale.

i. 0-1= 1 grade (No pain)

ii. 2-4= 2 Grade (Mild pain)

iii. 5-7= 3 Grade pain (Moderate pain)

iv. 8-10= 4 Grade pain (Sever pain)

18. Smell

i. **DAY1SML:** Smell of ulcer on 1 st day

ii. **DAY20SML:** Smell of ulcer on 20 th day

iii. **DAY40SML:** Smell of ulcer on 40 th day

iv. **DAY60SML:** Smell of ulcer on 60th day

Grading of smell.

i. No smell = 1 Grade

ii. Mild smell =2 Grade

iii. Intolerable smell=3 Grade

iv. Foul smell= 4 Grade

19. Healing outcome:

E= Complete Epithelization

C= Ulcer contracture but no Epithelization

Min A= Minor Amputation

Maj A=Major Amputation

D= Death of Patient.

The Statistical analysis is carried out using statistical package R-Programing is the first choice world-wide among researches. R is the authentic tool to standardise other statistical programs. R-3.3.2 which is advanced statistical programming language is used here. We have 260 patients sample size with 42 variables in it.

We have used descriptive statistics for summarising data. Welch two sample t-tests, Chi-square test for independence proportion test, and Willcoxrank test are of significance. Welch t-test is a modified version of student t-test, which is appropriate for mean comparison between two groups when data is numerical. Chi-square is used

to test independence, whenever to test dependency on some factor within two-way frequency table. Proportion test in a two-way table it is not possible to apply Chi-square test due to imbalance data Willcox test. It is non-parametric variant of t-test. Whenever we need to test two groups given in rank or order data. This test is appropriate choice.

A. Descriptive statistical assessment on initial status of both groups:

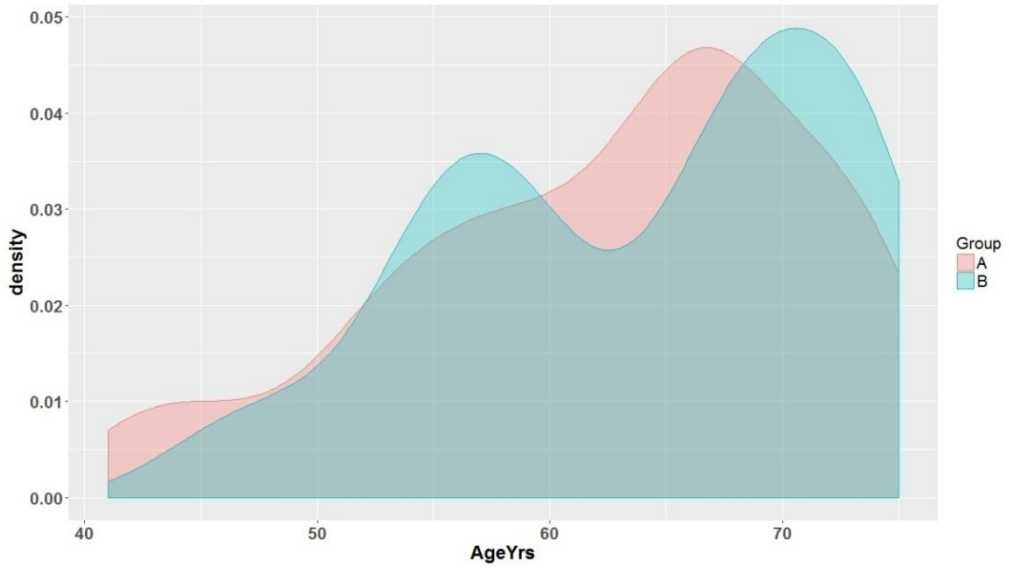
1. Age:

Table No. 59 - Distribution of 260 patients according to age:

Age in Yrs.	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Group A	41	57	64	62.41	68	75
Group B	44	57	66	63.89	70	75

In group A out of 130 patients minimum age is 41 yrs., maximum age is 75yrs, mean age is 62.41yrs and median of age is 64yrs, 1st Quarter is 57 yrs. 3rd Quarter is 68 yrs.

In group B out of 130 patients minimum age is 44 yrs., maximum age is 75 yrs., mean age is 63.89 yrs and median of age is 66yr, 1st Quarter is 57 yrs. 3rd Quarter is 70 yrs.



Graph No. 6: Distribution of 260 patients according to age

This is density plot age by parted between two groups. Graph is positively skewed. As we know that DM is dependent on age. So more patients are above 50 yrs age.

2. Gender:

Table No. 60 - Distribution of 260 patients according to Gender

Gender \ Group	Group A	Group B	Sum
Female	36	47	83
Male	94	83	177
Sum	130	130	260

In group A out of 130 patients male are 94 and 36 are female. In group B out of 130 patients male are 83 and 47 are female. Maximum male population is here.

With Yates' Correction: X-squared = 1.7698, df = 1, p-value = 0.1834. So we can conclude that patient allocation in both groups does not depend on gender so we can ignore gender effect in the study.

3. Occupation:

Table No. 61: Distribution of 260 patients according to the occupation:

Occupation\Group	Group A	Group B	Sum
Housewife	26	13	39
Sedentary	13	40	53
Worker	91	77	168

X-squared = 19.255, df = 2, p-value = 6.59e-05

Chi-square test for Occupation against Group is significant so we can conclude that housewives are more in Group A, Sedentary life-styled are more in Group B and Workers are more in Group A. Because the selection of Group (treatment type) was patient's choice and this evidencing that Housewives and workers believed in Ayurved and Sedentary patients are believed in modern medicine. In both group, maximum workers are there.

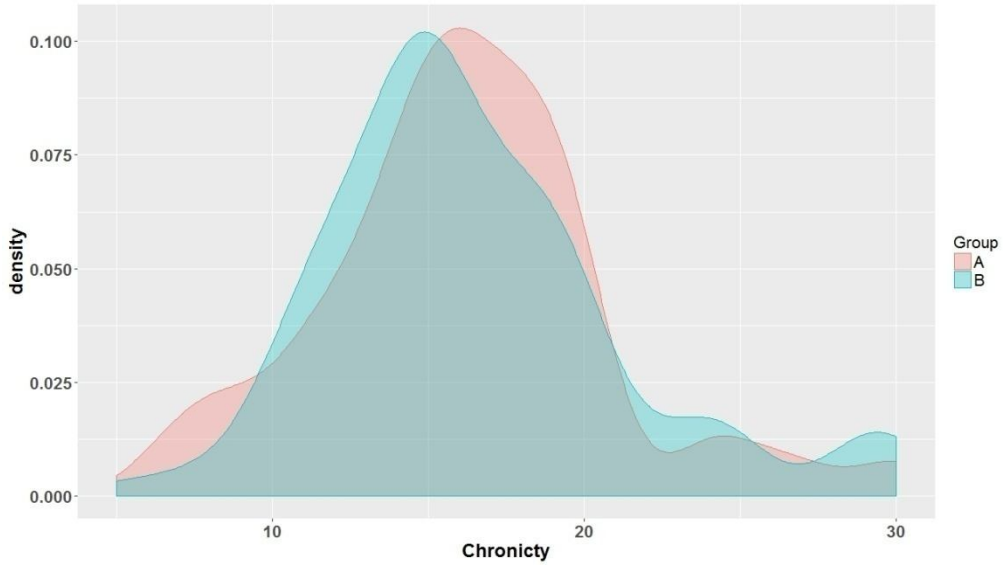
4. Ulcer chronicity:

Table No. 62: Distribution of 260 patients according to the chronicity of ulcer:

Chronicity	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
A	6	14	16	16.19	19	30
B	5	13.25	15	16.47	19	30

In group A out of 130 patients minimum chronicity is 6 weeks, maximum chronicity is30 weeks, mean chronicity is 16.19 weeks and median chronicity is 16 weeks, 1st Quarter is 14 weeks 3rd Quarter is 19 weeks.

In group B out of 130 patients minimum chronicity is 5 weeks, maximum chronicity is 30 weeks, mean chronicity is 16.47 weeks and median chronicity is 15 weeks, 1st Quarter is 13.25, weeks 3rd Quarter is 19 weeks.



Graph No. 7- Distribution of 260 patients according to the chronicity of ulcer:

To understand trends in distribution density plot is given.

5. Ulcer Part Involved:

Table No.63: Distribution of 260 patients according to the Ulcer Part Involved:

Part-involved	A	B	Sum
G	4	2	6
L	114	110	224
U	12	18	30
Sum	130	130	260

X-squared = 1.9381, df = 2, p-value = 0.3794

As Chi-square test is non-significant. So, arrangement of both groups is homogeneous.

In Both A and B Groups maximum number of patients are having lower leg ulcer i.e. Left leg.

6. Ulcer Grade:

Table No. 64: Distribution of 260 patients according to the ulcer Grade

Initial Ulcer grade	A	B	Sum
1	102	98	200
2	28	32	30
SUM	130	130	230

X-squared = 0.195, df = 1, p-value = 0.6588

As Chi-square tes t is non-significant. So, arrangement of both groups is homogeneous. In both Groups, maximum numbers of patients are having II Grade Ulcer

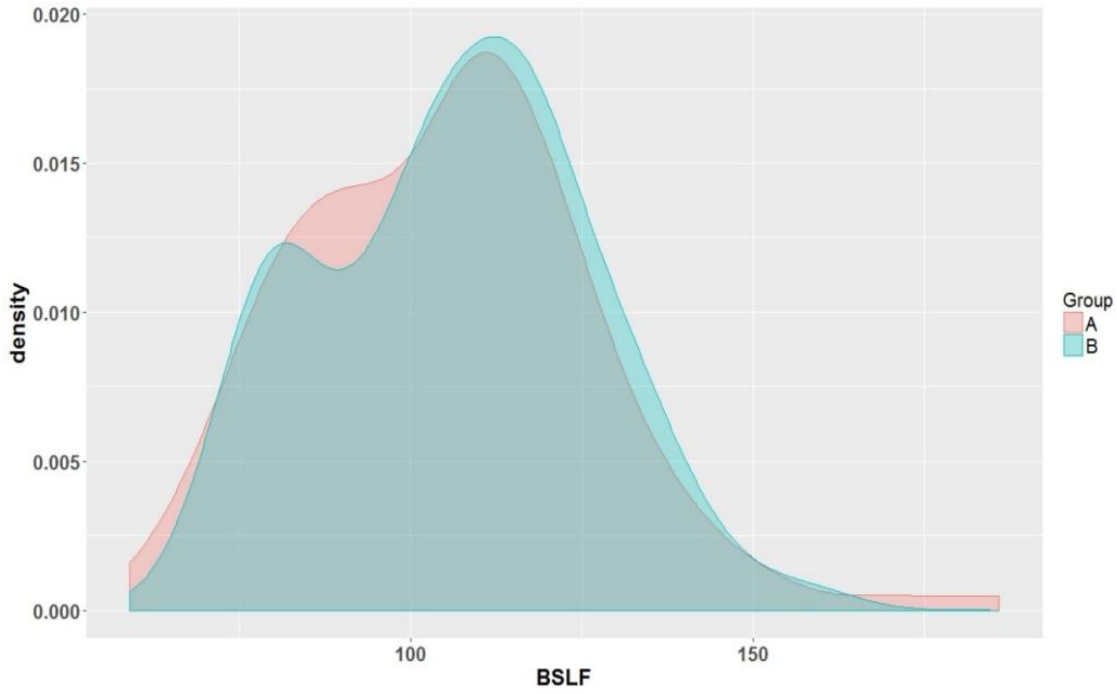
7. BSL Fasting:

Table No. 65: Distribution of 260 patients according to BSL Fasting

BSLF mg/dl	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Group A	59	90	108	105.1	118	186
Group B	68	92	108	106.4	119	160

In group A out of 130 patients minimum Fasting BSL is 59 mg/dl, maximum Fasting BSL is 186 mg/dl, mean Fasting BSL is 105.1 mg/dl and median Fasting BSL is 108 mg/dl, 1st Quarter Fasting BSL is 90 mg/dl 3rd Quarter Fasting BSL is 118 mg/dl.

In groupB out of 130 patients minimum Fasting BSL is 68 mg/dl, maximum Fasting BSL is 160 mg/dl, mean Fasting BSL is 106.4 mg/dl and median Fasting BSL is 108 mg/dl, 1st Quarter Fasting BSL is 92 mg/dl 3rd Quarter Fasting BSL is 119 mg/dl.



Graph No.8: - Distribution of 260 patients according to BSL Fasting

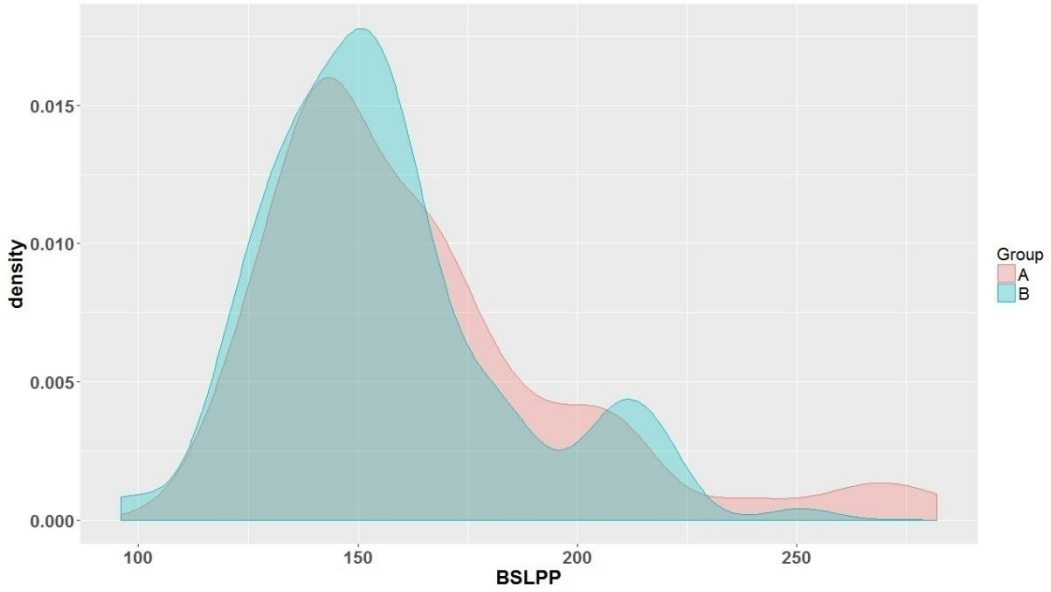
8. BSL Post parandial (PP)

Table No. 66: Distribution of 260 patients according to BSL post prandial.

BSLPP mg/dl	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Group A	110	139	152	163.3	176	282
Group B	96	136	152	155.8	166	251

In group A out of 130 patients minimum post prandial BSL is 110 mg/dl, maximum post prandial BSL is 282 mg/dl, mean post prandial BSL is 163.3 mg/dl and median post prandial BSL is 152 mg/dl, 1st Quarter post prandial BSL is 139 mg/dl 3rd Quarter post prandial BSL is 176 mg/dl.

In group B out of 130 patients minimum post prandial BSL is 96 mg/dl, maximum post prandial BSL is 251 mg/dl, mean post prandial BSL is 155.8 mg/dl and median post prandial BSL is 152 mg/dl, 1st Quarter post prandial BSL is 136 mg/dl 3rd Quarter post prandial BSL is 251 mg/dl.



Graph No. 9 - Distribution of 260 patients according to BSL post prandial

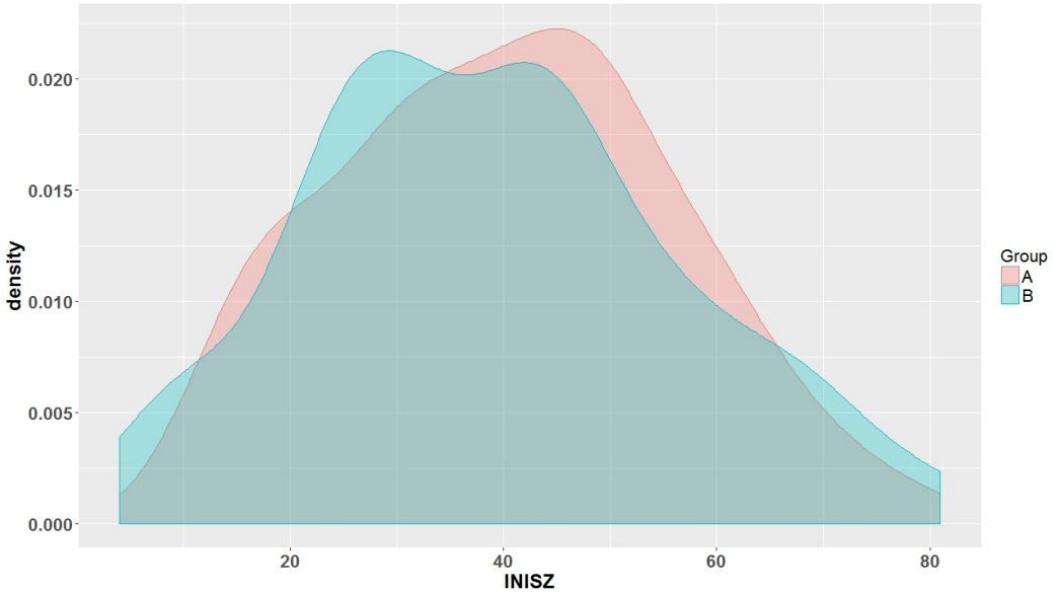
9. Initial Ulcer size:

Table No.67: Distribution of 260 patients according to initial Ulcer size:

Initial Size sq cm	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Group A	11	29	41	40.25	50	80
Group B	4	27	39	39.08	49.75	81

In group A out of 130 patients minimum initial size of Ulcer is 11 sq cm, maximum initial size of Ulcer is 80 sq cm, mean initial size of Ulcer is 40.25 sq cm and median initial size of Ulcer is 41 sq cm, 1st Quarter initial size of Ulcer is 29 sq cm 3rd Quarter initial size of Ulcer is 50 sq cm.

