“THE AETIOPATHOLOGICAL STUDY OF CHRONIC RENAL FAILURE WITH AYURVEDA PERSPECTIVE”

The Thesis of Ph.D. (Ayurveda)

In Subject of Kayachikitsa

Submitted by

Vd. Mrs. Swarupa M. Bhujbal

Under the Guidance of

Dr. Sadanand P. Sardeshmukh

A.V.P., Ph. D. (Ayu.)

Faculty of Ayurveda

TILAK MAHARASHTRA VIDYAPEETH, PUNE – 37

Duration

(September 2009 to March 2012)

DECLARATION

*I hereby declare that the thesis entitled "**The Aetiopathological Study Of Chronic Renal Failure With Ayurveda Perspective**" Completed and written by me has not previously formed the basis for the award of any degree or other similar title of this or any other University or examining body.*

Vd. Mrs. Swarupa M. Bhujbal

Ph. D. Scholar

Place:

Date:

Certificate

This is to certify that the thesis entitled, ***"The Aetiopathological Study of Chronic Renal Failure with Ayurveda Perspective"*** *which is being submitted herewith for the award of the Degree of Vidyavachaspati (Ph. D.) in Ayurveda of Tilak Maharashtra Vidyapeeth, Pune is the result of original research work completed by*

Vd. Mrs. Swarupa M. Bhujbal

Under my supervision and guidance. To the best of my knowledge and belief the work incorporated in this thesis has not formed the basis for the award of any Degree or similar title of this or any other University or examining body.

Place:

Date:

Dr. Sadanand P. Sardeshmukh
A.V.P., Ph. D. (Ayu.)
Name of the Guide

ACKNOWLEDGEMENT

I have great pleasure to acknowledge rather expressing my deep gratitude to those who have helped me in completing my dissertation work in different ways. I acknowledge sincerely though it is a formality but I think I must convey my regards to those who have helped me in this task of research work.

To me research or dissertation as truth finding or to see God face to face this I realized after observing the experience of Dr. S. P. Sardeshmukh who believes that service to man is to service to God. The inspiration behind research work is Dr. S. P. Sardeshmukh.

The immense service rendered by Kidney Care Clinic namely Dr. Abhay Sadre & his team; he supported research work by providing patients along with diagnostic analysis for further study. In the same way, Mr. Fathechand Ranka & Dr. Ramesh Ranka who equally took keen interest and gave me freehand for work in Saras Dialysis Centre, Pune.

I am grateful to Dr. S. M. Sathe and Dr. A. B. Dharmadhikari for their valuable suggestions.

I express my gratituded to Dr. Abhijit joshi for his constant moral support to complete the work in time.

I am thankful to Dr. Pankaj Wanjarkhedkar for his suggestions and encouragement throughout my research work. With great respect, I thank Dr. Mukund Sabanis for his valuable guidance.

I would like to thanks Dr.Mrs. Kanade for her excellence expertise in the field of statistics for research work.

I take an opportunity to thank my husband Dr.Milind Bhujbal who has been a constant pillar like support in completing my dissertation work. I thank my kids Arya & Shreeshail who allowed me to burn my midnight oil throughout my completion of dissertation. I am grateful to my parents-in-law, parents and members of the family.

I really thank Mr. Sachin Kadam who meticulously helped me in converting my data.

I would like to convey thanks Mr.Shailesh Patnakar, Mrs.Shruti Patnakar, Mrs.Jyostana Kher and Mrs.Nilima Mukherjee

I convey my gratitude to my colleagues Dr. Mrunalini Kulkarni, Dr.Abhijeet Saraf, Dr. Thorave, Dr. Kotashtane, Dr. Meena Sonawane and Dr. Mrs. Savitri Vasudevan for their kind co-operation. I would like to convey thanks to my student Dr. Bobade and Dr. Nerli for their help. I am grateful towards my friends for encouragement Mrs.Keskar, Mrs.Tupe and Miss Jadhav.

Finally yet importantly, I would like to convey my thanks to Mrs. Pathak, Mr. More and Mr. Gujar for their constant help.

Vd. Mrs. Swarupa M. Bhujbal

Research Scholar

INDEX

LIST OF ABBRIVATIONS

Texts

च सं	Ch. Sa.	Charak Samhita (चरक संहिता)
सु सं	Su. Sa.	Sushrut Samhits (सुश्रूत संहिता)
अ ह्र	Ah. Hr.	Ashtang Hridayam (अष्टांग ह्रदयम्)
अ सं	Ah. Sa.	Ashtang Sangraha (अष्टांग संग्रह)
भा प्र	Bh. P.	Bhavprakesh (भावप्रकाश)
यो र	Y. R.	Yogaratnakar (योगरत्नाकार)
मा नि	Ma. Ni.	Madhav Nidana (माधवनिदान)
शा सं	Sh. Sa.	Sharangdhara Samhita (शारंगधर संहिता)
भे सं	Bh. Sa.	Bhel Samhita (भेल संहिता)
ह सं	Ha. Sa.	Harita Samhita (हरीत संहिता)
रा नि	Ra. Ni.	Raj Nighantu (राज निघंठु)

Topics

सू स्था	Su.	Sutrasthana (सुत्र स्थान)
नि स्था	Ni.	Nidanasthana (निदान स्थान)
वि स्था	vi.	Vimansthana (विमान स्थान)
शा स्था	Sa.	Sharirasthana (शारिर स्थान)
चि स्था	Chi.	Chkitsasthana (चिकित्सा स्थान)

Statistical Details

1 Madhur, Lavana, Amala & Staging
2 Agni & Stage
3 Matra & Stage
4 Koshtha & Stage
5 Vidhi & Stage
6 Agni , Stage & Virya
7 Agni , Stage & Paka
8 Asyasukha & Stage
9 DM & Stage
10 HT & Stage
11 Anemia & Stage
12 Anemia , Stage , Anannabhilasha & Daurbalya
13 Anemia , Stage , Anannabhilasha & Shotha
14 DM , Stage , Anannabhilasha & Shotha
15 Anemia , Stage , Daurbalya & Shotha
16 DM , Stage , Daurbalya & Shotha
17 HT , Stage , Daurbalya & Prandusti
18 Anemia , Stage , Anannabhilasha & Chhardi
19 Anemia , Lavan & Amla
20 Amla , Lavan & Twakdry
21 Mamsarasa , Agni & Stage
22 Mamsarasa , Urine Protein & Stage
23 Mamsarasa & Urine Protein
24 Amla , Lavan & Muscle tone
25 Abhishyandi & mutra day frequency
26 Diwaswap & Cortico Medullar Differentiation
27 Mutra day frequency & USG
28 Chhardi & Anannabhilasha
29 Chhardi , Anannabhilasha & Anemia
30 Chhardi , Shotha & Abhishyandi
31 Chhardi , Anannabhilasha & Agnigp
32 Chhardi , Anannabhilasha & Abhyavaran Prakrut / Aprakrut
33 Addiction , Stage & Trushna
34 Diwaswap , Asyasukha & DM

Graphs Table Details

Flow Chart Details

1. Introduction:

Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiological processes associated with abnormal kidney function, and a progressive decline in glomerular filtration rate (GFR). Provides a widely accepted classification, based on recent guidelines of the National kidney foundation {Kidney Dialysis Outcome Quality Initiative (KDOQI)}, in which stages of CKD are defined according to the estimated GFR.

The term Chronic Renal Failure applies to the process of continuing significant irreversible reduction in Nephron number, and typically corresponds to CKD stages 3-5. The pathophysiological processes and adaptations associated with chronic renal failure will be the focus. The dispiriting term end-stage renal disease represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the uremic syndrome. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation.

It is important to identify factors that precipitate risk for CKD, even in individuals with normal GFR. Risk factors include hypertension, diabetes mellitus, autoimmune diseases, and older age, a family history of renal disease, a previous episode of acute renal failure, and the presence of proteinuria, abnormal urinary sediment, or structural abnormalities of the urinary tract.

CKD not only increases the mortality and morbidity due to its vascular complications resulting in cardiovascular- cerebrovascular events and CKD progression to end-stage kidney failure; but also because of its adverse impact on the economy of the country. [1]

This is now almost a global phenomenon and not restricted to India alone. Considering that prevalence of CKD in India is noted to be 13.8% which itself is very high; early detection, evaluation and preventive management will be the key to delay progression and to prevent adverse outcomes. In India ~ 90% patients cannot afford the cost. Over 1 million people worldwide live on dialysis or with a functioning graph shows incidence of CKD has doubled in the last 15 years.[2]

Epidemiology:-

Chronic kidney disease is a worldwide public health problem. There is a rising incidence and prevalence of kidney failure, with poor outcomes and high cost. There is an even higher prevalence of earlier stages of chronic kidney disease.

Increasing evidence, accrued in the past decades, indicates that the adverse outcomes of chronic kidney disease, such as kidney failure, cardiovascular disease, and premature death, can be prevent or delays. Earlier stages of chronic kidney disease can be detected through laboratory testing. Treatment of earlier stages of chronic kidney disease is effective in slowing the progression toward kidney failure. Initiation of treatment for cardiovascular risk factors at earlier stages of chronic kidney disease should be effective in reducing cardiovascular disease events both before and after the onset of kidney failure.[3]

Selection of Topic:

In Ayurveda practice, while treating CKD cases shows good result. In stage 1 to 3 disease may be reversed with regular Ayurveda treatment. The stage 4 and 5 which labeled as End Stage Kidney Disease, disease prognosis can be delayed with treatment all these better approach lead to CKD study.

While study CKD with Ayurveda it is must to understand CKD thoroughly means to understand the causes, lakashanas and samprapti. Ayurveda gives significant as appropriate knowledge of this disease. The fundamental principals related to Dinachrya, Rutucharya and the basic drugs are mentioned clearly in texts. Using all this principal the above disease can be treated properly.

The basic causes understanding in major aid for making awareness in society about the preventive tool those diseased with DM & HT. The awareness of kidney care can be possible with Ayurveda regime Dinacharya and Rutucharya modification in life style of the society.

Deceleration:-

While presenting the synopsis the title "**The Aetiopathological Study of Chronic Renal Failure with Ayurveda Perspective**" in 2009 but as per guidelines of National Kidney Foundation (USA), CRF term is change to CKD (Chronic Kidney Disease). The reason for changing name is given below.[4]

Nomenclature (Kidney Disease Outcome and Quality Initiative):-

The word ‘‘kidney’’ is of Middle English origin and is immediately understood by patients, their families, providers, health care professionals, and the lay public of native English speakers. On the other hand, ‘‘renal’’ and ‘‘nephrology,’’ derived from Latin and Greek roots, respectively, commonly require interpretation and explanation. The Work Group and the NKF are committed to communicating in language that can be widely understood, hence the preferential use of ‘‘kidney’’ throughout these guidelines.

National Kidney Foundation (USA) has formulated KDOQI (kidney disease outcome and quality initiative) guideline for all aspects of CKD. They have classified CKD in to stage 1- 5 depending on GFR level at detection. By applying these guidelines the term ‘chronic renal failure’ (CRF) has been removed and they get included in CKD. This has helped immensely to remove the fear and psychological impact on our CKD patients at large. However, it has not been able to sort out certain important major problems, particularly while trying to apply and adopt them to various ethnic populations including our own Indian population. KDOQI guidelines have staged the CKD based on GFR calculations done on estimations of creatinine clearance.[5]

2. Aim, Objective & Methodology

Aim:-

1. To study hetu, lakshana & samprapti of CRF with Ayurveda aspect.

Objective:-

1. To study 110 patients of CKD to understand Sroto dushti Causes, Lakashana and Samprapti.

Materials:

Well diagnosed patient of CKD

On the basis of hematological & biochemical as well as radiological tool

- CBC
- Blood Urea
- Sr. Creatinine
- Urine study
- Sonographical evidence
- Qualitative parameters as mentioned in case paper

Study Interest:-

1. The case study of 110 patients with different age group (Above 16 to 80), Gender, Occupation from Saras Dialysis Centre and Kidney Care Clinic, Pune were included in study.
2. Observation of different causes (hetu), symptoms and signs(lakashanas) and pathogenesis (samprapti)
3. To understand srotas involvement by distinguishing the observed hetus as well lakshana.
4. The statistical analysis of hetu, lakashanas for mentioning samprapti.

Sample Size:

- Number of patients – minimum 110 as per the rule of prevalence.
- Patients without dialysis & ongoing dialysis included.

Inclusion:

- Diabetes
- Hypertension
- Chronic Glomerular Nephritis
- Chronic Interstitial Nephritis
- Renal Vascular Sclerosis
- Vasculitis
- Nephrotic syndrome

Exclusion:

- Obstructive causes; urinary calculi, enlarged prostate
- Multiple myeloma
- Vesico urethral reflux
- Clinically non responding patients

Methodology:-

Hetu methodology: - The hetus are grouped into 1) *Aharia 2) Viharia 3) Manas 4) Viruddha*

Analysis will be

1. *Santarpan*
2. *Apatarpan*
3. *Vyadhi*
4. *Viruddha*

Ahara:-

Rasa: Taste

Madhur, (Sweet)

Amala (Sour)

Lavan (Salty)

Katu (Pungent)

Tikta (Belter)

Kashaya (Astrigent)

Vishamashana: - Irregular meal timing: - Present/Absent

Adhyashana: - Present / Absent

Paryusheet: - Stale food – Present/ Absent

Abhishyandi: - Present/ Absent

Vistambhi: - Present/ Absent

Mansa Ahara: - Present/ Absent

Pravar: -Brekfast /Lunch / Dinner- Excess

Avar: - Brekfast /Lunch/ Dinner - Less

Madhyam: - Brekfast /Lunch / Dinner - Moderate.

Viharia hetu methodology:-

1. *Exercise :* - Present/ Absent
2. *Nidra*: - Present/ Absent
3. *Diwaswap* : Present / Absent
4. *Jagaran*:- Present/ Absent
5. *Atap:*- Present/ Absent
6. *Upwas* :- Present/ Absent
7. *Rajasevana*:- Present/ Absent
8. *Bhashana*:- Present/ Absent
9. *Vegavrodha* :- Present/ Absent

Manas hetu methodology:-

1. *Krodha* – Present/ Absent
2. *Shoka*:- Present/ Absent

Viruddha hetu methodology:-

1. *Desh* :- Present/ Absent
2. *Kala* :- Present/ Absent
3. *Agni:*- Present/ Absent
4. *Matra* :- Present/ Absent
5. *Satmya viruddha*:- Present/ Absent
6. *Dosha Viruddha* :- Present/ Absent
7. *Sanskara*:- Present/ Absent
8. *Sanyog:*- Present/ Absent

9. *Virya viruddha*:- Present/ Absent
10. *Kostha viruddha*:- Present/ Absent
11. *Avastha viruddha*:- Present/ Absent
12. *Karma viruddha* :- Present/ Absent
13. *Parihara viruddha*:- Present/ Absent
14. *Pachana viruddha* :- Present/ Absent
15. *Paka viruddha* :- Present/ Absent
16. *Sanyog viruddha*:- Present/ Absent
17. *Hrud viruddha*:- Present/ Absent
18. *Sampat viruddha*:- Present/ Absent
19. *Vidhi*: - Present/ Absent.

Lakshana Methodology:-

1) *Srotodushti lakshana*
2) *Dosha lakshana*
3) *Dhatu dushti lakshana*
4) *Adhisthana lakshana*
5) *Avastha lakshana*
6) *Updrava lakshana*
7) *Bhahu dosha lakshana*

Samprapti methodology:-

Hetu and Lakshana correlation with *Adhisthana*

Thus, for above mentioned hetu lakshana references are as follows:-

i. अभ्यवरण - भोजनम् - सु. चि. २३ /४

ii. तृष्णा - सततं यः पिबेत वारि न तृप्तिमचिगच्छति । पुनः कांक्षति तोयं च तं तृष्णरितमदिशेत् ।। सु. ३४/३

iii. शोक - मनोभावः दुःखः । च. नि. ७/४
दैन्येनेन परीक्षयेत् । सु. चि. ३९१/४

iv. विरूध्द हेतु

यच्चापि देशकालाग्निमात्रासात्म्यानिलादिभिः। संस्कारतो वीर्यतश्च कोष्ठावस्थाक्रमैरपि ।।८६।।

परिहारोपचाराभ्यां पाकात् संयोगतोऽपि च। विरूद्धं तच्च न हितं हृत्संपद्विधिभिश्च यत् ।८७।।

विरूद्धं देशतस्तावद्रूक्षतीक्ष्णादि धन्वनि। आनूपे स्निग्धशीतादि भेषजं यन्निषेव्यते ।।८८।।

कालतोऽपि विरूद्धं यच्छीतरूक्षादिसेवनम् । शीत काले, तथोष्णे च कटुकोष्णादिसेवनम् ।।८९।।

विरूद्धमनले तद्वदन्नपानं चतुर्विधे। मधुसर्पिः समधृतं मात्रया तद्विरूध्यते ।।९०।।

कटुकोष्णादिसात्म्यस्य स्वादुशीतादिसेवनम् । यत्तत् सात्म्यविरूद्धं तु विरूद्धं त्वनिलादिभिः ।।९१।।

या समानगुणाभ्यास विरूद्धानौषधक्रिया । संस्कारतो विरूद्धं तद्यभोज्यं विषवभ्दवेत् ।।९२।।

एरण्डसीसकासक्तं शिखिमांसं यथैव हि । विरूद्धं वीर्यतो ज्ञेयं वीर्यतः शीतलात्मकम् ।।९३।।

तत् संयोज्योष्णवीर्येण द्रव्येण सह सेव्यते । क्रूरकोष्ठस्य चात्यल्पं मन्दवीर्यमभेदनम् ।।९४।।

मृदुकोष्ठस्य गुरू च भेदनीयं तथा बहु । एतत् कोष्ठविरूद्धं तु, विरूद्धं स्यादवस्थया।।९५।।

श्रमव्यवायव्यायामसक्तस्यानिलकोपनम्। निद्रालसस्यालसस्य भोजनं श्लेष्मकोपनम्।।९६।।

यच्चानुत्सृज्य विण्मुत्रं भुड्.क्ते यश्चाबुभुक्षितः। तच्च क्रमविरूद्धं स्याद्यच्चातिक्षुद्वशानुगः ।।९७।।

परिहारविरूद्धं तु वराहादीन्निषेव्य़ यत्। सेवेतोष्णं घृतादींश्च पीत्वा शीतं निषेवते ।।९८।।

विरूद्धं पाकतश्चापि दुष्टदुर्दारूसाधितम्। अपक्वतण्डुलात्यर्थपक्वदग्धं च यद्भवेत। संयोगतो विरूद्धं तद्यथाऽम्लं पयसा सह ।।९९।।

अमनोरूचितं यच्च हृद्विरूद्धं तदुच्यते । संपद्विरूद्धं तद्विद्यादसंजातरसं तु यत् ।।१००।।

अतिक्रान्तरसं वाऽपि विपन्नरसमेव वा ।। ज्ञेयं विधिविरूद्धं तु भुज्यते निभृते न यत् । तदेवंविधमन्नं स्याद्विरूद्धमुपयोजितम् ।।१०१।।

विरूद्धान्येवमादीनि वीर्यतो यानि कानिचित् । तान्वेकान्ताहितान्येव शेषं

विद्याद्धिताहितम् ।। Su. Su. 20/18

उत्क्लेश्य दोषान्न हरेत् द्रव्यं यत्तत्समासतः । विरूद्धं तद्धि धातूनां प्रत्यनीकतया स्थितम् ।। बलिनां मिथोगुणानां विषमतया समतयाप्युभयथापि । अ. सं. सू. ९/७

v. अभिष्यन्दी – दोषधातू मलस्त्रोतसां क्लेदः प्राप्तीजनकम्। सु. सू. ४६/५१

vi. पर्युषित - व्युष्टम् स्वकालं परित्यज अवस्थितम् । सु. उ. ५४/३

vii. आतपसेवन - आतपसेवनः सुर्यातपः । सु. चि. २४/८६
लंघनप्रकारः आतपसेवनम् । च. सु. १२/१८

viii. वेगावरोधः - गतिविरोधः तच्चाअनारोग्यकराणां श्रेष्ठम् । सं. सं. सु. ५/२६

ix. दिवास्वापः - दिवास्वप्न : । सु. शा. ४/ ४१

x. अंगमर्दः - अंगोद्वेष्टनम् इव वेदना । सु उ ५५/१६

xi. च्छर्दीः - आमाशय मुखमार्गेण दोषाणाम् बर्हिगमनम् । सु. चि. २०/५
हृल्लासः - हदयस्य उत्कलेशनम् । सु. उ. ५६/२१- २२

xii. अभिनन्दनम् न अपि । च. वि. ५/ १

xiii. जागरणः - निद्रायाः अभावः । च. नि. ९/३३

xiv. व्यायामः - शरीर आयासजनकं कर्म । वा. सू. २/१०

xv. विषमाशन - विषमद्रव्याणां अशनम् बहुस्तोक अक्राले वा अनादि अशनम् । च. नि. १/ ८९

xvi. पित्त कर्म अभिलाषा सं. सू. १९ /१,६

xvii. पांण्डुः - वर्णः श्वेतः । सु. सू. १३ /२३

xviii. अश्रध्दाः - रसप्रदोषेषु विकारेषु एकः। च. सु. २६

xix. मुर्च्छाः - चेतना च्युतिः । सु. नि. ९/२३

xx. भ्रमः - चक्रस्थितस्य एव संवेदनम्। च. चि. ३/३९

xxi. उपवास - भोजनपरित्यागः अस्यापि । सु. उ. ३९/ १३४

xxii. क्रोधः - मनोभाव कोपः । च. वि. ४/८
राजस गुणः । सु. शा. १/१८

xxiii. अध्यशन - पुर्वभुक्तेSपरिणते भोजनम् । च. चि. १५/२३६

3. Review of Literature:

3.1 Vedic literature:

The references from *Aranyka, Brahamana, Upanishadha* and *samhita* .as well as *Apadshautra Sutra, Bodhayan Shautra, Aukshaya Shautra Sutra* are enlisted below

Vedanga: In *Vedanga* the *mutra* word is observed with relation to excretion function and the diseases like ***Mutrameha*** in mentioned[6]

Mutra:

The words *Mutra, Mutra Kruchha, Mutra Purisha, Mutra purisha utsarga,* as well as *Mutra purisha* lohita reta are seen. In *Kausheya Shautrasutra Mutra meha, Mutra visarga, Mutra shakruta and Mutra kruchha* are seen.

Vrukka: In the Vedanga from *Kaushaye Shautra Sutra Vrukka, Vrukka Upchara, Vrukka meda* are seen

Yakruta: The word Yakruta along with *meda, loma* is seen.

Upanishadha: The word *Mutra, Mutra Drava, Mutra purisha* and *Mutra Shleshama, Rakta, Shukra* and *Sweda,* are observed.

Brahamana: The words are seen as *Vrukka, Mutra* and *Mutra purisha*.

Samhita:

The *Mutra* word is highlighted frequently. Apada Shautrasurta while performing the rituals, rule suggests prior urination to avoid further *Vega sandharana* of *adharniya Vega* to disciples before the *Yagnaya Karma.*

As well heart, tongue, sternum, liver, the two kidneys, left arm, both the thoracic walls, the right buttock one third of the rectum these are the limbs which are to be offered to the deities (the chief offerings).[7]

3.2 Ayurveda Literature Review:-

Ayurveda aspect to study CKD is to observe hetus; lakashanas (signs and symptoms) and correlate to understand samprapti (pathogenesis) .The basic need is to study formation of *Vrukka, Mutra* and its relation with *dosha* to understand the kidney disease. The srotas and dushti lakshana are reviewed .Thus, text reviewed for basic concepts which are involved in CKD aetiopathologenesis.

Content of Ayurveda review:-

1. *Vrukka*: - Relation with *Mahabhuta, Dosha, Dhatu, Mulsthana of Mutravaha Srotas, Mutra and Sweda.*
2. *Kleda: - Mutra Formation, Mutra Roga, E.g. Prameha and Vrukka Vikara.*
3. *Mutra: - Mutra and dosha relation, Mutra and Dhatu relation, Mutra and Kleda* relation.
4. *Srotas and dushti* lakashanani
5. Hetus causes mainly *Vegavrodha, Viruddha* reviewed.

Sharir – Vasti, Vrukka, Gavini, Mutravaha Srotas

As stated in *Khudika Garbhavkranti Adhyaya* formation of *Garbha* is due to *Shukra & Artava*. *Garbha* is *Matruja*. *Matruja Ahara* rasa nourishes *Garbha* eventually ends in the formation of individual.

Twak, Nabhi, Hridya, Kloma, Yakruta, Pliha, Vrukka, Vasti, Purishadhana, Amakshaya, Pakawakshaya, Uttar and adharguda, Shudrantra, & Vapa is maturja organs.[8]

***Vrukka – Matruja Avayav*:-**

The *twak lohit, mamsa, meda, nabhi Hrudya, klom, yakrut, pliha, vrukka, vasti and purishadhana* are maturjaavayuva.[9]

Vrukka and Mahabhuta Relation:-

Pruthavi	*Vrukka akara*
Jala	*Kleda and Mutra*
Tej	Tej mahabhuta for blood filtration with formation of mutra

Vayu	All functions of vrukka
Akasha	Maintains sachidrata –porosity.[10]

Vrukka and Dhatu: - *Vrukka* is formed from *Rakta and Meda dhatu.*

***Vrukka Mulsthana*: -** *Vrukka* is mulsthana for *Medavaha Strotas.* [11]

***Vrukka – Dosha*: -** *Tridosha* with *Kapha* dominances.

Strotas hetu and dushti lakshana:

***Medovaha Strotas*:** Day sleep, Fatty food and Alcoholic drinks.[12]

***Dushti Lakshana*:** Prameha Poorupa.

***Mutravaha Strotas*:** The basic site for mutravaha Strotas is *mutrakshaya and vankashana*.[13]

Dushti lakshana: *Bahumutrata, Alpamutrata, Mutra Avarodha, Buring Urination, Mutra Gandha, Varna* changes.

Purishavaha Strotas: *Pakawakshaya and guda*.[14]

PurishaStrotas Lakshana: *Shashoola, Sakashta, Sarakta, Pravartana.*

Swedavaha Strotas: *Meda and lomakup*

Dushti lakshana: *Daha, lomaharsha,* excess sweating, less sweating dryness of skin.[15]

Formation of Urine:-

Saman Vayu facilitate pachana with the help of *Kledaka Kapha* to reduce the Kledan karma facilitate Pachaka strava from *Amashaya, Pachamanasahya, Yakrut* and *Agnyashaya*. This helps in formation of *Teja, Drava* and *sukshma Ahara* and separation of *mala* into *Saara & kitta*. The ahara rasa absorbed by *Grahani* and forwarded towards heart (by *rasa dhamanya* for further body nourishment).[16]

This antrapachan gives rise to dosha formation. The dosha similar guna ghataka absorbed in rasa and nourishes for dosha. This is the vital function of *Samaan Vayu.*

Samaan Vayu Prakupit Vikaras are *Gulma, Atisaara and Agnimandya* etc.

Mutra Pravartara Excretion urine

The apana vayu site is *Vrushna, Vrukka, Vasti, Gavini, Nabhi, Uru & Guda*.[17]

When the bladder is completely filled with urine, it is excreted with the stimulus of *Apana Vayu.*

Apana Vayu Prakupita Vyadhi: The *apana vayu* vitiation leads to *Mutragrha, Garbhastrava and Bhagandra*.[18]

Dosha & Mutra:-

The *tridosha nirmiti* in the body is output of digestive process that is pachana. Here pachana means it is of the Ahara taken by individual for his daily energy requirement. The daily diet consumption is processed through *pachanasanstha*. This is directly related to the basic three avasthapakas respectively forming *prakrut Kapha, Pitta & Vata*. Further the remaining (Pachit Ahara rasa) part after formation of the *tridoshas* is directed / circulated throughout the body. During this process the particular *dhatus* take necessary part for their poshana individually.

This is the basic pachana process in digestive system labeled as three *avasthapaka* which is not possible without *kayagni*.

The rest remaing part is already having mala in *avyaktarupa* where *mutra* is also *avyakt* in *jalia* form as it is with the *dhatuposhanka ghatak.*

The same rasa circulating throughout the body nourishes the individual *dhatu* with their respective *ghatak.* Hence this is the *sukshma pachana* nourishing the *dhatus.* The *jalia* part again *in avyakta* form is given back to the circulating rasa.

Here during *sukshma pachana* meda *dhatu* is the chief among the *sevan dhatus* which contributes on higher side for *kleda* formation.

Thus *samanya pachana* is *pachana sanstha* level & *sukshma pachana* is at dhatu level, simultaneously form the *avyakta mutra* which at terminal stage is given *vykta mutra* form at *mutrakshaya* level. (*Vrukka – vasti*)

Pitta dosha:

Pitta vruddhi Lakshana – This leads to yellow coloration of mutra.[19]

Pittakshaya: The functions of *pachak* pitta is hampered due to which *Agnimandya* & *Anannabhilasha* and the whole body get pale due to *kkshaya* of *pitta*.

***Yakrut in Mutra Nirmiti*:**

Amashaya Grahani, Pakawashaya and their respective *avasthapaka* - *Madhur, Amla & Katu*. This ahara rasa formed in *avasthapaka* basically circulated from *Yakrut* to heart. The state of ahara rasa depending upon which type of ahara is taken is processed by *Yakrut*. The function of *Yakrut* is to maintain ahara rasa *Atisukshma & Saarabhut* in nature. Remaining portion which is not completely pachit is cleared by *Yakrutastha Agni*. The *kitta paramanu* formed in *Yakrut* mix with *kleda* and excreted as mutra.[20]

***Rakta Dhatu*:** Functions of *Rakta dhatu* are *Varna Prasadana, Indriya Prasadana, Indriya vishaya grahana* & timely excretion of mala.[21]

Meda dhatu:

The Meda dhatu nirmiti is followed by *mamsadhatu* with the help of *medadhatuagni*. *Meda dhatu* consists of *jal mahabhuta* dominant aliment and *sneha* dominant aliment. The *sira and snayu* are *updhatu* of meda.[22]

***Vrukka mutra and sweda*:** The *kapha* & meda properties are approximately same in nature. The kleda portion of kapha partly store in meda dhatus when needed used by sharir. As well if this kleda becomes excess stored in Vrukka and regulated as mutra & sweda. The sneha of majjadhatu keeps Twaka Varna in prakrutika condition. It helps in raktotpatti with Yakrut, Pliha & Sira.

Pachana and Mutra roga:

The Abhojana Ajeerna, Atibhojana, Vishmashna, Asatmya, Guru, Sheet, Rooksha bhojana leads to disturbances of Agni. The dushitagni is unable to digest laghuahara too; Disturbed pachana gives shuktatva to ahara forming Aharvisha. The portion leads to Mutrsanga and Mutravikara.[23]

The above mentioned hetus for Agni dushti are responsible for pachan vikruti. Also the dushti ahara rasa with vyana vayu circulated throughout –Sharir. The vikruti observed in sthana vaigunaya.[24]

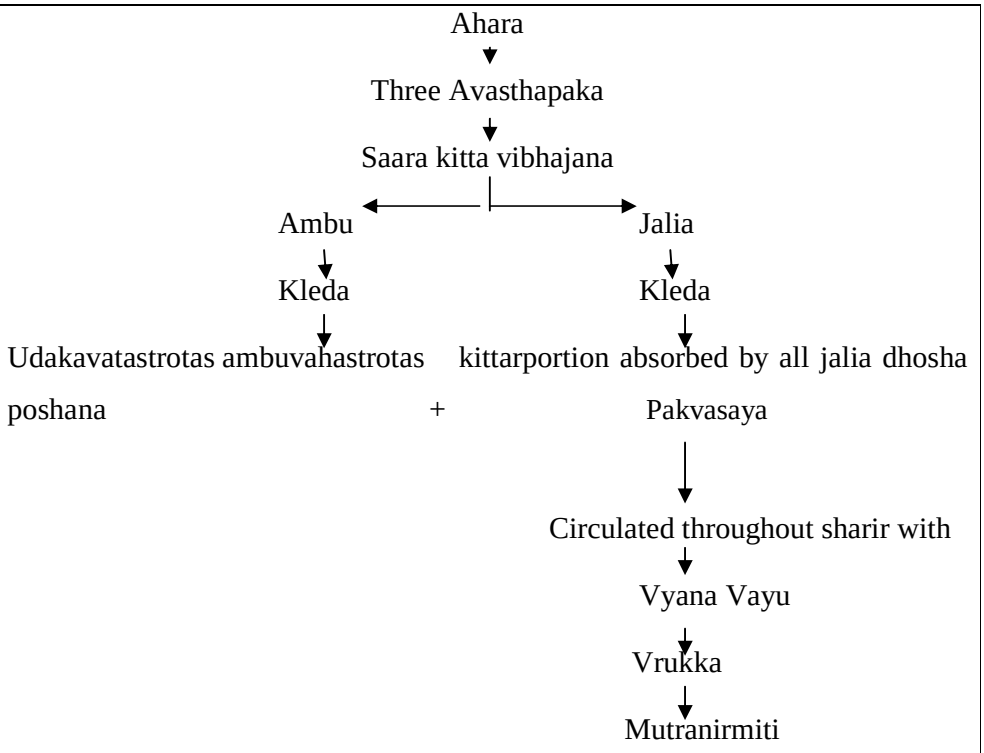
This leads to rasa dhatu dushti. This dushit rasa dhatu with vyana vayu circulated throughout body. It is lodged where basic vaigunaya i.e. Khavaigunya is present.

Krimi :–

The kshira, guda, tila, matsaya (anoop), pistanna, jreerna, puti, klinna, viruddha ahara, leads to shlemajakirmi. These krimi gives rise lakshanas such as Murchha, Jrumbha, Aanaha, Agmarda, Chhardi, Karshya and Parushya.[25]

Mutra and Kleda:-

The pachana of ahara is passed through Avasthapaka.



Flow Chart No. 1 Mutra and Kleda

Kleda and Ahara Parinamkarbhava:-

For proper *ahara pachana ushma, vayu, kleda, sneha, kala, samyog* all these factors are needed.[26]

Kleda and Prameha:-

The *sharistha kleda* is mixed with *shleshma* and meda. This is driven toward *mutrakshaya*. The properties such as *shewta, sheet, murt, pichil, snigdha, madhur, Sandra* leads to type of *Kaphajmeha.*[27]

Kleda and Vata rakta: - The *dushit kleda* and vata results in vata rakta.[28]

Kleda & Pitta: The *kleda* with *shonit* leads to *tanutva* of *rakta dhat*u. [29]

The *mamsa kleda* leads to *pittaroga*.[30]

Kleda Rasa: -

The *lavana* rasa with *dushit kleda* gives rise to *mutra daha*, as Lavana rasa is basically *ushna, tikshna and upa-kledi*.[31]

Kleda Madya:-

The excess *madya sevana* leads to *Vidahi, Vidagdha and Kleda* Vruddhi causing *ksharatva*.

Harita samhita:-

Harita samhita mentioned *Arishtas lakshanas of Ashamari, Prameha, and Pandu vyadhi.* The *Pandu vyadhi Arishtas are Shopha, Shwas, Pipasa, Shoola*. The Prameha *Aristas* are excessive *stravas* and Prameha *pitika*.[32]

Rajnighantu:-

Rajnighantu mentioned *mutra dosha –Prameha, Mutra Kruchha, Mutra Rodha* is labeled as *mutra ashmari*.[33]

Pakvasaya sharir:-

Pakvasaya is *matruja avayva* site of vata. The *dhamnaya* of *Pakvasaya* carry *Vata, Pitta & Kapha Dosha, Udaka, Mutra, Shuddha Rakta, Shukra, Artava and Purisha.* The dushit dosha in Pakvasaya leads to *Katistha Shotha*.[34]

The Pakvasaya is *udabhava sthana of hikka*. The *apana prana vayu* leads to *hikka.*

Vega: The *Vega vidharana of adharaniya vega* is one of the dushti cause of *mutravaha Strotas.*[35]

Dharaniya: The significance of *Dharana* –to settle, restore for some time is dharana.

It may be the *kayic, vachic and manasic*.

Adharaniya: There are thirteen *adharaniya Vegas* are mentioned. *Mutra, Mala,*

Shukra, Apana, Chhardi, Shvyathu, Udagara, Trishana, Shudha, Jrumbha Ashru, and

Nidra, Shramashwasa.

Mutra Vega vidharana lakshana: *Vasti, vinama, mutra krushratva, shirashoola and vanshana anaha.*

Mala Vega vidharana lakshana: *Shirashoola, Malavabaddhata, Pindikodweshatan, Adhamana.*

Apana vayu vidharana lakshana: *The Vata, Mutra Mala Sanga, Adhamana, Klama, Udarshoola and Vata Vikara.*

Chhardi Vega vidharana: *Kandu, Kotha, Aruchi, Vyanga, Shotha, Pandu, Jwara, Kushta, Hrullasa and Visaarpa*

Shvayuthu Vega vidharana lakshana: *Manyastambha, Shirashoola, Ardit, Ardhavabhedaka and Indriyadrubalya.*

Udagara Vega vidharana: *The Hikka, Kasa, Aruchi, Urogaurava* seen.

Jrumbha Vega vidharana: *Vinama, Akshepa, Sankocha, Supti, Kampa* seen

Shudha Vega vidharana: *Karhsya, Daruballya, Vaivaranya, Agagmarda, Aruchi, Bhrama* seen.

Trishana Vega vidharana Lakshana: *Kantha and Mukha Shosha, Badhiraya, Shrama, Angasada, Hrudavedana.*

Ashru Vega vidharana: *Pratishya, netravikara, hrudvikara as well aruchi, bhrama.*

Nidra Vega vidharana: *Jrumbha, Aganmarda, Tandra, Shirogaurava and Netra Gaurava.*

Shramashavasa: *Gulma, Hrudroga, Samhoha*

Dharaniya Vega:

- **Manasa Vega:** *Lobha, Shoka, Krodha, Ahankara, Irsha.*
- **Vachic Vega:** *Bhashana, lies* and all.
- **Sharirik Vega:** *Stealing,*

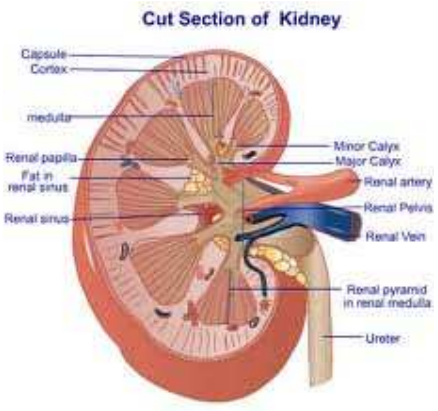
Thus, in above chapter Ayurveda aspect is reviewed. It significantly denotes the *vrukka, dosha, dhatu,* srotas and their relation with *mahabhut. Kleda* is also important component reviewed these all due to hetus giving rise to *vrukka vikara*.

3.3. Physiology Anatomy of the Kidneys:

Kidneys and Urinary tract:-

The two kidneys lie on the posterior wall of the abdomen, outside the peritoneal cavity (Figure). Each kidney of the adult human weighs about 150 grams and is about the size of a clenched fist. The medial side of each kidney contains an indented region called the helium through which pass the renal artery and vein, lymphatic, nerve supply, and urethra, which carries the final urine from the kidney to the bladder, where it is stored until emptied. The kidney is surrounded by a tough, fibrous capsule that protects its delicate inner structures.

If the kidney is bisected from top to bottom, the two major regions that can be visualized are the outer cortex and the inner region referred to as the medulla. The medulla is divided into multiple cone-shaped masses of tissue called renal pyramids. The base of each pyramid originates at the border between the cortex and medulla and terminates in the papilla, which projects into the space of the renal pelvis, a funnel-shaped continuation of the upper end of the urethra. The outer border of the pelvis is divided into open-ended pouches called major calyces that extend downward and divide into minor calyces, which collect urine from the tubules of each papilla. The walls of the calyces, pelvis, and urethra contain contractile elements that propel the urine toward the bladder, where urine is stored until it is emptied by micturition.[36]



Functions of Kidney:

Renal Blood Supply:-

Blood flow to the two kidneys is normally about 22 per cent of the cardiac output, or 1100 ml/min. The renal artery enters the kidney through the hilum and then branches progressively to form the interlobular arteries, arcuate arteries, interlobular arteries (also called radial arteries) and afferent arterioles, which lead to the glomerular capillaries, where large amounts of fluid and solutes (except the plasma proteins) are filtered to begin urine formation (Figure 26 – 3). The distal ends of the capillaries of each glomerular coalesce to form the efferent arteriole, which leads to a second capillary network, tubular capillaries, that surrounds the renal tubules.

Excretion of Metabolic Waste Products, Foreign Chemicals, Drugs, and Hormone Metabolites:-

The kidneys are the primary means for eliminating waste products of metabolism that are no longer needed by the body. These products include urea (from the metabolism of amino acids), creatinine (from muscle creatnine), uric acid (from nucleic acids), end products of hemoglobin breakdown (such as bilirubin), and metabolites of various hormones. These waste products must be eliminated from the body as rapidly as they are produced. The kidneys also eliminate most toxins and other foreign substances that are either produced by the body or ingested, such as pesticides, drugs, and food additives.

The Nephron is The Functional Unit of the Kidney:-

Each kidney in the human contains about 800,000 to 1 million nephrons, each capable of forming urine. The kidney cannot regenerate new nephrons. Therefore, with renal injury, disease, or normal aging, there is a gradual decrease in nephrons number. After age 40, the number of functioning nephrons usually decreases about 10 per cent every 10 years; thus, at age 80, many people have 40 per cent fewer functioning nephrons than they did at age 40. This loss is not life threatening because adaptive changes in the remaining Nephrons allow them to excrete the proper amounts of water, electrolytes, and waste products.

Micturition:-

Micturition is the process by which the urinary bladder empties when it becomes filled. This involves two main steps; first, the bladder fills progressively until the tension in its walls rises above a threshold level; this elicits the second step, which is a nervous reflex called the micturition reflex that empties the bladder or, if this fails, at least causes a conscious desire to urinate. Although the micturition reflex is an autonomic spinal cord reflex, it can also be inhibited or facilitated by centers in the cerebral cortex or brain stem.

Facilitation or Inhibition of Micturition by the Brain:-

The micturition reflex is a completely autonomic spinal cord reflex, but it can be inhibited or facilitated by centers in the brain. These centers include (1) strong facilitative and inhibitory centers in the brain stem, located mainly in the pons, and (2) several centers located in the cerebral cortex that are mainly inhibitory but can become excitatory.

The micturition reflex is the basic cause of micturition, but the higher centers normally exert final control of micturition as follows:

- The higher centers keep the micturition reflex partially inhibited, except when micturition is desired.
- The higher centers can prevent micturition, even if the micturition reflex occurs, by continual tonic contraction of the external bladder sphincter until a convenient time presents itself.
- When it is time to urinate, the cortical centers can facilitate the sacral micturation centers to help initiate a micturition reflex and at the same time inhibit the external urinary sphincter so that urination can occur.

Voluntary urination is usually initiated in the following way: First, a person voluntarily contracts his or her abdominal muscles, which increases the pressure in the bladder and allows extra urine to enter the bladder neck and posterior urethra under pressure, thus stretching their walls. This stimulates the stretch receptors, which excites the micturition reflex and simultaneously inhibits the external urethral sphincter. Ordinarily, all the urine will be emptied, with rarely more than 5 to 10 milliliters left in the bladder.[37]

Etiology and Epidemiology:-

TABLE 2 : Population at Risk for CKD
Age >65
Diabetes type 1 and 2
Family history of renal disease
Autoimmune disease
Systemic infections
Urinary tract infections/ stones
Urinary tract obstructions
Recovery of acute kidney injury
Hypertensive's
Drug abusers: Non-steroidal anti inflammatory drugs (NSAIDs), analgesics/ heroin
Neoplasia
Low birth weight
Reduced kidney mass
Low income
Low education
***Perhaps, an appropriate name for this group of patients would be CKD stage '0'.**

The most frequent cause of CKD is diabetic nephropathy, most often secondary to type 2 diabetes mellitus. Hypertensive nephropathy is a common cause of CKD in the elderly, in whom chronic renal ischemia as a result of small and large vascular disease may be under recognized. Progressive nephrosclerosis from vascular disease is the renal correlate of the same processes that lead to coronary heart disease and cerebro vascular disease. The increasing incidence of CKD in the elderly has been ascribed, in part, to decreased mortality from the cardiac and cerebral complications of atherosclerotic vascular disease in these individuals, enabling a greater segment of the population to manifest the renal component of generalized vascular disease. Nevertheless, it should be appreciated that overwhelmingly the vast majority of those with early stages of renal disease, especially of vascular origin, will

succumb to the cardiovascular and cerebro vascular consequences of the vascular disease before they can progress to the most advanced stages of CKD. The early stage of CKD, manifesting as albuminuria and even a minor decrement in GFR, is now recognized as a major risk factor for cardiovascular disease.

The striking inter individual variability in the rate of progression to CKD has an important heritable component, and a number of genetic loci that contribute to the progression of CKD have been identified. Similarly, it has been noted that women of reproductive age are relatively protected against progression of many renal diseases, and sex-specific responses to angiotensin II and its blockade have been identified.[37]

Pathophysiology of CKD:-

Chronic Kidney disease is any illness that has existed for > 3 months with either kidney damage or low GFR. Kidney damage manifests as abnormal gross or histopathological abnormality or investigation revel urinary abnormality, biochemical abnormality & imaging abnormality indicating kidney dysfunction.

Pathophysiology of Chronic Kidney Disease:-

The Pathophysiology of CKD involves two broad sets of mechanisms of damage:

(1) Initiating mechanisms specific to the underlying etiology (e.g., immune complexes and mediators of inflammation in certain types of glomerular nephritis, or toxin exposure in certain diseases of the renal tubules and interstitium).[1]

- Immune mechanism
- Inflammation
- Toxins

(2) A set of progressive mechanisms, involving hyper filtration and hypertrophy of the remaining viable nephrons, that are a common consequence following long-term reduction of renal mass, irrespective of underlying etiology. The responses to reduction in nephrons number are mediated by vasoactive hormones, cytokines, and growth factors. Eventually, these short- term adaptations of hypertrophy and hyper filtration become maladaptive as the increased pressure and flow predisposes to sclerosis and dropout of the

remaining nephrons. Increased interregnal activity of the rennin- angiotensin axis appears to contribute both to the initial adaptive hyper filtration and to the subsequent maladaptive hypertrophy and sclerosis, the latter, in part, owing to the stimulation of transforming growth factor β (TGF- β). This process explains why a reduction in renal mass from an isolated insult may lead to a progressive decline in renal function over many years.[38]

The Stages of CKD and Identification of At- Risk Populations:-

It is important to identify factors that increase the risk for CKD, even in individuals with normal GFR.

Risk factors:-

Hypertension, Diabetes mellitus, autoimmune disease, Older age, African ancestry, a family history of renal disease, h/o Acute Renal Failure, presence of proteinuria, abnormal urinary sediment, or structural abnormalities of the urinary tract.

Etiopathogenesis:-

All chronic nephropathies can lead to CRF. The diseases leading to CRF can generally be classified into two major groups: those causing glomerular pathology, and that causing tubulointerstitial pathology. Though this classification is useful to facilitate study, the disease rarely remains confined to either glomerular or tubulointerstitial tissue alone. In the final stage of CRF, all parts of the nephrons are involved.

Diseases causing glomerular pathology:-

A number of glomerular diseases associated with CRF have their pathogenesis in immune mechanisms (page 685). Glomerular destruction results in changes in filtration process and leads to development of the nephritic syndrome characterized by proteinuria, hypoalbuminaemia and edema. The important examples of chronic glomerular diseases causing CRF are covered under two headings: primary and systemic.

I. Primary glomerular pathology
II. Systemic glomerular pathology

Diseases causing tubulointerstitial pathology:

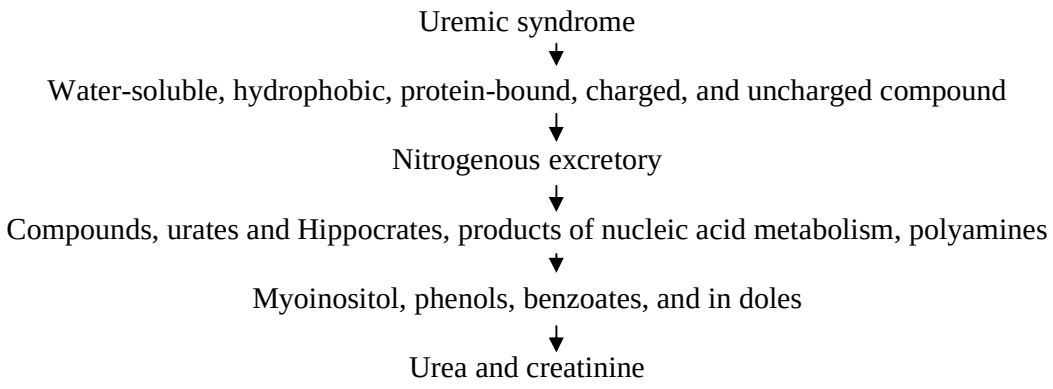
Damage to tubulointerstitial tissues results in alterations in reabsorption and secretion of important constituent's leads to excretion of large volumes of dilute urine. Tubulointerstitial diseases can be categorized according to initiating etiology into 4 groups: vascular, infections, toxic and obstructive.

I. Vascular causes: Long-standing primary or essential hypertension produces characteristic changes in renal arteries and arterioles referred to as nephrosclerosis. Nephrosclerosis causes progressive renal vascular occlusion terminating in ischemia and necrosis of renal tissue.
II. Infections causes: A good example of chronic renal infection causing CRF is chronic pyelonephritis. The chronicity of process results in progressive damage to increasing number of nephrons leading to CRF.
III. Toxic causes: Some toxic substances induce slow tubular injury, eventually culminating in CRF. The most common example is intake of high doses of analgesics such as phenacetin, aspirin and acetaminophen (chronic analgesic nephritis). Other substances that can cause CRF after prolonged exposure are lead, cadmium and uranium.
IV. Obstructive causes: Chronic obstruction in the urinary tract leads to progressive damage to the nephrons due to fluid back-pressure. The examples of this type of chronic injury are stones, blood clots, tumors, strictures and enlarged prostate.

Pathophysiology and Biochemistry of Uremia:-

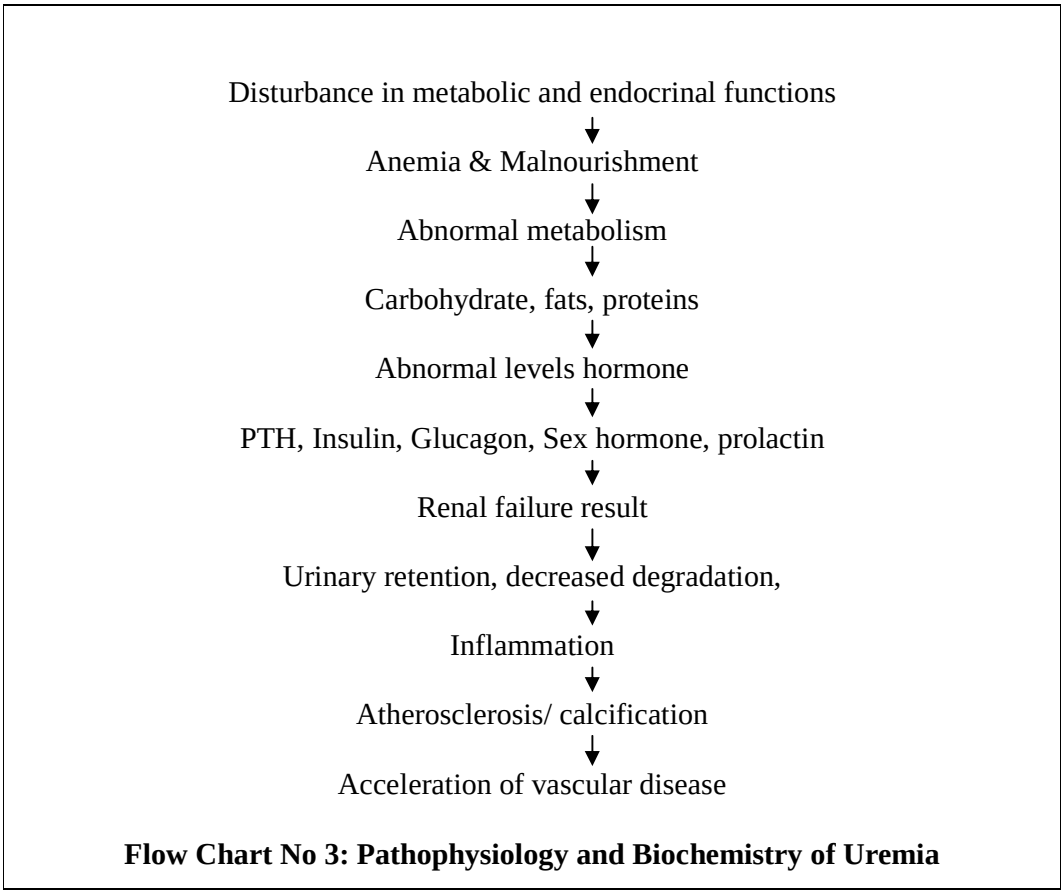
Although serum urea and creatinine concentrations are used to measure the excretory capacity of the kidneys, accumulation of these two molecules themselves do not account for the many symptoms and signs that characterize the uremic syndrome in advanced renal failure. Hundreds of toxins that accumulate in renal failure have been implicated in the uremic syndrome. These include water-soluble, hydrophobic, protein-bound, charged, and uncharged compounds. Additional categories of nitrogenous excretory products include guanido compounds, urates and

Hippocrates, products of nucleic acid metabolism, polyamines, myoinositol, phenols, benzoates, and in doles. Compounds with a molecular mass between 500 and 1500 Da, the so-called middle molecules, are also retained and contribute to morbidity and mortality. It is thus evident that the plasma concentrations of urea and creatinine should be viewed as being readily measured but incomplete, surrogate markers for these compounds and monitoring the levels of urea and creatinine in the patient with impaired kidney function represents a vast over-simplification of the uremic state.



Flow Chart No 2: Pathophysiology and Biochemistry of Uremia

The uremic syndrome and the disease state associated with advanced renal impairment involve more than renal excretory failure. A host of metabolic and endocrine functions normally undertaken by the kidneys are also impaired, and this results in Anemia, malnutrition, and abnormal metabolism of carbohydrates, fats, and proteins. Furthermore, plasma levels of many hormones, including PTH, insulin, glucagon, sex hormones, and prolactin, change with renal failure as a result of urinary retention, decreased degradation, or abnormal regulation. Finally, progressive renal impairment is associated with worsening systemic inflammation. Elevated levels of C-reactive protein are detected along with other acute-phase reactants, while levels of so-called negative acute-phase reactants, such as albumin and feting, decline with progressive renal impairment. Thus, renal impairment is important in the malnutrition-inflammation-atherosclerosis/ calcification syndrome, which contributes in turn to the acceleration of vascular disease and co morbidity associated with advanced renal disease.



Flow Chart No 3: Pathophysiology and Biochemistry of Uremia

In summary, the path physiology of the uremic syndrome can be divided into manifestations in three spheres of dysfunction:

1) Those consequent to the accumulation of toxins normally undergoing renal excretion, including products of protein metabolism.
2) Those consequent to the loss of other renal functions, such as fluid and electrolyte homeostasis and hormone regulation.
3) Progressive systemic inflammation and its vascular and nutritional consequences.

Chronic Kidney Disease:

Chronic kidney disease (CKD) encompasses a spectrum of different path physiologic processes associated with abnormal kidney function, progressive decline in glomerular filtration rate (GFR). Table1-1 provides a widely accepted classification, based on recent guidelines of the National Kidney foundation (Kidney Dialysis Outcomes Quality Initiative (KDOQI), in which stages of CKD are defined according to the estimated GFR.

The term chronic renal failure applies to the process of continuing significant irreversible reduction in nephrons number, and typically corresponds to CKD stages 3-5. The path physiologic processes and adaptations associated with chronic renal failure will be with the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the uremic syndrome. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation.

TABLE 1-1 CLASSIFICATION OF CHRONIC KIDNEY DISEASE (CKD)

Stage	GFR, ml/ min per 1.73 m^2
0	>90[α]
1	≥ 90[b]
2	60-89
3	30-59
4	15-29
5	<15

[α] with risk factors for CKD (see text)

[b] with demonstrated kidney damage (e.g. persistent proteinuria, abnormal urine sediment, abnormal blood and urine chemistry, abnormal imaging studies).

Note: GFR, Glomerular filtration rate.

Source: Modified from National Kidney Foundation. K/DOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, classification and stratification. Am J Kidney Dis 39: suppl 1, 2002.[3]

GFR Estimation:

Two equations commonly used to estimate GFR are shown in Table1-2 and incorporate the measured plasma creatinine concentration, age, sex, and ethnic origin reporting of estimated GFR, or "e-GFR," using one of these equations.

Table1-2

Recommended equations for Estimation of Glomerular Filtration Rate (GFR) Using Serum Creatinine Concentration (Pc_r), Age, Sex, Race and Body Weight

1. Equation from the Modification of Diet in Renal Disease study[α]

   Estimated GFR (mL/min per $1.73m^2$) = $1.86 \times (Pc_r)^{-1.154} \times (age)^{-0.203}$

   Multiply by 0.742 for women

   Multiply by 1.21 for African Americans

2. Cockcroft-Gault equation

   Estimated Creatinine Clearance (mL/min)

   $$= \frac{(140 - \text{Age}) \times \text{body weight, kg}}{72 \times PC\ r\ (mg/dL)}$$

   Multiply by 0.85 for women

[α] Equation is available in hand-held calculators and in tabular form.

Adapted from AS levey et al: Am J Kidney Dis 39(Suppl 1): S1, 2002, with permission.

GFR with Age and Creatinine level:

The normal annual mean decline in GFR with age from the peak GFR (~120mL/min per$1.73m^{2)}$ attained during the third decade of life is ~ 1 mL/ min per year per$1.73m^{2,}$ reaching a mean value of 70mL/min per$1.73m^2$ at age 70. The mean GFR is lower in women than in men. For example, a woman in her 80s with a normal serum creatinine may have a GFR of just 50mL/ min per$1.73m^{2.}$ Thus, even a mild elevation in serum creatinine concentration {e.g. 130 µmol/L (1.5mg/dL)}, often signifies a substantial reduction in GFR in most individuals.

Mechanisms:-

Raised intra- glomerular pressure:

- As nephrons scar and 'drop out,' remaining nephrons undergo compensatory adaptation, with blood flow per nephrons attempting to 'normalize' GFR (the Brenner Hypothesis).

- ↑ Glomerular capillary wall permeability is a feature of glomerular diseases.

- Renal vasodilatation may be an initiating event, with the glomerular exposed to a higher capillary pressure.

Glomerular damage:-

- Intra glomerular pressure -> wall stress and endothelial injury.
- Strain on mesangial cells -> matrix deposition mediated (in part) by angiotensin II and cytokine release (TGF – β, PDGF).

Proteinuria: - May be due to an underlying glomerular lesion, or result from raised intra glomerular pressure. Protein or factors bound to filtered albumin (such as fatty acids, growth factors or metabolic end- products) may lead to:

- Direct proximal tubular cell injury.
- Local cytokine synthesis (→ recruitment of interstitial inflammatory cells).
- Pro-fibrotic factors → interstitial scarring.
- Trans- differentiation of tubular cells into fibroblasts.

Tubulointerstitial scarring: - The degree of tubulointerstitial damage correlates better with long-term prognosis than glomerular damage. Proteinuria may itself be harmful to the tubulointerstitial, but chronic ischemic damage is also important: tissue oxygen tension is relatively low in the renal medulla, making tubules sensitive to hypoxic injury. Chronic ischemia occurs with:

- Damage to glomerular capillaries (glomerular sclerosis → altered per tubular perfusion).
- RAS activation → interregnal vasoconstriction.
- Intra tubular capillary loss and increased diffusion distance between capillaries and tubular cells, leads to vicious cycle of hypoxic.

Significance of Albuminuria, Proteinuria, Microalbumuria:

Measurement of albuminuria is also helpful for monitoring nephrons injury and the response to therapy in many forms of CKD, especially chronic glomerular diseases. While an accurate 24-h urine collection is the “gold standard” for measurement of albuminuria, the measurement of albumin-to- creatinine ratio in a spot first- morning urine sample is often more practical to obtain and correlates well, but not perfectly, with 24-h urine collections. Persistence in the urine of >17 mg of

albumin per gram of creatinine in adult males and 25mg albumin per gram of creatinine in adult females usually signifies chronic renal damage. Micro albuminuria refers to the excretion of amounts of albumin too small to detect by urinary dipstick or conventional measures of urine protein. It is a good screening test for early detection of renal disease, in particular, and may be a marker for the presence of micro vascular disease in general. If a patient has a large amount of excreted albumin, there is no reason to perform an assay for micro albuminuria.

Progression of Diseases:

Stages 1 & 2 CKD:

- Decreased GFR
- Renal parenchyma disease
- Poly cystic disease
- Glomerular nephritis
- Parenchyma and vascular diseases.
- Well preserved GFR.

The usually are not associated with any symptoms arising from the decrement in GFR. However, there may be symptoms from the underlying renal disease itself, such as edema in patients with nephritic syndrome or signs of hypertension secondary to the renal parenchyma disease in patients with polycystic kidney disease, some forms of glomerular nephritis, and many other parenchyma and vascular renal diseases, even with well-preserved GFR.

GFR progresses to stages 3 & 4:

- Organs affected
- Anemia
- Associated
- easy fatigability; decreasing appetite with progressive malnutrition
- Abnormality
- calcium, phosphorus, mineral regulating hormone, Parathyroid hormone
- sodium, potassium, water, and acid-base homeostasis

If the decline in GFR progresses to stages 3 & 4, clinical and laboratory complications of CKD become more prominent. Virtually all organ systems are affected, but the most evident complications include Anemia and associated easy fatigability; decreasing appetite with progressive malnutrition, abnormalities in calcium, phosphorus, and mineral – regulating hormones, such as 1,25 $(OH)_2$ D_3 (calcitriol) and parathyroid bromine (PTH); and abnormalities in sodium, potassium, water, and acid-base homeostasis.

Progresses to stage 5 CKD:

- Disturbance
- Nutritional status
- Water and electrolyte homeostasis

If the patient progresses to stage 5 CKD, toxins accumulate such that patients usually experience a marked disturbance in their activities of daily living, well-being, nutritional status, and water and electrolyte homeostasis, eventuating in the uremic syndrome. This state will culminate in death unless renal replacement therapy (dialysis or transplantation) is instituted.

CKD & Other disease (Anemia, Hypertension & diabetes)

Anemia of CKD

Erythropoietin (EPO) and the kidney:-

Red blood cell production is tightly regulated by a number of different growth factors. EPO is essential for the terminal maturation of erythrocytes, and differs from other growth factors in that it is produced by per tubular interstitial fibroblasts in the outer renal medulla and deep cortex of the kidney rather than the bone marrow. The kidney is ideally placed to regulate RBC production, as it is uniquely able to sense and control both O_2 tension and circulating volume (and differentiate between the two):

- Red cell mass is regulated by EPO.
- Circulating volume is regulated by salt and water excretion.
- The kidney maintains the haematocrit at 45% in normal conditions. (maximizing tissue O_2 delivery)

Chronic kidney disease, renal scarring → ↓ EPO synthesis, ↓ RBC production and anemia this occurs in most form of advanced CKD (e GFR < 35 mL/min), with a few exceptions:

- Adult polycystic kidney disease.
- Benign renal cysts.
- Renal cell carcinoma.

In these instances, EPO may be overproduced.

Differential diagnosis of anemia in CKD patients:-

EPO deficiency is not the only cause of ↓ Hb in CKD

Patients with CKD are susceptible to all other causes of anemia, so these should be actively sought in patients who appear disproportionately anemic or EPO resistant:

- Iron deficiency.
- Blood loss (Gl tract, haemodialysis)
- Foliate deficiency
- B12 deficiency
- Haemolysis
- Myelodysplasia
- Myeloma

Hypertension:-

Hypertension is the second leading cause of end-stage renal disease (ESRD). As an example, according to the United States Renal Data System (U. S. Renal Data System, 2009), about 51 -63% of all patients with CKD are hypertensive. This number grows to 90% in patients over 65years. In the corresponding general population the incidence of hypertension is 11 – 13% and 50%, respectively.

Hypertension causes a nephrosclerotic glomerulopathy characterized by:-

i. Renal vasculopathy affecting pre glomerular arteries and arterioles, resulting mainly from atherosclerosis, endothelial dysfunction, wall thickening and fibrosis.

ii. Microvascular disease of the glomerular tuft capillaries.

iii. Diffuse glomerular sclerosis and, less often, focal and segmental glomerular sclerosis (FSGS), involving damage to the filtration barrier constituents (Podocytes, mesangial cells and basement membranes.

iv. Interstitial fibrosis (Rosario & Wesson, 2006). Overall renal blood flow decreases as a consequence of arteriolar vasculopathy, vascular obstruction and decrease vascular density. However, GFR initially stays relatively constant. This is due to (i) increased glomerular capillary pressure resulting from deficient or upwardly reset renal auto regulation; and (ii) damage to the filtration barrier resulting in greater permeability. Subsequently, GFR decreases as a consequence of a progressive loss of surface area, mesangial hypertrophy and increasing glomerular and per tubular fibrosis. Concomitantly, basement membrane alterations produce albuminuria and protein hyper filtration.

	Pre glomerular arteries and arterioles affected ↓ Endothelial dysfunction
Renal vasculopathy	Wall thickening and fibrosis ↓
Micro vascular Disease	Glomerular tuft capillaries ↓
Filtration barrier damage	Podocytes mesangial cells and basement membrane ↓
Interstitial fibrosis	Arteriolar vasculopathy, vascular obstruction, depressed vascular density ↓
GFR decreased	loss of surface area, mesangial hypertrophy, increasing glomerular and per tubular fibrosis ↓
Alteration basement	proteinuria and alumunria

Flow Chart No. 4: Hypertension Nephropathy

Hypertension – associated CKD progression is highly dependent on

(i) Renal blood flow auto regulation and renal hemodynamic.

(ii) Artificial maneuvers or genetically-determined factors that modify renal function or renal tissue homeostasis, independently of their action on blood pressure or renal hemodynamic.

(iii) Genetic susceptibility factors. Renal auto regulation endows the kidneys with the capacity to maintain constant glomerular flow and pressure upon changes in systemic and renal perfusion pressure. Auto regulation is attained through vasoconstriction and vasodilatation of pre glomerular (afferent) arteries and arterioles. In addition to the insulation of renal function from the influence of fluctuations in systemic blood pressure, one of the most important physiological functions of auto regulation is believed to be the protection of renal tissues from mechanical overload derived from high blood pressure.[39]

Diabetic nephropathy:-

The diabetic nephropathy is leading cause of ESRD in the USA and Europe (Molitch et al., 2004). In fact, about 50% of ESRD patients (in the USA) are diabetic (U.S.Renal Data System, 2009). It is important to consider that hyperglycemia is primary initiator of diabetic nephropathy. In the absence of elevated glycemic, nephropathy does not develop. However, diabetic nephropathy holds a genetic component at two levels: first, the elevation of glycemic; and second, at establishing a genetic back- ground where nephropathy can occur (in the presence of hyperglycemia). Only 30% of patients with type 1, and 35 – 40% of patients with type 2 diabetes develop diabetic nephropathy irrespective of glycemic control (Diabetes Control & Complications, 1995). The clinical history of a typical patient starts with symptoms of hyper filtration (elevated values of GFR) and occasional microalbuminuria, which may last approximately 5 years. During the next – 20 years, microalbuminuria turns into progressively higher proteinuria, whereas GFR declines. Finally, the patient undergoes renal insufficiency with severe proteinuria, which eventually evolves towards ESRD (Schena & Gesualdo, 2005).

Diabetes patients

Type1-30%

Type2-35-40%

Hyper filtration (elevated values of GFR) 5years

Diagnosing CKD:-

Always assume a↓e GFR represents acute renal failure until proven otherwise. If uncertain, repeat within 5 day and refer as necessary.

TABLE 10: Manifestations Attributable to Uraemic Toxins

Cardiovascular system • **Atheromatosis** • **Arteriosclerosis** • **Cardiomyopathy** • **Decreased diastolic compliance** **Nervous system** • **Concentration disturbances** • **Cramps** • **Dementia** • **Depression** • **Fatigue** • **Headache** • **Seizures** • **Asterixis (flaps)** • **Motor weakness** • **Polyneuritis** • **Reduced sociability** • **Restless legs** • **Sleep disorders** • **Stupor, coma** **Haematological system** • **Bleeding** • **Hypercoagulability** **Immunological system** • **Inadequate antibody formation** • **Stimulation of inflammation** • **Susceptibility of cancer** • **Susceptibility of infection**	**Endocrinology** • **Dyslipidaemia** • **Glucose tolerance** • **Growth retardation** • **Hyperparathyroidism** • **Mpotence, diminished libido** **Bone disease** • **Adynamic bone disease** • **Amyloidosis** • **Osteitis fibrosa** • **Osteomalacia** • **Osteoporosis** **Skin** • **Melanosis** • **Pruritus** • **Uraemia frost** **Gastrointestinal system** • **Anorexia** • **Dyspepsia** • **Hiccups** • **Nausea, vomiting** • **Pancreatitis** **Pulmonary system** • **Pleuritis** • **Sleep apnoea syndrome** **Miscellaneous** • **Hypothermia** • **Thirst** • **Uraemic foetor** • **Weight loss**

Importance to identify with CKD:

- CKD predisposes to ↑CV risk. Modifying other CV risk factors (►↑BP) is likely to morbidity and mortality.

- Some patients will benefit from further investigation (e.g. renal biopsy).
- It may be possible to slow progression to ESRD.
- Complications of CKD (e.g. anemia and bone disease) can be identified and treated early.
- Those (relatively few) patients who will reach ESRD and require dialysis or transplantation need to be properly prepared.

Detection and quantification of proteinuria

- There is no need to perform 24^{th} urine collections for quantification of proteinuria.
- If dipstick ≥ 1 +, send an MSU to exclude UTI, and send a sample (preferable early morning) to clinical biochemistry (ideally 2 samples, 2 weeks apart): positive result:
    - Protein / creatinine ratio (PCR) ≥ 45mg/mmol.
    - Albumin / creatinine ratio (ACR) ≥ 30mg/mmol.
    - PCR is adequate in most instances. ACR is more sensitive for early disease screening in high risk groups (e.g. diabetes)
    - ACR ≥ 2.5mg/mmol (o) or 3.5mg/mmol (o) represents microalbuminuria.

Progression of CKD:-

Once CKD is established it tends to progress, regardless of underlying cause. Decline in GFR tends to be linear over time, unless clinical circumstances change. Progression of CKD is more often due to 2^{0} hemodynamic and metabolic factors, than underlying disease activity.