In group B out of 130 patients minimum initial size of Ulcer is 4 sq cm, maximum initial size of Ulcer is 81 sq cm, mean initial size of Ulcer is 39.08 sq cm and median initial size of Ulcer is 39 sq cm, 1st Quarter initial size of Ulcer is 27 sq cm 3rd Quarter initial size of Ulcer is 49.75 sq cm.



Graph No. 10 - Distribution of 260 patients according to initial Ulcer size

10.Pain :

Table No. 68: Frequency table for Group against pain

DAY1Pain	Group A	Group B	Sum
Grade 1	15	9	24
Grade 2	67	75	142
Grade 3	40	34	74
Grade 4	8	12	20

X-squared = 3.2372, df = 3, p-value = 0.3565

As Chi-square test is non - significant. So, arrangement of both group is homogeneous. Maximum patients are in grade 1st and grade 3 Category

11. Smell

Table No. 69: Assessment of smell of ulcer within group

DAY 1Smell	Group A	Group B	Sum
Grade 1	16	11	27
Grade 2	64	73	137
Grade 3	32	26	58
Grade 4	18	20	38

X-squared = 2.2431, df = 3, p-value = 0.5235 As Chi-square test is non-significant. So, arrangement of both groups is homogeneous. Maximum patients are in grade II category.

12. Size of the ulcer

Table No. 70: - Assessment of size of the Ulcer

DAY 1SZ	Group A	Group B	Sum
Grade 1	18	14	32
Grade 2	44	53	97
Grade 3	56	48	104
Grade 4	12	15	27
Sum	130	130	260

X-squared = 2.2838, df = 3, p-value = 0.5156

As Chi-square test is non-significant. So, arrangement of both groups is homogeneous. In both groups, maximum patients Ulcer size are in Grade 2 and Grade 3 before treatment

13. Secretion:

Table No. 71: Assessment of secretion within group

DAY1SEC	A	B	Sum
Grade 1	11	8	19
Grade 2	10	17	27
Grade 3	36	41	77
Grade 4	73	64	134
Sum	130	130	260

X-squared = 3.2044, df = 3, p-value = 0.3612

As Chi-square test is non-significant. So, arrangement of both groups is homogeneous. In both groups, maximum patient's secretions are in Grade 3 and Grade 4 before treatment.

14. Granulation:

Table No. 72: Assessment of Granulation tissue within group

Day 1 Granulation	A	B	Sum
Grade 1	1	2	3
Grade 2	127	124	251
Grade 3	2	1	3
Grade 4	0	3	3
Sum	130	130	260

>prop.test(n=251,127, p=124/251)

1-sample proportions test with continuity correction

data: 127 out of 251, null probability 124/251

X-squared = 0.099616, df = 1, p-value = 0.7523

alternative hypothesis: true p is not equal to 0.4940239

95 percent confidence interval: 0.4425380 0.5692283

sample estimates: p 0.5059761

Chi-square is not applicable to granulation data. As most of data accumulate in one category. So, we have used proportion test here. Maximum patients have grade II initial granulation.

15. Ulcer shape:

Table No. 73: Assessment of Ulcer shape within group

DAY 1Shape	Group A	Group B	Sum
Grade 1	64	67	131
Grade 2	44	40	84
Grade 3	22	23	45
Sum	130	130	260

X-squared = 0.2814, df = 2, p-value = 0.8687

As Chi-square test is non-significant. So, arrangement of both groups is homogeneous. In both groups, maximum patient's Ulcer shape are in Grade 1 and Grade 2 before treatment.

16. Ulcer bed

Table No. 74: Assessment of Ulcer bed within group

DAY1BD	A	B	Sum
Grade 1	0	0	0
Grade 2	22	15	37
Grade 3	10	8	18
Grade 4	98	107	205
Sum	130	130	260

X-squared = 1.9417, df = 2, p-value = 0.3788

As Chi-square test is non - significant. So, arrangement of both groups is homogeneous. In both groups, maximum patient's Ulcer bed are in Grade 4 and Grade 2 before treatment.

17. Corelation Matrix:

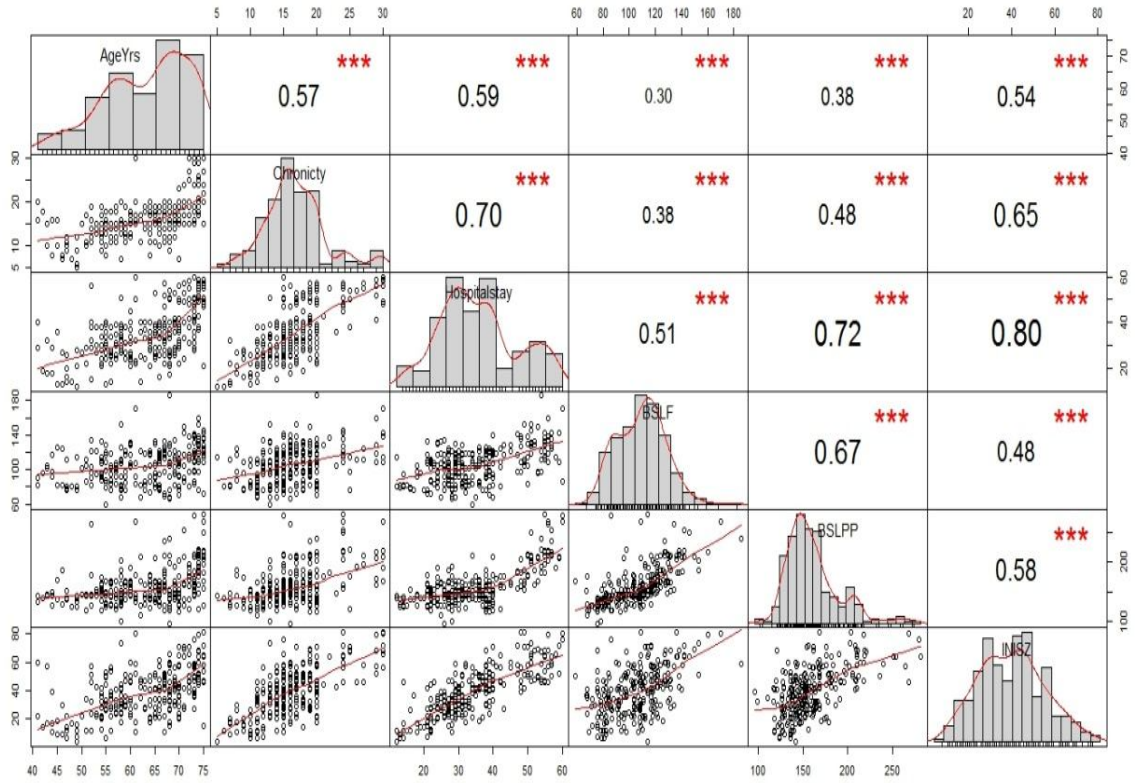


Figure No.11: correlation matrix graph.

The above graph is graphical representation of correlation matrix. The upper triangle contains correlation coefficient values i.e. R. The lower triangle contains scatter-plots. The purpose of graph is here to provide inter relations between all numerical variables considered in the study.

18 .T-test for initial status of patients in Numerical variables by Group:

Table No75: T-test Initial screening measured parameters compared according groups

By Group	Mean A	Mean B	Df	95%CI LL	95%CI UL	t value	P-value
Age	62.4077	63.8923	257.31	-3.58123	0.612	-1.3944	0.1644
Chronicity	16.1923	16.4692	257.46	-1.43187	0.878024	-0.4722	0.6372
Hospital Stay	35.6846	34.2	257.83	-1.35482	4.324047	1.0296	0.3042
BSLF	105.069	106.431	256.09	-6.39572	3.672642	-0.5326	0.5948
BSLPP	163.277	155.815	245.73	-0.28932	15.2124	1.8961	0.0591
Day 1 Size	2.47692	2.49231	257.97	-0.22105	0.190279	-0.1473	0.8830

All above t-test results are non-significant. As these variables are pre-treatment and non-significant so we can say that groups are randomly assigned. No sampling bias is there.

To avoid sampling bias, samples must be assigned equivalently in most of necessary aspects in both groups. The t test results are non-significant explaining that the group assignment is happened homogeneously and randomly.

19. T-test for initial status of patients in Numerical variables by Gender.

Table. No.76: T-test for initial status of patients in Numerical variables by Gender

By Gender	Mean A	Mean B	Df	95%CI LL	95%CI UL	t value	P-value
Age	63.36145	63.05085	169.19	-1.90501	2.526207	0.27674	0.7823
Chronicity	15.66265	16.64407	145.91	-2.26883	0.305997	-1.50660	0.1341
Hospital Stay	34.50602	35.14689	153.36	-3.75831	2.476569	-0.40613	0.6852
BSLF	104.1687	106.4915	163.52	-7.69208	3.046373	-0.85425	0.3942
BSLPP	158.6627	159.9605	153.96	-9.83459	7.238989	-0.30032	0.7643
*Day 1 Size	2.445783	2.502825	138.41	-0.29259	0.178511	-0.47881	0.6328

All above t-test results are non-significant. As these variables are pre-treatment and non-significant so we can say that samples are randomly assigned. No bias is there. No influence of gender is there.

In medical research, gender should not be ignored without evidence, because gender influences body parameter in many conditions. So, we tested that initial parameters are homogeneous according to gender. T-test non-significant results are sufficiently explaining that the role of gender may ignore here.

B. Observations for healing parameters made Before Treatment, during and After Treatment:

Frist day statuses for all healing parameters are discussed earlier. In this section, we are comparing progress of patients during treatment. The only numerical parameter is hospital stay. While pain, smell, size, secretion, granulation, Shape, bed is rank or category. So, mean of ordered categories and Wilcox test between group is applied

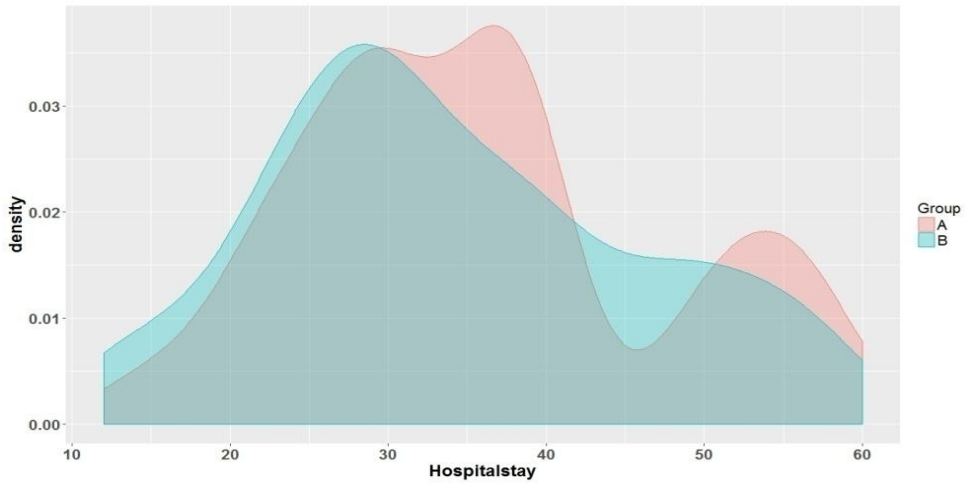
1. Hospital stay:

Table No. 77: Distribution of 260 patients according to total hospital stay:

Hospital stay	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
A	12	28	35	35.68	40	60
B	12	26	31	34.2	42	59

In group A out of 130 patients minimum hospital stay is 12 days, maximum hospital stay is 60 days, mean hospital stay is 35.38 days and median hospital stay is 35 days, 1st Quarter hospital stay is 28 days 3rd Quarter hospital stay is 40 days.

In group B out of 130 patients minimum hospital stay is 12 days, maximum hospital stay is 59 days, mean hospital stay is 34.2 days and median hospital stay is 31 days, 1st Quarter hospital stay is 26 days 3rd Quarter hospital stay is 42 days. It is homogeneously arranged. To understand trends in the distribution density plot.



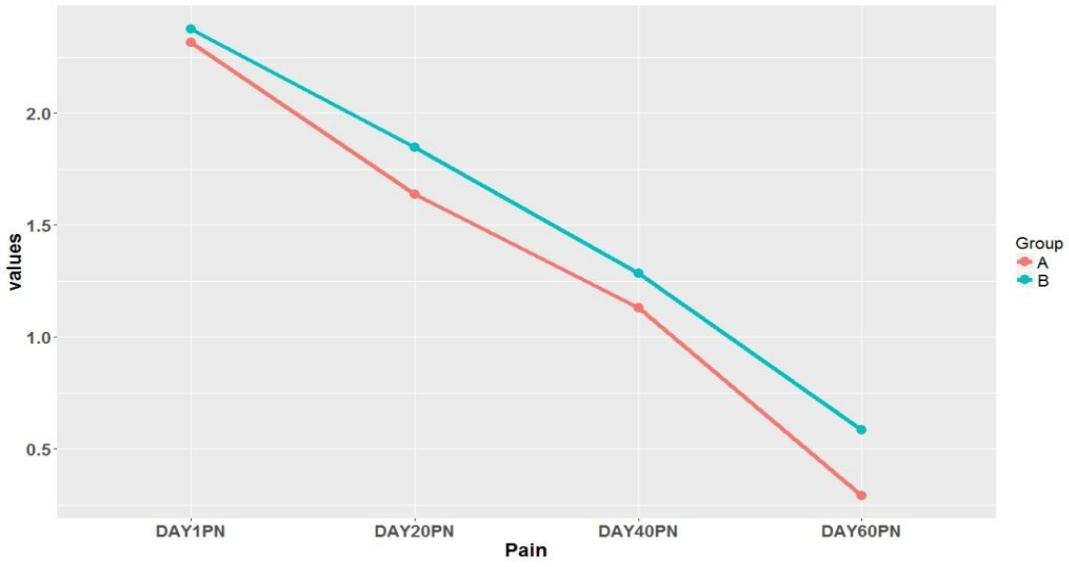
Graph No. 12: Distribution of 260 patients according to total hospital stay :

2. Pain:

Table No.78: Mean of ordered categories and Wilcox test between group results for Pain by day against Group

Pain\Group	A	B	Wilcox rank test value	P-Value
DAY1	2.315	2.377	8215.0	0.6679000
DAY20	1.638	1.846	7338.5	0.0457200*
DAY40	1.131	1.285	8031.5	0.3740000
DAY60	0.292	0.585	7732.5	0.1190000

In above table, column A and column B contains mean of ordered categories for pain level measurements on the day according to groups. We know that mean is not appropriate measure for ordered data, nevertheless, we used to mean here for conventions of understanding. Though we have used Wilcox rank test between both groups respectively pain measured on the day which is appropriate to compare ordered data. In above table only Day20 pain measured is significant which is mentioned with asterisk (*



Graph No. 13: Pain relif in 60 days between groups

Maximum patients are cured in between 20-40 days.

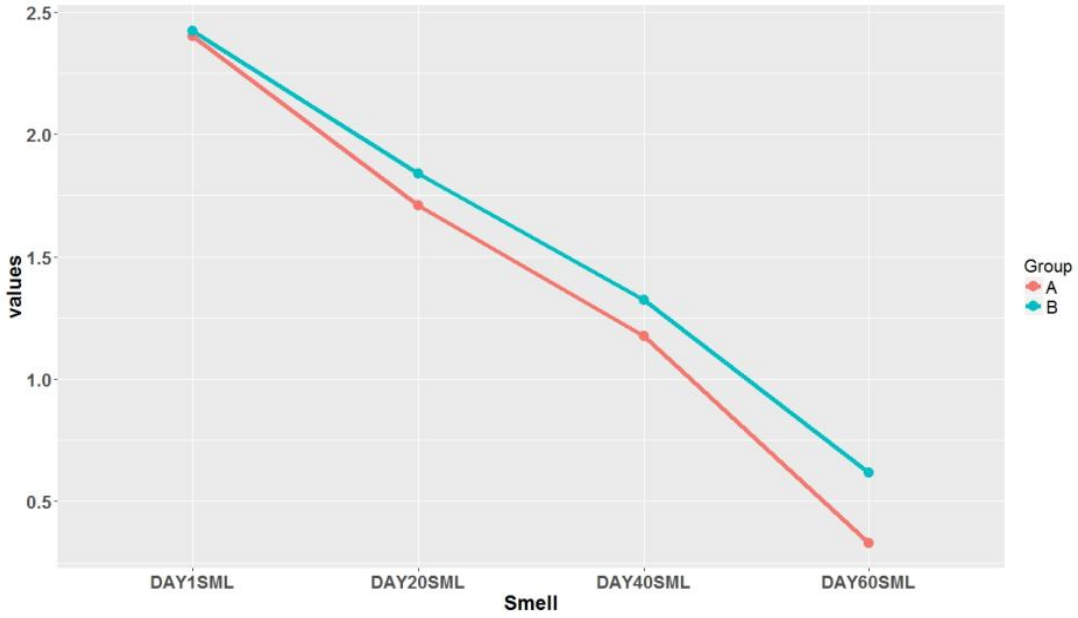
3. Smell:

Table No.79: Mean of ordered categories and Wilcox test between group results for Smell by day against Groups.

Group	A	B	Wilcox rank test value	P-Value
DAY1	2.400	2.423	8392.0	0.9175000
DAY20	1.708	1.838	8013.5	0.4359000
DAY40	1.177	1.323	8222.5	0.6448000
DAY60	0.331	0.615	7764.0	0.1363000

After observing the Wilcox rank test result the patient from group A have initial mean rank is 2.400 which is reduced up to 0.331 after treatment. From group B have initial mean rank is 2.423 which is reduced up to 0.615 after treatment. The gradual reduction in smell is observed in both group. The P-value is 0.9175000, 0.4359, 0.6448000, 0.1363000 which is greater than 0.005

The difference between two groups is not significant, which indicate that both groups have equal effect on smell.