Factors influencing progression of CRF

Non –modifiable

Underlying cause of kidney disease (tubulointerstitial disease tends to progress more slowly than glomerular disease).

- Race (progression faster in blacks).

Modifiable

- BP.
- Level of proteinuria
- Plus:
    - Nephrotoxic agents
    - Underlying disease activity (e.g. SLE, vacuities)
    - Further renal insults (superimposed obstruction, UTI)
    - Hypovolaemia or inter current illness
    - Dyslipidaemia
    - Hyper phosphataemia
    - Uncontrolled metabolic acidosis
    - Anemia
    - Smoking
    - Blood glucose control (if diabetic).

Conventional Management of CKD

General advice (all stages)

- Smoking cessation.
- Weight reduction if obese.
- Encourage aerobic exercise.
- Aspirin 75mg od if 10 year cardiovascular risk > 20 % (page. 298), as long as BP < 150/90.
- Check lipids and treat according to national guidelines.
- Avoid NSAIDs and other nephrotoxic drugs.
- Limit alcohol to <3 units per day (o) or 2 units per day (o).
- Vaccination against influenza and pneumococca**l.**

CKD stages 1-3

- Most of these patients will not progress to ESRD, so the emphasis should be on CV risk reduction.
- Can usually be effectively managed in primary care setting.
- Suggested criteria for referral to a specialist renal service are shown opposite.
- Stages 1-2 ; at least annual follow up:

- e GFR, urinalysis and PCR.
- Meticulous Bp control.

- Stages 3: at least 6 monthly follow up:
  - e GFR, urinalysis and PCR.
  - Meticulous BP control
  - If Hb <11 g/dL check ferritin (start on PO iron if < 100mg/dL), B12 and folate. Refer for IV iron ± EPO according to locally agreed protocols>
  - Annual check of serum calcium and phosphate.
  - Annual PTH and seek advice if > 70 pg/ml.

CKD stages 4-5

- Refer to a renal unit (►urgently if stage 5). Late referral of patients with advanced CKD is associated with poor outcomes.
- Full dietary assessment
- Optimize calcium, phosphate and PTH.
- Correct acidosis.
- Hepatitis B immunization.
- Information and discussion regarding future treatments (dialysis transplantation, or conservative/palliative treatment).

Complications of advanced CKD

Fluid overload:-

Salt and water overload is usual in advanced CKD. However, as tubule interstitial scarring progresses, loss of concentrating ability may fix (and often large) urine volumes and a relative salt-losing state. Such patients may be chronically hypo- rather than hypervolaemic, and require salt and water supplementation (e.g. $NaHCO_3$ 0.5 -1.5g tds and increased fluid intake).

Treating salt and water retention in CKD:-

The careful clinical assessment of volume status

- Dietary salt restriction.
- Fluid intake restriction.
- Start fursemide 40mg od and titrate as necessary (max 250mg daily).

- If poor response, consider thiazide diuretic (metolazone 2.5 -10mg od) for synergistic effect.Δ dieresis may be brisk. Beware ↓Na+, ↓K+ and volume depletion (consider admission).
- Monitor:
    - Daily weight – the best day to day guide of salt and water status. Ask the patient to keep a diary of their weight at home. Weight loss should generally be < 0.5 – 1kg/day.
    - BP
    - A rise in Ur ± Cr may restrict dose escalation. If Ur > 25 mmol/L consider dose reduction (or cessation), depending on clinical need.
    - Refractory volume overload may signal the need for renal replacement therapy.

Diet and Nutrition of CKD

Dietary advice is extremely important in the management of CKD and the maintenance of broader health in CKD patients.

Measurement of Nutritional status:

➔ No single parameter should be considered in isolation.

Assessment should include:

- History and examination to identify ongoing medical problems which may limit nutritional intake – psychosocial issues may be important.
- Dietary interview or diary: quantitative intake of nutrients.
- Subjective global assessment (SGA): is a simple scoring (subjective and objective) made on history and examination. It is well – validated in CKD, and powerful enough to predict outcome.
- Anthropometric measurements body mass index, skin-fold thickness, estimated percent weight loss, and mid-arm muscle circumference
- Serum albumin: reflects not only protein intake, but susceptible to changes with inflammation or infection. A strong predictor of future mortality in new starters on dialysis.
- Adequacy of dialysis: inadequate dialysis is a common contributing factor to malnutrition (uremic toxins are anorectic and pro-inflammatory). Dialysis adequacy should be assessed in conjunction with the normalized protein catabolic rate (n PCR), which is a

measure of the rate of urea formation. When any patient is in steady state, urea formation correlates with protein intake and protein breakdown.

Fluid restriction:

- CKD stages 4-5: fluid and salt restriction is often important to prevent volume overload.
- On dialysis: when the urine output drops, fluid restriction is vital to minimize weight gains. Aim for weight gains of 1-1.5 kg or less/day. In an uric patient, this means a fluid restriction of 750 – 1000ml. This must be combined with salt restriction.

Protein intake

- Intake averages ~80g/day in the developed world, although requirements may be only 50g.
- A low protein diet has been shown to slow the progression of renal failure in patients with CKD. Set against this is the danger of patients reaching dialysis with significant malnutrition. Most units advocate no more than moderate protein restriction. Daily protein targets:
    - 0.8g/kg per day for CKD stages 3-5
    - 1.2g/kg per day when on dialysis.
- Protein sources include meat, fish, eggs, milk, nuts, pulses, and beans.

Carbohydrate intake

- Adequate energy intake is essential for patient with CKD, especially those undergoing protein restrictions.
- Target 30-35kcal/kg per day.
- Source: mainly complex carbohydrates, some from mono- or poly-unsaturated fats. Dovetailing a diabetic diet with a renal diet can be difficult.
- Examples: sugar, jams, specialist high energy renal drinks.

Phosphate restriction

- The kidney is the main route of phosphate excretion. Current guidelines suggest a restriction of dietary phosphate of 0.8-1g/day if:
    - Serum phosphate >1.5mmol/L in CKD stages 3-4 or

- Serum phosphate >1.8mmol/L in CKD stage 5 or
- ↑PTH

- Prescribe phosphate binders if dietary restriction alone fails
- Phosphate-rich foods include all protein-containing foods, making phosphate restriction difficult to achieve. Examples: milk, cheese, custard, yogurt, ice cream, coal, chocolate drinks, beer, liver, baked beans, dried peas, and beans (e.g. chick peas), nuts, whole grain products, bran cereals, many convenience foods.

Potassium restriction

- Typical UK intake ~50-120mmol/day. With failing renal function, potassium excretion falls making potassium restriction necessary (esp. in patients taking ACEIs).
- K^+- rich foods include dairy products, potatoes (baked, chips, and crisps), some fruits (bannas, dried fruit, fresh pineapple),fresh fruit, juice, tomatoes, sweet corn, mushrooms, chocolate, and coffee.

Salt restriction

- This is a vast excess over physiological needs.
- Salt restriction is helpful if ↑BP ±volume overload. Aim for an intake of <5-6g/day.
- Na+ - rich foods include cheese, salted butter/margarine, salted meat (bacon, ham), tinned meat, vegetables and soups, packaged meals.

3.4. Medical Nutrition Therapy in Chronic Kidney Failure: Inter grating Clinical practice Guidelines:-

This review updates earlier published recommendations and integrates current clinical practice guidelines for disease as recommended by the National Kidney Foundation Kidney Dialysis Outcome Quality Initiative (K/DOQ) disease in adults prior to kidney failure (Stages 1-4), chronic kidney failure with hemodialysis or peritoneal dialysis, and management after kidney transplantation. Multiple diet parameters are necessary to provide optimal of calories, protein, sodium, fluid, potassium, calcium, and phosphorus, as well as other individualized nutrient care within changing kidney function and treatment modality status.[5]

Managing Anemia of Chronic Kidney Disease:-

Anemia begins early in the course of declining kidney function and is a frequent complication of chronic kidney disease are under diagnosed and undertreated. Anemia is associated with significantly increase including increased risks of left ventricular hypertrophy and heart failure. Although the detrimental effects of Anemia with advanced chronic kidney disease, it has been suggested that correcting Anemia in early stage kidney quality of life and also delay the progression to end – stage kidney disease. The identification of Anemia in early its aggressive management may also improve cardiovascular complications. Anemia of chronic kidney disease is abnormal erythropoietin production and iron deficiency. Anemia may be the result of kidney failure itself, metabolic and endocrine disorders. Guidelines and protocols for treating Anemia can assist practitioners in identifying patients evaluating response to treatment, and modifying treatment based on response. Erythropoesis stimulating agents in treat Anemia in pre dialysis and dialysis patients. Iron supplementation is usually required in patients or with iron deficiency. Successfully managing Anemia of chronic kidney disease with treatment patient lifestyle and improve compliance is paramount.

Growth and Body Composition in Children with Chronic kidney Disease:

Growth failure is a common yet complex problem of childhood chronic kidney disease caused by multiple factor disease or secondary to the renal impairment. This review seeks to describe the various pathophysiological failures in the various stages of

childhood with particular emphasis on nutritional problems and endocrine dysfunction these children. In addition, we shall examine the role of body composition in chronic kidney disease, their relation the potential effect of abnormalities in fat mass and lean mass on long-term morbidity and mortality.

A Challenge to Chronic Kidney Disease in Asia: The Report of the Second Asian Forum of Chronic Kidney

Background: The Asian Forum of Chronic Disease Initiative started in 2007 in Hamamatsu, Japan when together to facilitate collaboration in studying chronic kidney disease (CKD) in the Asia – Pacific region. Based the second meeting was organized as a consensus conference to frame the most relevant issues, and developer action plan. Proceedings: the meeting was held on 4 May 2008 as a pre-conference meeting to the 11th Asia Kuala Lumpur. This meeting consisted of three sessions: Session I was dedicated to the estimation of standardization of serum creatinine measurements. Session II discussed specific considerations in the etiology renal disease in Asia. We concluded that there were regional specific problems that might lead to a very disease. Session III discussed the issue of facilitation of coordination and integration of the CKD initiative developing countries in the Asia-Pacific region. Conclusion: The following action plans were formulated: (i) validating the hyper filtration rate equation or creating a new one using serum creatinine standardized by a central laboratory; registry to facilitate risk analysis of CKD and its morbidities; (iii) adapting existing clinical practice and management to address specific problems in this region; and (iv) working closely with other international manpower development and education in different aspects of CKD in developing countries.

Therapeutic Potential of Endothelial Receptor Antagonists for Chronic Proteinuric Renal Disease in Humans

Diabetes and arterial hypertension continue to be the main causes of chronic renal failure in 2010, with a rising prevalence in part due to the worldwide obesity epidemic. Proteinuria is a main feature of chronic renal disease and mediated by defects in the glomerular filtration barrier and is as a good predictor of cardiovascular events. Indeed, chronic renal disease due to glomerulosclerosis is one of the important risk factors for the development of coronary artery disease and stroke. Glomerulosclerosis develops in response to inflammatory activation and increased growth factor production. Preclinical

and first preliminary clinical studies provide strong evidence that endogenous endothelial1 (ET-1), a 21-amino-acid peptide with strong growth-promoting and vasoconstriction properties, plays a central role in the pathogenesis of proteinuria and glomerulosclerosis via activation of its ETA subtype receptor involving podocyte injury. These studies have not only shown that endothelial participates in the disease processes of hypertension and glomerulosclerosis but also that features of chronic renal disease such as proteinuria and glomerulosclerosis are reversible processes. Remarkably, the protective effects of endothelial receptors antagonists (ERAs) are present even on top of concomitant treatments with inhibitors of the rennin-angiotensin system. This review discusses current evidence for a role of endothelial for proteinuria renal disease and podocyte injury in diabetes and arterial hypertension and reviews the current status of endothelial receptor antagonists as a potential new treatment option in renal medicine.

Nutrition in Advanced Chronic Kidney Disease: Biomarkers and Body Composition Tools

Patients with stage 5 chronic kidney disease, also known as end- stage renal disease, must undergo a Hemodialysis is the most commonly used therapy. There have been many advances in this therapy over remain unacceptably high. Many of the known risk factors associated with hemodialysis are nutritionally related to have clinically meaningful ways to assess the protein and energy nutritional status of patients undergoing renal disease. Although there is no one “gold standard” method to assess nutritional status, there are concurrently for this purpose. This article provides descriptions of the most commonly used and readily available tools recommended by the Kidney Disease Outcomes Quality Initiative Clinical Practice Guidelines for Nutrition National Kidney Foundation.

4. Previous Work Done Review:

1. Research in Ayurveda: - Dr. M. S. Baghel.
2. To study the effect of Ayurveda treatment in cases of mootraghat and CRF – Vd. Mante Ganesh – 1994 – (Pune University).
3. The study of etiology of Shotha and nidana samprapti – Vd. Maheshavari K. – 1992 (Jaipur University).
4. A study of pathophysiology of urinary tract disorders w.s.r. to Mootravaha Strotas – Vd. Pattare A. G. – 1983 (Jamnagar).
5. Structures and functions of Urinary system with ref. to Mootravaha Strotas dushti and its principle of management by Ashmarihar Kwath Vd. Waghani C. M. – 1993 (Jamnagar).
6. Evaluation of Renal function test with concomitant use of Ayurvedic Mootral drugs in patients of C. R. F.
7. Study of Panchakarma w.s.r. to swedan therapy (Avgah) in nephrotic syndrome – Vd. Acharya Sripatilggo – (B.H.U.).

5. Observation:-

5.1 Frequency Table:

Trushna

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Prakrut	56	50.9	50.9	50.9
	Aprakrut	54	49.1	49.1	100.0
	Total	110	100.0	100.0	

Uvlua

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	48	43.6	43.6	43.6
	Elongated	53	48.2	48.2	91.8
	Other	9	8.2	8.2	100.0
	Total	110	100.0	100.0	

Jivaha

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Niram	16	14.5	14.5	14.5
	Sama	94	85.5	85.5	100.0
	Total	110	100.0	100.0	

Agni

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Sama	48	43.6	43.6	43.6
	Vishama	17	15.5	15.5	59.1
	Tikshna	11	10.0	10.0	69.1
	Manda	34	30.9	30.9	100.0
	Total	110	100.0	100.0	

Frequency Table

Abhyvaran

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Prakrut	82	74.5	74.5	74.5
	Aprakrut	28	25.5	25.5	100.0
	Total	110	100.0	100.0	

Twak normal

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	82	54.7	74.5	74.5
	Present	28	18.7	25.5	100.0
	Total	110	73.3	100.0	

Twakdry

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	39	26.0	35.5	35.5
	Present	71	47.3	64.5	100.0
	Total	110	73.3	100.0	

Twakscaly

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	87	58.0	79.1	79.1
	Present	23	15.3	20.9	100.0
	Total	110	73.3	100.0	

Twakscracthmark

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	92	61.3	83.6	83.6
	Present	18	12.0	16.4	100.0
	Total	110	73.3	100.0	

Twakpale

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	78	52.0	70.9	70.9
	Present	32	21.3	29.1	100.0
	Total	110	73.3	100.0	

Eyespallor

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	29	26.4	26.4	26.4
	Pallor	81	73.6	73.6	100.0
	Total	110	100.0	100.0	

Musclefatigue

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	37	33.6	33.6	33.6
	Present	73	66.4	66.4	100.0
	Total	110	100.0	100.0	

Nailspale

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	19	17.3	17.3	17.3
	Present	91	82.7	82.7	100.0
	Total	110	100.0	100.0	

Maladaily

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Daily	104	94.5	95.4	95.4
	With Medication	6	4.5	4.6	100.0
	Total	110	99.1	100.0	

Malakurchha

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Daily	79	71.8	71.8	71.8
	Kurchha	31	28.2	28.2	100.0
	Total	110	100.0	100.0	

Swaeda specific season

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	35	31.8	31.8	31.8
	Yes	75	68.2	68.2	100.0
	Total	110	100.0	100.0	

Sweda all season

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	101	91.8	91.8	91.8
	Yes	9	8.2	8.2	100.0
	Total	110	100.0	100.0	

Hypertension

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	36	32.7	32.7	32.7
	Present	74	67.3	67.3	100.0
	Total	110	100.0	100.0	

Frequency Table

DM

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	70	63.6	63.6	63.6
	Present	40	36.4	36.4	100.0
	Total	110	100.0	100.0	

HT + DM

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	83	75.5	75.5	75.5
	Present	27	24.5	24.5	100.0
	Total	110	100.0	100.0	

Auscultation

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	75	68.2	68.2	68.2
	Present	35	31.8	31.8	100.0
	Total	110	100.0	100.0	

Nasal

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	DNS	14	12.7	12.7	12.7
	Dryness	76	69.1	69.1	81.8
	Polyp	20	18.2	18.2	100.0
	Total	110	100.0	100.0	

Lips

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0 Normal	26	23.6	23.6	23.6
	1 Dryness	51	46.4	46.4	70.0
	2 scaly	33	30.0	30.0	100.0
	Total	110	100.0	100.0	

Soft palate

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	69	62.7	62.7	62.7
	Dry	41	37.3	37.3	100.0
	Total	110	100.0	100.0	

Oral cavity

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	45	40.9	40.9	40.9
	Less	62	56.4	56.4	97.3
	excess	3	2.7	2.7	100.0
	Total	110	100.0	100.0	

Pigmentation

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	73	66.4	66.4	66.4
	Present	37	33.6	33.6	100.0
	Total	110	100.0	100.0	

Frequency Table

Dialysis

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	73	66.4	66.4	66.4
	Present	37	33.6	33.6	100.0
	Total	110	100.0	100.0	

Sandhi kriyakashata

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	83	75.5	75.5	75.5
	Present	27	24.5	24.5	100.0
	Total	110	100.0	100.0	

Mala formed

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Daily	27	24.5	24.5	24.5
	Formed	83	75.5	75.5	100.0
	Total	110	100.0	100.0	

Mutra Day Frequency

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1 to 4 times	50	45.5	45.5	45.5
	4 to 8 times	57	51.8	51.8	97.3
	8 to 12 times	3	2.7	2.7	100.0
	Total	110	100.0	100.0	

Frequency Table

Mutra Night Frequency

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1 to 4 times	23	20.9	20.9	20.9
	4 to 8 times	76	69.1	69.1	90.0
	8 to 12 times	11	10.0	10.0	100.0
	Total	110	100.0	100.0	

Mutra Pain Burning

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	90	81.8	81.8	81.8
	Yes	20	18.2	18.2	100.0
	Total	110	100.0	100.0	

Stage

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	15	13.6	13.6	13.6
	2	10	9.1	9.1	22.7
	3	18	16.4	16.4	39.1
	4	31	28.2	28.2	67.3
	5	36	32.7	32.7	100.0
	Total	110	100.0	100.0	

5.2 Graphs Observation:

1. Age Profile Analysis

Table 1 Age Distribution of study group	
Age Groups	**Total No of Paitents**
Less than 30 yr	**20**
30-40 yr	**13**
40-50 yr	**23**
50-60 yr	**26**
60 -70 yr	**12**
above 70 yr	16

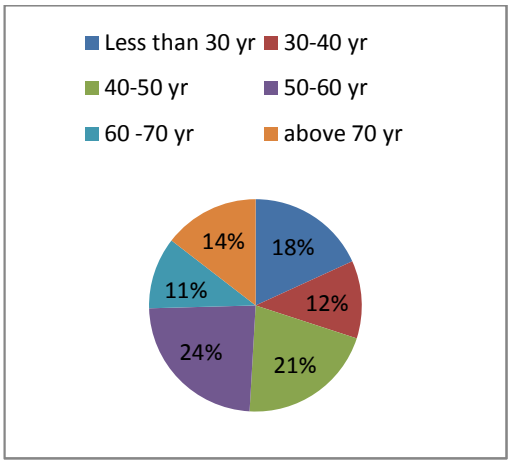


Figure 1
Age Distribution of Study Group

2. Occupation of Study Group:-

Table 2 Occupation of Study Group	
Occupation	Total No of Paitents
Students	**8**
HW & RETD	**34**
Service	**37**
Business	**14**
Heavy workers	**17**

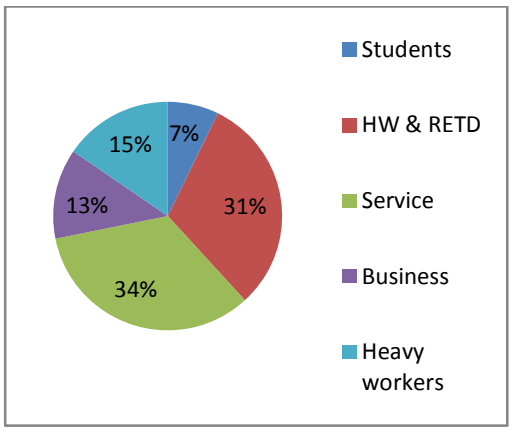


Table 2
Occupation of Study Group

3. Distribution of Gender:

Table 3 Gender Distribution	
Gender	**Total No.**
F	**41**
M	**69**

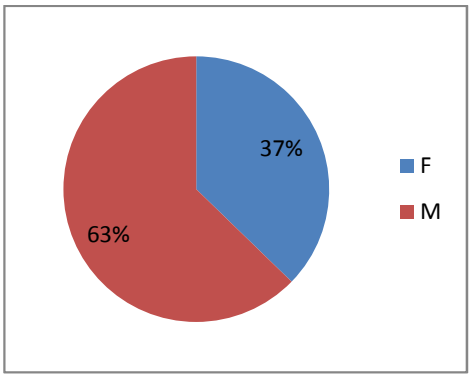


Table 3
Gender Distribution

4. Description of Stages in total:-

Table 4 Stages Distribution	
Stages	**Frequency**
1	15
2	10
3	18
4	31
5	36

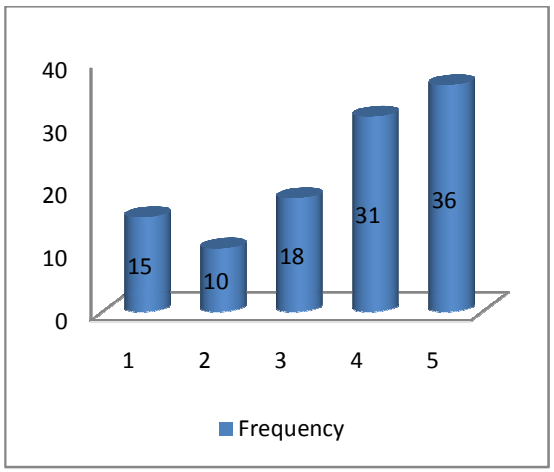


Table 4
Stages Distribution

5. Frequencies of DM:-

Table 5 Frequencies of DM	
DM	**Frequency**
0	**70**
1	**40**

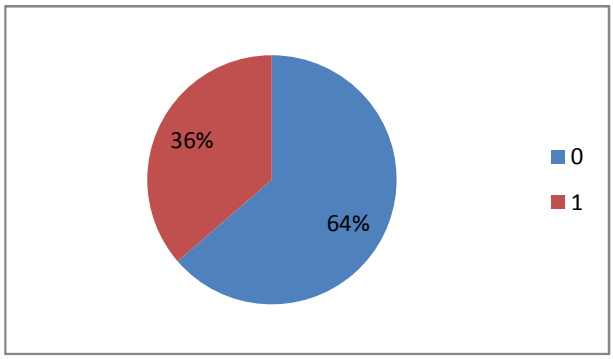


Table 5
Frequencies of DM

6. Frequencies of HT:-

Table 6 Frequencies of HT	
HT	**Frequency**
0	**36**
1	**74**

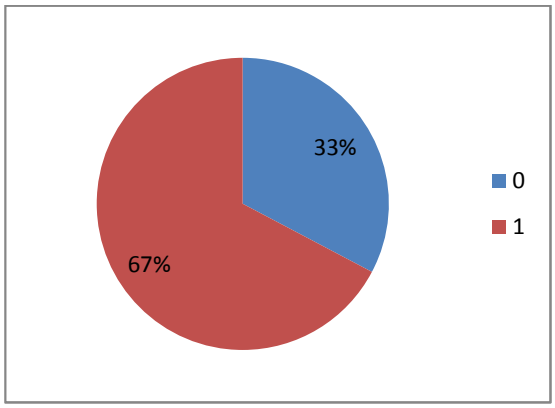


Table 6
Frequencies of HT

7. Frequencies of DM + HT:-

Table 7 Frequencies of DM + HT	
DM + HT	**Frequency**
0	**83**
1	**27**

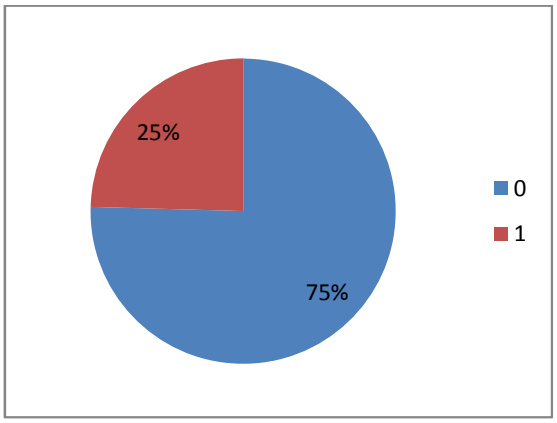


Table 7
Frequencies of HT

8. Frequencies of Anemia:-

Table 8 Frequencies of Anemia	
Anemia	**Frequency**
0	**59**
1	**51**

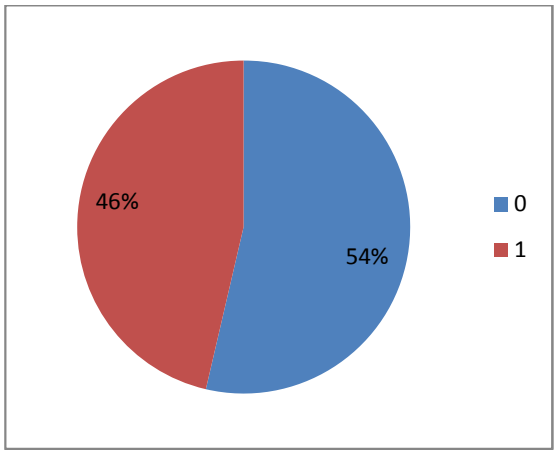


Table 8
Frequencies of Anemia

9. Frequencies of Dialysis :-

Table 9 Frequencies of Dialysis	
Dialysis	**Frequency**
Ab	**73**
P	**37**

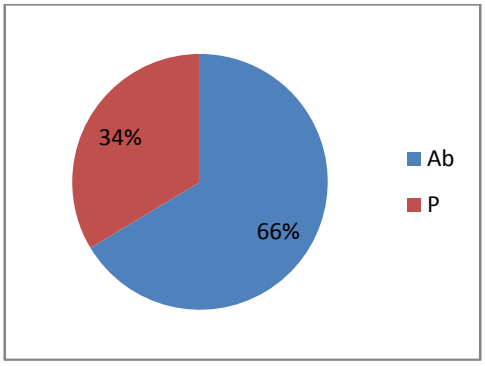


Table 9
Frequencies of Dialysis

10. Frequencies of Maternal:-

Table 10 Frequencies of Maternal	
Maternal	**Frequency**
Valid	83
Arthritis	2
Asthma	2
Cancer	3
DM	12
Heart Disease	1
HT	3
HT DM kidney disease	1
Kidney Disease	1
Liver Cirrhosis, Asicitis	1
polycystic kidney	1

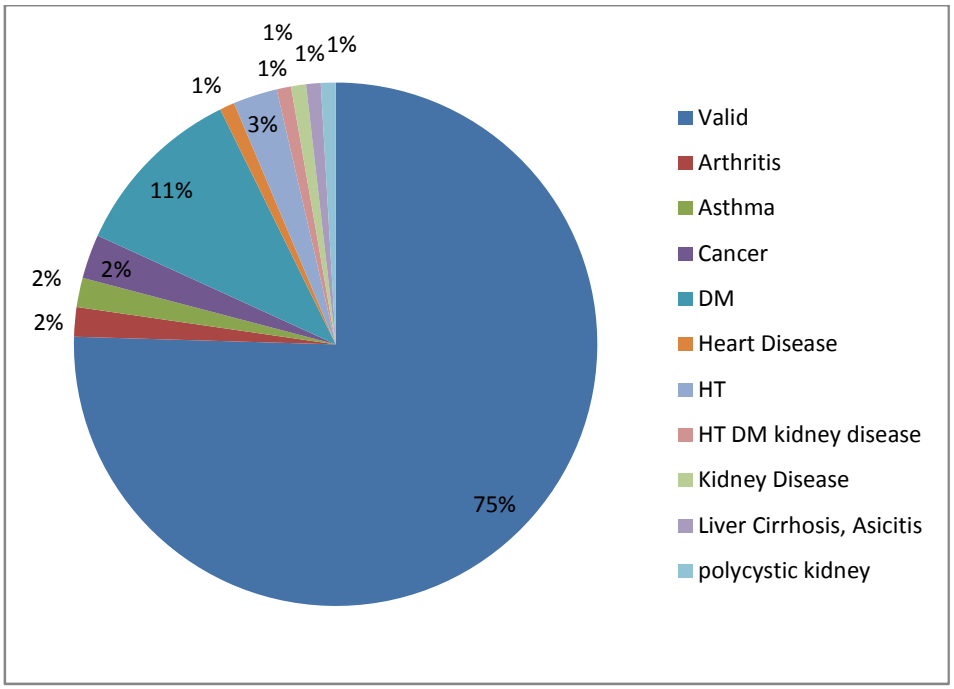


Table 10
Frequencies of Maternal

11. Frequencies of Paternal:-

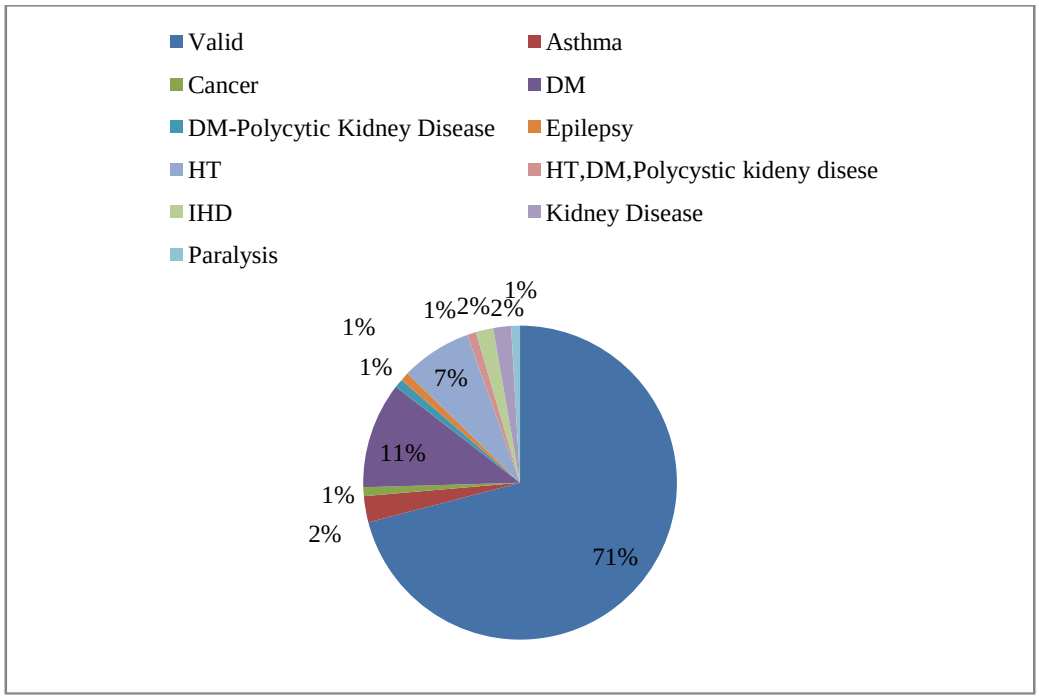


Table 11
Frequencies of Paternal

Table 11 Frequencies of Paternal	
Paternal	**Frequency**
Valid	**78**
Asthma	**3**
Cancer	**1**
DM	**12**
DM-Polycytic Kidney Disease	**1**
Epilepsy	**1**
HT	**8**
HT,DM,Polycystic kidney disese	**1**
IHD	**2**
Kidney Disease	**2**
Paralysis	**1**

12. Frequencies of Swakula :-

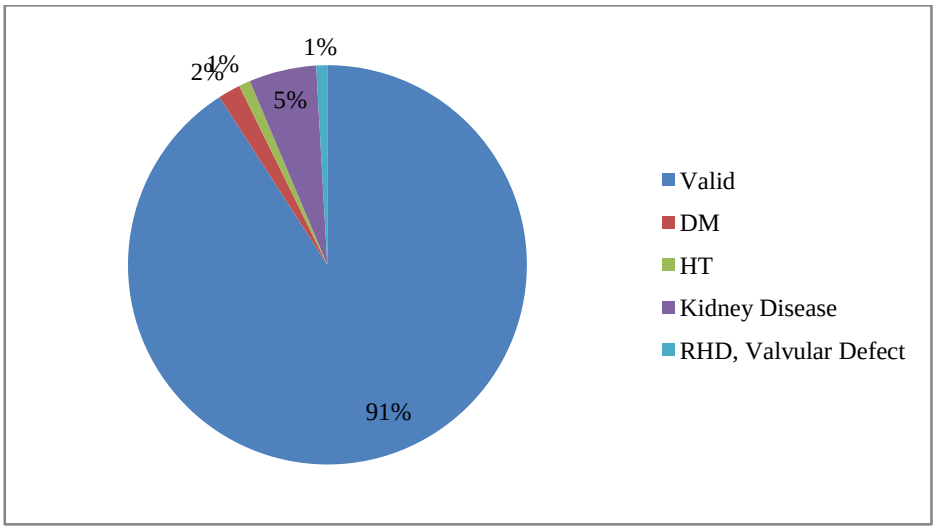
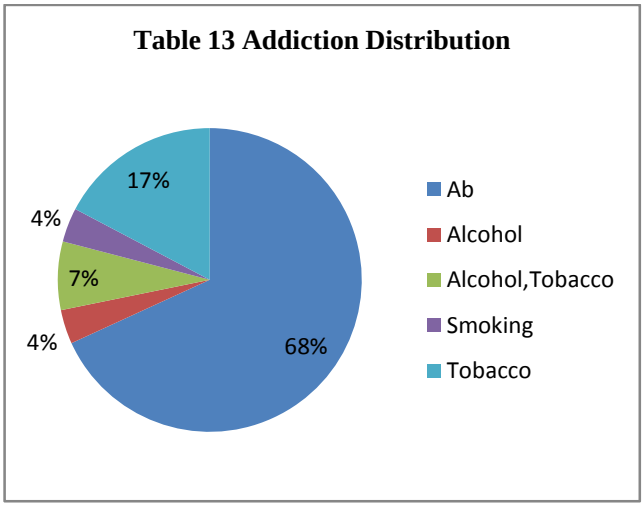


Table 12
Frequencies of Swakula

Table 12 Frequencies of Swakula	
Swakula	**Frequency**
Valid	**100**
DM	**2**
HT	**1**
Kidney Disease	**6**
RHD, Valvular Defect	**1**