Graph No. 14: Smell relief in 60 days between groups.

4. Size of the ulcer:

Table No. 80: Mean of ordered categories and Wilcox test between group results for Size by day against Group

Group	A	B	Wilcox rank test value	P-Value
DAY1	2.477	2.492	8579.0	0.8164000
DAY20	1.815	1.908	8160.5	0.5977000
DAY40	1.192	1.300	7646.0	0.1510000
DAY60	0.315	0.554	7877.0	0.2181000

After observing the Wilcox rank test result the patient from group A have initial mean rank is 2.477 which is reduced up to 0.315 after treatment. From group B have initial mean rank is 2.492 which is reduced up to 0.554 after treatment. The gradual reduction in Ulcer size is observed in both group. The P-value is 0.8164000, 0.5977000, 0.1510000000, 0.2181000 which is greater than 0.005 the difference between two groups is not significant which indicate that both groups have equal effect on Ulcer size.

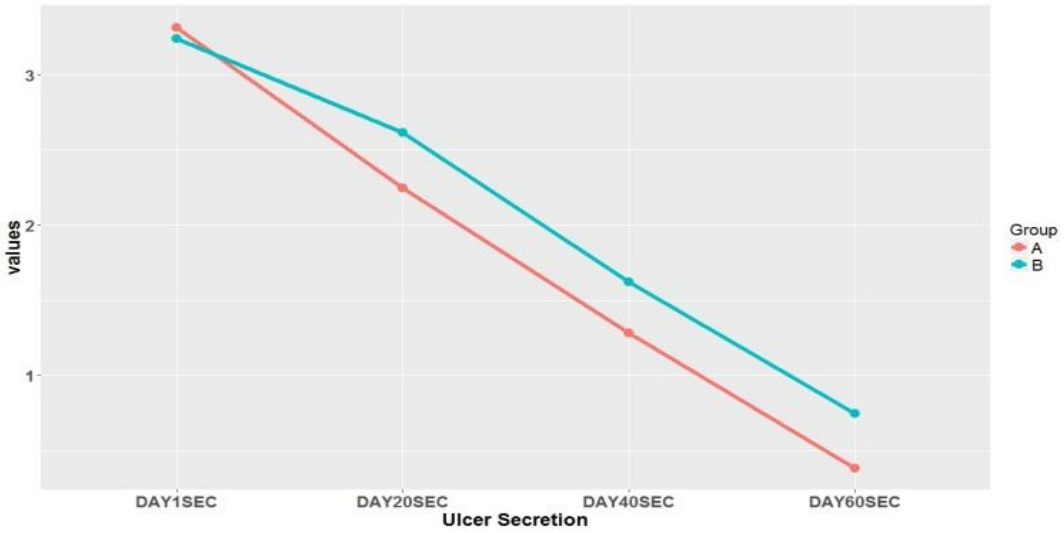
Graph No. 15: Size relief in 60 days between groups

5. Secretions:

Table No. 81: Mean of ordered categories and Wilcox test between group results for Secretion by day against Group

Group	A	B	Wilcox rank test value	P-Value
DAY1	3.315	3.238	8215.0	0.6679000
DAY20	2.246	2.615	7437.5	0.0684100
DAY40	1.285	1.623	8031.5	0.3740000
DAY60	0.385	0.746	7732.5	0.1190000

After observing the Wilcox rank test result the patient from group A have initial mean rank is 3.315 which is reduced up to 0.385 after treatment. From group B have initial mean rank is 3.238 which is reduced up to 0.746 after treatment. The gradual reduction in Ulcer size is observed in both groups.The P-value is 0.6679000, 0.0684100, 0.3740000, 0.1190000 which is greater than 0.005 the difference between two groups is not significant which indicate that both groups have equal effect on Ulcer secretion.



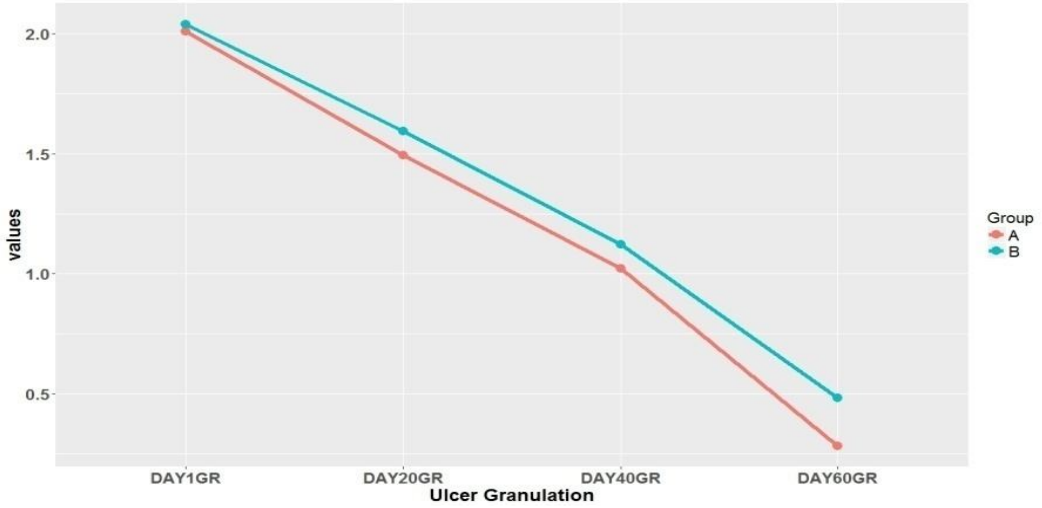
Graph No. 16: Secretion relief in 60 days between groups.

6. Granulation tissue:

Table No. 82: Mean of ordered categories and Wilcox test between group results for Granulation by day against Group

Group	A	B	Wilcox rank test value	P-Value
DAY1	2.008	2.038	9001.0	0.3178000
DAY20	1.492	1.592	6834.0	0.0048970*
DAY40	1.023	1.123	7505.5	0.0630900
DAY60	0.285	0.485	7818.5	0.1727000

After observing the Wilcox rank test result the patient from group A have initial mean rank is 2.008 which is reduced up to 0.285 after treatment. From group B patients have initial mean rank is 2.038 which is reduced up to 0.485 after treatment. The gradual improvement in Ulcer granulation tissue quality is observed in both group. The P-value is 0.3178000, 0.004100 (as Ayurved treatment group shows better granulation development during this period), 0.0630000, 0.1727000 which is greater than 0.005 the difference between two groups is not significantWhich indicate that both groups have equal effect on Ulcer granulation tissue improving quality



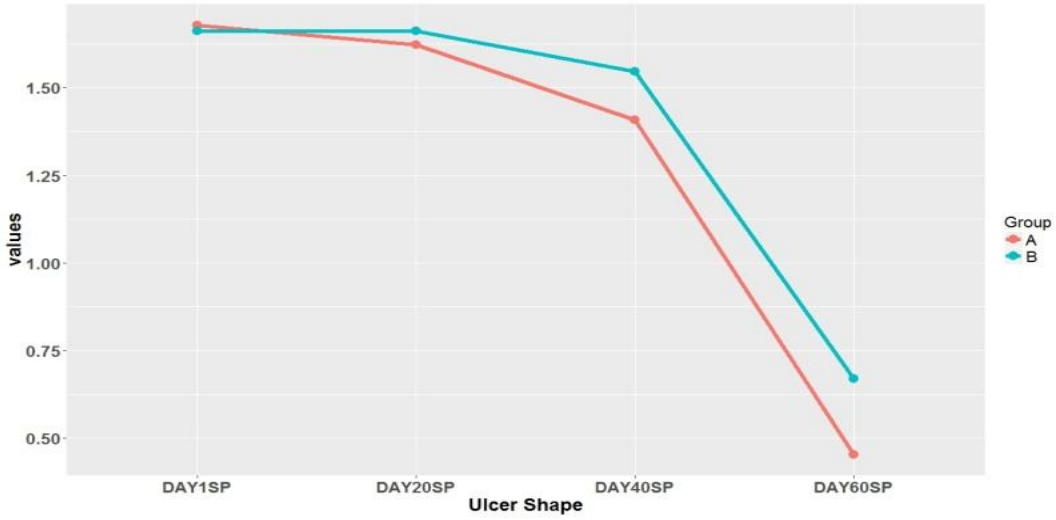
Graph No. 17: Granulation relief in 60 days between groups.

7. Ulcer shape:

Table No. 83: Mean of ordered categories and Wilcox test between group results for Shape by day against Group

Group	A	B	Wilcox rank test value	P-Value
DAY1	1.677	1.662	7852.0	0.1662000
DAY20	1.623	1.662	6763.0	0.0027330*
DAY40	1.408	1.546	6764.0	0.0012630*
DAY60	0.454	0.669	7746.5	0.1267000

After observing the Wilcox rank test result the patient from group A have initial mean rank is 1.677 which is reduced up to 0.454 after treatment. From group B patients have initial mean rank is 1.662 which is reduced up to 0.669 after treatment. The gradual improvement in Ulcer shape is observed in both group. The P-value is 0.1662000, 0.002700, 0.0012630, 0.1267000 which is greater than 0.005 the difference between two groups is not significant; which indicate that both groups have equal effect on Ulcer granulation tissue improving quality. At day 20 and day 40 p values is significant; as Ayurved treatment shows better result.

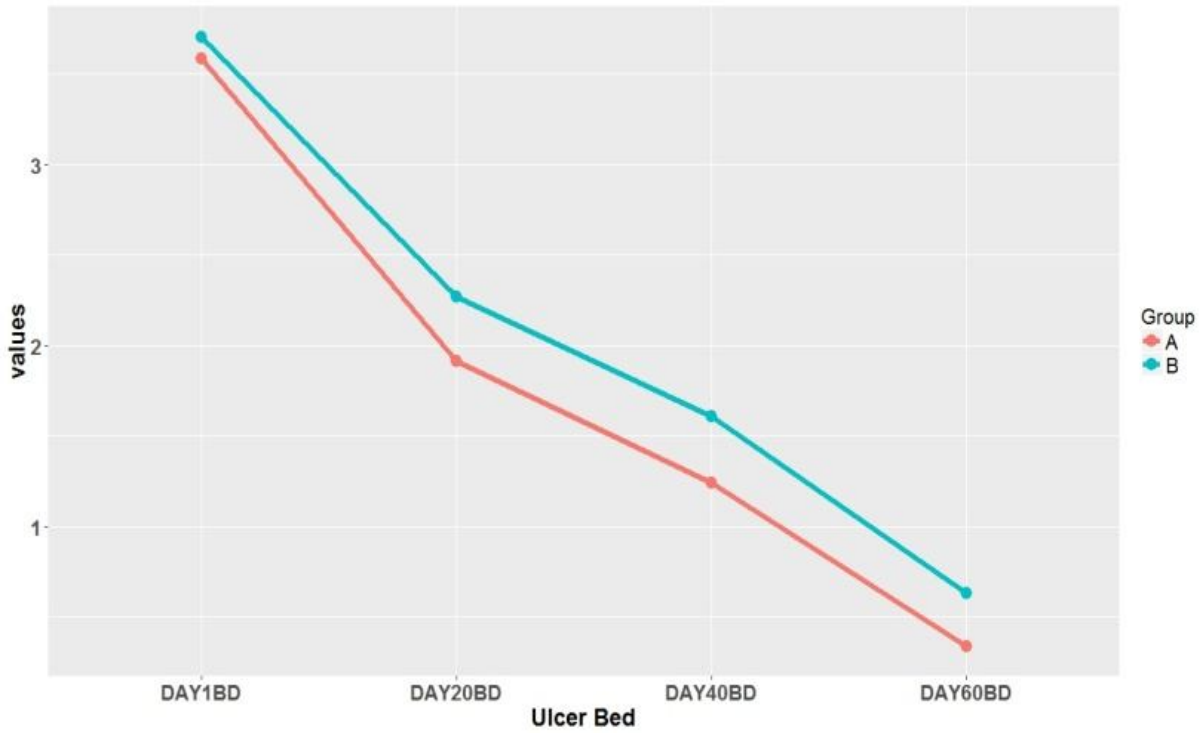


Graph No. 18: Shape relief in 60 days between groups

8. Ulcer Bed:

Table No. 84: Mean of ordered categories and Wilcox test between group results for Ulcer Bed by day against Group

Group	A	B	Wilcox rank test value	P-Value
DAY1	3.585	3.708	8382.0	0.7252000
DAY20	1.915	2.269	7585.0	0.1012000
DAY40	1.238	1.608	7630.0	0.0629300
DAY60	0.338	0.631	7714.5	0.1078000



Graph No. 19: Ulcer Bed relief in 60 days between groups.

9. Healing outcome in both groups:

Table No. 85: Healing outcome in both groups

Healing Outcome	Group A	Group B	Sum
Complete Epithelization	113	104	217
Contraction Without Epithelization	14	22	36
Minor Amputation	1	3	4
Major Amputation	2	1	3
Death	NAN	NAN	NAN
Sum	130	130	260

>chisq. Test (matrix(c(113,104,17,25),nrow=2,byrow=T))#Pulled table

Pearson's Chi-squared test with Yates' continuity correction

data: matrix(c(113, 104, 17, 25), nrow = 2, byrow = T)

X-squared = 1.4578, df = 1, p-value = 0.2273

Above table contains Chi-square test which results non-significant. The test is applied on pulled table to maintain continuity or accuracy, the pulling is done for major and minor amputation in to Contraction without Epithelization as frequency in both categories is less than 5 each.

The control group B is already accepted and proven treatment protocol for Ulcer in the Modern medicine.So, we can conclude that both groups are equally effective for the Ulcer treatment.

>prop.test(n=260,217,p=43/260)

1-sample proportions test with continuity correction

data: 217 out of 260, null probability 43/260

X-squared = 838.77, df = 1, p-value < 2.2e-16

alternative hypothesis: true p is not equal to 0.1653846

95 percent confidence interval:

0.7825509 0.8765266

Sample estimates: p 0.8346154

To speak for healing outcome, we need to test all outcomes within groups. But as chi-square test for Healing Outcome against Group is non-significant so we can consider that both methods of treatment are equally effective in the study. Due to small frequencies in minimum and major amputation in both groups we merge (pulling of data) amputation category in contraction to consider non-healing outcome in common and Epithelization is complete healed outcome.

In above single proportion test, we sum up all completely healed patients and compared with sum of all non-healed patients. The test is significant so we can conclude that both A and B treatment groups are 83.46% effective and 16.53% cases are non-healable with A or B treatment.

Chapter 6. Discussion

1. Prevalence of Diabetes:

As mentioned in Ayurveda causes of the Prameha are also seen in DM2. The main cardinal symptom of Prameha i.e. increased frequency of micturition, trishna, sweating, burning sensation and sweetness in mouth (M.ni. Pramehanidan 33/5-6)[328] is correlating with DM2. As the hetu's mentioned in classics (M.ni. Pramehanidan 33/1 [329] are increasing in day to day life style of current era. So ultimately the incidence of Prameha will also increasing. Prameha is also mentioned as Ashtomahagada; i.e. eight difficult diseases to treat, as prognosis of the disease are worst. (Su.Su.33/4-5).[330]

2. Prevalence of Ulcer in Diabetes:

In a study to determine prevalence of diabetic foot in India, the prevalence of infection noted was 6-11% and prevalence of amputation was 3% in type 2 diabetic patientsdue to nutritional poverty, lack of medical awareness about the diabetic ulcer. The patients could not able to recover his ulcer in time, so these ulcers land into chronic nonhealing ulcers. The fate of nonhealing ulcer is amputation or death.

Sushruta mention incidence of Asadhya Vrana in Madhumeha (Su.su.23/7)[331] Prameha is also mentioned as Ashtomahagadu.e. eight difficult diseases to treat, as prognosis of the disease are worst. (Su.Su.33/4-5). [332]

3. Prameha and Prameha Pidika:

In uncontrolled Diabetes vitiated vasa and meda with tridosha get aggravated and forms various types of Pidika type of Nij Vrana (Su.Ni.6/14)**,**[333] in which line of treatment should be done as per type of Diabetes. (Su.Ni.6/9)[334].Treatment of immature pidika is like shoph, and pakwapidika is like Vrana (Su.Chi.13/9)**.**[335]

4. Chronic Ulcers and Dushta Vrana:

Concept of Dustha Vrana and chronic ulcer is very well correlating (Su.Su. 22/7).[336] Charak and Sushruta have mentioned details of Ulcers in Vrana chapter, which are discussed in detail in Review of Vrana chapter.

5. Discussion on initial attributes of chronic ulcer:

1. Site of Ulcer:

The incidence of Ulcer site in present study was found more in lower and upper extremities of body which is due to stagnation of the blood against gravity. Samedescription was found in classics (Su.Chi.13/8)[337].This explains due to incompetence of Ras-Raktavaha Dhamani vitiated Ras, Pitta and Kapha get accumulated in extremities part of body. These ksheenRas-Raktta dhamani is not able to carry dosha in koshta. So, the Dosha get Adhogati and accumulate in shakha, which forms ulcer in extremities. Workers have maximum chances of injury to extremities. Offloading techniques to avoid pressure on ulcer plays important role in healing.