13 Addiction Distributions

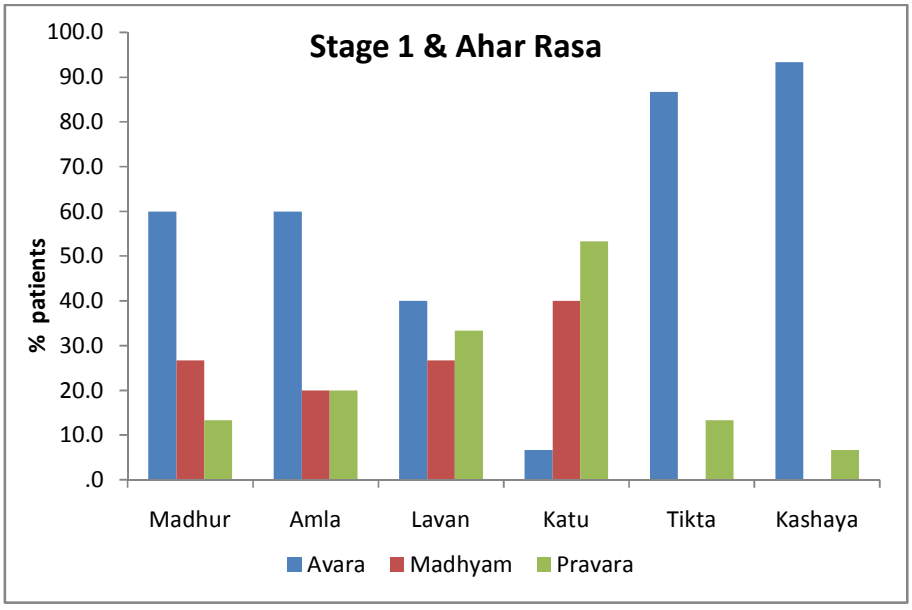
Table 13 Addiction Distribution	
Addiction	**Frequency**
Ab	75
Alcohol	4
Alcohol,Tobacco	8
Smoking	4
Tobacco	19

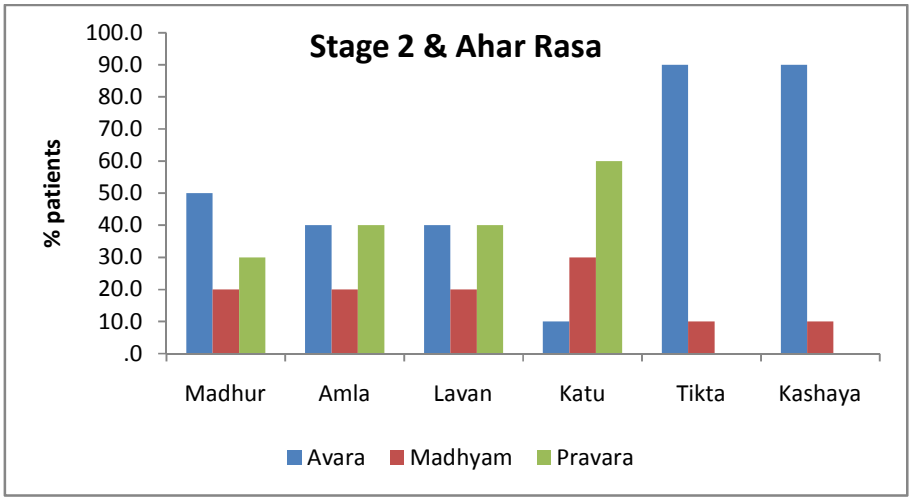


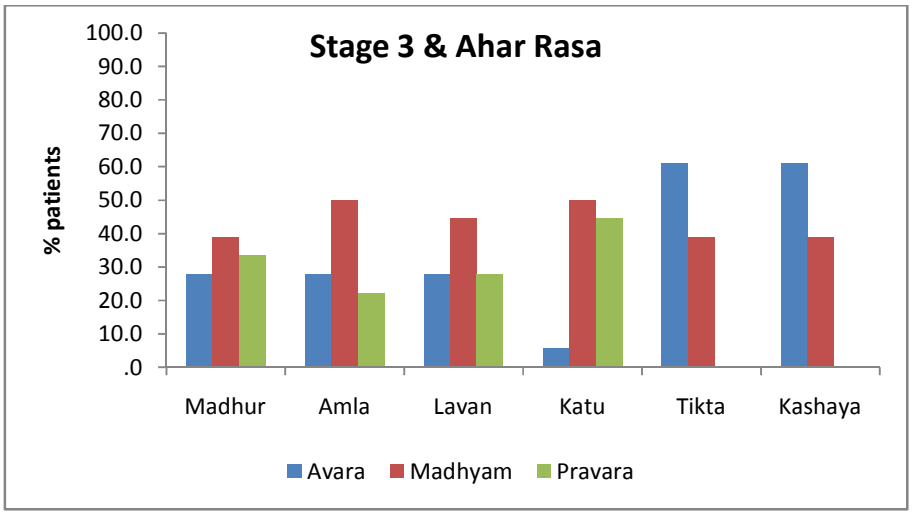
14 Stages & Ahar Rasa:-

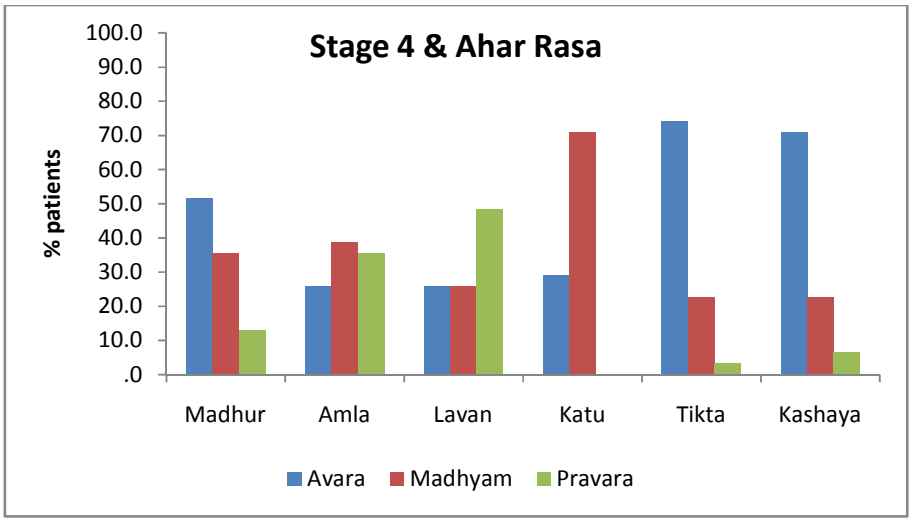
Table 14 Stage & Ahar Rasa

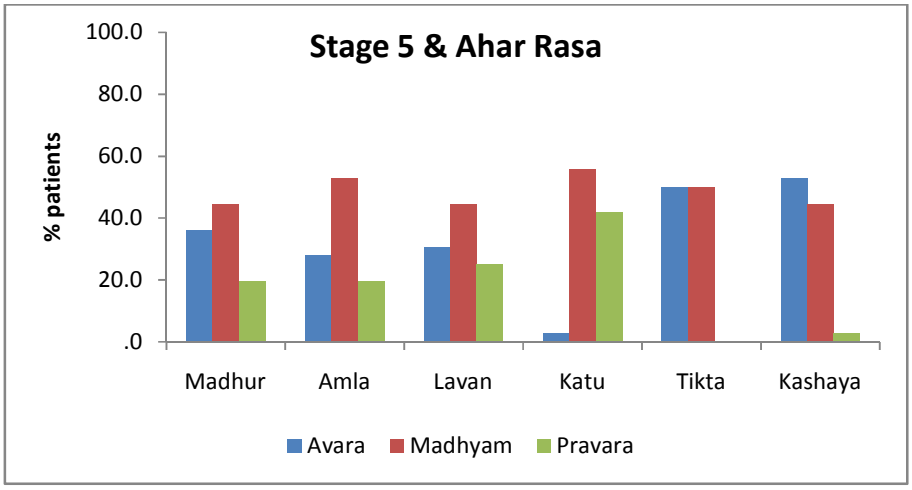
Stages	Ahar Rasa	Madhur	Amla	Lavan	Katu	Tikta	Kasaya
Stage 1	Avara	60.0	60.0	40.0	6.7	86.7	93.3
	Madhyam	26.7	20.0	26.7	40.0	.0	.0
	Pravara	13.3	20.0	33.3	53.3	13.3	6.7
Stage 2	Avara	50.0	40.0	40.0	10.0	90.0	90.0
	Madhyam	20.0	20.0	20.0	30.0	10.0	10.0
	Pravara	30.0	40.0	40.0	60.0	.0	.0
Stage 3	Avara	27.8	27.8	27.8	5.6	61.1	61.1
	Madhyam	38.9	50.0	44.4	50.0	38.9	38.9
	Pravara	33.3	22.2	27.8	44.4	.0	.0
Stage 4	Avara	51.6	25.8	25.8	29.0	74.2	71.0
	Madhyam	35.5	38.7	25.8	71.0	22.6	22.6
	Pravara	12.9	35.5	48.4		3.2	6.5
Stage 5	Avara	36.1	27.8	30.6	2.8	50.0	52.8
	Madhyam	44.4	52.8	44.4	55.6	50.0	44.4
	Pravara	19.4	19.4	25.0	41.7	.0	2.8




Stage 2 & Ahar Rasa
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Madhur
Amla
Lavan
Katu
Tikta
Kashaya
Avara
Madhyam
Pravara


Stage 3 & Ahar Rasa
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Madhur
Amla
Lavan
Katu
Tikta
Kashaya
Avara
Madhyam
Pravara

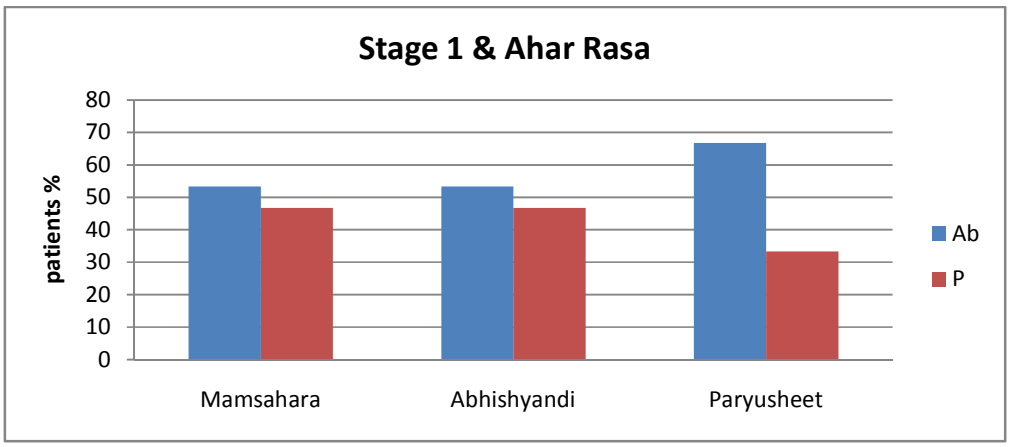

Stage 4 & Ahar Rasa
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Madhur
Amla
Lavan
Katu
Tikta
Kashaya
Avara
Madhyam
Pravara

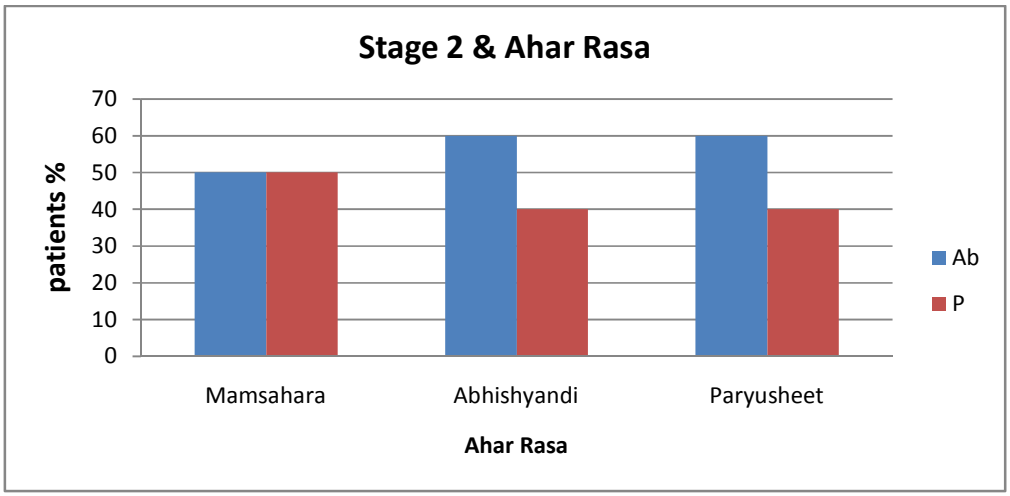


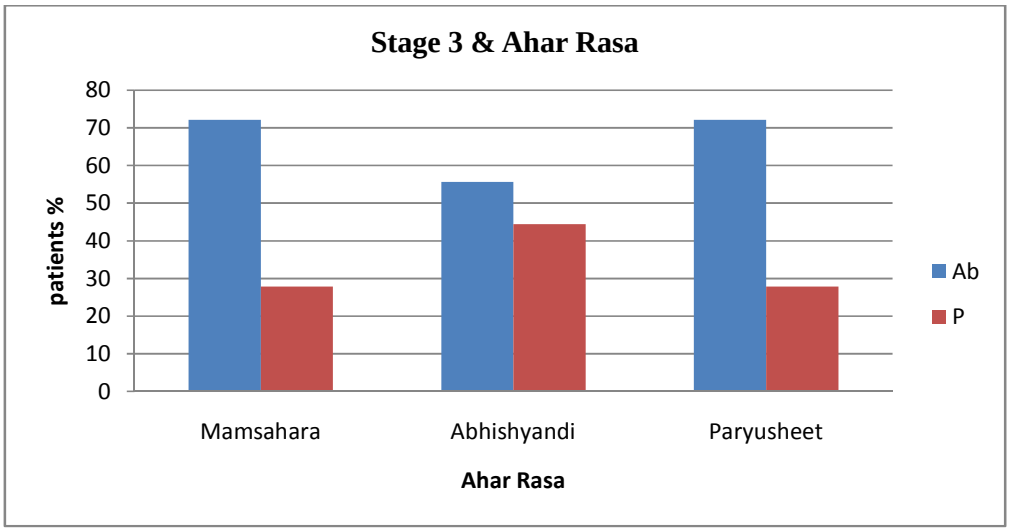
15 Stages & Ahar Rasa:-

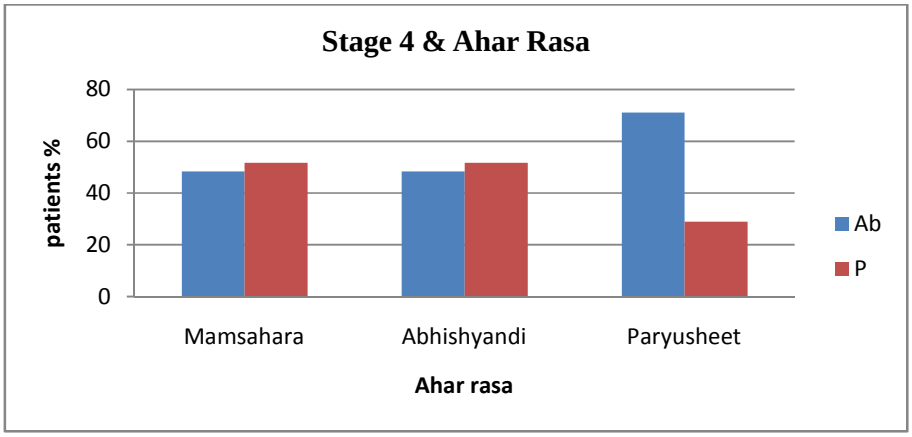
Table 15 Stage & Ahar Rasa

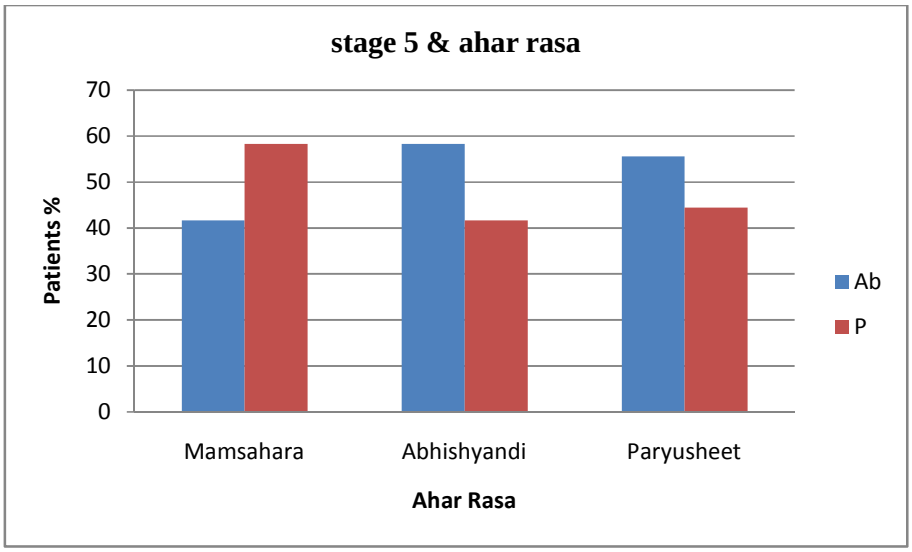
Stages	Ahar Rasa	Mamsahara	Abhishyandi	Paryusheet
Stage 1	Ab	53.3	53.3	66.7
	P	46.7	46.7	33.3
Stage 2	Ab	50.0	60.0	60.0
	P	50.0	40.0	40.0
Stage 3	Ab	72.2	55.6	72.2
	P	27.8	44.4	27.8
Stage 4	Ab	48.4	48.4	71.0
	P	51.6	51.6	29.0
Stage 5	Ab	41.7	58.3	55.6
	P	58.3	41.7	44.4


Stage 1 & Ahar Rasa
patients %
80
70
60
50
40
30
20
10
0
Mamsahara
Abhishyandi
Paryusheet
Ab
P


Stage 2 & Ahar Rasa
patients %
70
60
50
40
30
20
10
0
Mamsahara
Abhishyandi
Paryusheet
Ahar Rasa
Ab
P

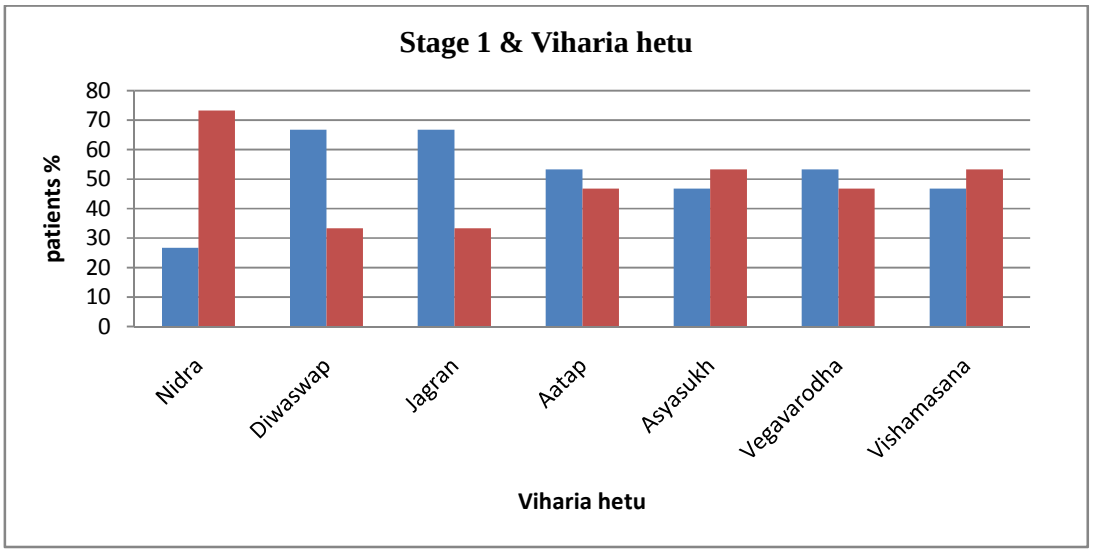

Stage 3 & Ahar Rasa
patients %
80
70
60
50
40
30
20
10
0
Mamsahara
Abhishyandi
Paryusheet
Ahar Rasa
Ab
P

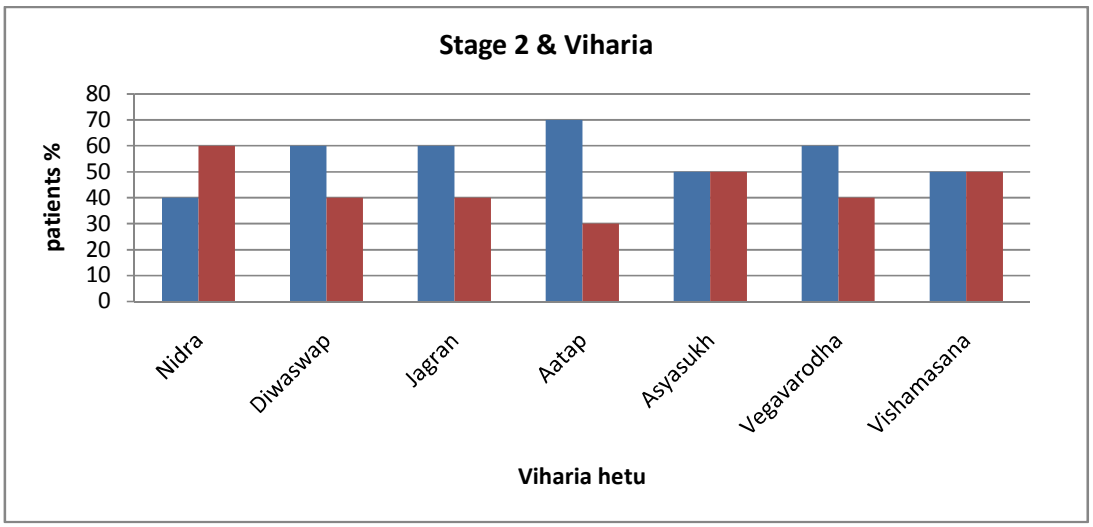

Stage 4 & Ahar Rasa
80
60
40
20
0
patients %
Mamsahara
Abhishyandi
Paryusheet
Ahar rasa
Ab
P

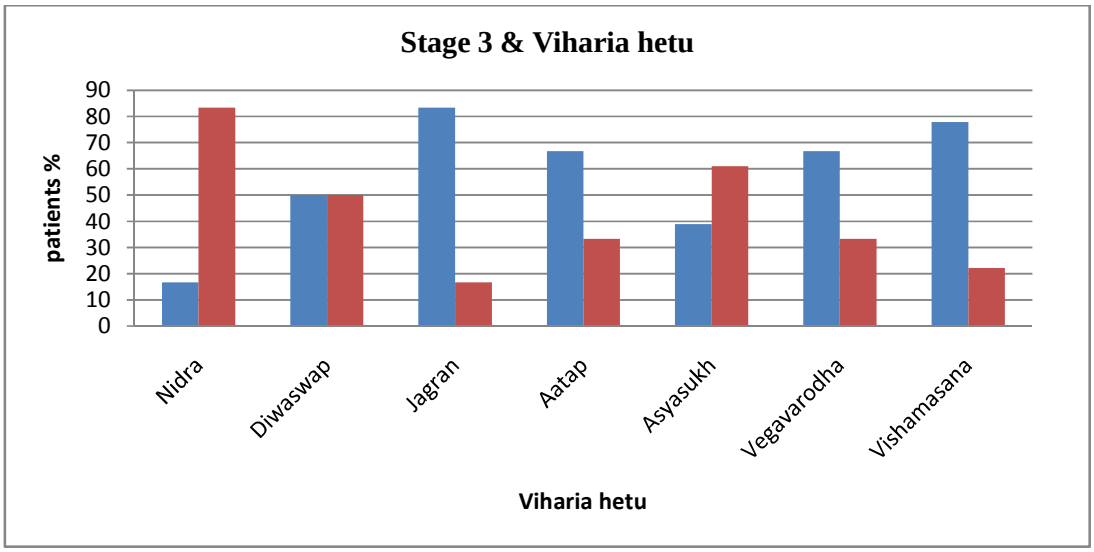

stage 5 & ahar rasa
70
60
50
40
30
20
10
0
Patients %
Mamsahara
Abhishyandi
Paryusheet
Ahar Rasa
Ab
P

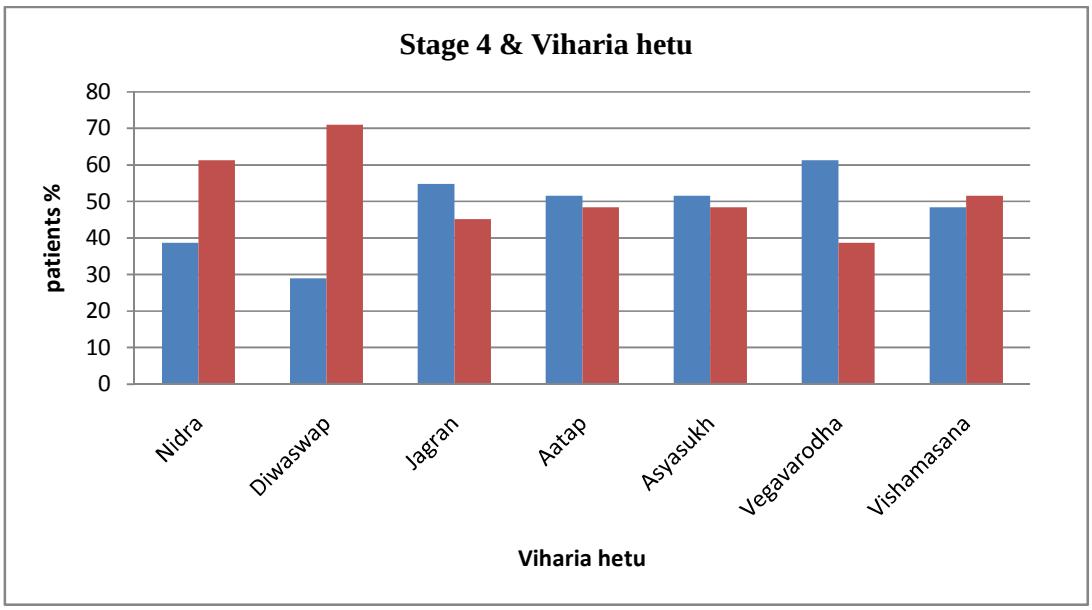
16 Stage & Viharia:-

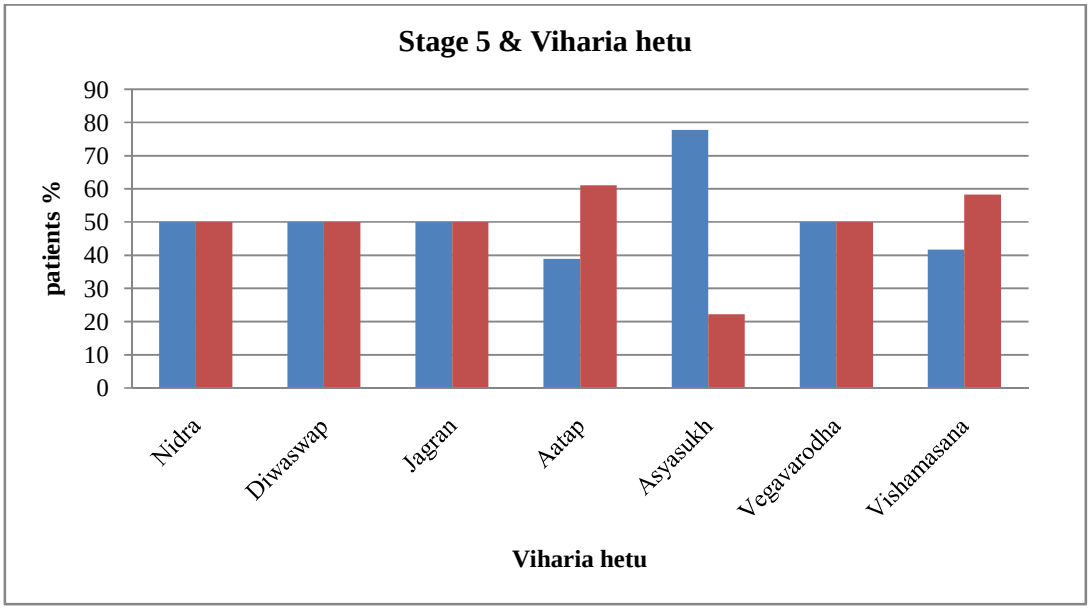
Table 16 Stage & Viharia								
Stages	Viharia	Nidra	Diwaswap	Jagran	Aatap	Asyasukh	Vegavarodha	Vishamasana
Stage 1	Ab	26.7	66.7	66.7	53.3	46.7	53.3	46.7
	P	73.3	33.3	33.3	46.7	53.3	46.7	53.3
Stage 2	Ab	40.0	60.0	60.0	70.0	50.0	60.0	50.0
	P	60.0	40.0	40.0	30.0	50.0	40.0	50.0
Stage 3	Ab	16.7	50.0	83.3	66.7	38.9	66.7	77.8
	P	83.3	50.0	16.7	33.3	61.1	33.3	22.2
Stage 4	Ab	38.7	29.0	54.8	51.6	51.6	61.3	48.4
	P	61.3	71.0	45.2	48.4	48.4	38.7	51.6
Stage 5	Ab	50.0	50.0	50.0	38.9	77.8	50.0	41.7
	P	50.0	50.0	50.0	61.1	22.2	50.0	58.3




Stage 2 & Viharia
patients %
80
70
60
50
40
30
20
10
0
Nidra
Diwaswap
Jagran
Aatap
Asyasukh
Vegavarodha
Vishamasana
Viharia hetu

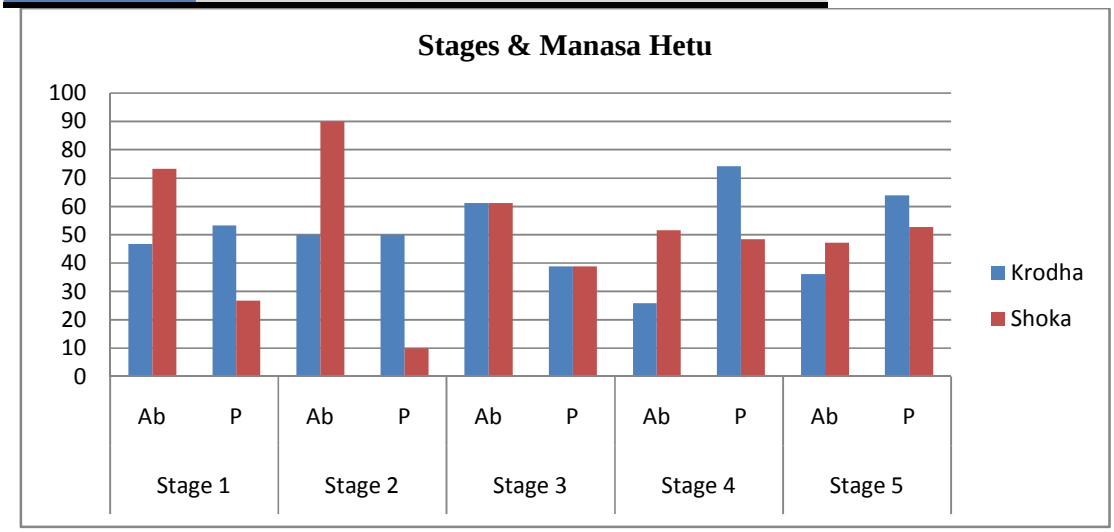

Stage 3 & Viharia hetu
patients %
90
80
70
60
50
40
30
20
10
0
Nidra
Diwaswap
Jagran
Aatap
Asyasukh
Vegavarodha
Vishamasana
Viharia hetu


Stage 4 & Viharia hetu
patients %
80
70
60
50
40
30
20
10
0
Nidra
Diwaswap
Jagran
Aatap
Asyasukh
Vegavarodha
Vishamasana
Viharia hetu


Stage 5 & Viharia hetu
patients %
90
80
70
60
50
40
30
20
10
0
Nidra
Diwaswap
Jagran
Aatap
Asyasukh
Vegavarodha
Vishamasana
Viharia hetu

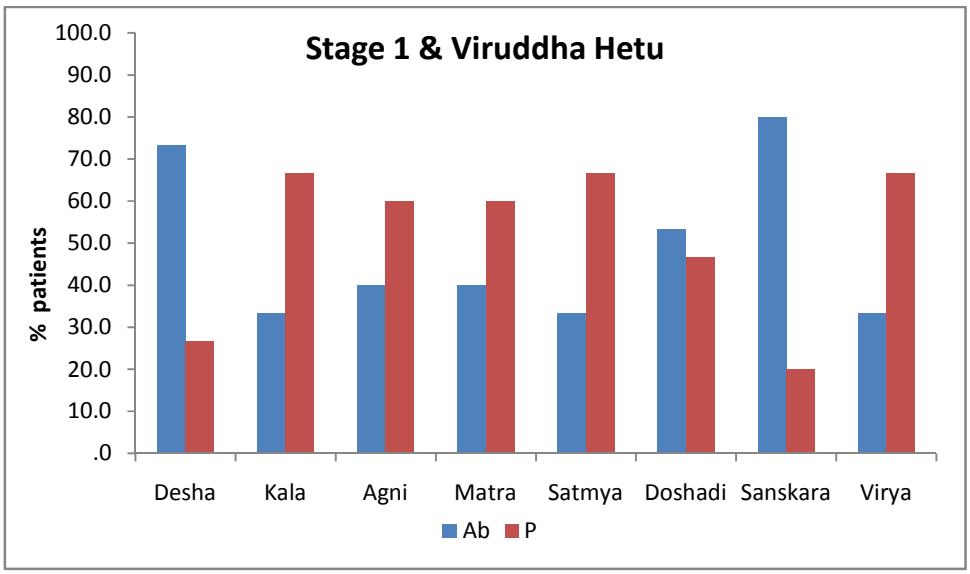
17 Stages & Manasa Hetu:-

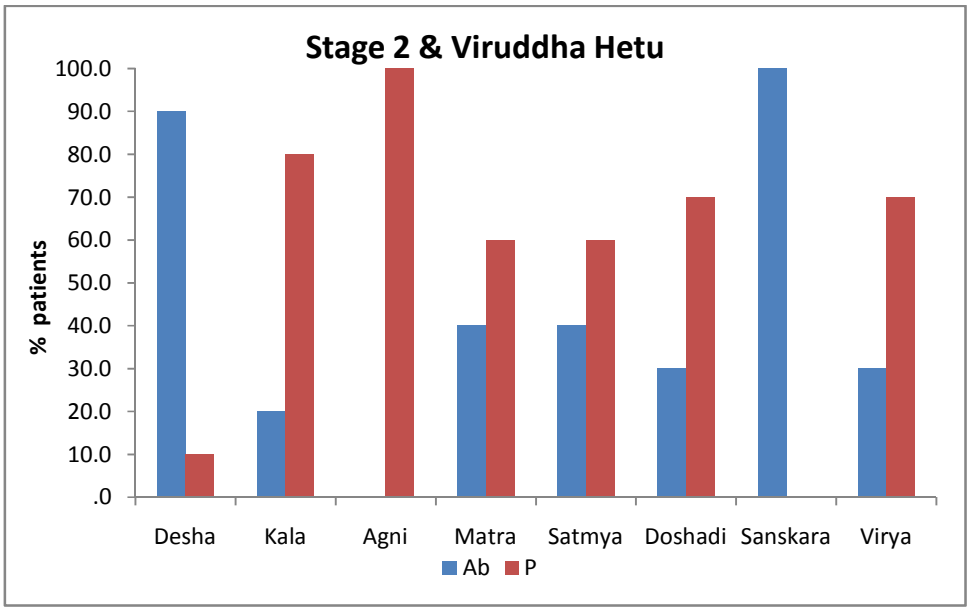
Table 17 Stages & Manasa Hetu			
Stages	Manasa Hetu	Krodha	Shoka
Stage 1	Ab	46.7	73.3
	P	53.3	26.7
Stage 2	Ab	50.0	90.0
	P	50.0	10.0
Stage 3	Ab	61.1	61.1
	P	38.9	38.9
Stage 4	Ab	25.8	51.6
	P	74.2	48.4
Stage 5	Ab	36.1	47.2
	P	63.9	52.8