2. Chronicity:

In correlation matrix graph we have observed, the grade of Ulcer, hospital stay, size, shape, healing outcome, pain, secretion, smell, and granulation is dependent on chronicity of Ulcer. As the chronicity increases the disease prognosisand grade of ulcer is also increases. The local, systemic damage and defense are also affected. This increases the chances of non-healingVrana in Madhumeha and it is mentioned as difficult to treat. (Su.Su.23/7)[338]. All diseases Asadhya after 1 year Chronicity. (Su.Su.10/6).[339] In Sadhyasadhyatwa Jirnavyadhi is also mentioned by Sushruta (Su.Su. 23/3,4,5,6,8).[340]

3. Age:

In correlation matrix graph we have observed, as the age increases the possibility of non-healing is also increases. As in senile age the recovery is slow due to reduce immunityandreduce production of essential nutrients and cells. In Sadhyasadhyatwa of Vrana the old age is mentioned as difficult to treat in Sushruta Samhita (Su.Su. 23/3, 4, 5, 6).[341]

4. Gender:

In correlation matrix graph we have observed, in male population the incidence of non-healing is more. The selection of cases is on willing and random criteria. So, we have not any evident data to explain this finding. One of reason may be the working stylein male dominating culture in this region, as more male are workers. Workers have maximum chances of injury.

5. Occupation:

In analysis of occupation graph and table we have observed, workers are more in numbers. One of reason may be the working style and male dominating culture in this region. So, more male are workers. Workers have maximum chances of injury also the nutrition is also not balance in them. The health awareness in these people is also low.

6. Hospital stay:

In correlation matrix graph, we have observed as the age, size of ulcer, BSL, chronicity all these increases the rate of healing is delayed. As the healing is delayed the hospital stay of patients is also increases.

7. BSL:

In correlation matrix graph we have observed, as thethe BSL level is increases the size of ulcer, chronicity, hospital stay are increases. As age increases then BSLlevel also increasing. In the prognosis of Diabetic Ulcer BSL control has direct effect on healing. The uncontrolled BSL level worse the pathology and increases complications. We have already discussed in Pathophysiology of Diabetic Ulcer.

8. Initial size of ulcer:

Minimum size in group A is 11sq/cm and in group B is 4sq/cm. It is an accidental finding due to sampling method. Maximum size is same. Most of patients from both groupare in between 20-60 sq/cm. In correlation matrix, the larger size has direct relation with age, chronicity, hospital stay, BSL level. The cause may be larger size of ulcer have maximum exposure area for infection, trauma, need more nutrient, reduced

immunity due to chronicity. This delays healing of ulcer. The non-healingtendency increases size of Ulcer.

9) Ulcer Grade:

Maximum patients of grade I ulcer are found in this study. The selection of cases is on willing and random criteria. Secondly only grade I and II ulcers are selected in this study. As grade III and grade IV ulcer need different treatment protocols and they are difficult to heal. So, they are excluded to avoid healing bias in study. So, we have not any evident data to explain this finding. Only early intervention for treatment after incidence of ulcer. As the mean chronicity for group A and B is 16.9 weeks and 16.47 weeks respectively. Sushruta has mention correlation of the site of ulcer and its severity according to Vrana-dhatugata adhisthan and its sadhyasadhatwa Su.Su.22/3.[342]

The t-test results are non-significant for initial parameters. So, we can say that groups equal for comparison are randomly assigned. No sampling bias is there.

6) Healing Parameters:

i) Size: In this study, we have minimum size is observed in pressure ulcer area of heel. These ulcers are very difficult to treat. While a patient is walking, his ulcer gets pressure effect and the less vascularity in this region. The maximum size is observed on anterior and posterioraspect of leg, sole area, and few gluteal areas. This is because of Chronicity of ulcer and most of patients have associated cellulitis in initial phase. The size of wound is directly proportion to age, chronicity, BSL level, nutrition, and infection.

ii) Shape: In Sushruta Samhita Su.Su.22/5[343] four shapes are mentioned. Among them we have not observed Triputak and Shesh shape in this study, as the sample size is limited. The original shape changes at the end of healing day's. As the oval and circular shapes remain till the end. Only irregular shape changes fast, changes to oval shape.

iii) Secretion: Maximum patients have grade 3 i.e. Hemosanginious and Grade 4 pus secretion initially. This is because all are chronic ulcer. Heavy infected ulcer shows pus discharge and unhealthygranulation and infected ulcer shows hemosanginious

discharge. This discharge is very well correlating to Sushruta's vran-stravamention in Su.Su.22/8[344]. The secretion shows gradual reduction in both groups.

iv) Smell: In Sushruta Samhita the details of Strava according to Dosha is given at Su.Su.22/10[345].But for practical and gradation purpose we have selected 4 grades. They are very well correlating to Sushruta. As the ulcer start healing the amount and quality of smell also decrease. The smell shows gradual reduction in both groups.

v) Granulation: In Sushruta Samhita the details of Granulation according to Dosha is given at Su.Su.22/7,12[346].He has mention various colour of Ulcers, but for practical and gradation purpose we have selected 4 grades. They are very well correlating to Sushruta. As the ulcer start healing the amount and quality of granulation is also improves. The granulation shows gradual improvement in both groups.

vi) Pain: The gradual and regular decrease in pain is observed in both groups. Only first 20days observation of Ayurved treatment group is showing little but fast decline in pain than group B. This may be due to early control of Vata dosha by drugs. The description about pain in Sushruta Samhita is in detail than modern science Su.Su.22/11.[347]

vii) Ulcer bed: In Sushruta Samhita the details of Ulcer bed colour according to Dosha is given at Su.Su.22/12.[348] He has mentioned various colour of Ulcers. But for practical and gradation purpose we have selected 4 grades. They are very well correlating to Sushruta's information. As the ulcer start healing, the amount and quality of Ulcer bed is also improves. The Ulcer bed colour shows gradual improvement in both groups.

Chi-square test has non-significant result. The test is applied on pulled table to maintain continuity or accuracy, the pulling is done for major and minor amputation in to Contraction without Epithelization as frequency in both categories is less than 5 each. Single proportion test, we sum up all completely healed patients and compared with sum of all non-healed patients. The test is significant so we can conclude that both A and B treatment groups are 83.46% effective and 16.53% cases are non-healable with A or B treatment.

The control group B is already accepted and proven treatment protocol for Ulcer in the Modern medicine. So we can conclude that both groups are equally effective for the Ulcer treatment.

7. Possible mode of action of Nimbadi Kalka:[349-53]

The Guna, Rasa, Veepak and Virya which normalize the vitiated dosha are given below.

1) According to Dosha Guna :

Table No. 86: Dosha Guna, Ras, Veepak and Veerya correlation: [350]

Dosha/ Guna	Guna	Rasa	Veepak	Virya
Vata	Ushna-DaruharidraTila Snigdha-Tila,Yashtimadhu Ghruta Sukshma-Tila Guru-Yashtimadhu Ghruta Mrudu-Yashtimadhu Ghruta	Madhur-Yashtimadhu, Ghruta,Tila, Madhu	Madhur-Yashtimadhu, Ghruta, Madhu	Ushna-DaruharidraTila
Pitta	Snigdha-Tila,Yashtimadhu Ghruta Sheet-Yashtimadhu Ghruta Guru-Yashtimadhu Ghruta Mrudu-Yashtimadhu Ghruta	Madhur-Yashtimadhu, Ghruta,Tila Madhu Tiktta- Nimba, DaruharidraTila Kashaya- Tila Madhu	Madhur-Yashtimadhu, Ghruta, Madhu	Sheet-Nimba Yashtimadhu, Ghruta, Madhu
Kapha	Ushna-DaruharidraTila Rukshya-Daruharidra Madhu Sukshma, tikshna-Tila Laghu-Nimba, Madhu Khara-Madhu	Tiktta- Nimba, DaruharidraTila Katu-DaruharidraTila Kashaya-Tila Madhu	Katu-DaruharidraTila	Ushna-DaruharidraTila

2) According to Rasa:

a) Tikta rasa:[349] Nimba, Daruharidra and Tila are acting as vishaghna (nullifies the toxic effects), krimighna (anti-bacterial), dahaprashamana (reduces burning sensation), kushthaghna (skin disease modifying agent), mansasthirakara (enhances granulation), deepana-pachana-shodhana (local tissue metabolism regulator), lekhana-chedana (debridement), and kledashoshana (absorb the unwanted secreation). With the effect of Ruksha property it works as Kleda-meda-lasika and puya (Pus) upashoshana.

b) Katu rasa:[349] Daruharidra acts as Kledahar (Reduces unwanted sticky secretions), kanduhara (anti-pruritic), krimihar (anti-bacterial), strotovivarana (enhances microcirculation and micro channels), shodhana (local tissue metabolism regulator), vishahara (nullifies the toxic effects), kushthahara (skin disease modifying agent), shwayathuvinashana (anti-inflamatory), shonitasanghatabheda (anti-coagulant) property which is necessary for wound healing it happens due to teekshna property of this drug.

c) Madhurarasa:[349] Yashtimadhu, Tila, Ghruta and Madhu act as vishaghna (nullifies the toxic effects), dahashamana (reduces burning sensation), twachya (skin tissue immune modulator, preenana (regulates, jeevana, tarpana, brimhana,ksheenakshat-ahitakara and sandhanakara (enhances healing stages) due to snigdha, sheeta, guru properties of these drugs.

d) Kashaya rasa:[349] Madhu and Tila acts as sandhankara, ropana, shoshana, peedana, stambhana, raktaprashamana, kleda-medashoshana, lekhana and twakvarnyakara due to rukhsh, laghu, sheeta and khara properties of these drugs.

3) According to Guna:

e) Guna of Nimbadi kalka and its action: [350]

1. Guru--This guna is essential for Bruhan of mamsa and other dathu. This is present in Yashtimadhu, Tila and Ghruta.This is helpful for Granulation and wound healing.

2. Laghu-- Langhan essential for pachan. It is Ropak, Vatkar, Khaphaghana. This is present in Nimba, Madhu.This is helpful in debridement.

3. Sheet—This guna has Stambhan property.It is Dhatuvrudhikar, Dahashamak,Vat-pittahar, Kaphavrudhikar, Bruhan, Rakttasthamban.This is helpful for Granulation and wound healing. It also stops bleeding, Dhatupakand burning sensation. This is present in Nimba, Yashtimadhu, Madhu and Ghruta.

4. Ushna--This guna has Swedan property. It is Pachak, Dahak, and Vat-Khaphahar. This is helpful in debridement, which is present inDaruharidra and til.

5. Snigdha-- This guna has Snehan property. It is Vathar, Kaphakar, Balya, Rasayan, Bruhan, Snehan, Mardavkar. This is helpful for Granulation. This is present Yashtimadhu, Ghruta and Til.

6. Ruksha-- This guna has Rukshan property. It is Vatkar, Kaphahar, Shoshak, Sthambak, and Khar. This is helpful for debridement and to stop excess secretions in Ulcer.This is present in Daruharidra and Madhu.

7. Mrudu-- This guna has Shlathan property. It is Vathar, Kaphakar.This is helpful for Granulation by providing moist and oily environment to Ulcer.This is helpful for Granulation. This is present in Til and Madhu.

8. Teekshana- -This guna has Shodhan property. It is Kaphahar, Pittakar, Dahan, Pachan, and Lekhan. This is helpful for debridement and to stop excess secretions in Ulcer.This is present in Daruharidra and Til.

9. Vishad--This guna has Skhalanproperty.It is Picchilnashak, Lekhan. This is helpful for debridement and to stop excess secretions in Ulcer.This is present in Daruharidra and Tila.

10. Khara- This guna has Lekhanproperty.It is kaphahar, Vatkar. This is helpful to stop excess secretions in Ulcer.This is present in Daruharidra and Madhu.

Sukshma- This guna has Veevaran property. This is helpful to propagate the drug at micro Chanel and cellular level. This is present in Tila.

11. Drava- This guna has Sandhan and Veelodan property. This is helpful to propagate the drug on Ulcer area. This is present in Madhu.

f) Various Karma of Nimbadi kalka for Vrana Ropan and Shodhan: [350-52]

After studying various references from Samhita and Nighantu the drug Nimbadi kalka has following Karma mentioned for Vrana Shodhan and Ropan in it.

Table No.87: Various karma of Nimbadi kalka

Karma/Drug	Daruharidra	Nimba	Yashti	Tila	Madhu	Ghruta
Vrana	+	+	+	+	+	+
Shotha	+	+	+		+	
Ruja	+					
Astra, Rakttapitta	+ Vatraktta				Rakttapitta	Rakttapitta
Daha			+	+		+
Krumi, Jantu Rakshoghna	+	+ +		+	+	
Veesha	+	+	+			+
Kushta Twakdosha Twachya	+ +	+	Varnya	+ +		
Veeshodhan Shodhan	+	+		+	+	
Pachan	+	+		+		
Lekhan	+			+	+	
Kandughna	+	+		+		
Sandhan			+		+	
Ropan	+	+	+	+	+	+
Balya			+	+	+	+
Bruhan			+	+		
Jeevaniya			+			
Snehan			+	+		
Sanskaranuvarti					+	+

8 Corelation of Karmas of Nimbadi kalka Gunapanchak with Modern science.[350-53]

- **Antimicrobial activity**: Krumighana, Vishahara, Shodhan, Pachan, Jwaraghana.
- **Anti-inflammatory**: Shothaghna, Shodhan, pachan, Jwaraghana**,** Vishahara, Rakttadosh.
- **Debridement**: Shoshan, Kledaghana, Lekhan, Deepan, Pachan, Shothaghna, Shodhan, Jwaraghana**,** Vishahara, Rakttadoshjeet.
- **Circulation**: Tarpan, Shodhan, Astrajeet, Rakttapittahar.
- **Nutrition**: Shodhan, Bruhan, Mansavruddhikar.
- **Disease modifying**: Mehaghna, Kushtaghana, Kandughna, Vranajeet.
- **Immune modulator and growth factor enhancer**: Rasayan, Jeevaniya, Bruhan, Shukral.
- **Soothing and moistening agent**: Snehan, Cledan, Tarpan, Sheet, Dahashaman, Drava

9. Apptavachan:

The formulation of Nimbadi kalka is mentioned in various Samhita and Nighantu.This is used in Vrana Chikitsa. Charak Samhita Chi.25/84,85 [354]**.** Sushruta Samhita Su.37/15-30 [355] Su Chi.1/76-79 [356].Ashtanga HridayaUtt.25/53, 54, 55 [357].Ashtanga SamgrahChi.14/29,31,62[358].SharangdharSamhitaDwi.2/5[359]Sha.Sam.M.9/55,56,[360]Sha .Sam.U.11/85,86,88.[361]BhaishajyaRatnavali47/45,32.[362]Yogratakar/Uttar/Varanashoth achikitsa- 1,2,3,4 [363]

Ahstanga Nighantu, Dhanvantri Nighantu, Shodhal Nighantu, Kaiyadev Nighantu,Raj Nighantu,Bhavprakash Nighantu,Nighantu Adarsha. In these Nighantu the Vranahar activity is mentioned. This is discussed earlier in drug review and probable mode of action of drug.

In this way, the activity of Nimbadi kalka for Chronic Diabetic Ulcer is justified. **10.**

10.Failure of cases:

As we see in correlation matrix graph healing of Ulcer is directly proportional to age, chronicity, BSL control, initial size of ulcer.

The patients underwent for minor and major amputations have age above 70 years. Chronicity of Ulcer above 25 weeks. BSL is not in total control. Initial ulcer size is

above70 sq/cm. All these parameters are on higher size in groups. The Dosha Dhatudushti is more. So these patients underwent for amputation to save their life.

Same explanation about non-healing of Ulcer are mentioned in Sushruta Samhita Su.Su.10/8,[364] Su.Su.23/3 [365],Su.Su.23/11[366],Su.Su.23/17 [367]. This is earlier discussed in initial attributes.

11. Untoword effect study:

In Ayurved treatment group, no any untoword effect is observed in patient tereated with ayurved treatment.

Chapter 7. Conclusion

This study conclude that the drug formulation, mentioned in ayurved text Sharangdhar samhita and Bhaishajya Ratnavali is equally effective with comparison to modern medicine Drug in the management of chronic DM IIulcer. This is tested on modern research clinical protocol. The concept of chronic ulcer can be very well correlated with Dushta Vrana concept in Ayurved text.

From resultwe can conclude that both A and B treatment groups are 83.46% effective and 16.53% cases are non-healable with A or B treatment. The control group B is already accepted and proven treatment protocol for Ulcer in the Modern medicine.

- **Scope, Limitation and further study:**

Local application of Nimbadi Kalka is effective in Chronic Diabetic Ulcers.But further studies are needed for commercial use on large scale population, as this study is on limited patient population. Also, the effect on Chronic Ulcers of other Etio-Pathogenasis is to be studies. The commercial ready preparations are also not available.

Chapter 8. Summary

The dissertation entitled Study of Vrana Shodhan and Ropan Karma of Nimbadi Kalka on Chronic Diabetic Ulcer was studied under the following sections.

Introduction: The selection of topic, failure of current treatment protocol, Need of Ayurved treatment in field of Chronic Ulcer. The selection of drug from Ayurved literature is explained.

Review of literature:

1) Review of Vrana and Dushta Vrana: In this chapter, the Concept and treatment of Vrana is studied From Veda period to Ayurveda Samhita. Vrana and dushta Vrana is thoroughly discussed. The definition; causes, types, management, complication etc. are explained from different Ayurved classical texts.

2) Review of Ulcer and Chronic Ulcer: The concept and treatment aspect of wound and ulcer from Egypt period and other part of world is explained. The definition, types, etiopathology, clinical features, complication and management of ulcers are described in this chapter. The chronic diabetic ulcers are also explained.

3) Review of Prameha: The disease Prameha, Prameha pidika are described here. The definition; causes, types, management, complication etc. are explained from different Ayurved classical texts.

4) Review of Diabetes**:** The definition, types, etiopathology, clinical features, complication and management of Diabetes is described in this chapter.