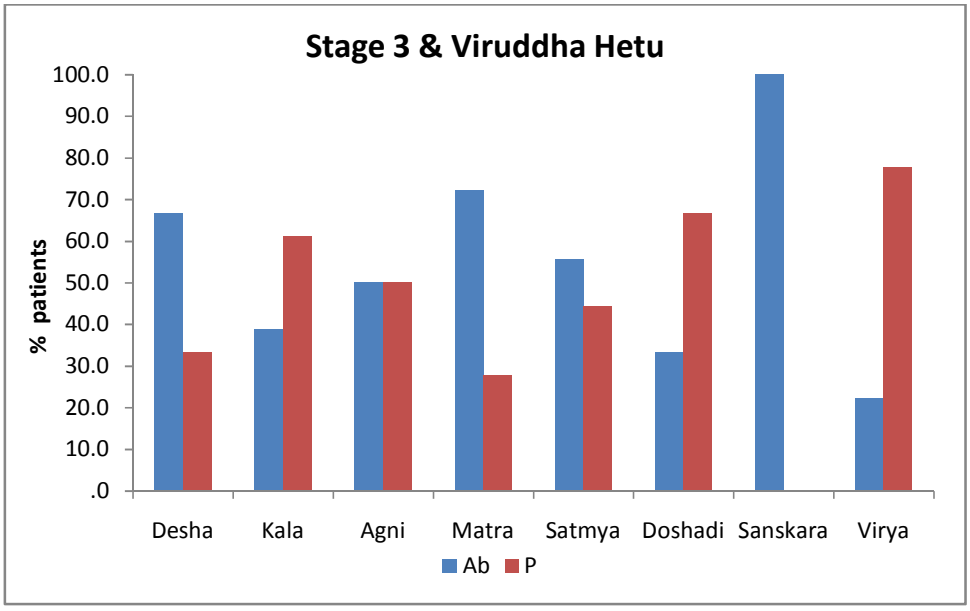


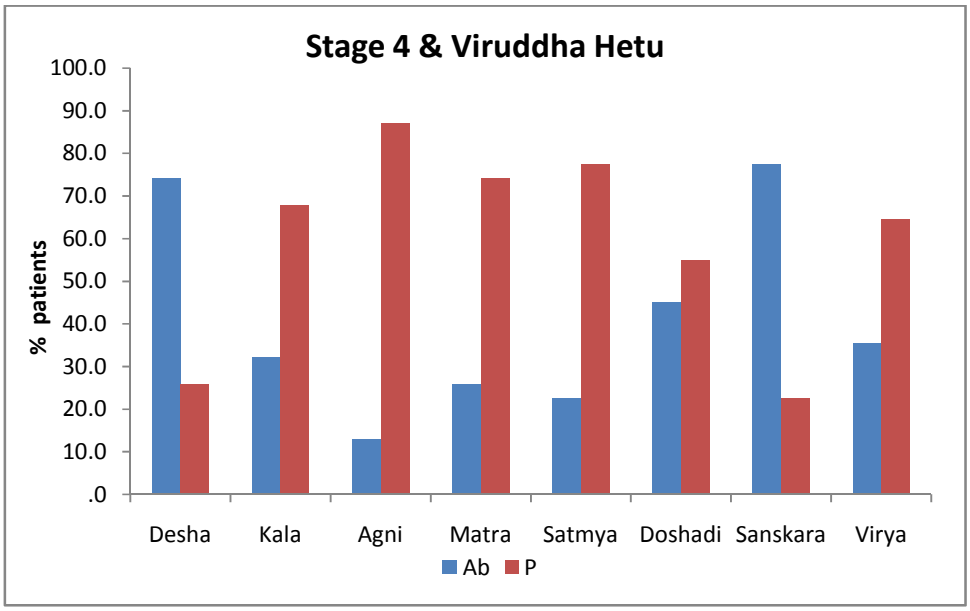
18 Stages & Viruddha Hetu

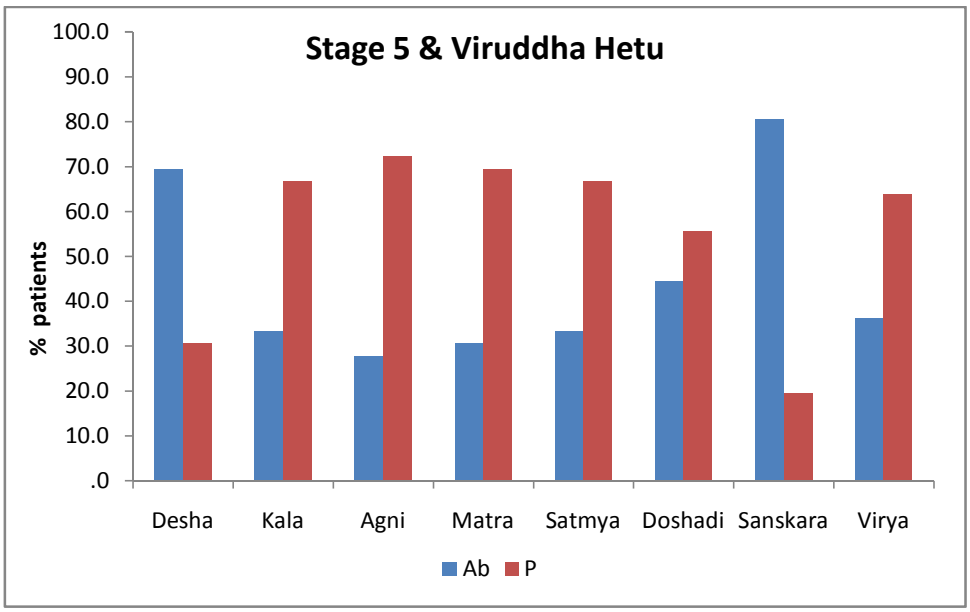
Table 18 Stage & Viruddha Hetu									
Stages	V. Hetu	Desha	Kala	Agni	Matra	Satmya	Doshadi	Sanskara	Virya
Stage 1	Ab	73.3	33.3	40.0	40.0	33.3	53.3	80.0	33.3
	P	26.7	66.7	60.0	60.0	66.7	46.7	20.0	66.7
Stage 2	Ab	90.0	20.0	.0	40.0	40.0	30.0	100.0	30.0
	P	10.0	80.0	100.0	60.0	60.0	70.0	.0	70.0
Stage 3	Ab	66.7	38.9	50.0	72.2	55.6	33.3	100.0	22.2
	P	33.3	61.1	50.0	27.8	44.4	66.7	.0	77.8
Stage 4	Ab	74.2	32.3	12.9	25.8	22.6	45.2	77.4	35.5
	P	25.8	67.7	87.1	74.2	77.4	54.8	22.6	64.5
Stage 5	Ab	69.4	33.3	27.8	30.6	33.3	44.4	80.6	36.1
	P	30.6	66.7	72.2	69.4	66.7	55.6	19.4	63.9


Stage 1 & Viruddha Hetu
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
% patients
Desha
Kala
Agni
Matra
Satmya
Doshadi
Sanskara
Virya
Ab
P


Stage 2 & Viruddha Hetu
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
% patients
Desha
Kala
Agni
Matra
Satmya
Doshadi
Sanskara
Virya
Ab
P


Stage 3 & Viruddha Hetu
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
% patients
Desha
Kala
Agni
Matra
Satmya
Doshadi
Sanskara
Virya
Ab
P

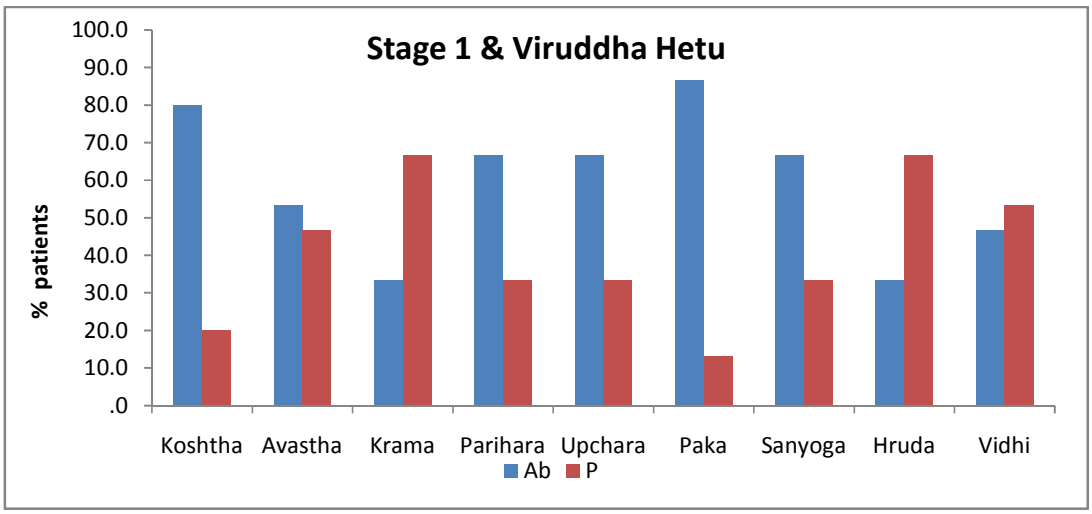

Stage 4 & Viruddha Hetu
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
% patients
Desha
Kala
Agni
Matra
Satmya
Doshadi
Sanskara
Virya
Ab
P

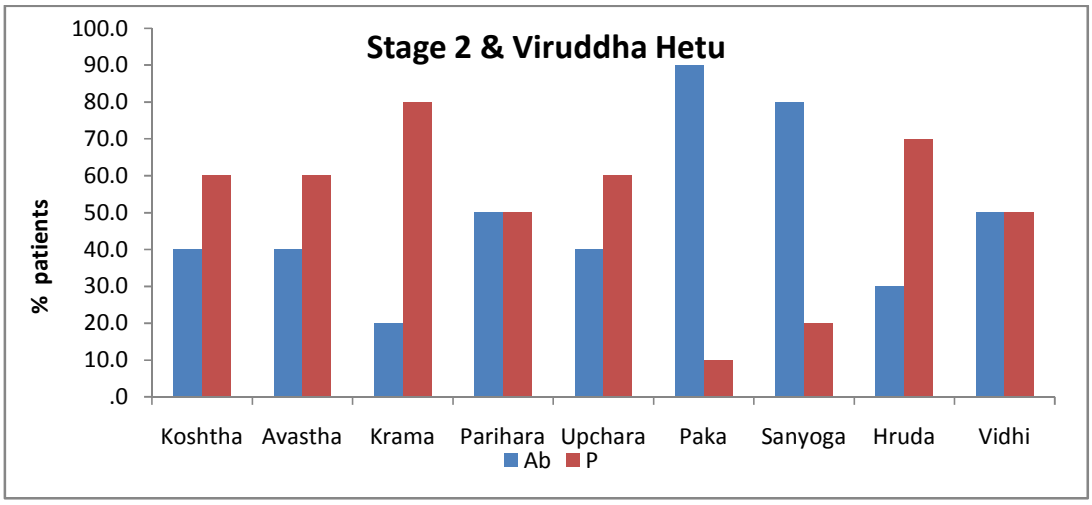


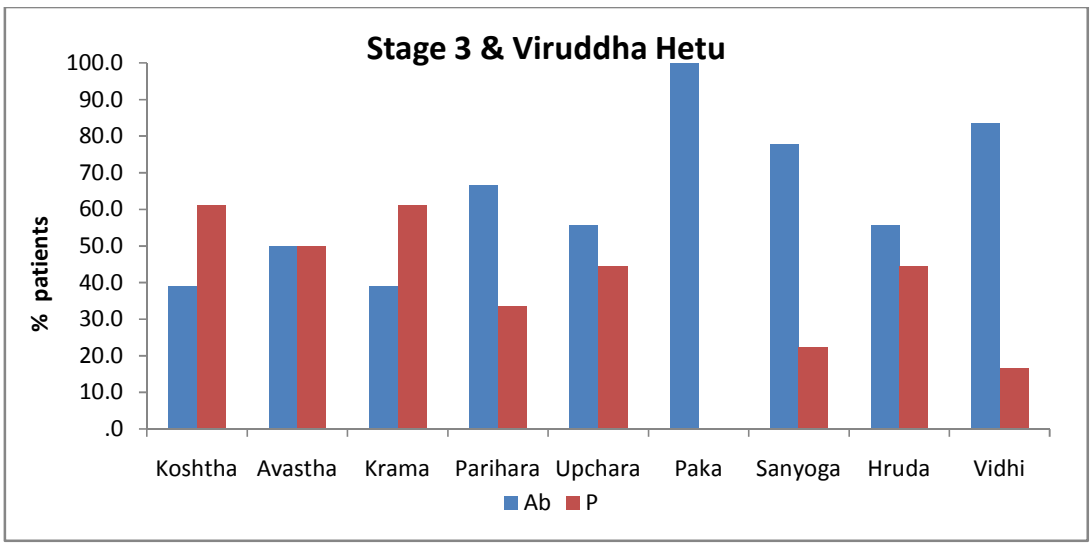
19 Stage & Viruddha Hetu

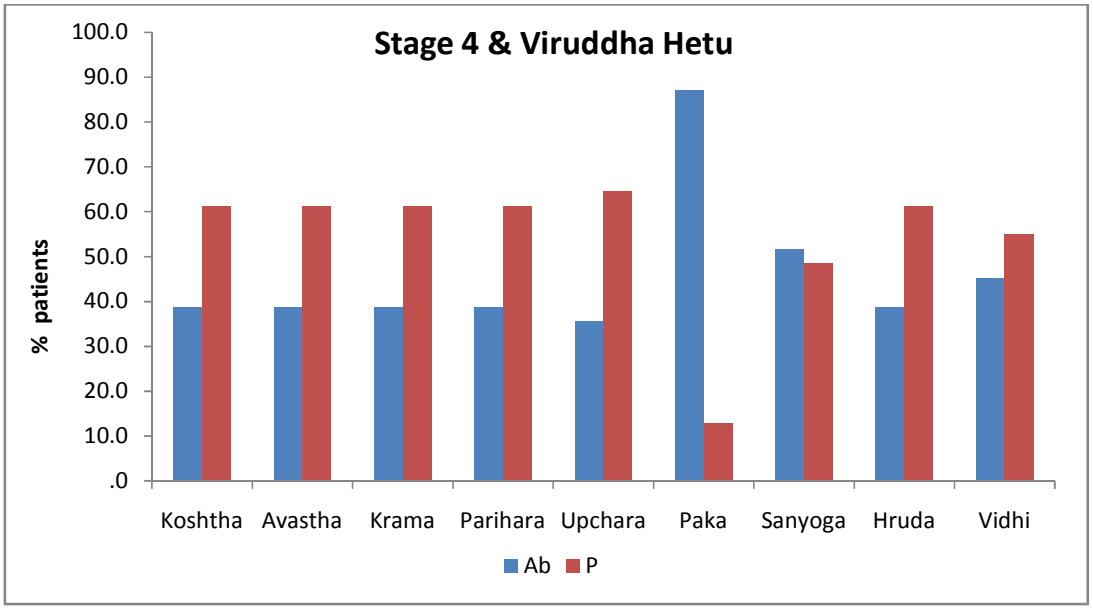
Table no 19 Stage & Viruddha Hetu

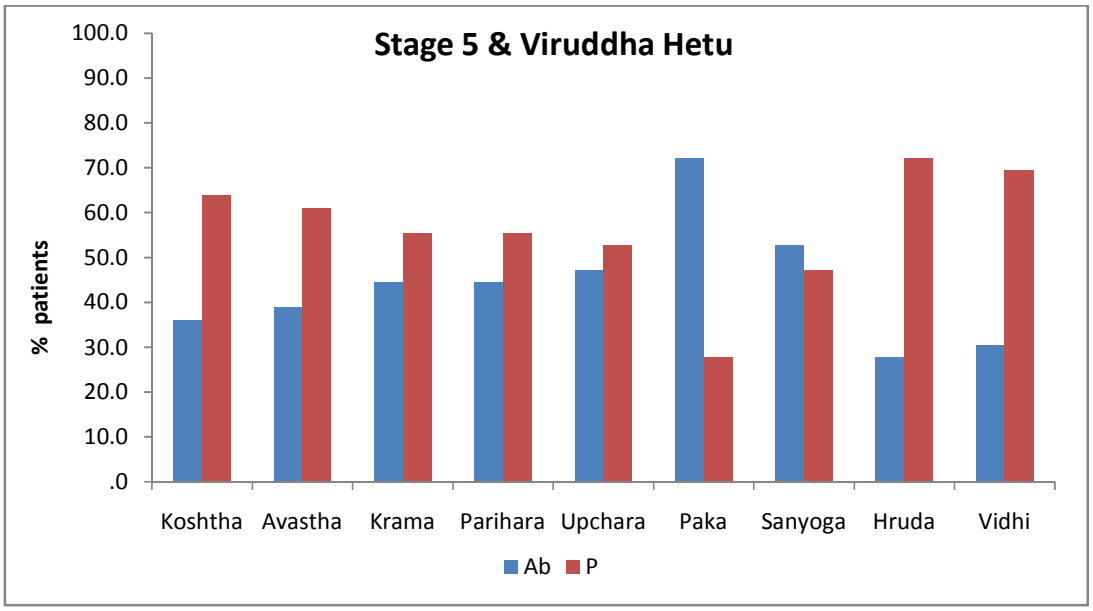
Stages	Hetu	Koshtha	Avastha	Krama	Parihara	Upchara	Paka	Sanyoga	Hruda	Vidhi
Stage 1	Ab	80.0	53.3	33.3	66.7	66.7	86.7	66.7	33.3	46.7
	P	20.0	46.7	66.7	33.3	33.3	13.3	33.3	66.7	53.3
Stage 2	Ab	40.0	40.0	20.0	50.0	40.0	90.0	80.0	30.0	50.0
	P	60.0	60.0	80.0	50.0	60.0	10.0	20.0	70.0	50.0
Stage 3	Ab	38.9	50.0	38.9	66.7	55.6	100.0	77.8	55.6	83.3
	P	61.1	50.0	61.1	33.3	44.4	.0	22.2	44.4	16.7
Stage 4	Ab	38.7	38.7	38.7	38.7	35.5	87.1	51.6	38.7	45.2
	P	61.3	61.3	61.3	61.3	64.5	12.9	48.4	61.3	54.8
Stage 5	Ab	36.1	38.9	44.4	44.4	47.2	72.2	52.8	27.8	30.6
	P	63.9	61.1	55.6	55.6	52.8	27.8	47.2	72.2	69.4


Stage 1 & Viruddha Hetu
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Koshtha
Avastha
Krama
Parihara
Upchara
Paka
Sanyoga
Hruda
Vidhi
Ab
P


Stage 2 & Viruddha Hetu
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Koshtha
Avastha
Krama
Parihara
Upchara
Paka
Sanyoga
Hruda
Vidhi
Ab
P

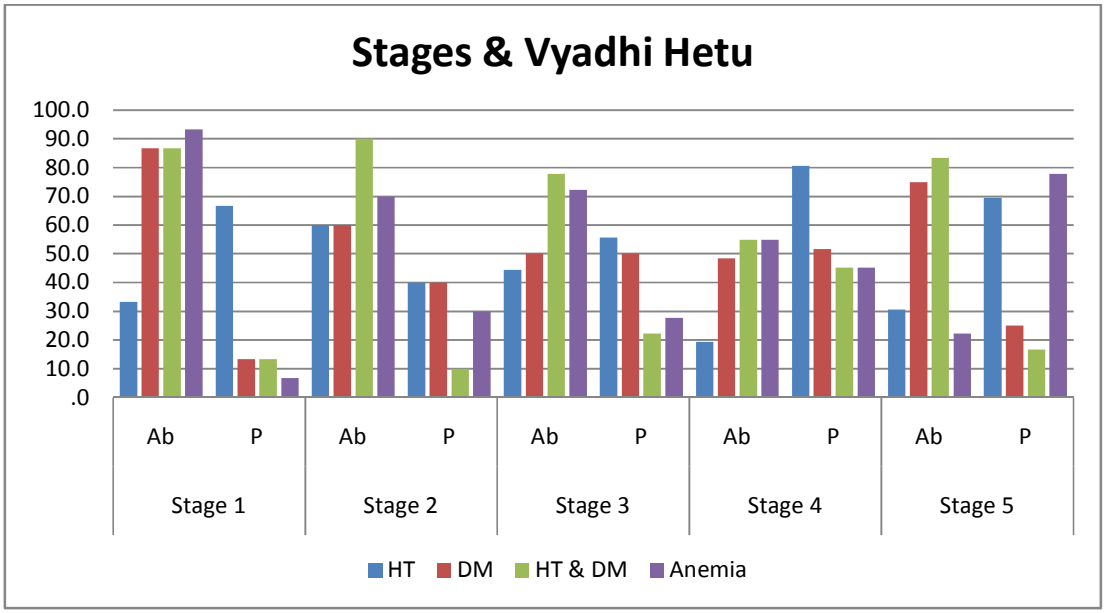

Stage 3 & Viruddha Hetu
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Koshtha
Avastha
Krama
Parihara
Upchara
Paka
Sanyoga
Hruda
Vidhi
Ab
P


Stage 4 & Viruddha Hetu
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Koshtha
Avastha
Krama
Parihara
Upchara
Paka
Sanyoga
Hruda
Vidhi
Ab
P


Stage 5 & Viruddha Hetu
% patients
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
.0
Koshtha
Avastha
Krama
Parihara
Upchara
Paka
Sanyoga
Hruda
Vidhi
Ab
P

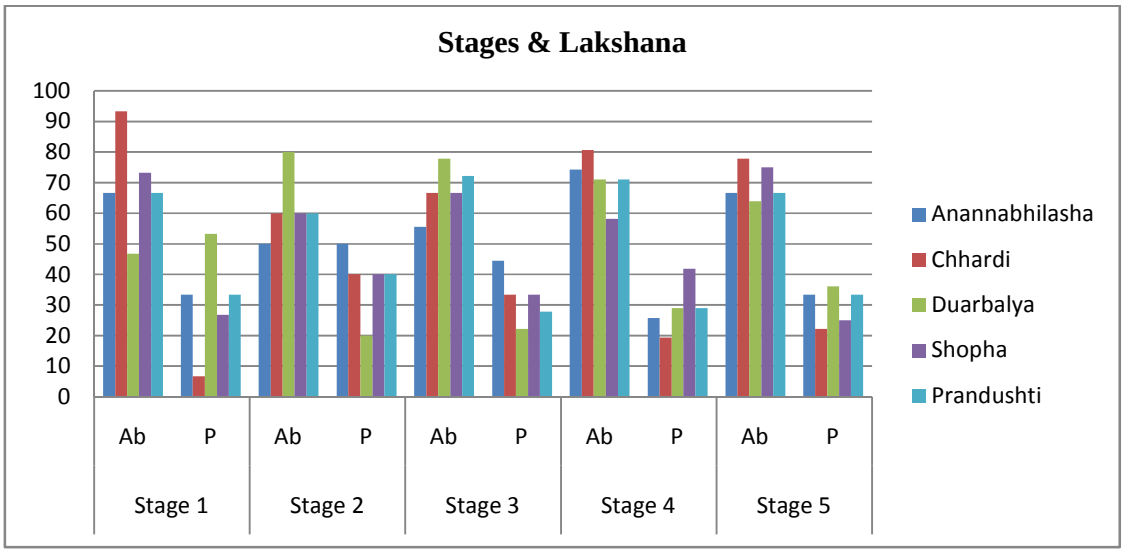
20 Stages & Vyadhi Hetu:-

Table 20 Stages & Vyadhi Hetu					
Stages	Hetu	HT	DM	HT & DM	Anemia
Stage 1	Ab	33.3	86.7	86.7	93.3
	P	66.7	13.3	13.3	6.7
Stage 2	Ab	60.0	60.0	90.0	70.0
	P	40.0	40.0	10.0	30.0
Stage 3	Ab	44.4	50.0	77.8	72.2
	P	55.6	50.0	22.2	27.8
Stage 4	Ab	19.4	48.4	54.8	54.8
	P	80.6	51.6	45.2	45.2
Stage 5	Ab	30.6	75.0	83.3	22.2
	P	69.4	25.0	16.7	77.8



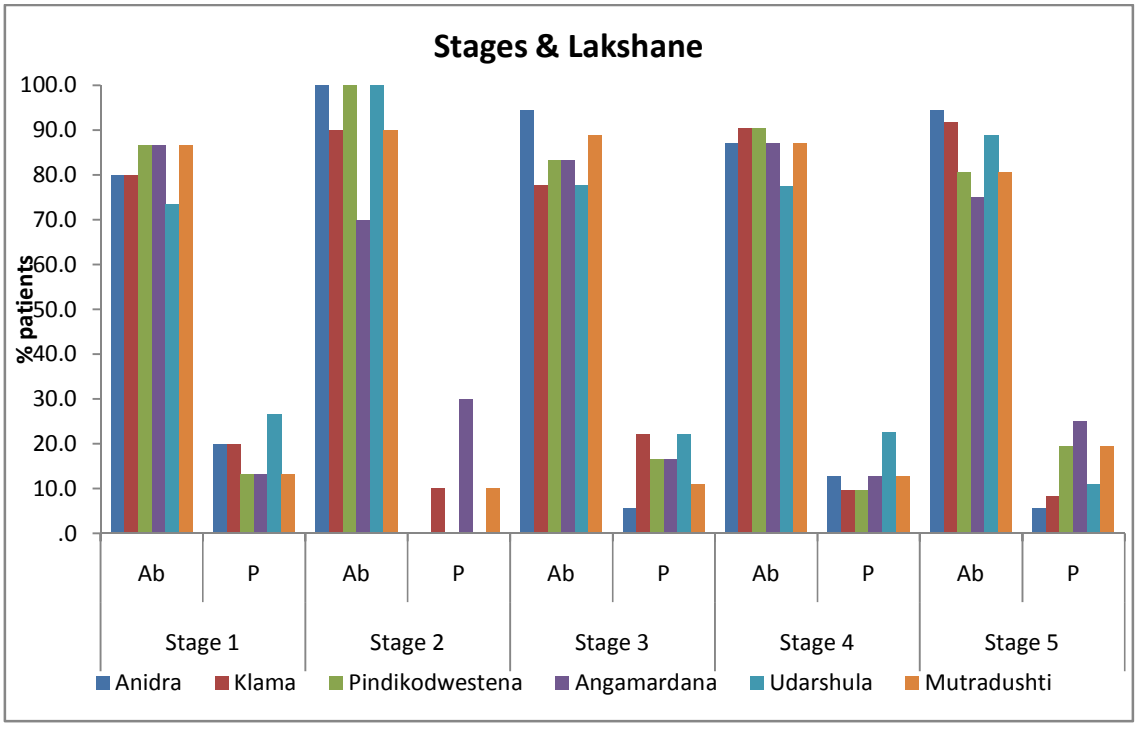
21 Stages & Lakshane

Table 21 Stages & Lakshana						
Stages		Anannabhilasha	Chhardi	Duarbalya	Shopha	Prandushti
Stage 1	Ab	66.7	93.3	46.7	73.3	66.7
	P	33.3	6.7	53.3	26.7	33.3
Stage 2	Ab	50.0	60.0	80.0	60.0	60.0
	P	50.0	40.0	20.0	40.0	40.0
Stage 3	Ab	55.6	66.7	77.8	66.7	72.2
	P	44.4	33.3	22.2	33.3	27.8
Stage 4	Ab	74.2	80.6	71.0	58.1	71.0
	P	25.8	19.4	29.0	41.9	29.0
Stage 5	Ab	66.7	77.8	63.9	75.0	66.7
	P	33.3	22.2	36.1	25.0	33.3



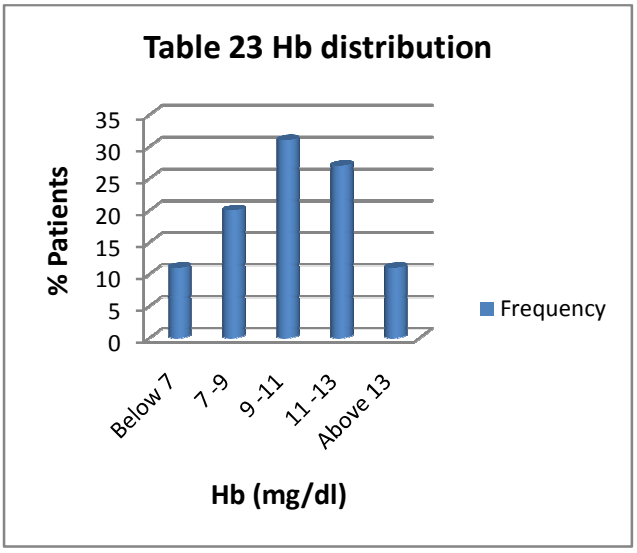
22 Stages & Lakshane

Table 22 Stages & Lakshane							
Stages		Anidra	Klama	Pindikodwestena	Angamardana	Udarshula	Mutradushti
Stage 1	Ab	80.0	80.0	86.7	86.7	73.3	86.7
	P	20.0	20.0	13.3	13.3	26.7	13.3
Stage 2	Ab	100.0	90.0	100.0	70.0	100.0	90.0
	P	.0	10.0	.0	30.0		10.0
Stage 3	Ab	94.4	77.8	83.3	83.3	77.8	88.9
	P	5.6	22.2	16.7	16.7	22.2	11.1
Stage 4	Ab	87.1	90.3	90.3	87.1	77.4	87.1
	P	12.9	9.7	9.7	12.9	22.6	12.9
Stage 5	Ab	94.4	91.7	80.6	75.0	88.9	80.6
	P	5.6	8.3	19.4	25.0	11.1	19.4



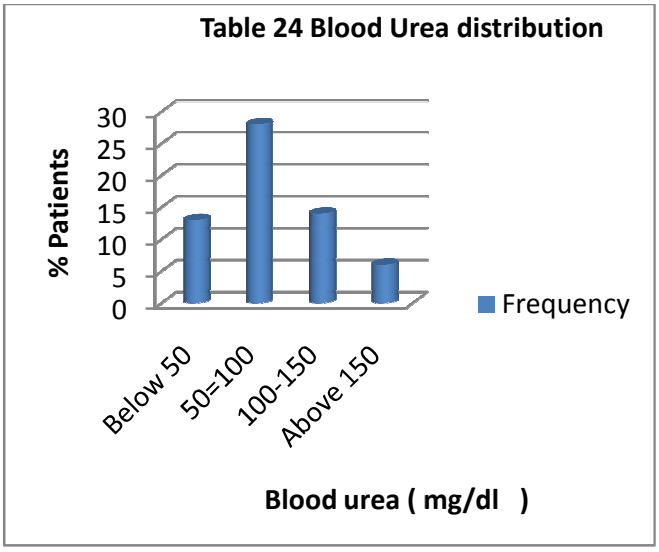
23 Hb distributions

Hb distribution		
		Frequency
Valid	Below 7	11
	7 -9	20
	9 -11	31
	11 -13	27
	Above 13	11
	Total	100
Missing	System	10
	Total	110



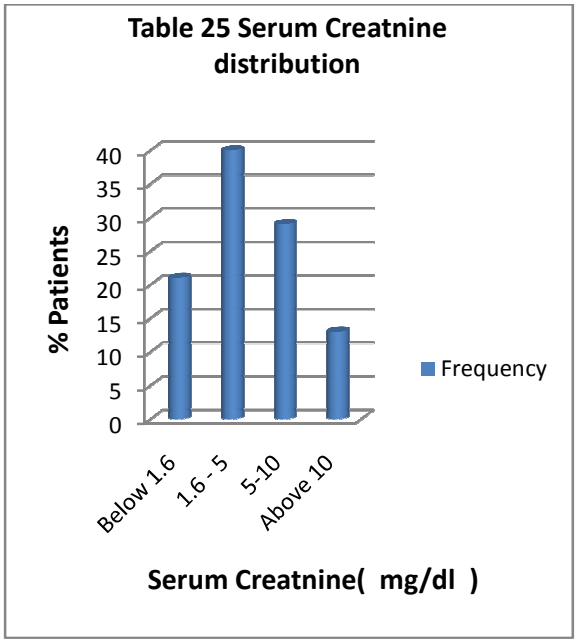
24 Blood Urea distributions

Blood Urea distribution		
		Frequency
Valid	Below 50	13
	50=100	28
	100-150	14
	Above 150	6
	Total	61
Missing	System	49
	Total	110



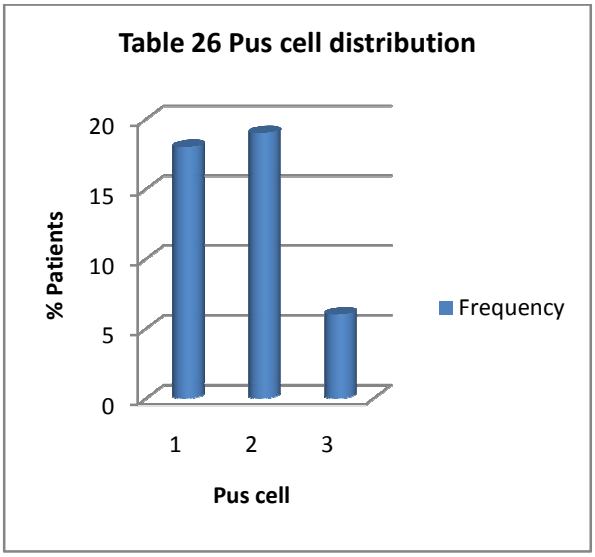
25 Serum Creatinine distributions

Serum Creatinine distribution		
		Frequency
Valid	Below 1.6	21
	1.6 - 5	40
	5-10	29
	Above 10	13
	Total	103
Missing	System	7
	Total	110



26 Pus cell distributions

Pus cell distribution		
		Frequency
Valid	1	18
	2	19
	3	6
	Total	43
Missing	System	67
	Total	110



27 Urine Proteins Distribution

Table 27 Urine Protein distribution		
		Frequency
Valid	0+	7
	1+	7
	2+	21
	3+	17
	4+	8
	5+	1
	6+	2
	Total	63
Missing	0	47
	Total	110