5) Review of Drug: The drug contains of Nimbadi Kalka is studied. Rasa, Guna, Veerya, Vipaaka and Karma etc according to Samhita, Nighantu of are presented in this chapter.

6) Review of previous work done: Regarding the topic Ayurved data base, and modern data base is studied for previous work done.

Clinical study:

Materials and methods: The objectives of the study are stated here. The collected drugs Authentication, method of preparation, Standardization of formulation and application are discussed. The inclusion criteria, exclusion criteria, assessment criteria etc. are presented.

Observation: The observations made during the study regarding incidence, subjective and objective criteria of ulcer in stated period is recorded. The observations are presented with the help of tables and graphs. The necessary statistical analysis is done.

Discussion: Discussion about observation, result and action of the drugs is discussed.

Conclusion: Here conclusion drawn on regarding the whole study.

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ANNEXURE - I

महाराष्ट्र विज्ञान वर्धिनी
आघारकर अनुसंधान संस्था
Maharashtra Association for the Cultivation of Science
AGHARKAR RESEARCH INSTITUTE
(An Autonomous Grant-in-Aid Institute under the Department of Science and Technology, Govt. of India)

November 2, 2011

AUTHENTICATION CERTIFICATE

Name of the party: - Dr. Satish B Bandgar

Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Azadiricta indica (Leaves sample)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with taxonomic, macroscopic and microscopic characters. We hereby authenticate that the sample belongs to leaves of *Azadirachta indica* A.Juss. (Family-Meliaceae).

This certificate is issued at his request and is given only for the academic use.

Upadhye
(A.S. Upadhye)

Scientist
Plant Drug Authentication Service
Botany Group
Plant Sciences Division

Auth.11-162.

आगरकर पथ, पुणे – 411 004, भारत, दूरभाष : (020) 2567 8916/17/18, 2565 4357, 2565 3680 फॅक्स : (020) 2565 1542
Agarkar Road, Pune - 411 004, India, Phone : (020) 2567 8916/17/18, 2565 4357, 2565 3680 Fax : (020) 2565 1542
Web : www.aripune.org E-mail : arimacs@pn2.vsnl.net.in

महाराष्ट्र विज्ञान वर्धिनी
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Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Azadiricta indica (Leaves sample)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with taxonomic, macroscopic and microscopic characters. We hereby authenticate that the sample belongs to leaves of *Azadirachta indica* A.Juss. (Family-Meliaceae).

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November 2, 2011

AUTHENTICATION CERTIFICATE

Name of the party: - Dr. Satish B Bandgar

Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Seasum indicum (Seed sample)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with macroscopic and organoleptic characters. We hereby authenticate that the sample belongs to seeds of *Sesamum orientale* L. {= *Sesamum indicum* L.}(Family – Pedaliaceae)

This certificate is issued at his request and is given only for the academic use.

Upadhye
(A.S. Upadhye)

Scientist
Plant Drug Authentication Service
Botany Group
Plant Sciences Division

Auth.11-163.

आगरकर पथ, पुणे – 411 004, भारत, दूरभाष : (020) 2567 8916/17/18, 2565 4357, 2565 3680 फॅक्स : (020) 2565 1542
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November 2, 2011

AUTHENTICATION CERTIFICATE

Name of the party: - Dr. Satish B Bandgar

Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Glycerzia glabra (root and stem pieces)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with macroscopic and microscopic characters. We hereby authenticate that the sample belongs to stem and roots pieces of *Glycyrrhiza glabra* L. (Family - Fabaceae).

This certificate is issued at his request and is given only for the academic use.

(A.S. Upadhye)

Scientist
Plant Drug Authentication Service
Botany Group
Plant Sciences Division

Auth.11-164

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November 2, 2011

AUTHENTICATION CERTIFICATE

Name of the party: - Dr. Satish B Bandgar

Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Barbaris aristata (stem pieces)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with macroscopic and microscopic characters. We hereby authenticate that the sample belongs to stem pieces of *Berberis aristata* DC. (Family- Berberidaceae).

This certificate is issued at his request and is given only for the academic use.

(A.S. Upadhye)

Scientist
Plant Drug Authentication Service
Botany Group
Plant Sciences Division

Auth.11-165.

आगरकर पथ, पुणे – 411 004, भारत, दूरभाष : (020) 2567 8916/17/18, 2565 4357, 2565 3680 फॅक्स : (020) 2565 1542
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Maharashtra Association for the Cultivation of Science
AGHARKAR RESEARCH INSTITUTE
(An Autonomous Grant-in-Aid Institute under the Department of Science and Technology, Govt. of India)

November 2, 2011

AUTHENTICATION CERTIFICATE

Name of the party: - Dr. Satish B Bandgar

Address: - Tilak Ayurvedic Mahavidyalaya, Rasta Peth, Pune 411 011

Reference: -Letter no. 564, dated 20/07/2011

Name of the sample: - Barbaris aristata (Flowering twig)

Sample size: - about 50g

Date of the receipt: - October 7, 2011

Report: -

The sample has been critically studied with taxonomical characters. We hereby authenticate that the sample belongs to *Berberis aristata* DC. (Family- Berberidaceae).

This certificate is issued at his request and is given only for the academic use.

Upadhye
(A.S. Upadhye)

Scientist
Plant Drug Authentication Service
Botany Group
Plant Sciences Division

Auth 11-166.

आगरकर पथ, पुणे – 411 004, भारत, दूरभाष : (020) 2567 8916/17/18, 2565 4357, 2565 3680 फॅक्स : (020) 2565 1542
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Web : www.aripune.org E-mail : arimacs@pn2.vsnl.net.in

ANNEXURE -II

Indian Drugs Research Association & Laboratory

561-B, Shivajinagar, Behind Congress Bhavan Lane, Pune - 411 005.
☎ : 25534018 / 25537875 E-mail : idralpune@gmail.com

Ref. No. Report No. 276 Date 1-3-2012

CERTIFICATE OF ANALYSIS
CONFIDENTIAL

Name of the Party	Dr. Satish B. Bandgar, Shankar-Niwas,Plot No.18, Off-'Ausa-Road,Latur, LATUR – 413 512.
Your Ref.No.	Dated 20-2-2012.
Type of the Sample.	Nimbadikalka.
Date of Receipt.	21-2-2012.
Batch No.	-
Quantity Received.	250 gms (app).

	Sample Drawn by Party
1 Description	Black Coloured Coarse, Sticky Powder with white coarse particles and characteristic odour.
2. pH (1%)	4.82
3. Fat %	26.858 %
4. Moisture	8.1928 %
5. Ash	11.557 %
6. Acid Insoluble Ash	0.8572 %
7. Water Soluble Extractive	30.93 %
8. Alcohol Soluble Extractive.	28.92 %
9. Sugar %	82 %

For I.D.R.A.& L.Pune.

ANNEXURE – III

Patient Information And Concent Sheet

You are being invited to participate in a research study. Before you take part in this research study, the study must be explained to you and you must be given the chance to ask questions. Please read carefully the information provided here. If you agree to participate, please sign the informed consent form. You will be given a copy of this document to take home with you

- **Protocol Title:**

  Study of Vrana Shodhan and Ropan Karma of Nimbadi Kalka on Chronic Diabetic Ulcer.

- **Principal investigator:** Dr. Satish B. Bandgar. (M.D. Ayurved)

  Mob. No.+91 9822071595 **E-mail** –satishbandgar76@gmail.com

- **Purpose of the research study:**

You are being invited to participate in a research work for Study of Vrana Shodhan and Ropan Karma of Nimbadi Kalka on Chronic Diabetic Ulcer. You are selected as a possible subject in this study because you are suffering from Chronic Diabetic Ulcer. The study drugs are being practiced since ancient times they are also mentioned in Ayurvedic classics for the treatment of Chronic Diabetic Ulcer. We hope that your participation will help us to generate data on its efficacy in treating Chronic Diabetic Ulcer. This study will recruit 260 patients of Chronic Diabetic Ulcer over a period of 3 year.

Any samples of tissues, blood or body fluids obtained during this study will be stored and analyzed only for the purposes of this study.

When your participation in the study is over, you will no longer have access to Diabetic non-healing ulcer treatment, unless special additional arrangements are made by the Principal Investigator.

- **Study procedures:**

If you agree to take part in this study, you will receive either of two treatments on your willingness. I.e. Nimbadi Kalka or Iodine, H_2O_2 for local ulcer dressing care.

Needed debridement will be done as per wound condition. Leg elevation, rest, offloading techniques etc. will be advice to you during treatment period.

If you agree to take part in this study, you will be asked to admit in hospital. Your participation in the study will last for maximum 60 days. Your ulcer local care is done daily with either by Nimbadi Kalka or Iodine, H_2O_2 for maximum 60 days.

- **Investigation:**

Blood & Urine – Hemogram, D.C., ESR, BSL, KFT, LFT, Urine routine and microscopic, etc.

- **Your responsibilities in this study:**

If you agree to participate in this study, you should follow.
Inform the Principal Investigator as soon as possible about any side effects that you may have encountered.
Be prepared to stay in hospital during said treatment period.
Follow all instructions given by doctor.

- **Withdrawal from study:**

You are free to withdraw your consent and discontinue your participation at any time without prejudice to you or effect on your medical care. If you decide to stop taking part in this study, you should tell the Principal Investigator.

Your doctor, the Principal Investigator of this study may stop your participation in the study at any time for one or more of the following reasons:

If you experience Serious Adverse Events.
Failure to follow the instructions of the Principal Investigator and/or study staff.
The Principal Investigator decides that continuing your participation could be harmful.
You need treatment not allowed in the study.
The study is cancelled.
Other administrative reasons.
Unanticipated circumstances.

- **Possible risks, discomforts and inconveniences:**

There are no known risks, discomforts and inconveniences associated with this research study.

- **Potential benefits:**

If you participate in this trial you may expect to benefit of relieving your suffering. There is no assurance you will benefit from this study. However, your participation may contribute to the medical knowledge about the use of these medicines in the management of Diabetic non-healing ulcers.

- **Subject's rights:**

Your participation in this study is entirely voluntary. Your questions will be answered clearly and to your satisfaction.

In the event of any new information becoming available that may be relevant to your willingness to continue in this study, you or your legal representative will be informed in a timely manner by the Principal Investigator.

- **Confidentiality of study and medical records:**

Information collected for this study will be kept confidential. Your records, to the extent of the applicable laws and regulations, will not be made publicly available. Only your Investigator will have access to the confidential information being collected.

However, Regulatory Agencies, Institution Ethics Committee will be granted direct access to your original medical records to check study procedures and data, without making any of your information public. By signing the Informed Consent Form attached, you or your legal representative is authorizing such access to your study and medical records.

Data collected and entered into the Case Report Forms are the property of Institution. In the event of any publication regarding this study, your identity will remain confidential.

- **Costs of participation:**

If you take part in this study, you will have to pay for the investigations if required.

- **Research related injury and compensation:**

The Hospital does not make any provisions to compensate trial subjects for research related injury. However, compensation may be considered on a case-by-case basis for unexpected injuries cause by the negligence of study team.

By signing this consent form, you will not waive any of your legal rights or release the parties involved in this study from liability for negligence.

- **For contact if you have queries:**

If you have questions about this research study and your rights or in the case of any injuries during this study, you may contact the Principal Investigator **Dr. Satish B. Bandgar** MD Ayurved Mob. No.09822071595 E-mail –satishbandgar76@gmail.com

If you have questions about the study or your rights as a participant, you can call the Institutional Ethics Committee which is the committee that reviewed and approved this study, the telephone number is **Dr.B.S. Nagoba** Ph. 9423075786, working hours 10 am to 5 pm.

Informed consent form
Details of research study:
Protocol title: Study of Vrana Shodhan and Ropan Karma of Nimbadi Kalka on Chronic Diabetic Ulcer. **Principal investigator:** Dr. Satish B. Bandgar. MD Ayurved **Mob. No:** 09822071595 **E-mail**–satishbandgar76@gmail.com
Subject's particulars
Name : **Address :** **Age :** **Sex :** **Date of Birth :**

Contact No. :

Part – I to be filled by patient

I, _____

(Name of patient)

agree to participate in the research study as described and on the terms set out in the Patient Information Sheet. The nature of my participation in the proposed research study has been explained to me in

_____ by Dr**. Satish B. Bandgar**

(Language) (Name of Principal Investigator)

I have fully discussed and understood the purpose and procedures of this study. I have been given the Patient Information Sheet and the opportunity to ask questions about this study and have received satisfactory answers and information.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reasons and without my medical care being affected.

I also give permission for information in my medical records to be used for research. In any event of publication, I understand that this information will not bear my name or other identifiers and that due care will be taken to preserve the confidentiality of this information.

_____

[Signature / Thumb print (Right / Left) of patient] (Date of signing)

Part – II to be filled by parent / legal guardian, where ever applicable

I, _____

(parent / legal guardian) hereby give consent for the above patient to participate in the proposed research study. The nature, risks and benefits of the study have been explained clearly to me and I fully understand them.

_____

[Signature/Thumbprint (Right / Left) of parent /legal guardian] (Date of signing)

Witnessed by :

(Name of witness) (Designation of witness)

(Name of witness) (Designation of witness)

Part Iv– Investigator's Statement

I, the undersigned, certify to the best of my knowledge that the patient legally acceptable representative signing this informed consent form had the study fully explained and clearly understands the nature, risks and benefits of his/her word's &participation in the study.

Dr. Satish B. Bandgar

Name of Investigator Signature Date

ANNEXURE - IV

MAEER PUNE'S
MAHARASHTRA INSTITUTE OF MEDICAL SCIENCES AND RESEARCH, LATUR INDIA
(MEDICAL COLLEGE & HOSPITAL)
ISO 9001 : 2000 Recognised
ETHICS COMMITTEE
MIMSR
Vishwanathpuram, Ambajogai Road, Latur - 413 531, INDIA
Tel : + 91-02382-227424, 227028
Fax : +91-02382-227246, 228939
email : mimsr@mitpune.com
website : www.mimsr.net

Ref :

REPORT OF ETHICS COMMITTEE

Date:

Reference No. MIMSR/EC/ 13/2011

Date: 30/04/2011

To,

Dr. Satish B. Bandgar

Department of Ayurveda,

MIMSR Medical College, Latur.

Subject: Regarding your research proposal Entitled "To study the Shodhan and Ropan Karma of Nimabadi Kalka on Chronic Diabetes Ulcer"

Ref:- :- Your letter dated 12/04/2011

Dear Dr. Bandgar,

The above mentioned research proposal was discussed in the Ethics Committee meeting held on 29/04/2011 at our college.

Ethical Committee has unanimously approved your research proposal. You are hereby suggested/advised to report any changes in the study protocol to the ethical committee.

ETHICS COMMITTEE LATUR M.I.M.S.R. LATUR

Dr. B. S. Nagoba,

Secretary, Ethics Committee

MIMSR Medical College, Latur

Secretary
ETHICS COMMITTEE
M.I.M.S.R. Medical College
LATUR - 413 531

ANNEXURE -V

Case Record Form (CRF)

- **Title of the Research Project**

To study the shodhan and Ropan karma of Nimbadi kalka on chronic diabetic Ulcers.

Date:

1. Name:

2. Address:

3. Age/Gender: **4. OPD NO:**

5. IPD NO:

6. Occupation:

7. Present complaints:

8. OPD of ulcer:

When was it is notice:
How doses ulcer starts:
Cause of ulcer:
Treatment taken for ulcer:
Outcome of treatment:

9. Family history:

10. Past medical history:

DM with treatment:
HTN
Previous ulcer: Surgery:
Trauma: Amputation:
Allergies: Immune compress:
Autoimmune: Other:

11. Habits:

Alcohol: Tobacco:Smoking:

12. Food:

13. General examination:

Conscious/semiconscious/unconscious

Pulse: /min

BP: / mm of Hg

Temp:

Anemia: Edema: Icterus:

Skin: ENT:

Toung: Nutrition:

Stool: Urine:

Sleep: Appetite:

14. Systemic examination:

CVS:

S1: S2:

Peripheral pulse:

CRT: Local temp:

R/S:

P/A:

CNS:

Sensory:

Motor:

Autonomous:

Feet examination;

Shape: Deformity:

Callosity:

15. Ulcer examinations:

Location:

Shape:

Size:

Depth:

16. Pain: 1---10 Grade scale

17. Surrounding skin:

Normal/Edematous/Erythematic/Scaling/Exzematic/Dark colour/Maceration

18. Margin of Ulcer:

Slopping/Punched out/Everted/

19. Ulcer bed:

Colour: Black/Yellow/Pink/Red/

20. Necrotic tissue:

21. Granulation:

22. Ulcer discharge: Pus/Serous/Hemosangionus

23. Odour:

24. Maggots: Yes/No

25. Ulcer grade:

26. Investigations:

Heamogram with CBC:

BSL: Fasting: mg/dl pp: mg/dl

Urine Routine and Microscopic:

Culture report of ulcer:

Others:

Name & Signature of Doctor:

Dr. SATISH B. BANDGAR

M.D. (Ayurved)

ANNEXURE – VI

Diabetic Ulcer grades classification

According to Wagner's & Armstrong University of Texas:

Grades	0	1	2	3	4	5
A)	Callus or Scar	Superficial Infection	Wound up to Tendon/Capsule	Penetrating to Bone /Joint	Necrotic foot	Entire foot Necrosis
B)	Ischemia	Ischemia	Ischemia	Ischemia	Ischemia	Ischemia
C)	Infection	Infection	Infection	Infection	Infection	Infection
D)	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection	Ischemia + Infection