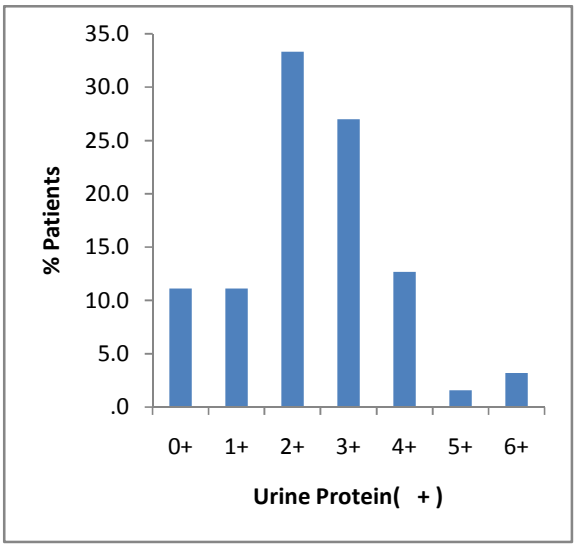


Table 27
Urine Protein Distribution

5.3 Statistical Results observation:

Software used for statistical analysis SPSS software, IBM Version 17.0

S. N.	Association	Chi- squared test	The P Values
1	Association of Madhur Lavana and Amla with staging	Chi- squared: 10.370	P < 0.03
2	Association of Agni with Staging	Chi- squared: 13.127	P < 0.01
3	Association of Matra with Staging	Chi- squared: 11.770	P < 0.01
4	Association of Koshatha with Staging	Chi- squared: 9.418	P < 0.05
5	Association of Vidhi with Staging	Chi- squared: 13.514	P < 0.00
6	Association of Agni, Virya with Staging	Chi- squared: 12.162	P < 0.01
7	Association of Agni, Paka with Staging	Chi- squared: 12.301	P < 0.01
8	Association of Asyshukha with Staging	Chi- squared: 9.983	P < 0.04
9	Association of DM with Staging	Chi- squared: 10.066	P < 0.03
10	Association of HT with Staging	Chi- squared: 10.533	P < 0.03
11	Association of Anemia with Staging	Chi- squared: 27.387	P < 0.00
12	Association of Anemia, Anannabhilasha & Daurbalya with Staging	Chi- squared: 12.407	P < 0.01
13	Association of Anemia, Anannabhilasha & Shotha with Staging	Chi- squared: 19.946	P < 0.00
14	Association of DM, Anannabhilasha & Shotha with Staging	Chi- squared: 9.143	P < 0.05

15	Association of Anemia, daurabalya & Shotha with Staging	Chi- squared: 19.140	P < 0.00
16	Association of DM, daurabalya & Shotha With staging	Chi- squared: 7.996	P < 0.04
17	Association of HT, daurabalya & Prandusti With staging	Chi- squared: 11.980	P < 0.01
18	Association of Anemia, Anannabhilasha & Chhardi With staging	Chi- squared: 17.664	P < 0.00
19	Association of Anemia, Lavan & Amla With staging	Chi- squared: 5.758	P < 0.05
20	Association of Amla, Lavan & Twakdry With staging	Chi- squared: 10.695	P < 0.03
21	Association of Mamsa ahara & Agni With staging	Chi- squared: 8.371	P < 0.00
22	Association of Mamsa ahara & Urine Protein With staging	Chi- squared: 7.873	P < 0.04
23	Association of Mamsa ahara * Urine Protein With staging	Chi- squared: 12.505	P < 0.05
24	Association of Amla, Lavan & Muscle tone With staging	Chi- squared: 15.718	P < 0.00
25	Association of Abhishyandi & mutra With staging	Chi- squared: 6.079	P < 0.04
26	Association of Diwaswap & CMDcode with Staging	Chi- squared: 9.034	P < 0.00
27	Association of mutra & USG With staging	Chi- squared: 7.357	P < 0.02
28	Association of Chhardi & Anannabhilasha With staging	Chi- squared: 6.667	P < 0.01
29	Association of Chhardi, Anannabhilasha & Anemia With staging	Chi- squared: 9.450	P < 0.00

30	Association of Chhardi, Shotha & Abhishyandi With staging	Chi- squared: 4.635	P < 0.03
31	Association of Chhardi, Anannabhilasha & Agni with Staging	Chi- squared: 7.085	P < 0.00
32	Association of Chhardi, Anannabhilasha & Abhyvaran with Staging	Chi- squared: 12.666	P < 0.00
33	Association of Addiction & Trushna with Staging	Chi- squared: 35.420	P < 0.00
34	Association of Diwaswap, Asyshukha & DM With staging	Chi- squared: 4.427	P < 0.03

6. Discussion:-

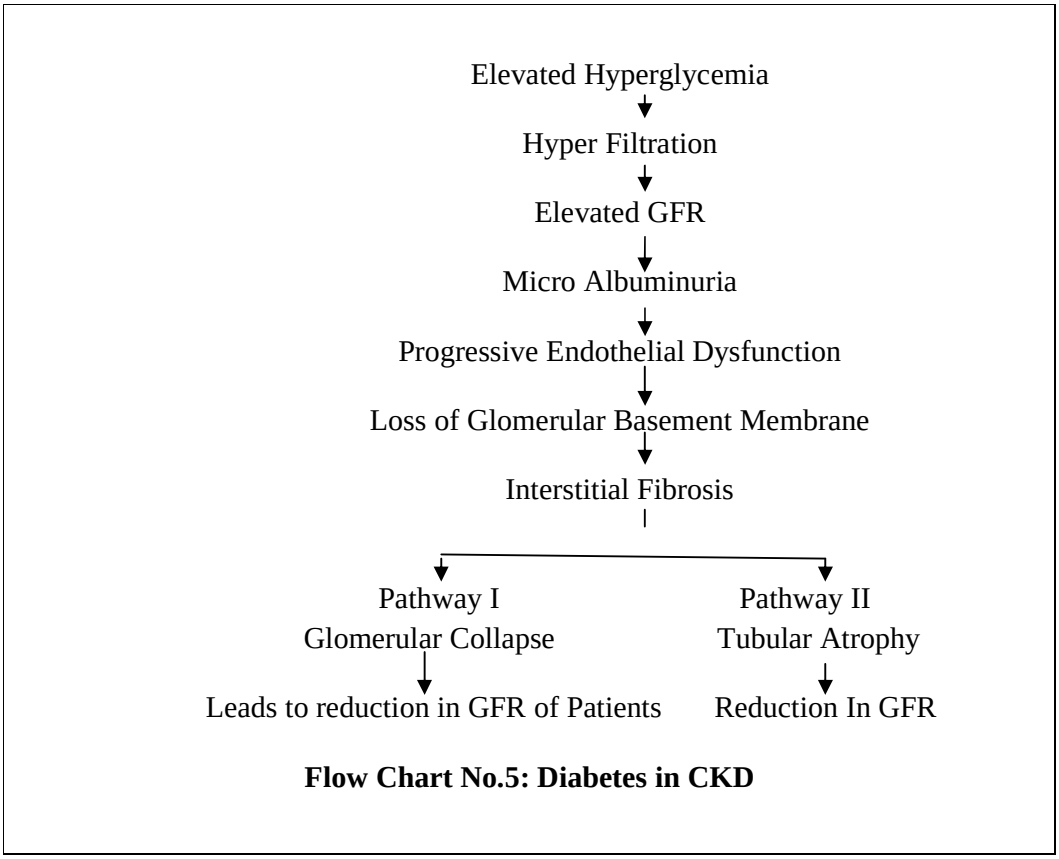
6.1 Modern Literature Discussion

In this chapter, Diabetes and Hypertension are discussed as per modern science. The urea and creatinine are the basic biochemical components of kidney disease. The creatinine and urea pathway are discussed. The CKD attributes as per modern science compared to Ayurveda srotas. The complication of CKD is mainly anemia. Thus anemia is explained.

This chapter is followed by Ayurveda literature discussion hetus, lakashanas to mention probable samprapti of CKD.

1) Diabetes in CKD:-

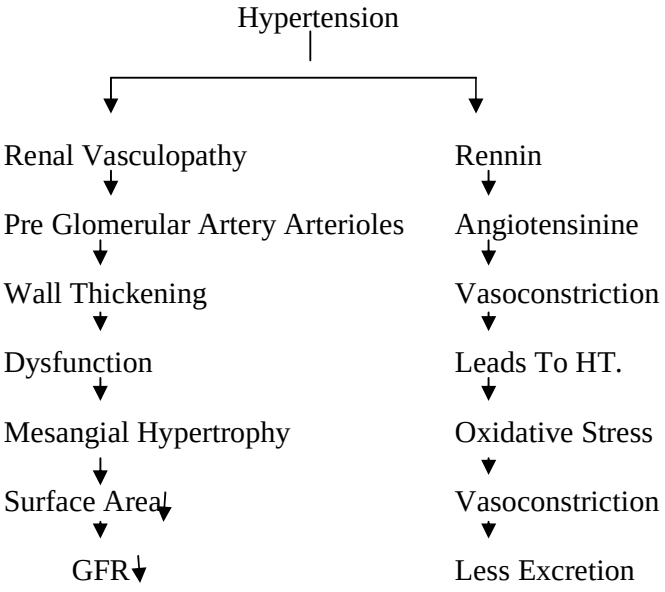
Diabetes is the cause for CKD in long term or ongoing treatment patients the pathogenesis mentioned in literature is as below and as well observed in patients of CKD.[40]



Flow Chart No.5: Diabetes in CKD

2) Hypertension:-

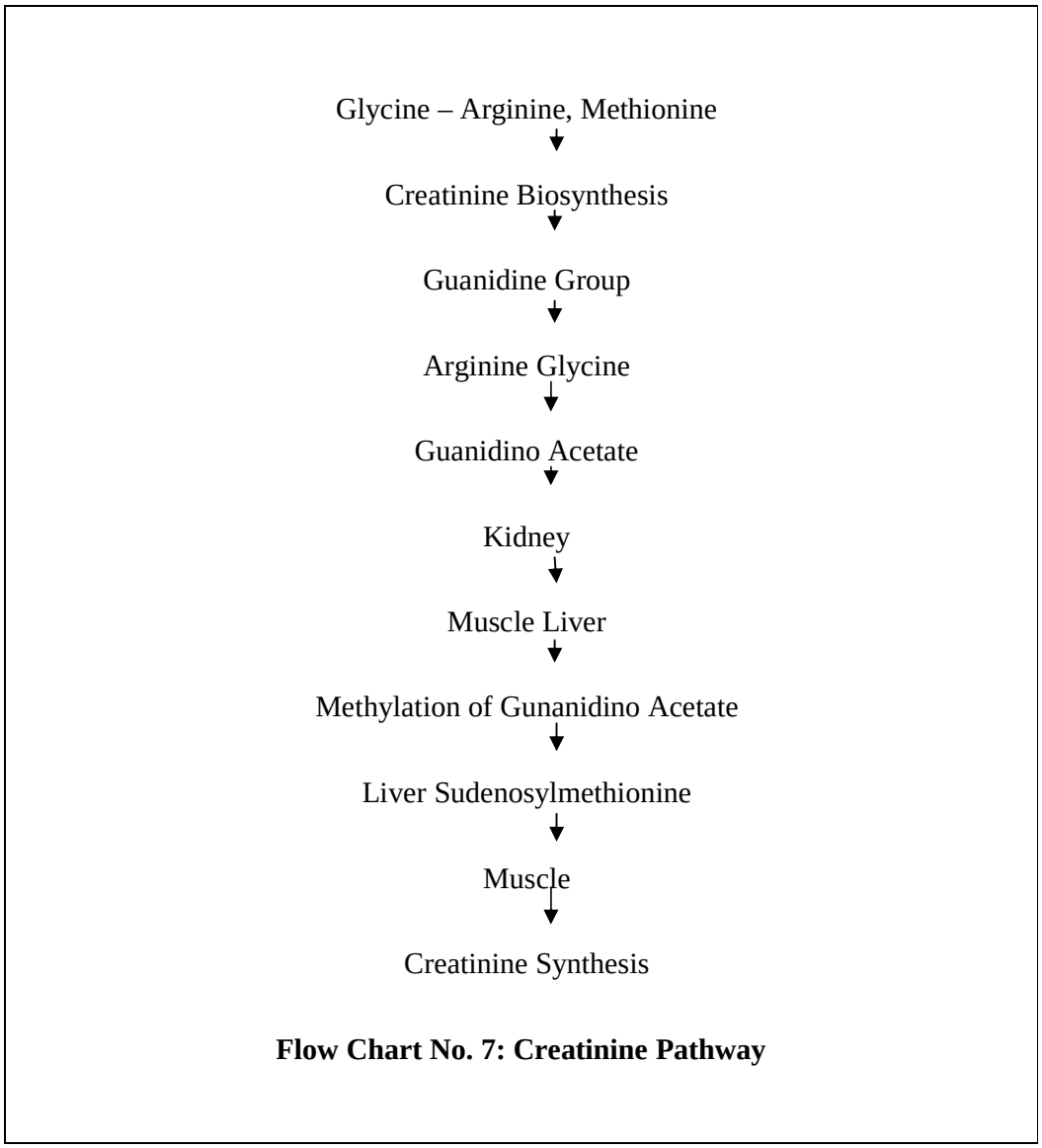
The hypertension is observed as vyadhi hetu for CKD all stages.



Flow Chart No. 6: Hypertension

3) Creatinine Pathway:-

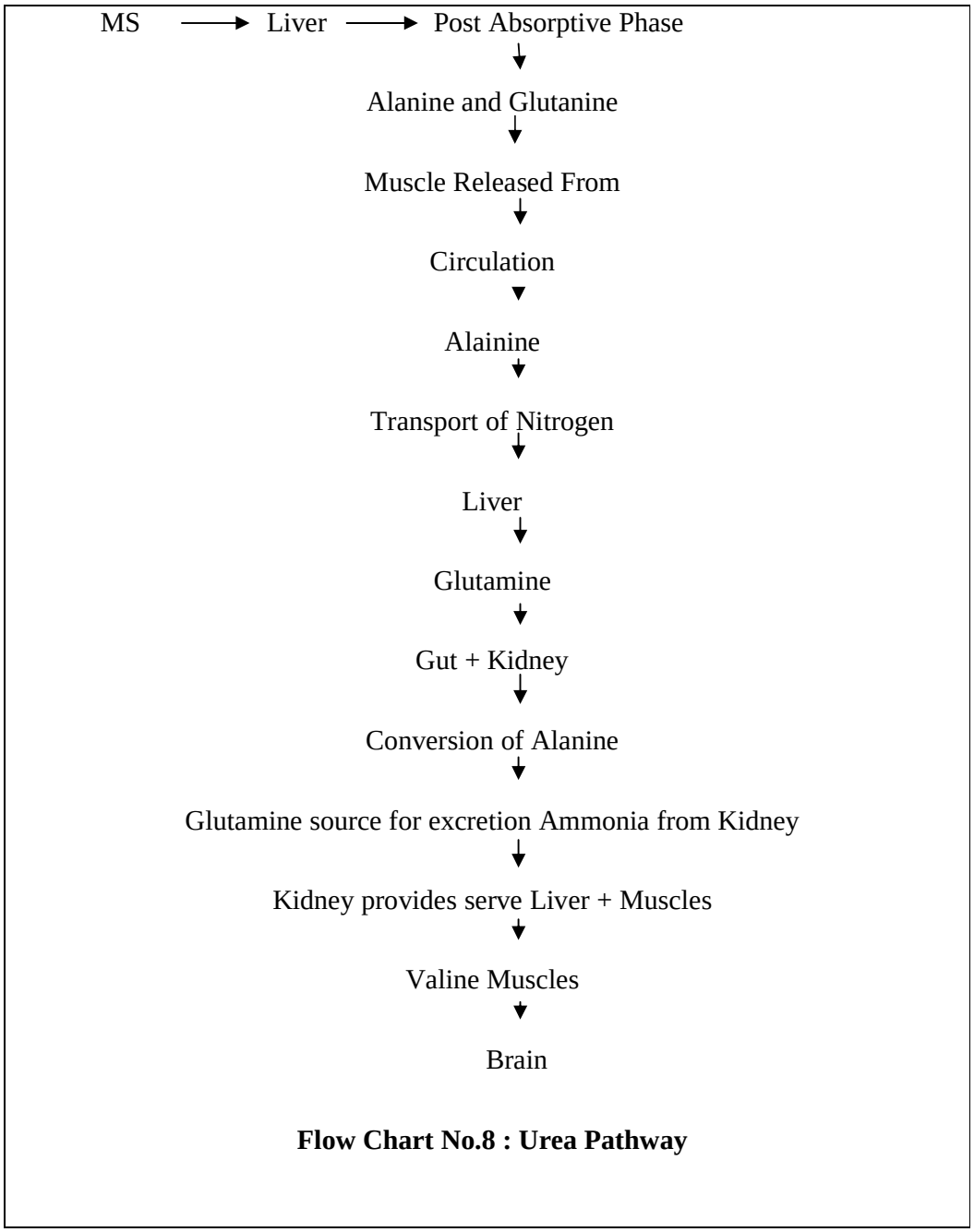
Creatinine excretion is function of muscle mass. Both creatinine and its energy reserve from phosphor creatinine are present in muscle, brain and blood. Creatinine (creatinine anhydrase) is formed in muscle form creatinine phosphate by irreversible non enzymatic dehydration and loss of phosphate. The 24 hours excretion of creatinine in the urine of the given subject is reasonably constant from day to day and proportionate to muscle mass. Traces of creatinine also normally occur in urine.[41]



Flow Chart No. 7: Creatinine Pathway

4) Urea Pathway:-

Urea is the major end product of nitrogen catabolism in human, urea synthesis in the liver released into blood and cleared from kidneys, constituted 80 to 90 % of the nitrogen excreted.[42]



Flow Chart No.8 : Urea Pathway

5) Manifestation of attributes of CKD are:

Modern	Ayurveda
1) Cardiovascular system Cardiomyopathy	1. Pranvaha Strotas Ayasena-shawas Shawaskashta
2) Nervous system Cramps Fatigue Headache Sleep disorders	2. Mamsavaha Strotas Pindikodweshatana Klama Shirshoola Anidra
3) Hematological system Bleeding	3. Raktavaha Strotas Shotha, pale skin
4) Immunological Stimulation of inflammation	4. Shotha
5) Bone disease Osteomalacia	5. Bone disease Sandishoola, kriyakashtata
6) Skin Purities Melanosis	6. Rasavaha Strotas Kandu Vaivarnya
7) Gastrointestinal Anorexia Nausea Hiccups Nausea & Vomiting	7. Anavaha Strotas Anannabhilasha Hrullas Hikka Chhardi
8) Miscellaneous Thirst	8. Udakavaha Trishna

Diabetic Nephropathy Pathway	Ayurveda Prameha Pathway
Hyper filtration ↓ Elevated GFR ↓ Microabuminimia ↓ Progressive endothelial dysfunction ↓ Loss of glomerular basement membrane ↓ Interstitial fibrosis ↓ Glomerular collapse Tubular atrophy	Hetu Sevana ↓ Kleda dushti ↓ Dushit Mutra nirmiti ↓ Mutrakshaya khavaigunya ↓ Kleda dushti upalepana of vrukka ↓ Karya dushti of meda & rakta dhatu ↓ Mutra Sangraha Mutrabahulya

6.2 Observation:-

Ayurveda Literature Discussion

This chapter mainly focuses on concepts of CKD where filtration is hampered. So, Ayurveda literature review is discussed related to its by Hetus *Santarpans, Apatarpan and Ajeernajnya* hetus for pathogenesis. In CKD, Diabetes and HT are vyadhi hetu so Prameha is mentioned in literature discussion and *pandu* is discussed.

Content of topic:-

1. *Hetu – Santarpan/ Apatarpana - Ajeernajnya*
2. *Parameha & Pandu*

Santarpan hetu:

In Charak samhita Santarpaniyadhya- *Snigdha, Madhur, Guru, Pichhil, Navanna, Madya, Anup mamsa* all these hetus are causative for the Santarpan vydhi. Basically *Santarpanotha* vyadhi are those caused due to assimilation of dosha dushti in *Strotas causing Indriyalepana*. The diseases caused by the Santarpan hetus are followed *Prameha, Kandu, Pandu, Aamadosha, Jwara, Tandra, Athisthoulya, Aalasya, Mutrakruchhra and Shotha*.[43]

Santarpan Hetus

↓

Madhur, Snigdha, Guru, Pichhil, Navana, Madya

↓

Agni dushti

↓

Vyakta vyadhi

Diseases: Prameha, Kandu, Pandu, Jwara, Mutrakruchtra, Atistholya, Aalasya, Shotha

Flow Chart No. 9: Santarpan hetu

Apatarpan hetu:-

The hetu *Katu, Kakshaya, Tikta and Paryusheet ahara sevana* observed. The *aatap, jagaran, vyayama, krodha, shoka are apatarpan hetu.*[44]

Apatarpan hetus

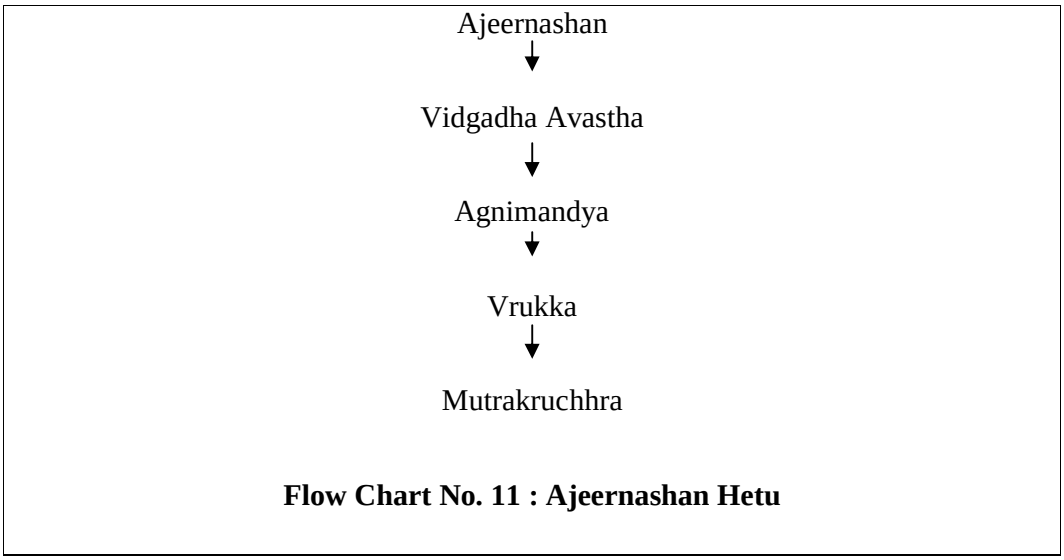
Ahara - Katu, Kakshaya, Tikta
↓
Vihara - Aatap, Jagaran
↓
Manasa - Krodha, Shoka
↓
Guna - Rooksha, Ushna
↓
Rasa Mahabhuta – Agni + Vayu
↓
Sthana Grahani -
↓
Agni Dushti
↓
Mutrasangrha
↓
Karnadushti (lakashana of end stage)

Flow Chart No. 10: Apatarpan hetu

Ajeernashan Hetu:-

The state of *ajeernashan i*s that when previous ahara is not digested well, then it leads to *Ajeerna*. The *ajeernashan* causes vidagdha avastha, leading to *tridosha prakopa*. *The prakopita doshas* are causing *avarodha of Strotas* leading to diseases.[45]

The *apakva ahara* rasa leads to *tridosha prakopa* causing *agnimandya and Stroto avarodha* especially *mutravaha Strotas*. The *dushit kleda* leads to *mutrakruchhra.* The diet according to state of Agni is important feature of CKD.



Flow Chart No. 11 : Ajeernashan Hetu

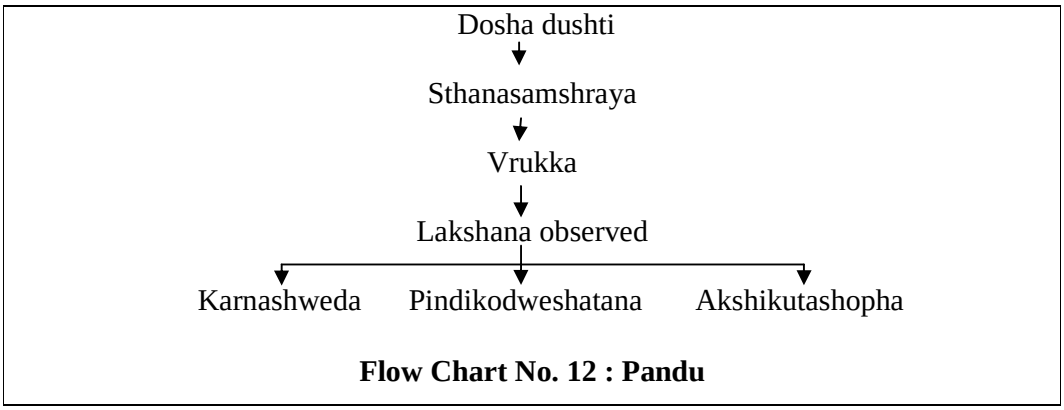
Pandu:-

The *Pandu* can be apparently a hetu for CKD and dominantly complication of kidney disease. The *Santarpan, Apatarpan and Viruddha* are basic hetu for CKD also these hetus attributes for Pandu.[46]

In this process because of hetus dosha dushti is *vata*, leading to *rasa dushti* along with *Agni*. Thus in turn disturbs *rakta* and *meda* dhatu. Consequently *rakta mala – Pitta & sneha* of the *meda dhatu* are disturbed/ hampered. These again contribute to vata dushti as well as present them in the *dushit* form as *Pandu.*

Apatarpan /Ajeerna samprapti

Viruddha / Diwaswap / Krodha / Shoka/ Vishamashana



Flow Chart No. 12 : Pandu

Prameha:-

In CKD Prameha is observed as a vyadhi hetu for many cases. In the Prameha vyadhi both *Apatarpan and Santarpana* hetus are observed in samprapti of Prameha.

Prameha in CKD:

1. Santarpana

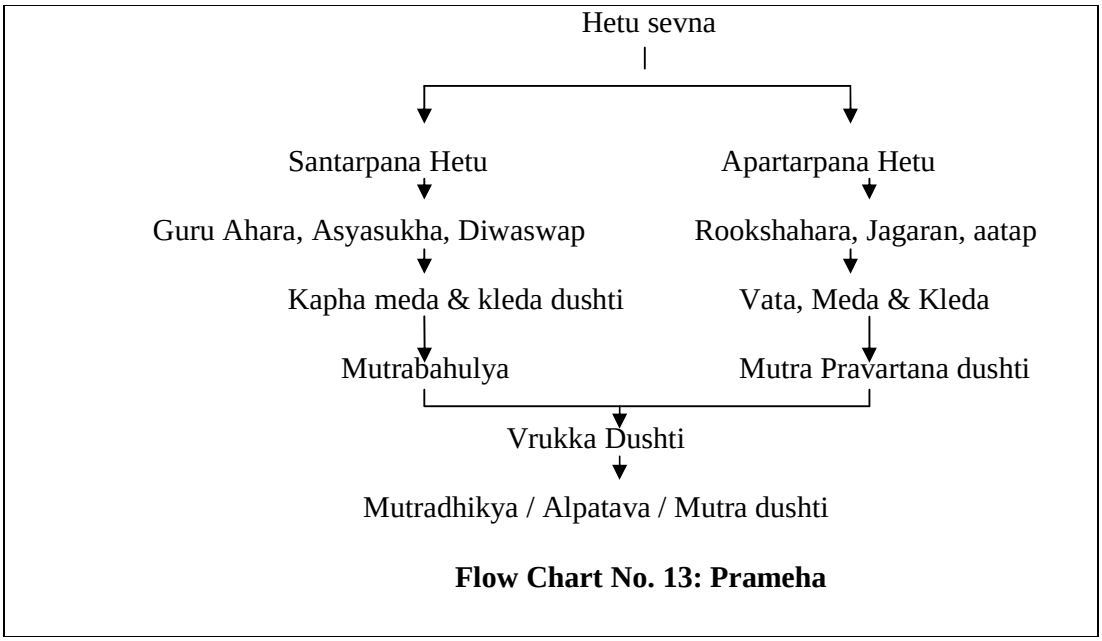
2. Apatarpan

Prameha in CKD by Santarpana Hetu:

The *santarpana hetu sevana Diwaswap, Abhishyandi, Guru Ahara* etc leads to *kapha dushti*. Mainly *shleshamika guna* is aggravated disturbing further meda *dhatu*; both contribute for *dushit kleda* vruddhi. This *kleda* vruddhi in turns results in *mutrabahulya*.[47]

Prameha in CKD by Apatarpan Hetu:

The *Apatarpana* hetu such as *Rooksha, Katu ahara, Jagarana, Aatapa* etc. leads to *Kapha meda, & kleda dushti & Vata, Meda & Kleda* dushti respectively leading to *vrukka dushti* and *mutra dushti*.[48]



Flow Chart No. 13: Prameha

Prameha as *vyadhi hetu, Prameha samprapti* (kleda dushti) leads to changes in *karya* (function) and *sharir* (structure) of *vrukka.* This leads to deranged function of excretion.[49]

6.3 Clinical observation discussion:

In this chapter, lakashana methodology assessment is discussed. The lakashana methodology i.e. *dosha lakashana, srotodushti lakashana adhistana doshaka lakashanas* and clinical findings are discussed in CKD.

1. **Srotos lakashana/ Clinical finding: -**

Annavaha srotas-

Jivha parishana (Tounge):-

The Sama and Nirama Jivaha observed in patients .The *Niram jivaha* observed in 14.5% patients, indicating normal pachana. The *Sama jivaha* is observed in 85.5% patients. Thus the *Sama jivaha* indicates pachana is disturbed.

Agni Parishana:-

The *Agni pariksahna* in patients of CKD is as follows *the Sama Agni* 43.6%, *vishama Agni* 15.5%, *tikshana Agni* 10.0% & *manda Agni* 30.9% are observed. The *manda Agni* actually is the main cause for disease.

Abhyvaran Shakti Parishana:-.

The *Abhyavaran Shakti* of patients of CKD is observed .The *prakrut abhyavaran* 74.5% and the *aprakrut abhyavaran* 25.5% is observed. Thus pachana is disturbed in such patients.[i]

2. **Uadakavaha srotas:-**

Trishna (Thirst):-

The trishna lakashana observed as *Prakrut* lakashana in 50.9% and the *Aprakrut trishna* observed in 49% patients.[ii]

Soft palate (Talu)

The soft palate normal 62.7% patient and dry palate 37.3% patients observed. The *talu kloma* are *the mula sthana* of *Udakavaha Strotas.* Hence, dushti of *udakavaha strotas* were observed.

Salivation, Lips (Lalastrava, Ostha)

The lips normal, dry and scaly are observed in patients .The percentage of normal lips patient is 23.6% .The dry 46.4% and scaly 30.0 %.

The normal salivation 40.9%, less salivation patients 56.4% and excess salivation 2.7 % were observed.

3. **Mamsa:-**

Muscle fatigue (Mamsa daurbalya)

The muscle fatigue absent in 33.6% & present in 66.4% is observed. The uvula normal in 48.3%, the elongated uvula in 48.2% & others 8.2% patient's are observed. Thus the elongated uvula shows *Mamsavaha Strotas* dushti and *Medovaha Strotas* dushti.[50]

4. **Prana vaha:-**

Auscultation

The normal auscultation observed is 68.2 % and the abnormal auscultation 36.81%. Here *Pranavaha Strotas* dushti is observed.

Nasa (Nasal examination)

The distribution of percentage observed in patients of DNS- 12.7%, dryness 69.1%and polyp 18.2%. The nasal dryness and polyp is the cause for dushti of *kapha & vata* dosha.

5. **Meda/ Avastha lakashana:-**

Vrukka (Kidney)

The renal angle tenderness is observed in 10% patients and the non tenderness observed in 90% patients.

6. **Rakta:-**

Netra (Eyes)

The percentage of eyes pallor is 73.4%.The non pallor eyes in 26.4%.The pale eyes are suggestive of *rakta dhatu* dushti in CKD.

Pigmentation

The pigmentation absent in 66.4% and the pigmentation present in 33.6% are observed. However, *Raktavaha Strotas* dushti lakshana were observed here. [51]

7. **Asthivaha srotas:-**

Nakha (Nails)

The percentage of pale, clubbing, koilonychias and normal conditions of nails observed. The pale nails observed in 82.7% patients. Clubbing 10.9% & the koilonychias 2.7% are observed.

Hair fall (Kesha shatana)

The hair fall observed in 48.2% patients .The hair greying observed in 57.2% patients. Hair fall observed as swedvaha stroto dusti lakshana in patients 48.2%. The hair greying is observed as Pitta & Rasa dushti lakshana.

Sandhi Kriya kastha (Jointpain)

The *sandhi kriyakashtata* observed in 24.5%. The *sandhi* is the basic site for *majja dhatu*. The majja is having *medaposhakansha* in it. The correlation of *majja dhatu* in CKD is observed as the (*sarakta sneha*) majja dhatu dushti is observed with disease progression from stage 1 to stage 5.

8. <u>Swedavaha:-</u>

Skin (Twak)

The normal skin 18.7%, dry skin 47.3%, scaly 15.3%, scratch mark 12.0% & pale skin 21.3% are observed. The twak is vata sthana (*sparshnendriya*) and its dushti lakshanas are observed in patients. The twak is *updhatu of mamsa dhatu*; which signifies twak dushti suggestive of mamsa dushti in CKD patients

Sweda pravartana (Perspiration)

The sweda *pravartana* observed in summer 68.2% ,sweda absent in 31.8% while sweda absent in all season 91.8% and sweda present in all 8.2% patients. The sweda nourishes twak; roma. The disturbances in sweda pravartana are suggestive of kleda dushti. The *swedapravartana* absent in 31.5% this shows that twakgat sneha is dusht. This indicates dushit kleda directly disturbed twak sneha representing lakshanas dry skin & itching.[52]

9. <u>Mutravaha:-</u>

Daily frequency of urine (Mutra frequency)

The daytime frequency of urine 1 to 4 times 45.5%, 4 to 8 times 51.8%, 8 to 12 times 2.7%. The urine frequency at night 1 to 4 times 20.9%, 4 to 8 times 69.1% & 8 to 12 times 10.0% are observed. The urine burning and pain in lower abdomen patients observed were 18.2%.

10. <u>Purisha:-</u>

Mala pravartana(Bowel habit)

The daily mala pavarthana 94.5%, mala a pavarthana 4.5 %, formed mala 75.5%, kruchhratava 28.2% is observed. The bowel habit in present data is non significant due to sweda & mutradushti mainly. Hence it

indicates mutra & sweda dominantly hampered but invariably purisha is mostly normal in CKD.

Other observation:-

11. Vyadhi lakashana:-

Hypertension & DM

The hypertension absent in 32.7% and hypertension present in 67.3% patients. 63.6% were Diabetic where as 36.4% were non DM. The numbers of patients observed HT+DM were 24.5% .The vydhi hetu as DM+HT were observed.

12. Dialysis & staging

The numbers of patients undergoing dialysis were observed 66.45% and the 33.6%non dialysis observed .The dialysis is conventional treatment for end stage.

The staging observed were stage 1st -13.6%, 2nd -9.1%, 3rd -16.4%, 4th -28.2% and 5th 32.7%.

13. Hereditary feature

The diseases observed were Asthma, HT, DM, Kidney disease and polycystic disease of kidney. The DM – 12, HT- 8, Kidney Disease -4, IHD -2, Asthma -3, and Cancer-1 observed. The percentages of DM & HT disease were mainly seen. The kidney disease 6, DM -2, HT- 1, IHD -1 patient observed. The hereditary cause is dominant in swakul history. It shows that bijadushti is main reason for disease occurrence.

6.4 Observational discussion:-

The observational discussion is based upon the finding of hetus and lakashanas mainly. The classifications of hetus to understand its effect in CKD staging are given below:-

Hetu observational discussion:-

1) *Santarpana*
2) *Apatarpan*
3) *Viruddha*
4) *Vyadhi hetu*

Lakashana observation:-

1) *Strodushti lakashana*
2) *Dosha lakashana*
3) *Dhatu dushti lakashana*
4) *Avastha lakashana*
5) *Updrava lakashana*

Srotas observation:-

Srotas dushti and staging

Hetu discussion:-

To study importance of hetus:-

1. By knowing hetu “Nidanaparivanjana” can be achieved.
2. Treatment is against the hetus hence knowledge of hetus is important.
3. Hetu involved gives knowledge of dosha characteristics responsible in pathogensis.
4. Dosha ability for disease process is understood detail concept (swantantra and partantra).
5. Disease gravity (vyadhi bala) can be judged properly with hetus.
6. Involvement of dosha i.e. *ansha-ansha* aspect is *sansarg or sannipat* can be understood with hetu.

The Case Record From for study prepared on the basis of daily routine of patient – with different histories family, drugs, addiction etc. thus; hetus are *aharia viharia, viruddha and manas.* These hetus are classified into *Santarpana, Apatarpan, Viruddha and Vyadhi hetu.*

1) ***Santarpan hetu:-*** *Madhur, Amala, Lavana, Ahara, Abhishyandi, Asyasukha, Diwaswap are Santarpan hetu. These hetus are Kapha prakopa hetu.*
2) ***Apatarpana hetu****:- Katu, Tikta, Kashaya, Jagaran, Aatap, Vyayama are considered. These hetus leads to vata dushti.*
3) ***Viruddha hetu****:- Vegavarodha, Vishamashana are vidhi viruddha, parihara viruddha from desh to sampat all types of viruddha considered for observationl discussion.*[iv]
4) ***Vyadhi hetu****:- the cause for disease/ vyadhi is DM & HT are vyadhi hetu*

Stage1 Apatarpan / Santarpan Hetus

Santarpana Hetu – *Amala,*[53] *Lavana,*[54] *Mamsa Ahara, Abhishyandi,*[v] *Payrusheet,*[vi] *Asyasukha*
Apatarpana Hetu – *Aatap.* [vii]
Agnidushtijanya / Viruddha Hetu- *Vegavarodha,*[viii] *Virya, Krama, Hrud, Agni, Matra & Vidhi*
Vyadhi Hetu:- *HT*

The *Santarpan and Apatarpana* hetus are observed in stage 1. These hetus leads to *agnidushti, vata & Kapha dushti* is observed. These hetu leads to probable changes in filtration GFR.

Stage 2 Hetus:- (Santarpana & Apatarpan)

Santarpana Hetu – *Amala, Lavana, Mamsa Ahara, Abhishyandi, Payrusheet,*
Apatarpana Hetu – *Katu*[55]
Agnidushtijanya / Viruddha Hetu- *Krama, Hrud, Kala, Agni, Dosha, Virya*
Vyadhi Hetu:- *DM & HT*

The combination of altogether *santarpana apatarpan hetus* affects the functions of dosha *dhatu and agni*. The *agnidushti* disturbs pachana and absorption at *grahani* and *pakavashaya* respectively leading to changes in filtration rate. As

changes in Sr. Creatinine, Blood urea and urine protein observed in laboratory finding.

Stage 3:-

Santarpana Hetu – *Madhur,*[56] *Lavana, Abhishyandi, Asyasukha*
Apatarpana Hetu – *Katu*
Agnidushtijanya / Viruddha Hetu- *Dosha and Virya*
Vyadhi Hetu:- *HT & DM*

In stage 3, *santarpana* hetus are present disturbing *Kapha dosha and kleda* while *apatarpana* hetu affects *vata dosha*. Thus in stage 3 GFR reductions can be observed.

Stage 4:-

Santarpana Hetu – *Amala , Lavana, Madhur, Mamsa Ahara, Abhishyandi, Diwaswapa*[xi]
Apatarpana Hetu –
Agnidushtijanya / Viruddha Hetu- *Kala, Agni, Matra, Satmya, Virya*
Vyadhi Hetu:- *HT & DM*

The End Stage Kidney Disease with Ayurveda aspect observed hetus are *santarpana and apatarpan* observed. The dominance of dosha with above hetus is *vata & Kapha* mainly. The *vata dushtikara Apatrapan* hetus are dominant in stage 4. The combination of aharia and viharia hetus observed.

Thus, these hetus affects the absorption phase as well as excretory phase

Stage 5:-

Santarpana Hetu *– Amala, Lavana, Mamsa Ahara, Abhishyandi, Payrusheet,*
Apatarpana Hetu – Katu, Aatap
Agnidushtijanya / Viruddha Hetu *– Kala, Agni, Matra, Satmya*
Vyadhi Hetu:- *HT, Anemia*

The hetus for stage 5 are *santarpana & aapatarpana*. These hetus directly affect on *vata & Kapha* dushti leading to dosha dushti. These hetus probably affects filtration rate i.e. reduction in GFR is observed.

From stage 1 to hetus are in different combination with vyadhi/viruddha hetu. The combination of *ahara, vihara* is observed it suggest that from Ayurveda aspect the regulation for *ahara & vihara* is important for prevention of further progression of disease in stage 4 & 5 i.e. End Stage Renal Disease.

Stages and Mansa Hetus:-

The manas hetus observed in study are *krodha and shoka*. The krodha hetu is significantly observed. This leads to pitta dushti hetu as alone but as it in the combination of hetus observed. Thus in combination with other hetus *agnidushti* is observed. This will affect *pachana and sara kitta vibhajana* in CKD cases.[iii]

Lakashana Observation:-

The methodology for lakashana interpretation is *dosha lakshana, vyadhi lakshana, samavastha, niramavastha, updrava, srotas dushti, dhatudushti, adhisthanadushti lakashana.*

The study of lakashana gives dosha and its *ansha-ansha* aspect in detail. The lakshana denotes *vydhi udabhava sthana to prasara sthana* the combination of dosha dushti and dhatu dushti is expressed with the lakshana. The *srotodusti* identified with lakshanas. Thus, it will help in identifying dosha, *sthana* and srotas.

The lakashana are classified as

1) *Srotodushti lakashana*
2) *Dosha lakashana*
3) *Dhatu dushti lakashana*
4) *Adisthana lakashana*
5) *Avastha lakashana*
6) *Updrava lakashan*
7) *Bhahu dosha lakashana*

The studies of 110 cases with different lakashanas observed are grouped. Lakashan are grouped according to srotas dushti and stages. This gives understanding regarding involvement of srotas in that particular stage. The lakashanas are probably dosha lakashana as patient observed once and other lakashana are sroto dushti lakashana. Here, *prandushti lakashana, avasthadustika*

1. *Prandushti Lakashana:- Ayasenashwas, Shawaskastata, Kasa, Hikka.*[57]
2. *Shoth:- Pada, Mukha Shopha, Sarvangashopha, And Akshikutshopha.*[58]
3. *Angamarda:- Angamarda, Jadata, Sarvangshool, Anshshoola.* [x]
4. *Udarshoola:- Udaradhamana, Udarshoola, Urodaha, Malavahabhadhata.*
5. *Mutradushti:- Sarkta Mutra, Spitmutra, Alpamutra, Bahumutra.*
6. *Chhardi :- Hrullas.*[xi]
7. *Trushana :- Shosha.*[ii]
8. *Anannabhilasha: - Aruchi.* [xii]

These lakshana are grouped for statistical analysis for better assessment of lakshana using Chi-square test.

Stage 1 & Lakashana:-

The probable Dosha lakashanas are *Vataj – Ayasen-shwas, Daurbalaya, Katishoola etc. Pittaj* lakashanas observed are *Urodaha and Kaphaj* lakashana observed are *Shotha, Hrullas, and Anannabhilasha*. The probable dhatu lakashanas are *rasa and rakta*. *Avastha lakashana are Angamarda and Agnim*andya, these are *Sama lakashana. The Sroto Dusti* lakashanas observed are

Anannabhilasha – Rasavaha[59]*, Annavaha*[60]

Hrullas – Rasavaha

Prandushti – Pranavaha,[61] *Annavaha, Udakavaha*[62]

Stage 2 & Lakashana:-

In the stage 2 Dosha lakashana observed in patients are *Vataj – Malavabadhata, Pindikodwestana, Daurbalaya, Pittaj – Sarakta Mutra Pravartana, Kaphaj – Anannabhilasha, Hrullas and Shotha. The Avasthadarshan* lakashanas observed are *Angamarda, Mandajawara are Sama* lakashana. The *Srotodushti* lakashanas are observed

Prandushti – Pranavaha, Annavaha, Udakavaha

Anannabhilasha – Annavaha

Chhardi, Hrullas – Rasavaha

Mutralakashana- Mutravaha[63]

The *Dhatudusti* lakshana are *Daurabalaya, Pindikodwestana are Mamsa, Rakta Dhatu dushti* lakashana.

Stage 3 and Lakashana:-

The dosha lakshana for Vataj dosha lakashanas are *Daurbalya, Anidra, Malavabhadhata while Pittaj* lakshana are *Trishna, Amlapitta and Kaphaj* lakashana are *Shotha, Bahumutrata*. The *Avastha lakashana* are *sama* lakashana *Trishna* and *Amalpitta*. The dhatudushti lakashanas are *Daurbalaya and Pindokodwestana*. The srotas dushti lakashana

Prandushti – Pranavaha, Annavaha, Udakavaha

Mutralakashana- Mutravaha

Anannabhilasha - Annavaha

Hrullas – Rasavaha

Stage 4 & Lakashana:-

The Dosha lakshanas for stage 4 are as follows:-

The dosha lakashanas are *Vataj – Anidra, Pindikodwestana, Pittaj – Trushna, Bhram and Kaphaj – Kandu.* The Avastha Lakashana observed *Ajeerna and Aruchi*. The *Dhatu Dushti* lakshana is *Pindikodwestan and Dauarbalya*. The Srotodushti laskshana

Anannabhilasha – Annavaha, Rasavaha

Prandushti- Pranavaha, Annavaha, Udakavaha

Stage 5 & Lakashana:-

In stage 5 lakashanas observed are as follows:-

The dosha lakashan are *Vataj – Daurbalaya, Anidra, Katishoola, Pittaj lakashana are Amalapitta, Sarvangadaha and Kaphaj lakashana are Chhardi, Udarjadata.* The *Avasthadarshak* lakashana observed are *Akshikutshopha, Aruchi, and Aarti*. The Dhatu dushti Lakashana is dauarbalya.

Anannabhilasha – Annavaha, Rasavaha

Prandushti- Pranavaha, Annavaha, Udakavaha

Updrava lakashna: In CKD signifiacant Updrava lakshana observed is Pandu (Anemia)

Other lakshana Observed:

1. Antardaha and shitkamitava: The shitkamitva and Antardaha is observed in patients of CKD.

2. *Karnashweda:* The karnashweda is observed in CKD patients which can be probable lakshana of *Pandutava or vatavruddhi.*

Limitation for Dosha lakashana:-

Dosha & Avastha:-

The study is based upon one time examination dosha lakashana require *bahukalika parishana* (frequent follow up). The dosha lakashana are observed in relation with hetus, its *sansarg and sannipat*. In this study, probable dosha lakashanas are mentioned.

Bahudosha lakashana:-

These are lakashanas are not mentioned only probability mentioned in the given study. As these lakashanas are for essential for treatment.

Limitation for gradation of patient's lakashana and causes:-

1. Only dietary hetus are classified as *Pravar/ Avara/ Madhyam* on the basis of daily/ twice- thrice week and once in week the servings.
2. For other causes patient's interrogation is difficult as the disease is critical.
3. As the study is based on 110 patients if gradation done then only few cases for lakashana e.g. 2 to 3 were expressed so in the study only 0 – 1 present / absent. Scale is used as per research protocol.
4. Onetime study pattern followed:-

   As the disease CKD is defined only after ≥ 3 months duration, GFR raised it suggests that the disease is well establish in patients so the case assessed once in each stages.
5. *Vyadhi pratgatmika lakshana*:- to get the precise knowledge of lakashana larger sample size required.

6.5 Samprapti discussion-

Samprapti means disease pathogensis from Ayurveda aspect. In observed cases, samprapti methodology is the correlation between hetus, lakshanas and adhisthana i.e. causes sign & symptoms and organ involved. The observed data is based on clinical as well laboratory finding to understand the appropriate lakashana related to the stages.

Samprapti is the process to understand dosha dushti due to respective hetus (causes) and its spread (*Urdhava, Adho & other*) in sharir (body).

To Understand Samprapti In CKD:-

1. ***Sankhya Samprapti*** denotes involvement of dosha i.e. *vata, pitta, Kapha, dwandaja* or sanipathik combination.
2. ***Pradhyna Samprapti***: - it gives knowledge about dosha dushti with their proportionate guna involvement respectively – *Vrudhatara or Vrudhatam*a.
3. ***Vidhi Samprapti*** indicates *Nija & Agantuja vyadhi*.
4. ***Vikalpa Samprapti***: - Vikalpa samprapti gives knowledge of *ansha-ansha* aspect of *dosha, dhatu & their features.*
5. ***Bala Samprapti*** (as per kala):-

   This samprapti is related with *Hetu, Poorupa and Roop* means causes, *prodormal* symptom and expressed symptom are established in total completely or partially. This gives *bala* strength of the disease vice versa if hetus are not dominant as well *pooroop* then vyadhi is also with less gravity.

Stage 1

The Santarpan hetus observed are

- *Santarpana hetu:- Madhur, Amala, Lavana, Abhishyandi, Adhyshana, Mamsarasa, Diwaswap.*
- *Apatarpan Hetu:- Katu, Tikta, Kashaya, Jagaran,*[xii] *Vyayama*[xiv]
- *Agnidaestijynya Hetu:- Vishmashana,*[xv] *Paryusheet, Vegavarodha,*

Stage 1:-

The santarpana hetus observed are excess *Madhur, Mamsa, Abhishyandi Ahara, Asyasukha and Diwaswap.* While Aapatarpana hetus are excess *katu ahara, jagaran and aatap.* The vyadhi hetus are DM & HT. With santarpana, aapatarpana along with *vishamashana, adhyshana, vegavarodha* leads to vataprakopa leading to agnidusti. The dushit agni causing tridosha prakopa. This, prakopita tridosha disturbs function of *Pranavaha, Annavaha, Udakavaha, Rasavaha, Raktavaha*[66] *and Mamsavaha*[67] srotas mainly. The prakupit dosha gets sthanashavashrya in vrukka disturbing its function. Due to this Lakashanas observed in study are *Daurbalya, Anannabhilasha, Pranadushti, Shotha and Udarshool* in stage 1.

Stage 2:-

Hetus observed are santarpana hetus excess Madhur, Lavan, Mamas Ahara & Asysukha. The Aapatarpana hetus excess *Katuahara, vyayama and jagarana,* vyadhi hetu observed are DM & HT. The vegavarodha & vishamashana hetus are vidhi, Agni & pariahara viruddha. These hetus leads to vata, Kapha prakopa. The prakupita dosha and agnidushti leads to rasa, rakta and mamsa dushti. The dosha & dushya dushti is circulated throughout sharir, by means of vyan vayu disturbing srotas. The srotas dushti observed is *Anna, Prana, udaka, Rasa and Mutra vaha* srotas. The dushti lakashani for stage 2 in CKD are *Anannabhilasha, Chhardi, Hrullas, Shotha, Pranadushti, anngamarda and mutra dushti.*

Stage 3:-

In stage 3 santarpana hetus are excess *Madhur, Abhishyandi Ahara and Asyasukha.* While apatarpan hetus are katu Ahara and vyayama vyadhi hetu are DM & HT. viruddha hetus *vidhi, Krama, Parihara, Dosha and Virya* are observed leading to agni dushti however, *agnidushti leads to tridosha prakopa.* The prakupita dosha leads to dhatu dushti i.e. *rasa, mamsa dhatu both prakupita* dosha and dhatu by means of vyana vayu are circulated throughout body terminally sthanasamshraya in vrukka. Lakashanas observed are *Anannabhilasha, Chhardi, Shotha, Pranadushti and mutradushti.*

Stage 4:-

Santarpana hetus are excess *Amla, Lavana, Abhishyandi, Mamsa Ahara, Diwaswap and Asyasukha apartana* hetus are excess *Katu, Vyayma And Jagaran*. The vyadhi hetu is DM & HT. The viruddha hetu observed are *vishmanhana and vegavarodha.* The agnidushti leads to *tridosha prakopa*. The prakopita dosha and dushya by means of vyana circulated throughout sharir and *sthansmashraya* in *vrukka*. The prakupit dosha leads to srotas dushti lakashanani *Shotha, dauarbalya, pranadushti and Anannabhilasha*. Srotas involved are *anna, prana, udaka, rasa, rakta and mamsavaha srotas.*

Stage 5:-

The hetus observed are apartana, Santarpan, viruddha and mainly vyadhi hetu. The santarpana hetu excess *amla, Abhishyandi, Mamsarasa and Diwaswapa leads to Kapha dushti and kleda* dushti while apartana hetus *Katu, Vyayama and Jagaran* are responsible for vata dushti. Thus, combination of all hetu leads to vata Kapha dushti mainly with viruddha hetu *rasadi dhatu* dushti occurred. The dhatu dushti with agni dushti observed. The dushit dosha circulated by *vyana vayu* throughout sharir disturbing *Anna, Prana, Udaka, Rasa, Rakta and Mamsavaha srots*. The srotodushti lakashanas are *Daurbalya, Anannabhilasha, Pranadushti, Shotha and Anngamarda.*

<u>Samanya Samprapti:-</u>

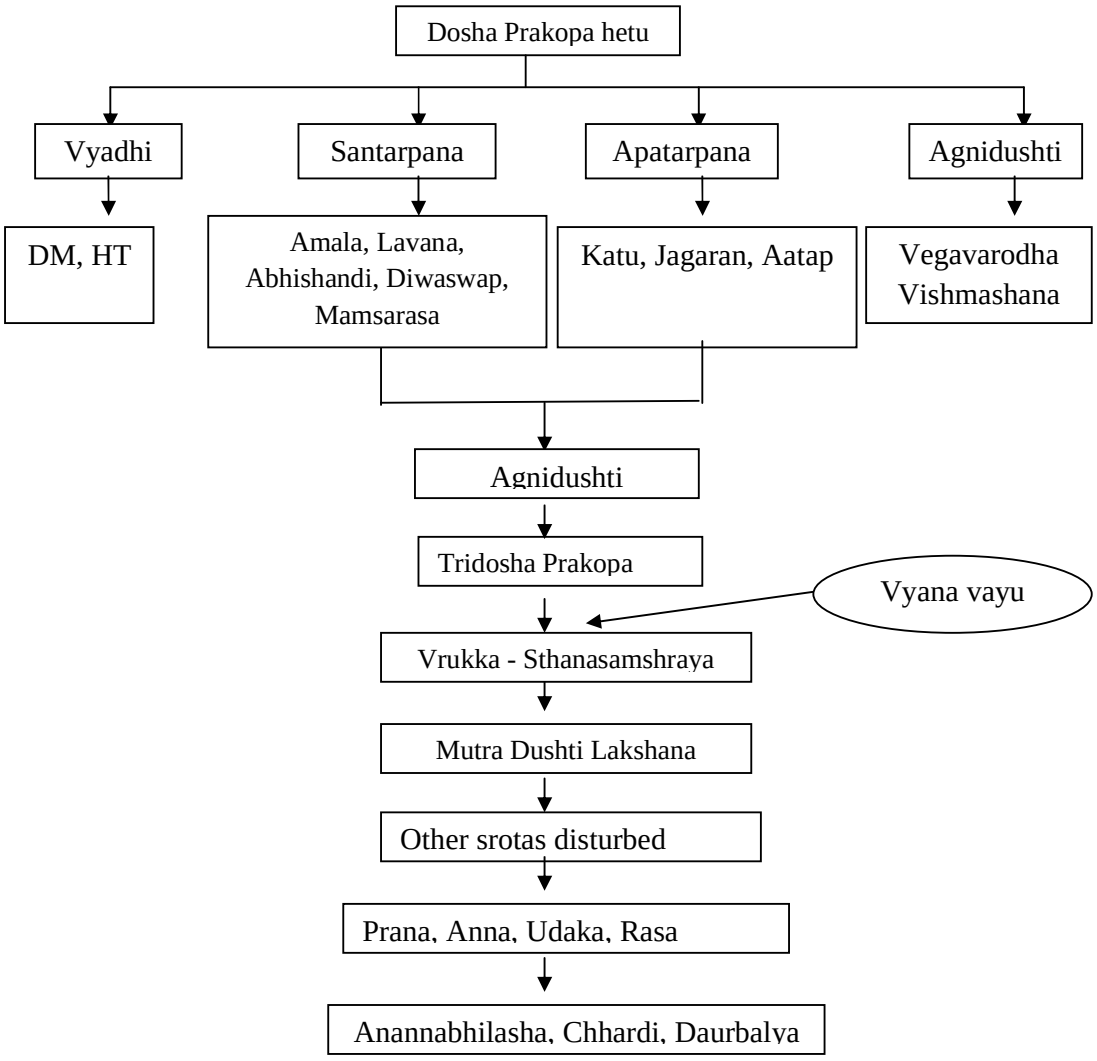
प्रतिरोगमिति क्रुध्दा रोगाधिष्ठानगामिनीः। रसायनीः प्रपद्याशु दोषा देहे विकुर्वते ।। अ. सं. नि. १ /१८

The dosha dushti caused by different hetus, instantly these dushti dosha are spread in the body through *Rasayanis* and reaches up to *Adhisthana* resulting in vyadhi nirmiti.

व्यानेन रसधातुर्हि विक्षेपोचितकर्मणा । युगपत् सर्वतोऽजस्रं देहे विक्षिप्यते सदा।। Ch. Chi. 15/36, page 516.

Prakrut karma of *vyana vayu is vishepana of rasadi dhatu* throughout body. In *vyadhi nirmiti the khavaigunya* – one of the important component of vyadhi is already present respective organs. Here, due to *khavaigunya* gets obstructed giving rise to vyadhi.

The hetus observed are *santarpana, aapatarpana, agnidushtijanya and vyadhi* hetu. Due to this hetus *agnidushti* accrued. The agni dushti leads to *tridosha prak*opa the prakupit dosha with *vyana vayu* circulated throughout sharir gets *sthanashavashrya* in vrukka resulting in *mutravaha and other srotodushti*. This is *samanya samprapti* for CKD in given study.



Flow Chart No.14 Samanya Sanprapti

6.6 Investigation discussion :-

In this chapter investigation required for CKD such as Hb % blood urea Sr. Creatinine and urine analysis (pus cells and protein) are discussed.

1. Hb distribution: - The Hb % grouped under the criteria of mild, moderate and severe Anemia. The patients observed in mild Anemia are 38, moderate Anemia – 51 and severe Anemia - 11. In CKD Anemia is observed as lakashana in previous history and complication. The Rasa & Rakta dhatu dushti is mainly observed.

2. Blood urea distribution:-The urea below 50 is 13, between 50 to 100 - 28, 100 to 150 - 14 and above 150-6 patients observed. The disturbed Saara kitta vibhajana is significantly observed in end stages.

The urea is the outcome saara kitta vibhajana in initial pachana. It is nitrogenous waste product i.e. mala is to be expelled out at mutrashaya level. In CKD mentioned hetus leads to dosha dushti basically Pitta dominance, Kapha and Vata. Also Rakta, Meda & Mamsa dhatus both uniformly disturbs the kidney tissues i.e. the vrukka dushti.

3. Serum creatinine distribution:-The normal value of serum creatinine is 1.2(units) above the normal value that is considered pathological in the study below 1.6 – 20, between 1.6 to 5- 40 were observed, 5 to 10- 29 and above 10 – 13 patients were observed.

The creatinine is liberated from muscle mass. The level of creatinine is important in major GFR in CKD cases. Thus nephron level filtration hampered may be because of DM, HT & anemia origin which may differ but the severity of damage of Nephron is measured with creatinine only.

4. Pus cells:-The pus cells 1 to 4 - 18 observed, 4 to 8 – 19 observed & 8 to 12 – 6 patients are observed.

5. Urine protein distribution:-Trace urine protein present in 7 patients, 1+ urine protein present in 7, the 2+ in 21, 3+ in 17, 4+ in 8, 5+ in 1 & 6+ in 2 patients observed.

6.7 Statistical Discussion:-

Aharia Hetu:-

The cross table for *Amla rasa and Madhur rasa* is non significant with staging. The staging is assessed with GFR, for that aharia hetu are not observed significantly. The *Madhur and Lavana rasa* are responsible for *kleda vruddhi,* as well as up lepana. The *Amlarasa* is causative factor for *mamsadaha & shaithilya*.[55]

The dosha dominance form stage 1 to 5 is *kapha,* the lakshanas were observed mainly *Anannabhilasha, Chhardi, Shopha, Pranadushti Lakshana and Daurbalya.* The *Amla & Madhur* are *kapha prakopa and vata shamak* (pacifier).

The shaethilya is seen in mamsa dhatu agni dushti leading to disturbances in creatinine level.

Shoka (grief):-

The shoka is manasabhava. It is feature of *meda, shaya and vata vruddhi and kleda dushti*. This leads to disturbing pachana, shoshana karya of pitta as well of Agni parinamana. Thus shoka and staging is significantly observed. The Shoka observed as swedavaha and twak dushti lakshana.

Koshtha Viruddha stage:-

The koshtha viruddha is significantly $P<0.05$ observed. The Koshtha viruddha ahara sevana leads to disturbed Saara kitta vibhajana. This gives accumulation kitta and causing disturbances in excretion.

Agni Viruddha and Stage:-

The patients observed Agni viruddha were $P< 0.01$ significantly. It means the ahara is not taken as per Agni status. The patients were observed with Manda, Tikshna and Vishama Agnis. The diet taken by they were of atimatra, singdha, katurasa sevana. The ahara in accordance with Agni gives proper digestion (pachana). In each stage Agni viruddha is observed which denotes that Agni viruddha ahara sevana leads to dosha dushti leading to improper digestion (pachana). The agni viruddha as per the staging is Stage 1 –60%, Stage 2 – 100%, Stage 3 -50%,

Stage 4 – 87%, & Stage 5 – 72.2%. Thus improper digestion at the end part of organ means filtration level is deranged. This is labeled as pachana dushti at vrukka level by viruddha hetus.

Matra Viruddha & stage:-

The Matra viruddha and staging P< 0.01 were significantly observed. The Matra (Rashi) is mentioned for individual as per individual Agni. Those who were not following the Matra towards Agni are causes vata vitiation along with pitta dushti, in them. These vitiation leads to reduction in *Saara & kitta vibhjana*. This may reduce filtration and staging.

Paka Viruddha and Staging:-

If properties and procedure mentioned as per texts are not followed or altered. It disturbs pachana with Agni dushti.

Vidhi Viruddha & stages:-

It is P < 0.00 significant. The ahara vidhi gives bala, Varna and good health. The vidhi viruddha hetu signifies that absorptive & post absorptive phase of ahara sevana and dispersion of ahara rasa improper formation of (dosha, dhusya and mala) from pachana, leading to disease.[64]

Asyasukha & stage:-

It is significant P < 0.04. It leads to kapha dushti further leading to kleda vruddhi or dushit kleda into mutra dushti.

DM & staging:-

The DM is the disease of *kapha & kleda dushti* leading to progression of disease (P< 0.039).

HT& staging:-

HT is cause for CKD from the vyadhi hetu in each stage HT is observed to be present in each stage of CKD (P < 0.03).

Anemia & staging:-

The Anemia in the CKD apparently cause and complication. In anemia pitta dushti along with rakta & meda dhatu is observed. The anemia is observed significantly. Anemia is present in CKD ($P < 0.00$)

Daurbalya and Anannabhilasha:-

The *Anannabhilasha* is Strotas dushti lakshana for Annavaha Strotas and *Daurbalya* lakshana are observed in stage 1 to 5 significantly. The *Daurbalya* present and *Anannabhilasha* absent in staging are observed ($P < 0.03$).

Diwaswap & cortico medullar differentiation:-

The *Diwaswap* absent is significant $P< 0.04$ for cortico medullar differentiation deranged this leads to Varna formation in vrukka leading to structural changes. The *Diwaswap* leads to kapha dosha dushti and kleda dushti.

Abhishyandi & Mutra day frequency:-

The *abhishyandi* ahara sevana is significant $P< 0.04$ to *mutra* frequency. The *abhishyandi ahara* sevana leads to *kleda dushti* leading to disturbed mutra frequency.

Multivariant Correlation:-

Agni Viruddha, Virya Viruddha & stage:-

It is significant in staging $P < 0.01$. The Agni & virya in combination worked together, it will affect the pachana and the formation of dosha. Agni viruddha observed leading to ajeerna etc. In the above mentioned group vriya viruddha absent and Agni viruddha were present.

Agni Viruddha, Paka Viruddha & staging:-

The Agni viruddha creates tridosha dushti dhatu dushti. It is significantly observed in 5th stage. This tridosha dushti leads to reduction in GFR significantly ($P < 0.01$).

Hetu, lakshana & staging:-

The correlation of hetu to lakashana and staging assessment between Aatapa sevana and patient suffering from *Anannabhilasha* are non significant. It shows that viharia hetu i.e. *Aatapa* is not hetu for *Anannabhilasha* in CKD. The *Anannabhilasha* present and Aatapa hetu absent is observed in staging of CKD. As Anannabhilasha is *Agni dushti* lakshana due to *samana vayu & disturbed pachak pitta.*

Previous History of DM, Anannabhilasha & Shotha:-

The previous history of Diabetics to *Anannabhilasha and Shotha* is significant ($P < 0.05$).

Anemia, Daurbalya & Shotha:-

The *Shotha* absent and *Daurbalya* present were ($P < 0.03$) observed in staging. While *Shotha* and *Daurbaly*a in Anemia were reaches to significance ($P < 0.08$).

Previous history of HT, Daurbalya & Pranadushti:-

In staging *Daurbalya* absent and *Pranadushti* present is ($P < 0.01$) observed. The *dosha dushti* leads to *Pranvaha Strotas* dushti showing *Aysena-shawas, shawaskashta.*

Anemia, Lavan & Amla:-

The *Amla and Lavana* rasa are responsible for *pitta & rakta dhatu dushti*. It can affect the GFR directly with other hetus ($P < 0.05$).

Amla, Lavan & Twakdry:-

Twak dryness is observed in patients. The *Amla Lavana rasa causes rasa, rakta dushti and pitta dosha dushti* leading to disturb state of twak and dryness observed ($P< 0.03$).

Mamsahara, Agni & Staging:-

The mamsahara taken by patients in *Agni viruddha* state leads to *tridosha prakopa and rasa dhatu dushti* ($P < 0.00$).

Mamsahara, Urine Protein & Stage:-

The *mamsahara* and protein loss is significantly observed in stage 5. The *mamsahara* is not significant for urine protein in 1 to 4 stages in patients observed ($P< 0.04$).

Mamsahara, Agni & Staging:-

While stage 1 to 4 the test is not significant. As the state of *agni & dhatu kshaya* is in progression with agni dushti. This is observed in stage 5 significantly.

Chhardi, Anannabhilasha & staging:-

The *Chhardi Anannabhilasha* is significant for 2 to 4 stages $P< 0.01$ while stage 1 to 5 *Chhardi Anannabhilasha* is not significant.

Chhardi, Shotha & Abhishyandi:-

The *abhishyandi* sevana is significantly present in *Chhardi & Shotha*. The *abhishyandi is dushti hetu* for *kapha and pitta dosha* ($P < 0.03$).

Chhardi, Anannabhilasha & Agni:-

The *Sama Agni* patient *Anannabhilasha* is significant $P < 0.00$ while *manda agni* patients *Anannabhilasha* is $P< 0.01$ significantly observed.

Chhardi, Anannabhilasha & Abhyvaran:-

When *Abhyvaran Shakti is prakrut, Chhardi and Anannabhilasha* is $P< 0.00$.

Madhur, Lavana, Amla and Staging:-

In stage 5, Amla and Lavana rasa are observed significant $P<0.03$. However it suggests kaphakara ahara leads to the GFR reduction the Lavana rasa is for pitta dushti and rakta dushti as well Amla rasa is causative factor for mamsadaha and shaethilaya.

Anemia, Shotha, Anannabhilasha & stage:-

The Shotha absent and Anannabhilasha present group is significant $P < 0.04$. The rakta dhatu dushti along with rasa dhatu is observed. The rasa mala kapha and rakta mala pitta with Agni dushti is observed for shotha Anannabhilasha lakshna.

Anemia, Anannabhilasha, Chhardi & stage:-

The *Chhardi* absent and *Anannabhilasha* present is significantly observed in Anemia and CKD staging $P < 0.02$. The *Chhardi and Anannabhilasha* are not significant in Anemia CKD staging 0.18.

7. Conclusion:-

1. The aetiopathological study of CKD with Ayurveda perspective gives hetus with dosha dushti to understand the diseases. It is beneficial in preventive aspect of diseases for 'Nidana Parivarjana".
    - DM, HT, Anemic patient should avoid Amla & Lavan rasa in excess.
    - Antagonistic food should be avoided i.e. viruddha ahara.
    - Proper regime should be followed by individual as per their agni. Hetus observed were *Amla, Lavana dominant rasa, Abhishyandi, Mamsahara, Vishamashana, Diwaswap, Asyasukha* etc.

2. The Viruddha hetu /antagonistic food in the form of *Matra, Vidhi, Kala, Agni, Karma* are the causes for diseases progression. This antagonistic food has proved to be one of the important causes for reduction of GFR.

3. The Santarpana hetus such as excessive intake of *Amla and Lavana rasa* individually or in combination by patients of mainly DM, HT and Anemia causes further disturbances in the states of *Pitta, Rakta and Mamsa* leading to dhatu paka avastha.

4. The individual must avoid Santarpana hetu such as *Asyasukha and Diwaswap*. As the *Asyaskuha and Diwaswap* disturbs the state of pachana leading to kapha dushti in turn producing kleda dushti by further precipitation.

5. The Santarpana hetu such as *Abhishyandi ahara and Mamsaahara* in combination should be avoided this gives *kleda dushti, kapha dosha dushti*.

6. Regular evaluation should be followed to prevent terminal symptoms such as *Anannabhilasha, Chhardi and Hrullas* through investigation of proteinuria & micro albuminuria. These are probable dosha lakashana.

7. The staging as per Ayurveda aspect is on the basis of tridosha dushti. The *Vata, Kapha, Agni* has proved to be the main cause.

8. CKD has expressed itself as Bahu (multi) strotas disorder:
    - *Annavaha, Pranavaha, Udaka, Rasa, Mamsa*
    - *Anavaha, Pranavaha, Udaka, Rasa, Mutra*
    - *Anavaha, Pranavaha, Udaka, Rasa, Mutra*
    - *Anavaha, Pranavaha, Udaka, Rasa, Rakta, Mamsa*
    - *Anavaha, Pranavaha, Udaka, Rasa, Rakta, Mamsa*

The kha-vaigunya occurs in mutravaha strotas.

9. The emphasis should be on tikta and kasaya rasa since they have property of vishada & vishodana of mutra. Leading to healthy state of individual.

10. The viharia hetu alone are not significant. It should be avoided in association of above mentioned hetus.

11. In CKD probable lakshana sammuchya is lasting feature of stages as per Ayurveda rather than GFR estimation parameter as mentioned in modern medicine.
    - *Anannabhilasha, Daurbalya, Prandushti, Shoth*
    - *Anannabhilasha, Chhardi, Shotha, Prandushti, Mutra dushti*
    - *Anannabhilasha, Chhardi, Shotha, Prandushti, Mutra dushti*
    - *Anannabhilasha, Shotha, Daurbalya, Pranadushti*
    - *Anannabhilasha, Daurbalya, Pranadushti, Shotha lakshanas are there.*

12. The vyadhi hetu observed mainly HT & DM.

13. *Vrukka Virkruti / Dushti* observed as *Premhajyna, Shothajyna, Panduprabala, Yakrut Dushtijyna, Medopradosha vrukka dosha*, this can be treated as avasttika dushti of vrukka.