II) RNAO Guideline Development Panel, 2005

Non limb threatening ulcer Grade-I	**limb threatening ulcer Grade-II**	**limb threatening ulcer Grade-III**
Superficial infection	**Pain (in a previously insensate foot)**	**Systemic infection**
Bright red granulation tissue	Deep wound infection	In addition to deep wound infection: Fever
Non-healing	Erythematic (> 2 cm)	Rigors
Friable and exuberant granulation	Wound breakdown	Hypotension
New areas of breakdown or necrosis	Increased size or satellite areas	Multi-organ failure
Increased exudates	Undermining or tunneling	
Bridging of soft tissue	Probing to bone	
Foul odour	Flu-like symptoms	
	Anorexia	

ANNEXURE – VII

Key to Master-chart Variable description and data coding

1. Sr.No.: Serial Number

2. Group: A= Ayurvedic treatment group, B=Allopathic treatment group

3. Occupation: W= Worker, H =Housewife, S=Sedentary work

4. Gender: M=Male, F= Female

5. Age : Age in years

6. Chronicity: Chronicity of ulcer in weeks

7. Hop.stay: Hospital stay in days

8. Part involved: Ulcer part involved U=Upper limb ,
L=Lower limb,
G= Gluteal area

9. Initial Ulcer grde: Initial ulcer grade on 1 day According to its classification.

1= Ulcer grade 1

2= Ulcer grade 2

10. BSL F: Blood sugar level fasting mg/dl

11. BSL PP: Blood sugar level post prandial mg/dl

12. Ulcer Size:

i. **Day1 SZ:** Ulcer size on 1 day in square centimeter
ii. **Day20 SZ:** Ulcer size on 20 day in square centimeter
iii. **Day40 SZ:** Ulcer size on 40 day in square centimeter
iv. **Day60 SZ:** Ulcer size on 60 day in square centimetre

13. Grading of Ulcer size:

1-20 sq. cm = 1 grade

21-40 sq. cm = 2 grade

41-60 sq. cm = 3 grade

>61 sq. cm = 4 grade

14. Ulcer Shape

i. **Day 1 SP:** Ulcer shape on 1 day
ii. **Day 20 SP:** Ulcer shape on 20 day
iii. **Day 40 SP:** Ulcer shape on 40 day
iv. **Day 60 SP:** Ulcer shape on 60 day

15. Grading of shape

O = Oval shape

C = Circular

I = Irregular

16. Ulcer Bed:

i. **DAY 1 BD:** Day 1 ulcer bed
ii. **DAY 20 BD:** Day 20 ulcer bed
iii. **DAY 40 BD:** Day 40 ulcer bed
iv. **DAY 60 BD:** Day 60 ulcer bed

17. Grading of Ulcer bed colour:

Pink colour=1 Grade

Ischemic =2 Grade

Black colour = 3 Grade

Yellow/Green= 4 Grade

18. Granulation:

i. **Day 1 GR :** Granulation on 1 day
ii. **Day 20 GR :** Granulation on 20 day
iii. **Day 40 GR :** Granulation on 40 day
iv. **Day 60 GR :** Granulation on 60 day

17. Grading of granulation tissue colour:

Pink colour = 1 Grade

Red Ischemic = 2 Grade

Absent = 3 grade

Over granulation = 4 Grade

18. Secretion:

i. **Day 1 SEC:** Secretion on 1 day
ii. **Day 20 SEC:** Secretion on 20 day
iii. **Day 40 SEC:** Secretion on 40 day
iv. **Day 60 SEC:** Secretion on 60 day

19. Grading of secretion:

No secretion = 1Grade

Serus secretion = 2 Grade

Hemosangionus =3 Grade

Pus secretion = 4 Grade

20. Pain:

i. **DAY 1 PN**: Day 1 pain

ii. **DAY 20 PN**: Day 20 pain

iii. **DAY 40 PN**: Day 40 pain

iv. **DAY 60 PN**: Day 60 pain

21. Grading of pain on pain scale:

Pain 0-1= 1 grade (No pain)

2-4= 2 Grade (Mild pain)

5-7= 3 Grade pain (Moderate pain)

8-10= 4 Grade pain (Sever pain).

22. Smell:

i. **DAY 1 SML:** Smell of ulcer on 1 day

ii. **DAY 20 SML:** Smell of ulcer on 20 day

iii. **DAY 40 SML:** Smell of ulcer on 40 day

iv. **DAY 60 SML:** Smell of ulcer on 60 day

23. Grading of smell:

No smell = 1 Grade

Mild smell = 2 Grade

Intolerable smell =3 Grade

Foul smell = 4 Grade

24 Healing outcome:

E = Complete Epithelization

C = Ulcer contracture but no Epithelization

Min A = Minor Amputation

Maj A = Major Amputation

D = Death of Patient

ANNEXURE-VIII MASTER CHART

SR. NO	Name	Group	Occu-pation	Gender	AgeYrs	Chronicty	Hospi-talstay	Partinolved	Ini Ulcergrd	BSLF	BSLPP	DAY1SZ	DAY20SZ	DAY40SZ	DAY60SZ	DAY1SP	DAY20SP	DAY40SP	DAY60SP	DAY1BD	DAY20BD	DAY40BD	DAY60BD	DAY1GR	DAY20GR	DAY40GR	DAY60GR	DAY1SEC	DAY20SEC	DAY40SEC	DAY60SEC	DAY1PN	DAY20PN	DAY40PN	DAY60PN	DAY1SML	DAY20SML	DAY40SML	DAY60SML	HEALING OUTCOME
1	UTTAM RAMBHAU JADHAV	A	W	M	65	16	25	L	1	132	165	28-II	1	0	0	O	O	0	0	2	1	0	0	2	1	0	0	2	0	0	0	2	1	0	0	2	1	0	0	E
2	VISHWANTH KISAN WAGHMARE	A	W	M	52	11	32	L	1	90	145	32-II	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	3	2	1	0	3	2	0	0	E
3	KESHAV NAGNATH GAIKWAD	A	W	M	63	19	40	L	1	82	128	42-III	2	1	0	C	O	0	0	4	2	1	0	2	2	1	0	4	3	1	0	2	1	1	0	2	2	1	0	E
4	JAMUNABAI SUDAM SHINDE	A	H	F	54	12	38	L	1	94	134	38-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
5	MAHATAB BADUL PATHAN	A	S	M	48	7	14	U	1	81	124	17-I	1	0	0	I	O	0	0	4	1	0	0	2	1	0	0	1	0	0	0	1	1	0	0	1	1	0	0	E
6	SUBHAS ILKANT DHAKANE	A	W	M	52	16	29	L	1	96	142	24-II	1	0	0	O	O	0	0	2	1	0	0	2	1	0	0	3	1	0	0	2	1	0	0	2	1	0	0	E
7	LAXMAN PIRAJI GHODAKE	A	W	M	68	18	37	L	1	108	144	46-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
8	JOTIRAM LAXMAN NIKAM	A	W	M	72	30	58	L	2	115	190	66-IV	4	3	2	I	I	I	O	4	4	2	1	2	2	2	1	4	4	3	1	4	3	2	1	4	3	2	1	C
9	LAXMIBAI VITTHAL PATIL	A	H	F	56	12	34	L	1	118	167	56-III	2	1	0	O	O	O	0	3	2	1	0	2	1	1	0	4	2	1	0	3	2	1	0	2	1	1	0	E
10	HUNAMANT RAVJI NALWADE	A	W	M	65	18	39	L	1	107	152	52-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	2	1	0	2	1	1	0	E
11	KASHIBAI VISHWATH MANE	A	H	F	58	7	32	U	1	82	148	18-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
12	VAIJANATH VENKATRAO BADURE	A	W	M	68	20	50	L	2	130	210	58-III	3	2	1	C	C	O	O	4	4	3	1	2	2	1	1	4	4	2	2	3	3	2	1	4	3	2	2	C
13	RAJABHAU KASHINATH PANDIT	A	W	M	69	15	27	L	1	82	148	30-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
14	MUKTABAI VISHWANTH KENDRE	A	H	F	61	15	22	L	1	107	138	11-I	1	1	0	C	C	0	0	2	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	E
15	UTTAM SHIDRAM BHOSLE	A	W	M	71	19	53	L	2	150	209	69-IV	3	2	1	I	I	I	I	4	2	1	1	2	2	1	1	4	3	2	1	3	2	1	1	3	2	1	1	E
16	NIVRUTTTI ASHRUBA GHULE	A	W	M	67	14	39	L	1	124	168	44-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	2	1	0	2	2	1	0	E
17	BHIMRAO MARUTI SONTAKKE	A	W	M	73	15	50	L	2	138	212	50-III	3	2	1	I	I	I	I	4	4	2	1	2	2	1	1	4	3	2	1	3	2	1	1	4	3	2	1	C
18	LAXMIBAI BABURAO KENDRE	A	W	F	56	18	28	L	1	88	152	34-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	2	1	1	0	E
19	SUSHANT MAHATO	A	W	M	68	14	40	L	1	68	118	41-III	2	1	0	C	C	C	0	2	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
20	GOROBA EKNATH SITAPURE	A	S	M	49	9	28	U	2	76	132	16-I	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	1	1	1	0	E
21	RAMKISHAN SHYAM PHAD	A	W	M	67	18	52	L	2	132	186	48-III	2	1	1	O	O	C	C	4	2	1	1	2	1	1	0	4	3	1	1	3	2	1	1	3	2	2	1	E
22	UTTAM KINDIBA HADBE	A	W	M	74	26	54	L	2	112	192	46-III	2	1	1	C	I	C	C	4	2	1	1	2	2	1	1	4	2	2	1	2	2	1	1	2	2	1	1	E
23	ASHOK MANIKRAO PANCHAL	A	W	M	64	18	36	L	1	122	168	50-III	2	1	0	O	O	O	0	3	1	1	0	2	1	1	0	3	2	1	0	3	2	1	0	3	2	1	0	E
24	KANDABAI GOVIND MORLWAR	A	H	F	54	14	30	L	1	96	146	36-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	4	1	1	0	2	1	1	0	3	1	1	0	E
25	LAXMIBAI SHIVRAM PHAD	A	H	F	58	13	28	L	1	77	127	25-II	1	1	0	O	C	C	0	4	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	2	1	1	0	E
26	BHANUDAS LIMBAHI LATPATE	A	W	M	61	17	23	L	1	108	146	29-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	2	1	1	0	E
27	NASAHEB BALBHIM KALE	A	S	M	49	6	12	G	1	80	138	12-I	1	0	0	O	O	0	0	2	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
28	KALUBAI SAKHARAM KENDRE	A	W	F	75	27	57	L	2	124	210	58-III	3	2	1	C	I	O	O	3	2	1	1	2	1	1	1	4	2	1	1	4	3	2	1	4	3	2	1	C
29	RAVSAHEB NARHARIRAO HANDE	A	W	M	66	18	29	L	1	78	132	30-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
30	MURLIDHAR DNYNOBA SURVASE	A	W	M	68	20	54	L	1	186	240	59-III	3	2	1	C	O	O	O	4	2	1	1	2	2	1	1	4	4	2	1	3	2	1	1	4	3	2	1	E
31	VAIJANATH GURUBASAPPA PATIL	A	W	M	52	19	40	L	1	121	178	51-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	3	1	1	0	E
32	SUGRABAI EKNATH SURVASE	A	H	F	58	14	38	L	1	92	138	31-II	1	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
33	NARHARI LAXMAN DAHIPALE	A	W	M	70	20	31	L	1	81	139	37-II	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
34	FULAJI MARUTI SAGAR	A	W	M	44	16	18	U	1	92	142	17-I	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
35	KISAN DNYNOBA MANE	A	S	M	71	24	57	L	2	142	254	64-IV	3	2	1	I	I	O	O	4	3	1	1	2	2	1	1	4	3	2	1	4	3	2	1	4	3	2	1	C
36	HARIBA MARUTI BIRAJDAR	A	W	M	54	14	22	L	1	108	149	28-II	1	1	0	C	C	O	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
37	ANJJANBAI DATTU NARAYANKAR	A	H	F	68	16	28	L	1	67	152	37-II	1	1	0	C	C	O	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
38	SANTOSH DURGADAS VISHNA	A	W	M	57	15	30	L	1	92	168	30-II	1	1	0	C	C	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
39	SHEELABAI AJPAL PATIL	A	H	F	64	8	21	U	1	117	179	18-I	1	1	0	O	C	O	0	4	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	1	1	1	0	E
40	BAHURAO DAJIBA JAGTAP	A	W	M	72	27	55	L	2	112	196	46-III	3	2	1	O	O	O	O	3	2	2	1	2	2	1	1	4	4	3	1	3	2	1	1	4	3	2	1	C
41	RAJENDA SHIVAJI JADHAV	A	W	M	67	17	22	L	1	72	126	34-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	2	1	1	0	E
42	FAIMA ELAHI SHIKH	A	H	F	58	13	35	L	1	68	176	37-II	1	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	4	1	0	3	2	1	0	2	1	1	0	E
43	MAHDAVRAO SONERAO DESHMUKH	A	S	M	66	18	38	L	1	108	159	30-II	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	3	1	0	1	1	1	0	1	1	1	0	E
44	SHALEN DATTARYA UJLAMBE	A	H	F	54	9	24	L	1	96	174	20-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	1	1	1	0	2	1	1	0	2	1	1	0	E
45	PANDURANG HARIBA SHINDE	A	W	M	46	8	27	L	1	124	186	30-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
46	GONDERAO GOVINDRAO SONPHULE	A	W	M	68	19	51	L	2	130	198	60-III	3	2	1	I	I	I	I	4	4	2	1	2	2	2	1	4	4	3	2	3	2	2	1	3	3	2	1	C
47	RAGHVENDRA ASHOK BHOSLE	A	W	M	73	30	48	G	2	109	168	56-III	3	2	1	I	I	I	I	3	2	2	1	2	2	1	1	4	4	2	1	3	2	2	1	3	2	2	1	E
48	GANGADHAR T SHINDE	A	W	M	64	20	36	L	1	86	162	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	3	1	0	3	2	1	0	2	2	1	0	E
49	SANGEETBAI B KARAD	A	H	F	52	12	38	L	1	103	179	46-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	3	1	0	2	2	1	0	2	2	1	0	E
50	RAJABHAU J NIKAM	A	W	M	62	17	40	L	1	121	208	48-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
51	VITHOBA S CHITTADE	A	W	M	65	19	29	L	1	130	168	52-III	2	1	0	C	C	0	0	4	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
52	SANGITA B KAMBLE	A	H	F	68	11	21	L	1	108	146	18-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	E
53	ABHIMANYU N KOLI	A	W	M	56	15	25	L	1	118	164	39-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
54	JANARDHAN P MUNDE	A	S	M	74	20	57	L	2	136	210	64-IV	3	2	1	I	I	I	I	4	3	2	2	2	2	2	1	4	4	2	2	4	3	2	1	4	3	3	2	C
55	RAM K CHAVAN	A	W	M	70	20	50	L	2	104	176	58-III	3	2	1	O	I	C	C	4	3	2	1	2	2	1	1	4	3	2	1	3	2	1	1	3	2	2	1	E
56	DADASAHEB L GAVALI	A	W	M	66	14	29	L	1	114	144	40-II	2	1	0	O	O	O	0	4	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
57	GANGUBAI K THKUR	A	H	F	47	9	16	U	1	78	136	12-I	1	0	0	C	O	0	0	2	1	0	0	2	1	0	0	3	1	0	0	2	1	0	0	2	1	0	0	E
58	BHAGWAT N DONGRE	A	W	M	65	17	53	L	2	96	206	50-III	2	2	1	O	O	O	O	3	3	2	1	2	2	1	1	4	4	1	1	3	2	2	1	4	3	2	1	E
59	GOPAL V BURANDE	A	W	M	65	11	38	L	1	91	138	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
60	RAMKISHAN D DAPKAR	A	W	M	63	19	33	L	1	102	146	47-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
61	ANNAPURNA K RAJE	A	W	F	59	11	31	L	1	85	165	32-II	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	4	1	0	2	1	1	0	2	1	1	0	E
62	GANPAT K CHILLE	A	W	M	75	15	55	L	2	122	196	54-III	3	2	1	I	I	I	O	4	4	2	1	2	2	1	1	4	4	2	1	3	2	1	1	4	3	2	1	E
63	DHANAJI R MULE	A	W	M	61	16	32	L	1	92	144	59-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
64	BHUBURAO B KAMBLE	A	W	M	55	13	34	L	1	59	168	50-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	1	1	0	3	2	1	0	E
65	LALITABAI G THOMBRE	A	W	F	60	16	21	L	1	117	164	27-II	1	1	0	C	O	O	0	2	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	3	1	1	0	E
66	RAMDEV D PRASAD	A	W	M	59	13	24	L	1	98	159	36-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	3	1	1	0	E
67	BABRUVAN H SHINDE	A	W	M	75	24	55	L	2	142	219	65-IV	3	2	1	I	I	I	I	4	4	3	1	2	2	1	1	4	3	2	1	4	3	2	1	4	3	2	1	C
68	MANGAL P SUGARE	A	H	F	55	8	27	L	1	87	142	26-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	2	1	0	3	2	1	0	2	1	1	0	E
69	PRAKASH C SELKAR	A	W	M	43	10	18	U	1	104	146	17-I	1	0	0	C	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
70	SURYAKANT S JADHAV	A	W	M	64	11	32	L	1	74	122	30-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	1	1	0	3	2	1	0	E
71	SANTOSH B GAIKWAD	A	S	M	66	16	36	L	1	110	160	43-III	2	1	0	C	O	O	0	3	2	1	0	2	2	1	0	4	3	1	0	2	1	1	0	2	2	1	0	E
72	VISHWANATH M BIRAJDAR	A	W	M	54	15	30	L	1	114	152	38-II	1	1	0	C	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	3	2	1	0	2	1	1	0	E
73	LATIKABAI N MOTADE	A	H	F	67	16	39	L	1	120	162	49-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	2	2	1	0	2	1	1	0	E
74	DATTATRYA R CHAME	A	W	M	68	14	33	L	1	118	152	32-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	3	2	1	0	3	2	1	0	E
75	ANNARAO N PATIL	A	W	M	61	30	60	L	2	170	268	80-IV	4	3	2	I	I	I	I	4	4	4	2	2	2	2	2	4	4	4	3	4	3	2	2	4	3	2	2	C
76	MAMATHAPPA G SHETE	A	W	M	53	13	27	L	1	94	142	22-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	0	0	E
77	MAHLING M LENKIBALY	A	S	M	63	14	24	L	1	86	138	27-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
78	RAKAMABAI S NEHAR	A	H	F	74	17	51	L	2	119	198	52-III	3	2	1	C	O	O	O	3	3	2	1	2	2	2	1	4	4	3	1	2	2	2	1	3	2	2	1	E
79	ASLAM N SHIKH	A	W	M	42	18	19	U	1	96	138	14-I	1	0	0	C	C	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
80	DNYANOBA V MAGAR	A	W	M	64	13	28	L	1	122	176	21-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	1	1	0	3	2	1	0	2	1	1	0	E
81	JANAK J WAKURE	A	W	M	65	19	36	L	1	108	157	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	2	1	1	0	3	2	1	0	E
82	RADHABAI M GADE	A	H	F	58	20	38	L	1	126	186	42-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	3	1	0	2	1	1	0	2	1	1	0	E
83	SATYAWAN S GITTE	A	W	M	75	24	56	L	2	109	268	46-III	2	2	1	C	O	O	O	4	4	2	1	2	2	1	1	4	4	2	1	2	2	2	1	4	3	2	1	E
84	BASAWARAJ S SWAMI	A	W	M	66	16	40	L	1	94	142	48-III	2	1	0	O	O	O	0	4	3	1	0	2	2	1	0	4	2	1	0	2	2	1	0	3	2	1	0	E
85	ADIKA B BHARADE	A	H	F	67	12	31	G	1	118	152	22-II	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	2	1	0	3	1	1	0	2	1	1	0	E
86	BHAGWAN B JOSHI	A	W	M	45	16	36	L	1	126	170	19-I	1	1	0	C	O	O	0	4	2	1	0	2	2	1	0	4	3	1	0	2	1	1	0	2	1	1	0	E
87	SHIVAJI S SURYAWANSHI	A	S	M	60	15	37	L	1	108	142	50-III	2	1	0	O	`O	O	0	3	2	1	0	2	2	1	0	4	4	1	0	2	1	1	0	2	1	1	0	E
88	VIDYAVATI V ALGUDE	A	H	F	68	14	26	L	1	92	138	33-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	2	1	0	3	2	1	0	2	2	1	0	E
89	DILIP R BHALERAO	A	W	M	61	17	50	L	2	84	179	52-III	3	2	1	C	O	O	O	4	4	3	1	2	2	1	1	4	4	2	1	2	2	2	1	4	3	2	1	E
90	DNYAOBA B SOMVANSHI	A	W	M	73	15	28	L	1	98	146	37-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	3	2	1	0	2	2	1	0	E
91	PANDURANG P MUNDE	A	W	M	60	18	40	L	1	108	142	60-III	2	1	0	O	O	O	0	4	4	1	0	2	1	1	0	4	2	1	0	2	2	1	0	3	2	1	0	E
92	USHA M KEDAR	A	H	F	70	7	22	U	1	112	138	20-I	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	1	1	1	0	E
93	BHIMRAO M GAIKWAD	A	W	M	64	19	34	L	1	92	138	41-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	4	1	0	2	2	1	0	2	2	1	0	E
94	PRABHU R GAIKWAD	A	W	M	70	20	37	L	1	111	126	21-II	1	1	0	O	O	O	0	2	2	1	0	2	2	1	0	4	4	1	0	3	2	1	0	2	2	1	0	E
95	RUKMINBAI S MUNDE	A	W	F	72	17	48	L	2	127	182	47-III	2	1	1	I	I	I	O	4	4	3	1	2	2	1	1	4	4	2	1	2	2	2	1	3	3	2	1	E
96	NARAYAN H CHIGALE	A	W	M	68	9	27	L	1	109	122	15-I	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	E

ANNEXURE-VIII MASTER CHART

SR. NO	Name	Group	Occu-pation	Gender	AgeYrs	Chronicty	Hospi-talstay	Partinolved	Ini Ulcergrd	BSLF	BSLPP	DAY1SZ	DAY20SZ	DAY40SZ	DAY60SZ	DAY1SP	DAY20SP	DAY40SP	DAY60SP	DAY1BD	DAY20BD	DAY40BD	DAY60BD	DAY1GR	DAY20GR	DAY40GR	DAY60GR	DAY1SEC	DAY20SEC	DAY40SEC	DAY60SEC	DAY1PN	DAY20PN	DAY40PN	DAY60PN	DAY1SML	DAY20SML	DAY40SML	DAY60SML	HEALING OUTCOME
97	NARAYAN P JADHAV	A	W	M	69	18	53	L	2	98	168	49-III	3	2	2	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	2	2	2	1	3	3	2	2	C
98	SUJANBAI P SHIRSAT	A	W	F	62	11	25	L	1	68	110	35-II	1	1	0	I	I	I	0	4	1	1	0	2	1	1	0	4	1	1	0	3	1	1	0	2	1	1	0	E
99	ANGAD A PATIL	A	W	M	67	19	57	L	2	127	236	50-III	3	2	2	I	I	I	I	4	4	4	2	2	2	2	2	4	4	4	3	3	3	2	2	4	4	3	3	C
100	SARAJABAI N GARULE	A	H	F	75	24	55	U	2	114	282	64-IV	3	3	3	I	I	I	I	4	4	4	4	3	3	3	3	4	4	4	4	4	4	4	3	3	4	4	3	4	Maj A
101	GOPIRAJ B SURYABANSHI	A	W	M	68	12	25	L	1	108	126	26-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
102	PRALHAD S INDRALE	A	W	M	44	16	29	L	1	117	149	39-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	3	2	1	0	E
103	CHANDRAKANT G JAMODAR	A	W	M	65	16	35	L	1	104	168	42-III	2	1	0	C	C	O	0	4	2	1	0	2	1	1	0	4	3	1	0	2	2	1	0	2	2	1	0	E
104	ADNYNBAI N SHINDE	A	S	F	52	14	15	U	1	82	127	12-I	1	0	0	O	O	0	0	4	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
105	SHIVAJI G YARULE	A	S	M	71	17	40	L	2	117	162	71-IV	4	3	0	I	I	I	0	2	2	2	0	2	2	0	0	4	4	4	0	3	3	2	0	2	2	2	0	C
106	DONDIRAM E CHAMLE	A	W	M	67	18	36	L	1	113	160	51-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
107	MUKUND S PAWAR	A	W	M	43	15	32	L	1	102	143	57-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	3	2	1	0	E
108	CHAYA D LOMTE	A	H	F	54	19	28	L	1	92	164	32-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
109	AYYUB H PATHAN	A	W	M	74	25	38	L	2	76	121	68-IV	2	1	0	I	I	I	0	3	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
110	NAGNATH U CHAPKE	A	W	M	61	20	23	L	1	83	133	18-I	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	1	1	1	0	E
111	ABASAHEB N WAGHMARE	A	S	M	72	15	38	L	1	117	169	58-III	2	1	0	O	O	O	0	2	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
112	SHIVKANYA M MALWAD	A	W	F	57	11	31	L	1	108	138	33-II	1	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
113	RAVSAHEB V BHISE	A	W	M	41	16	40	L	1	90	133	60-III	2	1	0	O	O	O	0	2	1	1	0	2	1	1	0	4	2	1	0	3	2	1	0	3	1	1	0	E
114	SAMBHAJI B NARSALE	A	W	M	72	17	42	L	1	80	120	47-III	2	1	1	O	O	O	O	4	2	1	1	2	2	1	1	4	2	1	1	2	2	1	1	2	2	1	1	E
115	NAGARBAI B JADHAV	A	H	F	58	14	30	L	1	111	146	42-III	2	1	0	C	C	C	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	2	1	0	E
116	MOHAN K KAMBLE	A	W	M	63	18	37	L	1	76	139	47-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
117	DNYNOBA S RAJE	A	W	M	73	16	40	G	1	140	208	41-III	2	1	0	C	C	C	0	2	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
118	BEBEYSAYARA S SHIKH	A	W	M	57	19	24	L	1	104	126	28-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	E
119	GANGADHAR K AYCHIT	A	W	M	41	20	29	L	1	82	142	22-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
120	BAPURAO S ALANE	A	S	M	75	15	21	U	1	72	126	15-I	1	1	0	C	C	C	O	4	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	1	1	1	0	E
121	SAROJA R DHOKE	A	H	F	51	17	29	L	1	104	146	34-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
122	RAMCHANDRA V GUNJALE	A	W	M	73	16	36	L	1	110	154	49-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
123	RATNAPPA G DHUMAL	A	W	M	67	17	38	L	1	118	162	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
124	MANGALBAI R MASKE	A	H	F	57	15	35	L	1	102	154	41-III	2	1	0	O	O	O	0	4	1	1	0	2	1	1	0	4	2	1	1	3	2	1	0	2	2	1	0	E
125	MARUTI V GHANDURE	A	W	M	74	18	37	L	1	111	147	49-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	2	2	1	0	E
126	SUBHASH S BIRADAR	A	W	M	66	19	39	L	1	122	168	61-IV	2	1	0	I	I	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
127	SHIKH B S	A	W	F	58	13	51	L	1	153	203	73-IV	3	3	2	I	I	I	I	4	4	4	3	2	2	2	2	4	4	4	3	3	3	3	2	2	2	2	1	C
128	DHONDIBA G SURYAVANSHI	A	W	M	71	20	33	L	1	86	136	29-II	1	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	3	2	1	0	E
129	VITTHAL S BOINWAD	A	W	M	52	16	56	L	1	134	265	57-III	3	3	3	I	I	I	I	4	4	4	4	2	2	2	2	4	4	4	4	3	3	3	2	4	4	4	3	Min A
130	KAMALBAI J VIDHUR	A	W	F	73	15	60	L	2	86	280	76-IV	4	4	4	I	I	I	I	4	4	4	4	3	3	3	3	4	4	4	4	4	4	4	4	4	4	4	4	Maj A
131	DESHMUKH A TUKARAM	B	S	M	75	24	51	L	2	146	251	64-IV	3	3	2	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	3	3	3	2	4	4	4	3	C
132	KENDRE V D	B	S	F	74	29	56	L	2	160	218	79-IV	4	3	2	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	3	3	3	3	4	4	4	3	C
133	UTTAM D KOLI	B	W	M	44	8	18	L	1	98	126	11-I	1	0	0	O	O	0	0	2	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
134	NAMDEV G DUVAGE	B	W	M	47	12	20	U	1	76	138	14-I	1	0	0	O	O	0	0	2	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
135	SUNDARBAI N CHAVAN	B	S	F	70	23	36	L	1	104	148	43-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
136	CHANDRAKAT N MAGAR	B	W	M	64	17	40	L	1	118	152	47-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	3	1	0	2	2	1	0	3	2	1	0	E
137	TUKARAM B MANE	B	S	M	56	14	38	L	1	76	127	54-III	2	1	0	O	O	O	0	3	2	1	0	2	2	1	0	4	2	1	0	2	2	1	0	3	2	1	0	E
138	HARUBAI S RAJMANE	B	S	F	65	15	27	L	1	92	136	24-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
139	BHABU M SHIKH	B	W	M	73	18	34	L	1	84	142	38-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
140	SHANKAR T GUDEWAR	B	W	M	58	13	39	L	1	80	137	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	4	1	0	3	2	1	0	3	2	1	0	E
141	SUKSALBAI M KSHIRSAGAR	B	S	F	66	15	26	L	1	76	127	21-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
142	VAIJNATH B KAMBLE	B	W	M	59	19	31	L	1	92	146	29-II	2	1	0	C	C	O	0	4	2	1	0	2	1	1	0	3	2	2	0	2	1	1	0	2	1	1	0	E
143	GUNALE S G	B	S	F	75	30	59	G	2	142	208	72-IV	4	4	3	I	I	I	I	4	4	4	4	2	2	2	2	4	4	4	4	4	3	3	3	4	4	4	3	C
144	KASHINATH K PHAD	B	W	M	67	20	26	L	1	117	162	32-II	2	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
145	ASHA P KULKARNI	B	S	F	68	16	26	L	1	76	96	39-II	2	1	0	C	C	O	0	4	2	1	0	2	1	1	0	2	2	1	0	2	1	1	0	2	2	1	0	E
146	SHRIMANT G SUTAR	B	W	M	60	13	26	U	1	104	144	22-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
147	KUSUM A YAMGAR	B	H	F	54	12	22	U	1	110	156	13-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	1	1	1	0	E
148	MATHURABAI G PAWAR	B	H	F	72	17	48	L	2	138	182	43-III	2	1	1	O	O	O	O	3	2	2	1	2	2	1	1	4	4	2	1	2	2	1	1	3	2	1	1	E
149	HARICHANDRA G PAWAR	B	S	M	69	19	36	L	1	82	136	42-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
150	ABU B SHIKH	B	S	M	70	20	39	L	1	71	127	29-II	2	1	0	C	C	C	0	4	2	1	0	2	1	1	0	4	3	1	0	3	2	1	0	3	2	1	0	E
151	SARASVATI B DATAL	B	W	F	55	14	39	L	1	99	130	53-III	2	1	0	C	C	O	0	4	2	1	0	2	1	1	0	4	3	1	0	3	2	1	0	2	2	1	0	E
152	VAIJABAI M BHENDE	B	H	F	73	15	47	L	2	141	184	58-III	2	2	1	O	O	O	O	4	2	2	1	2	2	1	1	4	3	2	1	2	1	1	1	2	1	1	1	E
153	SHANTA G DHUMAL	B	W	F	68	11	17	L	1	77	136	37-II	1	0	0	C	C	0	0	4	1	0	0	2	1	0	0	3	1	0	0	2	1	0	0	2	1	0	0	E
154	TUKARAM B DEVKAR	B	W	M	53	15	38	L	1	122	154	43-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	3	2	1	0	E
155	HAMUMANT G CHINALE	B	W	M	70	17	31	L	1	107	138	36-II	2	1	0	I	I	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
156	SUNITA D PARATBONE	B	H	F	74	19	57	L	2	130	214	60-III	3	2	2	O	O	O	O	4	4	4	3	2	2	2	2	4	4	4	3	3	3	2	2	2	2	2	2	C
157	SHIVHAR M AGHAV	B	W	M	51	11	22	U	1	111	142	34-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
158	RAVSAHEB M SHYAMGIRE	B	W	M	57	14	26	L	1	100	146	25-II	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
159	MUKUND K KALE	B	W	M	70	20	56	L	2	118	202	57-III	2	1	1	O	O	O	O	4	2	1	1	2	2	1	1	4	3	2	1	3	2	1	1	2	2	1	1	E
160	CHANDRAKANT N MAGAR	B	W	M	58	13	28	L	1	78	99	32-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	1	1	0	E
161	PRALHAD M UTAGE	B	W	M	69	15	39	L	1	115	154	40-II	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
162	JOTIRAM L NIKAM	B	S	M	74	24	57	L	2	127	218	82-IV	4	4	4	I	I	I	I	4	4	4	4	2	2	2	2	4	4	4	4	4	4	4	3	4	4	4	4	Min A
163	DADARAO L SAGAR	B	W	M	71	19	49	L	2	104	164	42-III	2	2	2	I	I	I	I	4	4	4	3	2	2	2	2	4	4	4	4	4	4	4	4	4	4	4	4	C
164	TUKARM B MANE	B	S	M	74	29	52	L	2	128	162	68-IV	3	3	2	I	I	I	I	3	3	2	2	2	2	2	2	4	4	4	3	4	4	4	3	4	4	4	4	C
165	HANMANT S KOPARKAR	B	S	M	75	24	48	L	2	136	204	81-IV	4	4	3	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	4	4	3	3	4	4	3	3	C
166	HARUBAI H RAJE	B	H	F	70	20	38	U	1	76	117	42-III	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	4	3	2	0	2	2	1	0	3	2	1	0	E
167	BALASHAEB V JADHAV	B	W	M	46	7	13	L	1	82	129	9-I	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	11	0	0	1	1	0	0	E
168	KISAN G MANE	B	S	F	73	19	43	U	2	117	158	27-II	2	2	1	C	C	C	C	4	4	2	1	2	2	1	1	2	2	2	1	3	2	2	1	2	2	1	1	E
169	RAJARAM S MANE	B	W	M	50	15	27	L	1	102	146	22-II	1	1	0	O	O	O	0	4	2	1	0	2	2	1	0	2	2	1	0	2	2	1	0	2	2	1	0	E
170	ANTIKA G POTBHARE	B	S	F	68	12	22	L	1	108	152	27-II	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
171	UDHAV O BHURE	B	W	M	64	14	29	L	1	92	136	20-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	2	1	1	0	E
172	SAKSHI J GUNALE	B	W	F	51	10	22	L	1	86	118	12-I	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
173	VIDYA G JAGTAP	B	W	F	53	14	30	L	1	121	149	24-II	1	1	0	C	C	C	0	4	2	1	0	2	2	1	0	2	2	1	0	2	2	1	0	2	2	1	0	E
174	KESHAV N GAIKWAD	B	W	M	67	11	28	L	1	69	118	11-I	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	2	1	0	1	1	1	0	1	1	1	0	E
175	RAMA B HIPPARGE	B	W	M	66	13	33	L	1	82	147	34-II	2	1	0	O	O	O	0	2	2	1	0	2	1	1	0	3	2	1	0	2	2	1	0	3	2	1	0	E
176	KODABAI G MORLAWAR	B	W	F	54	17	34	L	1	108	152	21-II	1	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	3	2	1	0	E
177	JAMNBAI S SHINDE	B	S	F	72	25	50	L	2	114	176	71-IV	3	2	1	I	I	I	I	4	4	2	1	2	2	1	1	4	4	3	1	3	3	2	1	4	3	2	1	E
178	RAMRAO S BIRADAR	B	S	M	75	22	55	L	2	137	182	64-IV	2	2	1	I	I	I	I	3	3	2	1	2	2	1	1	4	3	2	1	3	2	1	1	4	3	2	1	E
179	HARUN B SHIKH	B	W	F	55	18	40	L	1	120	147	33-II	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	3	2	1	0	3	2	1	0	2	2	1	0	E
180	VISHWNATH S SHANDASHIRE	B	W	M	63	15	33	L	1	119	162	28-II	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	3	2	0	2	1	1	0	2	1	1	0	E
181	VIMALABAI A BANSUDE	B	W	F	66	10	16	L	1	79	154	26-II	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	2	1	0	0	2	1	0	0	2	1	0	0	E
182	SHIDRAM R KAJLE	B	W	M	56	14	26	L	1	84	126	37-II	2	1	0	O	O	O	0	2	1	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
183	BHANUDAS L LATPATE	B	S	M	72	20	48	G	2	98	159	39-II	2	2	1	C	C	C	C	4	4	4	2	2	2	2	2	4	4	4	4	3	3	3	2	3	3	2	2	C
184	GAYABAI B GAIKWAD	B	H	F	74	19	54	L	2	121	182	48-III	3	2	2	O	O	O	O	4	4	4	3	2	2	2	2	4	4	4	4	3	3	3	2	3	3	3	2	C
185	ASHOK U SONAWANE	B	W	M	57	15	39	L	1	97	154	24-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	4	1	0	2	2	1	0	2	2	1	0	E
186	SANJAY M VANGATE	B	W	M	62	11	14	L	1	82	127	46-III	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	2	1	0	0	1	1	0	0	E
187	SHRINIVAS R KULKARNI	B	W	M	69	14	29	L	1	103	140	44-III	2	1	0	C	C	C	0	4	1	1	0	2	1	1	0	4	2	1	0	2	2	1	0	2	1	1	0	E
188	DHONDABAI G CHANDE	B	W	F	58	16	37	L	1	111	152	42-III	2	1	0	C	C	C	0	4	2	1	0	2	2	1	0	4	2	1	0	3	2	1	0	3	2	1	0	E
189	BHAGA B SOMVANSHI	B	H	F	72	19	31	L	1	97	136	50-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
190	RAJABHAU K PANDIT	B	S	M	70	20	22	L	1	109	153	44-III	1	1	0	O	O	O	0	4	1	1	0	2	1	1	0	3	1	1	0	2	1	1	0	2	1	1	0	E
191	BHIMRAO M SONTAKKE	B	W	M	60	17	27	L	1	117	146	47-III	1	1	0	C	C	C	0	4	1	1	0	2	1	1	0	3	2	1	0	2	2	1	0	2	1	1	0	E
192	NILESH L BHOSLE	B	S	M	74	29	59	U	2	109	206	52-III	3	3	3	O	O	O	O	4	4	4	4	2	2	2	2	4	4	4	4	3	3	3	3	3	3	3	3	Min A
193	UTTAM K HADBE	B	S	M	73	18	29	L	1	99	151	59-III	2	1	0	C	C	C	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E

ANNEXURE-VIII MASTER CHART

SR. NO	Name	Group	Occu-pation	Gender	AgeYrs	Chronicty	Hospi-talstay	Partinolved	Ini Ulcergrd	BSLF	BSLPP	DAY1SZ	DAY20SZ	DAY40SZ	DAY60SZ	DAY1SP	DAY20SP	DAY40SP	DAY60SP	DAY1BD	DAY20BD	DAY40BD	DAY60BD	DAY1GR	DAY20GR	DAY40GR	DAY60GR	DAY1SEC	DAY20SEC	DAY40SEC	DAY60SEC	DAY1PN	DAY20PN	DAY40PN	DAY60PN	DAY1SML	DAY20SML	DAY40SML	DAY60SML	HEALING OUTCOME
194	DIGAMBAR G KULKARNI	B	W	M	60	15	34	L	1	117	168	27-II	1	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
195	UTTAM R MANE	B	S	M	75	20	55	U	2	128	218	47-III	3	3	2	C	C	C	C	4	4	4	3	2	2	2	2	4	4	4	4	4	4	3	3	4	4	3	3	C
196	MUKIND R RATHOD	B	W	M	60	12	23	L	1	105	153	46-III	2	1	0	C	C	C	0	4	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
197	HAJI H SAYYAD	B	W	M	47	10	26	L	1	112	152	22-II	1	1	0	O	O	C	0	2	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
198	SINDHU M ANDLE	B	H	F	75	24	47	L	2	117	156	72-IV	4	3	3	I	I	I	I	4	4	4	4	2	2	2	2	4	4	4	4	4	4	3	3	3	3	3	3	C
199	BAPURAO P JAGTAP	B	S	M	74	30	56	L	2	138	219	68-IV	4	4	4	I	I	I	I	4	4	4	4	2	2	2	2	4	4	4	4	4	4	4	4	4	4	4	4	Min A
200	PRAKASH C SELKAR	B	W	M	49	5	12	U	1	114	142	4-I	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
201	SUMITRA R NARWADE	B	W	F	66	14	38	L	1	117	157	57-III	2	1	0	C	C	C	O	4	2	1	0	2	2	1	0	4	3	1	0	2	2	1	0	2	1	1	0	E
202	LAXMIBAI A KAMBLE	B	W	F	68	15	35	L	1	105	136	53-III	2	1	0	O	O	O	0	4	4	1	0	2	2	1	0	4	4	1	0	2	2	1	0	3	2	1	0	E
203	RAMAKANT S GUDE	B	W	M	56	11	14	U	1	94	140	12-II	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
204	KAVITA P RAJE	B	S	F	70	17	32	L	1	81	162	24-II	1	1	0	C	C	C	0	4	2	1	0	2	1	1	0	3	2	1	0	3	2	1	0	2	2	1	0	E
205	BAPURAO K MADANE	B	W	M	69	16	29	L	1	111	154	32-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
206	VISHWESHWAR N BAPTE	B	W	M	58	15	27	L	1	108	133	28-II	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
207	BANU S SHIKH	B	H	F	70	19	42	L	2	109	176	65-IV	2	1	1	I	I	I	I	3	2	2	1	2	2	1	1	4	4	2	1	3	2	1	1	4	3	2	1	E
208	JAARDHAN M INGALE	B	S	M	73	18	51	L	2	131	209	47-III	3	2	2	I	I	I	I	4	4	4	3	2	2	2	2	4	4	4	3	2	2	2	2	2	2	2	2	C
209	GANAPAT K TAWADE	B	W	M	55	14	24	L	1	117	128	27-II	1	1	0	C	C	C	0	4	2	1	0	2	1	1	0	2	2	1	0	2	1	1	0	2	1	1	0	E
210	GARAKH S GULBILE	B	S	M	74	21	43	L	2	128	176	44-III	2	1	1	C	C	C	C	4	2	2	1	2	2	1	1	4	4	1	1	3	2	1	1	3	2	1	1	E
211	FULABAI S SHINDE	B	W	F	54	13	21	U	1	132	151	42-III	1	1	0	O	O	O	0	4	2	1	0	2	1	1	0	2	2	1	0	2	1	1	0	2	1	1	0	E
212	NAJEERSAHEB M SAYYED	B	W	M	65	15	27	I	1	84	133	34-II	1	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
213	SEEMANTABAI R DAHIGARE	B	W	F	57	12	30	L	1	97	121	41-III	2	1	0	O	O	O	0	4	2	1	0	2	2	1	0	4	3	1	0	3	2	1	0	2	1	1	0	E
214	MANOHAR V SURYAVANSHI	B	S	M	73	29	41	L	2	119	172	57-III	3	2	2	I	I	I	I	3	3	2	2	2	2	2	2	4	4	4	3	3	3	2	2	4	3	3	2	C
215	KANTABAI M MANE	B	S	F	74	25	54	L	2	98	221	67-IV	3	3	2	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	4	3	2	2	4	3	3	2	C
216	SONJI M INGALE	B	W	M	55	16	31	L	1	129	148	44-III	2	1	0	C	C	C	0	4	2	2	0	2	2	1	0	4	4	1	0	2	1	1	0	3	2	1	0	E
217	KANTA S CHAME	B	S	F	69	12	26	L	1	134	154	30-II	1	1	0	O	O	O	0	2	2	1	0	2	1	1	0	3	2	1	0	2	2	1	0	2	2	1	0	E
218	NARAYA H CHIGALE	B	W	M	60	17	24	L	1	130	166	32-II	1	1	0	C	C	C	0	4	2	1	0	2	1	1	0	2	2	1	0	2	2	1	0	2	1	1	0	E
219	SADIPAN Y KAMBLE	B	S	M	70	15	29	L	1	107	141	29-II	2	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	2	0	2	2	1	0	2	2	1	0	E
220	GANGABAI R SAVTE	B	S	F	70	16	30	L	1	92	128	43-III	2	1	0	C	C	C	0	4	2	2	0	2	2	1	0	4	2	2	0	2	2	1	0	2	2	1	0	E
221	RAJKUMAR S DAKE	B	W	M	60	13	24	L	1	131	191	34-II	2	1	0	O	O	O	0	4	2	2	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
222	RADHABAI S JAKKALWAD	B	W	F	55	10	13	U	1	79	124	7-I	1	0	0	O	O	0	0	4	1	0	0	2	1	0	0	2	1	0	0	2	1	0	0	1	1	0	0	E
223	MANGAL G GADE	B	W	F	54	11	22	L	1	87	118	20-I	1	1	0	O	O	O	0	2	1	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
224	GANI K TOWAR	B	S	M	71	15	24	U	1	91	126	27-II	1	1	0	O	O	O	0	4	2	1	0	2	2	1	0	2	2	1	0	2	2	1	0	2	1	0	0	E
225	JAGANATH K KAGALE	B	W	M	60	18	45	L	2	128	166	60-III	2	2	2	I	I	I	I	4	4	4	3	2	2	2	2	4	4	4	4	3	3	3	2	3	3	2	2	C
226	MUKUND P PAWAR	B	S	M	73	15	31	L	1	117	153	36-II	1	1	0	C	C	C	0	4	2	2	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
227	KJAKHAN S PATHAN	B	S	M	74	17	49	L	2	153	187	51-III	2	2	1	I	I	I	I	4	4	2	2	2	2	1	1	4	4	3	1	2	2	1	1	3	2	1	1	E
228	RAVINDRA S SATPUTE	B	W	M	62	16	36	L	1	121	168	43-III	2	1	0	O	O	O	0	4	4	1	0	2	2	1	0	4	4	2	0	3	2	1	0	3	2	1	0	E
229	MOHAN P PAWAR	B	W	M	64	15	31	L	1	104	142	33-II	2	1	0	O	O	O	0	4	2	2	0	2	1	1	0	3	3	1	0	2	2	1	0	2	2	1	0	E
230	MINAKSHI A ADE	B	S	F	74	19	55	L	2	132	211	68-IV	4	4	4	I	I	I	I	3	3	3	3	3	3	3	3	4	4	4	4	4	4	4	4	4	4	4	4	Maj A
231	GANGA V GAIKWAD	B	W	F	47	11	14	U	1	81	136	7-I	1	0	0	O	O	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
232	KAVITA P RATHOD	B	H	F	75	30	49	L	2	109	168	81-IV	4	3	3	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	4	3	2	3	4	4	3	3	C
233	RAJU S DOKE	B	W	M	45	10	13	U	1	82	136	9-I	1	0	0	O	O	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	E
234	DILIP S GOLKUNDE	B	W	M	56	12	22	U	1	102	129	22-II	1	1	0	C	C	C	0	2	1	1	0	2	2	1	0	2	2	1	0	2	1	1	0	2	1	1	0	E
235	RAMAKANT M PAWAR	B	W	M	66	19	29	L	1	118	162	26-II	1	1	0	O	O	O	0	4	2	1	0	2	1	1	0	3	2	1	0	2	2	1	0	2	1	1	0	E
236	BHIMA S DEVKAR	B	W	M	67	17	42	L	1	124	157	46-III	3	2	2	C	C	C	C	3	3	2	2	2	2	2	3	3	3	3	2	2	2	2	3	3	2	2	C
237	NAYUM A SHIKH	B	W	M	57	18	31	L	1	83	144	28-II	2	1	0	O	O	O	0	4	2	1	0	4	2	1	0	4	3	1	0	3	2	1	0	2	2	1	0	E
238	LALUSINGH G CHITOD	B	S	M	67	16	47	L	1	97	182	52-III	3	2	2	O	O	O	O	4	4	4	2	2	2	2	2	3	3	2	2	2	2	2	2	4	3	2	2	C
239	SAROJA R DAKE	B	S	F	69	17	42	L	1	103	161	56-III	3	2	2	O	O	O	O	4	4	4	2	2	2	2	2	4	4	4	3	3	3	2	2	3	3	2	2	C
240	RAMAKANT M PAWAR	B	W	M	58	13	34	L	1	110	163	44-III	2	1	0	C	C	C	0	4	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	3	2	1	0	E
241	P L KODPHUGE	B	H	F	68	15	51	L	2	127	210	66-IV	3	2	2	I	I	I	I	4	4	4	3	2	2	2	2	4	4	4	3	3	3	2	2	4	3	2	2	C
242	ANWAR I BAGWAN	B	W	M	59	14	38	L	1	76	136	47-III	2	1	0	O	O	O	0	4	2	2	0	2	2	1	0	3	2	1	0	2	1	1	0	3	1	1	0	E
243	DHONDIBA G SURYAVASHI	B	W	M	55	15	36	L	1	85	142	49-III	2	1	0	O	O	O	0	4	4	2	0	2	1	1	0	4	4	1	0	3	2	1	0	2	1	1	0	E
244	SIMANTHA P JADHAV	B	H	F	69	19	40	L	1	94	153	42-III	2	1	1	C	C	C	C	4	4	2	2	2	2	1	1	3	3	2	1	2	2	1	1	2	2	1	1	E
245	ISMIL R SHIKH	B	W	M	60	15	31	L	1	103	158	24-II	2	1	0	O	O	O	0	4	2	2	0	2	2	1	0	3	3	1	0	2	1	1	0	2	1	1	0	E
246	SUBHASH S GARJE	B	S	M	70	20	49	L	2	112	154	71IV	4	3	2	I	I	I	I	4	4	3	2	2	2	2	2	4	4	4	3	3	2	2	1	4	4	3	3	C
247	S B NARHARE	B	W	M	59	12	27	L	1	121	168	30-II	2	1	0	C	C	C	0	4	2	2	0	2	2	1	0	4	3	1	0	3	2	1	0	2	2	1	0	E
248	ASHOK J PANCHAL	B	W	M	58	14	29	L	1	68	129	24-II	1	1	0	O	O	O	0	4	2	2	0	2	1	1	0	4	4	1	0	3	2	1	0	2	1	1	0	E
249	UMAKANT S BAHETI	B	W	M	65	15	24	L	1	79	136	21-II	1	1	0	C	C	C	0	2	2	1	0	2	1	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
250	SUGADHA C CHAWAN	B	W	F	56	15	27	L	1	104	165	29-II	1	1	0	O	O	O	0	4	2	2	0	2	2	1	0	3	2	1	0	2	1	1	0	2	1	1	0	E
251	NARAYAN R PATEL	B	S	M	67	18	32	L	1	118	178	32-II	2	1	0	O	O	O	0	4	2	2	0	2	2	1	0	4	4	1	0	3	2	1	0	2	2	1	0	E
252	DATTU M KADAM	B	W	M	54	13	21	U	1	110	161	22-II	1	1	0	O	O	O	0	2	2	1	0	4	2	1	0	2	2	1	0	1	1	1	0	2	1	1	0	E
253	BAPURAO D GAIKWAD	B	W	M	69	15	28	L	1	121	192	47-III	2	1	0	C	C	C	0	4	2	2	0	2	2	1	0	3	3	1	0	2	2	1	0	2	1	1	0	E
254	MUKIDA G SWAMI	B	W	M	67	17	34	L	1	117	210	42-III	2	1	0	C	C	C	0	4	4	2	0	2	2	1	0	4	4	1	0	3	2	1	0	2	1	1	0	E
255	SUVARNA D MULE	B	H	F	70	19	39	L	1	93	117	32-II	2	1	0	O	O	O	0	4	2	2	0	2	2	1	0	3	3	1	0	3	2	1	0	3	2	1	0	E
256	GONGADHAR T SHINDE	B	W	M	55	13	30	L	1	109	149	31-II	2	1	0	O	O	O	0	4	2	2	0	4	2	1	0	4	3	1	0	2	1	1	0	2	1	1	0	E
257	SEETABAI S PURI	B	W	F	59	15	31	L	1	117	151	51-III	2	1	0	O	O	O	0	4	2	2	0	2	2	1	0	4	3	1	0	3	2	1	0	2	1	1	0	E
258	SHRIRANG S KALE	B	W	M	56	15	27	L	1	105	142	27-II	1	1	0	C	C	C	0	4	2	2	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E
259	YASHODABAI B UTGE	B	S	F	73	24	45	L	2	121	162	56-III	3	2	2	I	I	I	I	4	3	2	2	2	2	2	2	4	4	4	3	4	3	2	2	4	3	2	2	C
260	MANGAL J GORULE	B	W	F	49	12	21	U	1	76	124	8-I	1	1	0	O	O	O	0	2	2	1	0	2	1	1	0	4	2	1	0	2	1	1	0	2	1	1	0	E