8. Scope for further Study

1. To standardize a preventive tool, this will prevent disease progression instead of landing into ESRD.
2. Further study can be possible to set a precise evidence based treatment as per the samprapti & its stages.
3. To prevent dialysis with the Ayurvedic treatment such as to develop shodhana treatment plan like raktamokshana, nasya etc as the treatment.
4. Specified Ayurvedic Diet treatment for CKD is needed.
5. To reduce the economic burden and to enhance quality of life of ESRD patients with standardized protocol to maintain diseases like DM, HT and Anemia.
6. Large sample size is needed to define pratyatmika lakshana and standardization of symptom.
7. Enough sample size is required to explore ansha-ansha samprapti of stages.
8. Further study is needed related to viruddha Ahara leading to end stage renal disease along with vyadhi hetu since significant statistical values were observed.
9. The involvement of mutravaha srotas in CKD and its effects on other srotas are studied in large sample size.
10. The patients should be observed longer period to assess for gradation of dosha lakashana, Sama lakashana and srotas involvement.
11. The involvement of srotas mutravaha or annavaha priorly is to be studied in larger sample size.

9. Bibliography

1. API Textbook of Medicine, 7th Edition, 2003
2. Ashtang Hrudaya, Kaviraj Atrideo Gupta, 20th Edition, 1997 Choukhamba Publication
3. ***Astangahrdaya of Vagbhata*** – edited by Pt. Hari Sadasiva Sastri Paradakara Bhisagacarya, the Chaukhamba Ayurvijana GranthAmla 54, publishers – Chaukhambha Orientalia, Varanasi -221001.
4. ***Astangahrdayam of Vagbhata-*** by Kaviraja Atrideva Gupta, edited by vaidya Yadunandana Upadhyaya, the kashi Sanskrit series 150, publishers – Chaukhambha Sanskrit Sansthan, Varanasi -221001.
5. Ayurvediya Shabdkosh Mahakosh, Venimadhav Shastri, N. H. Shastri, M. R. Sahitya & Sanskruti Mandal, 1968
6. Ayurvediya Triskandh Hetukosh Prathamkhand, Vaidya D. P. Gadgil, T. M. V. Edition 2004
7. Charaksamhita Chakarapani Tika, Vd. Yadavji Trikamji Acharya, Choukhamba Publication, Edition 2001
8. Charaksamhita, Acharya Vidyadhar Shukla, Ravidatta Tripathi, Choukhamba Publication 5th Edition 2001.
9. ***Chkradatta of Sri Chakrapanidatta*** – by Dr. Indradeva Tripathi, the kashi Sanskrit series 252, publishers – Chaukhambha Sanskrit Sansthan, Varanasi -221001.
10. Davidsons Principles & Practice of Medicine, 16th Edition ELBS
11. ***Food Science & Nutrition*** – by Sunetra Roday, Published in India by Oxford University Press 2007.
12. ***Handbook Nephrology and Hypertension*** – indan edition, edited by Simon Steddon, Neli Ashman, Alistair Chesser, John Cunningham, Published in india by Oxford University Press 2006.
13. ***Harita samhita***: - text with Asha Hindi commentary by Ramavdlamba Shastri M.A. Ph.D. Govt. Ayurvedic college, Varanasi Praechyw Prakashanas Varanasi -221002, 1985.

14. Harper's Biochemistry, 25th Edition, by Robert K. Murray, Daryl K. Granner, Peter A. Mayes, and Victor W. Rodwell.
15. Harrison's Principles of Internal Medicine, 17th edition, volume II, 2008.
16. Human Physiology, Dr. C. C. Chaterjee, Medical Allied Agency, 1997 Edition
17. ***Kasyapa Samhita*** –by Nepal Rajaguru Pandit Hemaraja Sarma, the kashi Sanskrit series 154, publishers – Chaukhambha Sanskrit Sansthan, Varanasi - 221001.
18. ***Madhavanidanam of Sri Madhavakara*** - by Sri. Sudarsana Sastri, the kashi Sanskrit series 158, publishers – Chaukhambha Sanskrit Sansthan, Varanasi - 221001.
19. ***Madhavnidan Madhukoshtika*** – 1, Raghunandan Upadhyaya, Choukhamba Publication 2003
20. ***Nighantu Tatha Nirutksti:*** - Nirudshana, Bhashavigan evam shabdarthvigan ka praobitam bhartiga &rathas (Hindi Anuvala peristritha) bhvashana yogi and shashikumar publisher motilal Banarsitas.
21. Principles and Practice of Medicine, 7th edition, edited by C.R.W.Edwards/ I. A. D. Bouchier/ C. Haslett/ E. R. Chilvers.
22. Raj Nighonta of pandit Narahari edited with 'Dravyaunaprakasika' Hindi commentary by Dr. Indradeo Tripathi, B.I.M.S.D.Sc. An Introduction by Alarya viswanatha Dwivedi krishnadas academy Varanasi 1998.
23. ***Sarngadhara – Samhita*** – by Pandit Sarngadharacharya son of Pandit Damodara, Jaikrishnadas Ayurveda Series No. 53, publishers – Chaukhambha Orientalia, Varanasi -221001, Fifth edition 2002.
24. ***Sarth Charaksanhita***, Late Shankar Dajishastri Pade, Choukhamba Publication Khand- 1.
25. ***Sarth Waghbhat***, Dr. Ganesh Garde, Anmol Publication
26. ***Sushrutsamhita***, Dr. B. G. Ghanekar, Mar. 2004, Meherchand
27. ***Synchro Destiny*** – by Dr. Deepak Chopra, Author of the million- copy bestseller Ageless Body, Timeless Mind.
28. ***Text book of Medical Physiology***, Guyton & Hall, 10th Edition
29. ***Textbook of API Medicine, 8th edition , Volume 1***, editor in chief Siddharth N. shah, Published by The Association of Physicians of India, 2008.

30. ***Textbook of Medical Physiology, 11th edition***, Arthur C. Guyton, M.D.
31. ***Textbook of Pathology***, Jaypee Brothers Medical Publishers (P) LTD, New Delhi.
32. ***The Savagdhara*** – samhita by pandit saaragadhoracharya son of pandit Damodra with the commentary Adhamalla's Dipika and Kasivama's Guddhartha Dipika edited with foot notes by pandit Parasuram Sastri, Vidyasagar publishers chaukhambha orientalia post Box No.12 C8032 varanasi 221001 (U.P.) India fourth edition.

33. Common pathophysiological mechanisms of chronic kidney disease:

Therapeutic perspective:- Jos'e M. Lopez-Novoa[b,d], Carlos Martinez-Salgado[a,b,d,]*, Ana B. Rodriguez-Pena[c], Francisco J. Lopez Hernandez [a,b,d,]*, Unidad de invesrigacion, Hospital Universitario de Salamanca, Salamanca, Spain, Unidad de fisiopatologia Renal y Cardiovascular, Departamento de Fisiologia y Farmacologia, Universidad de Salamanca, Salamanca, Spain, National institutes of Health, Bethesda MD, USA, Instituto Reina Sofia de Investigaci'on Nefrol'ogica, Fundaci'on figo alvarez de Toledo, Spain

34. ***Medical Nutrition Therapy in Chronic Kidney Failure: Inter grating Clinical practice Guidelines :-*** Judith A. Beto; Vinod K. Bansal, Nutrition sciences, Dominican University, 7900 W. Division, River Forest, IL 60305. Journal of the American Dietetic Association Vol.: 104, No.: 3, March 2004.

35. ***Managing Anemia of Chronic Kidney Disease:-*** Susan A. Krikorian, Department of Pharmacy practice, School of Pharmacy, Massachusetts College of Pharmacy, American Journal of Lifestyle Medicine Vol.:3, No.: 2, April 2009 (Page 135-146)

36. ***Growth and Body Composition in Children with Chronic kidney Disease:*** R. Rashid; E.Neill; H. Maxwell; S. F. Ahmed, Bone & Endocrine Research Group, British Journal of Nutrition Vol.: 97, No.: 2, February 2007(Page 232-238)

37. ***Renal Osteodystrophy in Children:*** A Systemic Disease Associated with Cardiovascular Manifestations, Craig B. Langman; Ellen R. Brooks, Children's

Memorial Hospital, 2300 Children's Plaza, MS #37, Chicago, IL 60614, University 3930; fax: +1 773 327 5072. Growth Hormone & IGF Research Vol.: 16, No.: S1, July 2006 (Page 79-83)

38. ***Application of Branched- Chain Amino Acids in Human Pathological States:*** Renal Failure, Nowl J. M. Cano; Denis Fouque; and Xavier M. Leverve, INSERM- EO 221, Universit'e Joseph Fourier, Grenoble, France, Journal of nutrition Vol.: 136, No.: 1, January 2006 (Page 299-307)

39. ***A Challenge to Chronic Kidney Disease in Asia:*** The Report of the Second Asian Forum of Chronic Kidney, Yusuke Tsukamoto ; Vivekanand Jha; Gavin Becker; Hung Chun Chen; Vlado Perkovi; Tungsanga; Haiyan Wang; Zaki Morad., Dr. Yusuke Tsukamoto, Shuwa General Hospital, Department of Nephrology, 1200 ya-Saitama-ken 344- 0035, Japan. American Naturalist Vol.: 157, No.: 5, May 2001 (Page 248-252)

40. ***Therapeutic Potential of Endothelin Receptor Antagonists for Chronic Proteinuric Renal Disease in Humans:*** Matthias Barton, Molecular Internal Medicine, University of Zurich, LTK Y 44 G 22, Winterthurer Strasse 190, CH-8057 Zrich, Switzerland., Biochimica et Biophysica Acta: Molecular Basis of Disease Val.: 1802, No.: 12, December 2010 (Page 1203-1213)

41. ***Nutrition in Advanced Chronic Kidney Disease:*** Biomarkers and Body Composition Tools, Mary B. Sundell; Lara B. Pupim; T. Alp Ikizler, Nutrition Today Val.: 42, No.: 1, February 2007 (Page 22-27)

42. ***Kidney Disease and the Risk of End Stage Renal Disease Versus Death:*** Lorien S. Dalrymple; Ronit Katz; Bryan Kestenbaum; Michael G. Shlipak; Mark J. Sarnak; Catherine Stehman- Breen; David Siscovick; Anne B. Newman; Linda Fried, Dept. of Medicine University of California Davis 4150 V Street, # 3500 PSSB Sacramento CA 95817 USA, Journal of General Internal Medicine, Vol. 26, No. 4, April 2011 (page 379-385).

43. ***Catabolic State after an Overnight Fast in Patients with Chronic Renal Failure:*** Yutaka Nakaya; Takaaki Shimohata; Sayaka Haraguchi; Toshiyuki Nakao; Jun Minaguchi; Haruo Sumitani; Nagakatsu Harada; Sakaue, Dept. of Nutrition and Metabolism, the University of Tokushima Graduate School, Tokushima, Japan Division of Nephro Tokyo Medical University, Tokyo, Japan Kawashima Hospital, Tokushima, Japan, Nutrition vol. 27, No. 3, March 2011, (page 329-332)

44. ***Thology of Chronic Tubular, Glomerular and Renovascular Nephropathies:*** Clinical Implications: Jose M Lopez-Novoa; Ana B Rodriguez-Pena; Alberto Ortiz; Carlos Martinez- Salgado; Francisco J Lopez Hernandez, Instituto de Estudios de Ciencias de la Salud de Castilla y Leon (IECSCYL), Soria, Spain, Journal of Translational Medicine vol.9, No. 13, January, 20 2011.

45. ***Tic potential of Endothelin Receptor Antagonists for Chronic Proteinuric Renal Disease in Humans;*** Mattias Barton, Molecular Internal Medicine, University of Zurich, LTK Y 44 G 22, Winterthurer Strasse 190, CH – 8057 Zrich, Switzerland, Biochimica et Biophysica Acta: Molecular Basis of Disease vol. 1802, No. 12, December 2010 (page 1203-1213).

46. Mani Mk. Chronic renal failure in India. Nephrol Dial Transplant 1993;8;684-9; discussion 683.

47. Sakhuja V, Jha V, Ghosh Ak, Ahmed S, Saha Tk, Chronic renal failure in India. Nephrol Dial Transplant 1994;9;871-2.

48. Khan IH, Catto GR, Edward N, Macleod AM. Chronic renal failure: Factors influencing nephrology referral. QJ Med 1994;87;559-64.

49. Landray MJ, Thambyrajah J, McGlynn FJ, Jones HJ, Baigent C, Kendall MJ, et al. Epidemiological evaluation of known and suspected cardiovascular risk factors in chronic renal impairment. Am J Kidney Dis 2001;38;537-46.

50. ***Barsoum RS, End-*** Stage renal disease in North Africa. Kidney Int Suppl 2003: S111-S114.

51. ***Standring S, Gray's anatomy:*** the anatomical basis of clinical practice. 39th ed. London: Churchill Livingstone; 2005:1269.

52. Levey As, Greene T, Kusek JW, Beck GJ. A simplified equation to predict glomerular filtration rate from serum creatinine. J Am Soc Nephrol 2000;11;155A.

53. Agarwal SK. Chronic kidney disease and its prevention in India. Kidney Int Suppl 2005;68;S41-S45.

54. Pfister M, Jakob S, Frey FJ, Niederer U, Schmidt M, Marti HP. Judgment analysis in clinical nephrology. Am J Kidney Dis 1999;34;569;-75.

55. Ball S, Cook T, Hulme B, Palmer A, Taube D. The diagnosis and racial origin of 394 patients undergoing renal biopsy: An association between Indian race and interstitial nephritis. Nephrol Dial Transplant 1997;12;71-7.

56. Balakrishnan N, John GT, Korula A, Visalakshi J, Talaulikar GS, Thomas PP, et al. Spectrum of biopsy proven renal disease and changing trends at a tropical tertiary care centre, 1990-2001. Indian J Nephrol 2003;13;29-35.

57. Valderrabano F, Gomez-Campdera F, Jones EH. Hypertension as cause of end-stage renal disease: Lessons from international registries. Kidney Int Suppl 1998;68;S60-S66.

58. ***Ation of Renal Function and Disease in Patients with Cirrhosis:*** Claire Francoz, Denis Glotz, Richard Moreau, Francois Durand, Hepatology and Liver intensive Care Unit, Hospital Beaujon, Clichy; INSERM, U773, Centre de Recherche Biomdicale Bichat Beaujon, Hospital Beaujon, Clichy, France, Journal of Hepatology vol. 52, No.4, April 2010.

59. ***World Health Organization:*** Preventing Chronic Disease: A Vital Investment. Geneva, WHO, 2005.

60. Grassmann A, Gioberge S, Moeller S, et al: ESRD patients in 2004: global overview of patient numbers, treatment modalities and associated trends. Nephrol Dial Transplant 2005;20:2587–2593.

61. ***Center for Disease Control and Prevention (CDC):*** Prevalence of chronic kidney disease and associated risk factors – United States, 1999–2004. MMWR Morb Mortal Wkly Rep 2004;56:161–165.

62. Hamer RA, El Nahas AM: The burden of chronic kidney disease. BMJ 2006;332:563–564.

63. Agarwal SK, Dash SC, Irshad M, et al: Prevalence of chronic renal failure in adults in Delhi, India. Nephrol Dial Transplant 2005;20:1638–1642.

64. Mani MK: Prevention of chronic renal failure at the community level. Kidney Int 2003;63(suppl 83):S86–S89.

65. Mani MK: Experience with a program for prevention of chronic renal failure in India. Kidney Int 2005;67(suppl 94):S75–S78.

66. Modi GK, Jha V: The incidence of end-stage renal disease in India: a population-based study. Kidney Int 2006;70:2131–2133.

67. Mani MK: Nephrologist sans frontières: preventing chronic kidney disease on a shoestring. Kidney Int 2006;70:821–823.

68. Agarwal SK, Dash SC: Spectrum of renal diseases in India in adults. J Assoc Physicians India 2000;48:594–600.

69. Mani MK: Chronic renal failure in India. Nephrol Dial Transplant 1993;8:684–689.

70. Mittal S, Kher V, Gulati S, Agarwal LK, Arora P: Chronic renal failure in India. Ren Fail 1997;19:753–770.

71. Sakhuja V, Jha V, Ghosh AK, Ahmed S, Saha TK: Chronic renal failure in India. Nephrol Dial Transplant 1994;9:871–872.

72. Dash SC, Agarwal SK: Incidence of chronic kidney disease in India. Nephrol Dial Transplant 2006;21:232–233.

73. Agarwal SK: Chronic kidney disease and its prevention in India. Kidney Int 2005;98:S41–S45.

10. Reference Review

[1] Harrison's internal medicine 17th edition II, 1762

[2] Large scale population studies oxford journal – medicinenephrology Dialysis transplantation volume 21 issues Page No232-233.

[3] NKF KDOQI GUIDELINES

[4] API text book of Medicine, 8 editions, Shahils Clinical Medicine

[5] National Kidney Foundation Kidney Dialysis Outcome Quality Initiative (K/DOQ)

[6] Vaidika – Pada- Anukrama- Dosa – VVRL S. V. Series, Vol. 10, Sec 4&5,

[7] Vedic word concordance part II, page 1, 170, 835

[8] माता - वृक्कयोः गर्भे ।। Ch. Sh.3/6, page310
रक्तमेदः प्रसादाद् वृक्कौ। As. Sa. 5/28

[9] प्रसादः रक्तस्य च मेदसःच गर्भे - वृक्कयोः गर्भे Su. Sa. 4/31

[10] तत्र द्रव्यावि गुरूखरकठिनमन्दस्थिरविशदसान्द्र। रथूलगन्ध गुणबहुलानि पार्थिवानि।। Ch. Su. 26/11, page 138

[11] वृषणौ बस्तिमेढ्रं च नाभ्युरू वंक्षणौ गुदम् । Ch. Chi. 28/10, page 616

[12] मेदोवहानां स्त्रोतसां वृक्कौ मूलं वपावहनं च! Ch. Vi. 5/8, page 251

[13] अतिसृष्टमतिबद्धं प्रकुपितमल्पाल्पमभीक्ष्णं वा बहप्रदुष्टानीति विद्यात् । च. वि. ५/८

[14] कृच्छ्रेणाल्पाल्पं सशब्दशूलमतिद्रवमतिग्रथितमतिबहु चोपविशनतं द्यष्ट्रा पुरीषवहान्यस्य स्त्रोतांसि प्रदुष्टानीति विद्यात् । च. वि. ५/८

[15] दुष्टि : स्त्रोतसः - स्वेदवहस्य परूषत्वस्य Ch. Vi. 5/8,14-16, page 250,251

[16] समानोद्यग्निसमीपस्थः कोष्ठे चरति सर्वतः। अन्नं गृहाति पचति विवेचयति मुञ्चति ।। Va. Su. 12/8, page 55

[17] शुक्रार्तवशकृन्मूत्रगर्भनिष्क्रमणक्रियः । Va. Su. 12

[18] क्रुद्धश्च कुरूते रोगान् घोरान् बस्तिगुदाश्रयान् । Su. Ni. 1

[19] पीतविण्मूत्रनेत्रत्वक् क्षुत्तृड्दाहाल्पनिद्रताः। पित्तम् लिङग क्षीणेद्यनिलेद्यङग स्य सादोद्यल्पं भाषितेहितम्। संज्ञामोहस्तथा श्लेष्मवृद्धयुक्तामयसम्भवः।। Va. Su. 11/7, page 52

[20] Doshadhatu malavidgyna page 102.

[21] प्रसन्नवर्णेन्द्रियमिन्द्रियार्था - निच्छन्तमव्याहतपक्तृवेगम् । सुखान्वितं तु (पु) ष्टिबलोपपन्नं विशुद्धरक्तं पुरूषं वदन्ति।। Ch. Su. 24/24

[22] मेदसः स्नेहमादाय सिरास्नायुत्वमाप्नुयात् । Su. Sh.

[23] मूत्ररोगांश्चमूत्रस्थं कुक्षिरोगान् शकृद्वतम् । रसादिभिश्च संसृष्टं कुर्याद्रोगान् रसादिजान् ।। Ch. Chi. 15/49, page 517

[24] व्यानेन रसधातुर्हि विक्षेपोचितकर्मणा । युगपत् सर्वतोद्गजस्रं देहे विक्षिप्यते सदा।। Ch. Chi. 15/36, page 516.

[25] श्लेष्मजाःक्षीरगुडतिलमत्स्यानूपमांसपिष्टान्नपरमान्नकुसुम्भस्नेहाजीर्णपूतिक्लिन्नसंकीर्णविरूद्धासात्म्य भोजनसमुत्थानाः! प्रभावो - ह्रल्लासः, आस्यसंस्रवणम्,अरोचकाविपाकौ, ज्वरः, मूर्च्छा, जृम्भा, क्षवथुः, आनाहः, अङ्गमर्दः, छर्दिः, काश्र्यं, पारूष्यं, चेति।। Ch. Vi. 7/12

[26] आहारपरिणामकरास्त्विमे भावा भवन्ति । तद्यथा - उष्मा, वायुः, क्लेदः, स्नेहः, कालः, समयोगश्चेति ।। Ch. Su. 6/14, page 332

[27] क्लेद : - प्रमेहस्य (सं) Ch. Chi. 6/51

[28] शोथः क्लेदसहितः - वातरक्तस्थाने वातरक्तस्य रक्ताधिकस्य ।। Ch. Chi 29/27, page 629

[29] पित्तरोगः - शोणितक्लेद :।। As. Sa. Su. 20/10

[30] पित्तरोगः - शोणितक्लेद :।। Ch. Su. 20/14

[31] प्रलीयन् क्लेदविष्यन्दमार्दवं कुरूते मुखे। यः शीघ्रं लवणो ज्ञेयः स विदाहान्मुखस्य च ।। Ch. Su.26/67

[32] पाण्डुदन्त नखो यश्च पाण्डूनेत्रश्च मानवः। पाण्डूSघातवांश्चैव पाण्डूरोगी विनश्यति।। Ha. 2/4124

[33] मूत्रदोषस्तु विज्ञेयः प्रेमेहो मेह इत्यादि । कृच्छ्रं तु मुत्रकृच्छ्रं स्यात् मुत्ररोधोअश्मरी च सा ।। Ra. Ni.

[34] आश्रयः पक्वाशयः - दोषाणाम् शोथस्य मध्यस्थितस्य (सं) Su. Chi. 23/6

[35] न वेगान् धारयेद्वीमात्र्जातान् मूत्रपुरीषयोः । न रेतसो न वातस्य न छर्द्याः क्षवथोर्न च ।। Ch. Su. 7/ 3

[36] Text book of Medical Physiology 11th edition 308-309.

[37] API text book of Medicine, 8th edition, vol. I, page 733.

[38] The Text book of Pathology-Harsha mohan –chapter 20 The Kidney and Lower urinary Tract.

[39] Pharmacology & Therapeutics, Common path physiological mechanisms of chronic kidney disease: therapeutic perspective.

[40] Handbook of Nephrology, J. M. Lopez – Novoaetal / Pharmacology and therapeutics 128 (2010) 61-81

[41] J. M. Lopez – Novoaetal / Pharmacology and therapeutics 128 (2010) 61-81. Harrison's internal medicine 17th edition II, 1762

[42] Harper's Biochemistry, 25 editions, Robert K. Murray.

[43] संतर्पयति यः स्निग्धैर्मधुरैर्गुरूपिच्छिलैः । कुष्ठान्यामप्रदोषाश्च मूत्रकृच्छ्रंमरोचकः।। Ch.Su.23/3-6

[44] वक्ष्यन्ते सौषधाश्चोर्ध्वमपतर्पणजा गदाः। विण्मूत्रसंग्रहः शूलं जङ्घेरूत्रिकसंश्रयम्।। Ch. Su. 23/26-28, page 123.

[45] अभोजनादजीर्णातिभोजनाद्विषमाशनात् ।अपच्यमानं शुक्तत्वं यात्यन्नंविषरूपताम् ।। Ch. Chi 15/42-44

[46] किट्टमन्नस्य विण्मूत्रं रसस्य तु कफोद्भसृज । परस्परोपसंस्तम्ब्धाधातुस्नेहपरम्परा। Ch. Chi. 15/18-19

[47] शरीरक्लेदं पुनर्दूषयन् मूत्रत्वेन परिणमयति। Ch. Ni. 4/8

[48] शरीरक्लेदस्तु श्लेष्मामेदोमिश्रः प्रविशन् मूत्राशर्यं मूत्रत्वमापद्यमानः श्लेष्मिकैरेभिर्दशभिर्गुणैरूपसृज्यते वैषम्ययुक्तैः। Ch. Ni. 4/9

[49] त्रिदोषकोपनिमित्ता विंशतिः प्रमेहा भवन्ति विकाराश्चापरेद्मपरिसंख्येयाः। इति सर्वविकारविघातभावाभावप्रतिविशेषाभिनिर्वृत्तिहेतुर्भवत्युक्तः।। Ch. Ni. 4/3-4, Page 212.

[50] संहननतच्श्रेति संहननं । च. वि. ८/११६

[51] वक्ष्यन्ते रक्तदोषजाः। नीलिका कामला व्यङग पिप्ल्पवस्तिलकालकाः। Ch. Su. 28/12

[52] स्वेदस्य केशविधृतिः Va. Su. 11/5

[53] अम्लो रसो रक्तं दूषयति, मांसं विदहति, कायं शिथिलीकरोति। Ch. Su. 26/ 2- Page 329

[54] लवणः स्तम्भसङघतबन्धविध्मापनोऽग्निकृत् । सोऽतियुक्तोऽस्रपवनं खलति पलितं वलिम्। A. Ha. Su. 10/ 12- 13, page 175-176.

[55] रसः कटुः - क्लेदस्य शमस्य ।। Ch. Su. 26/43-44, page 143-45,

[56] तत्र, मधुरो रसः शरीरसात्म्याद्रसरूधिरमांसमेदोस्थिमज्जौजः शुक्राभिवर्धन आयुष्यः । च. सू. २६/१

[57] मारूतः प्राणवाहीनि स्त्रोतांस्याविश्य कुप्यति । उरःस्थः कफमुद्धूय हिक्काश्वासान् करोति सः । च. चि. १७/१७

[58] बाह्याः सिराः प्राप्य यदा कफासृक्. पित्तानि संदूषयतीह वायुः । तैर्बद्धमार्गः स तदा विसर्पन्नुत्सेधलिग्ङं ।। च. चि. १२/८

[59] रसवहानां स्त्रोतसां ह्रदयं मूलं दश च धमन्यः । च. वि. ५

अश्रद्धा चारूचिच्श्रास्यवैरस्यमरसज्ञता। ह्रल्लासो गौरवं तन्द्रा साङगमर्दो ज्वरस्तमः ।। च. सू. २८ /९

[60] अन्नवहानां स्त्रोतसामामाशयो मूलं वामं च पार्श्व, प्रदुष्टानां तु खल्वेषामिदं विशेषविज्ञानं भवति; तद्यथा - अनन्नाभिलषणमरोचकविपाकौ छर्दि च द्यष्टावऽन्नवहान्यस्य स्त्रोतांसि प्रदुष्टानीति विद्यात् । च. वि. ५

[61] तत्र प्राणवहानां स्त्रोतसां ह्रदयं मूलं महास्त्रोतश्च, प्रदुष्टानां तु खल्वेषामिदं विशेषविज्ञानं भवति; तद्यथा - अतिसृष्टमतिबद्धं कुपितमल्पाल्पमभीक्ष्णं वा सशब्दशूलकुच्छ्सन्तं द्यष्टवा प्राणवहान्यस्य स्त्रोतांसि प्रदुष्टानीति विद्यात् । च. वि. ५

[62] उदकवहानां स्त्रोतसां तालुमूलं क्लोम च, प्रदुष्टानां तु खल्वेषामिदं विशेषविज्ञानं भवति; तद्यथा - जिह्रातालवोष्ठकण्ठक्लोमशोषं पिपासां चातिप्रवृद्धां द्यष्टवेदकवहान्यस्य स्त्रोतांसि प्रदुष्टानीति विद्यात् च. वि. ५

[63] अतिसृष्टमतिबद्धं प्रकुपितमल्पाल्पमभीक्ष्णं वा बहुप्रदुष्टानीति विद्यात् । च. वि. ५/८

[64] सात्म्यतोऽल्पतया वाऽपि दीप्ताग्नेसतरूणस्य च । स्निग्धव्यायामबलिनां विरूद्धं वितथं भवेत् ।। Su. Su. 20/22

65 शोणितवहानां स्त्रोतसां यकृन्मूलं प्लीहा च। च. वि. ५

66 मांसवहानां च स्त्रोतसां स्नायुर्मूलं त्वक्र् च । च. वि. ५

67 पित्ते मन्देऽनलः शीतं प्रभाहानिः। Va. Su. 11/16

68 व्यायामाच्च विहाराच्च विक्षिप्तत्वाच्च चेतसः । न क्लेदमुपगच्छन्ति दिवा तेनास्य धातवः।। Ch. Chi. 15/239

69 स्वेदे रोमच्युतिः स्तब्धरोमता स्फुटनं त्वचः ।। Va. Su. 11/22

70 विट्स्वेदमूत्राम्बुवहानि वायुः स्त्रोतांसि संरूध्य यदोर्ध्वमेति । Ch. Chi. 20/16,page 556

71 स्वेतेजोऽम्बुगुणस्निग्धोद्रिक्तं मेदोऽभिजायते । Ch. Chi. 15/30

72 रोगाः सर्वेऽपि मंदेऽग्नौ। As. Ha. Ni. 12/1

73 मज्जवहानां स्त्रोतसामस्थीनि मूलं सन्धयश्च । Ch. Vi. 5/8

74 रक्तवाहीनि दुष्यन्ति भजतां चातपानलौ। मांसवाहीनि दुष्यन्ति भुक्त्वा च स्वपतां दिवा ।। मेदोवाहीनि दुष्यन्ति वारूण्याश्चातिसेवनात् ।। Ch. Vi. 5/14-16

75 निद्रायत्तं सुखं दुःखं पुष्टिः काश्र्ये बलाबलम् । A. Ha. Su. 7/53,page 141

76 किट्टमन्नस्य विण्मूत्रं रसस्य तु कफोद्मसृज । Ch. Vi. & Ch.Chi. 15/18

77 रसाद्रक्तं ततो मांसं मांसान्मेदस्ततोद्मस्थि च। अस्थो मज्जा ततः शुक्रं शुक्राद्वर्भः प्रसादजः Ch. Chi. 15/16

78 संतर्पयति यः स्निग्धैर्मधुरर्गुरूपिच्छिलैः । नवान्नैर्नवमद्यैश्चमांसैश्चनूपवारिजैः ।। च. सू. २३/३

79 वक्ष्यन्ते सौषधाश्चोर्ध्वमपतर्पणजा गदाः ।। विण्मूत्रसंग्रह : शूलं जघ्ङोरूत्रिकसंश्रयम् ।। च. सू. २३ /२६ -२७

80 पुरीषर्निग्रहज लक्षणभूतं पिण्डिकाया उद्वेष्टनम् । अशीतिवातविकारे ष्वेकः ।। च. सू. २०/११

81 उद्वेष्टनम् उद्वेष्टमानायामिव दुःखम् । अ. ह. सू. ४/३

82 मेषजैर्विनिवर्तन्ते विकाराः साध्यसंमताः ।। च. सू. १/ ६२

83 तत्रानुबन्ध्यानुबन्धकृतो विशेषः- स्वतन्त्रो व्यक्तलिङगे यथोक्तसमुत्थानप्रशमो भवत्यनुबन्ध्यः, तद्विपरीतलक्षणस्त्वनुबन्धः । च. वि. ६/११

84 तत्र भूमिपरीक्षा आतुरपरिज्ञानहेतोर्वा स्यादौषधपरिज्ञानहेतोर्वा । च. वि. ८/ ९३

85 अग्नि जरणशक्त्या परीक्षेत बलं व्यायामशक्त्या । वायुः सामो विबन्धाग्निसादतन्द्रान्त्रकूजनैः।। वेदनाशोथनिस्तोदैः क्रमशोऽङगनि पीडयेत् । वा. सू. १३

86 दुर्गन्धं हरितं श्यामं पित्तमम्लं स्थिरं गुरू ।। आविलस्तन्तुलः स्त्यानः कण्ठदेशेऽवतिष्ठते ।। शरीरगुणाः पुनर्द्विविधाः संग्रहेण - मलभूताः, प्रसादभूताच्श्र । तत्र मलभूतास्ते ये शरीरस्याबाधकराः स्युः । च. शा. ६/१७

87 विट्स्वेदमूत्राम्बुवहानि वायुः स्त्रोतांसि संरूद्ध यदोर्ध्वमेति । च. चि. २० /१६

88 रोगोत्पादकहेतुनिदानम् । यथा दुष्टेन दोषेण यथा चानुविसर्पता । निर्वृत्तिरामयस्यासौ संप्राप्तिर्जातिरागतिः ।। वा. नि. अ. १

संख्याविकल्पप्राधान्यबलकालविशेषतः । सा भिद्यते यथाऽत्रैव वक्ष्यतेऽष्टौ ज्वरा इति ।। वा. नि. अ. १

सर्वेषामेव रोगाणां निदानं कुपिता मलाः । तत्प्रकोपस्य तु प्रोक्तं विविधाहितसेवनम् ।। वा. नि. अ. १

89 तत्प्रकोपस्य तु प्रोक्तं विविधाहितसेवनम् । मा. नि. १/१४

90 तद्यथा ज्वरसन्तापाद्रक्तपित्तमुदीर्यते । क्षयो रोगस्य हेतुत्वे शोषस्याप्युपजायते ।। मा. नि. १/ १५- १८

91 दोषे विबद्धे नष्टेऽग्नौ सर्वसंपूर्णलक्षणः। सन्निपातज्वरोऽ साध्यः, कृच्छ्रसाध्यस्ततोऽ न्यथा ।। मा. नि. २/ २४

92 हृत्पीडा-काश्र्य-दौर्बल्यं वैरस्यं परिकार्तिका ।मृद्धिः सर्वरसानां च मनसः सदनं तथा ।। मा. नि. ४/ ८

93 आमं विदग्धं विष्टब्धं कफ-पित्तानिलैस्त्रिभिः। अजीर्णे केचिदिच्छन्ति चतुर्थे रसशेषतः।। मा. नि. ६/ ५

94 तत्रामे गुरूतोत्क्लेदः शोथो गण्डाक्षिकूटगः। उद्गारश्च यथाभुक्तमविदग्धः प्रवर्तते।। मा. नि. ६/१०

95 कफादामाशये जाता वृद्धाः सर्पन्ति सर्वतः । मूर्च्छा-च्छर्दि-ज्वरानाह-काश्र्य-क्षवथु- पीनसान्। मा. नि. ७/७- १०

96 व्यायाममम्लं लवणानि मद्यं मृदं दिवास्वप्नमतीव तीक्ष्णम्। निषेवमाणस्य प्रदूष्य रक्तं दोषास्त्वचं पाण्डुरतां नयन्ति।। मा. नि. ८/२

97 वात-मूत्र-पुरीषाणां सग्डो ध्मानं क्लमो रूजा। जठरे वातजाश्चान्ये रोगाः स्युर्वातनिग्रहात् ।। मा. नि. २७/२

98 वात-मूत्र-पुरीषासृक्कफ- मेदोवहानि वै। स्त्रोतांस्युदावर्तयति पुरीषं चातिवर्तयेत् । मा. नि. २७/ १४

99 उत्क्लेदः ष्ठीवनं तोदः शूलं हृल्लासकस्तमः। अरूचिः श्यावनेत्रत्वं शौथश्च क्रिमिजे भवेत्।। मा. नि. २९/६

11. Annxure:

Srotas Causes and lakashana distribution

Conventional	Ayurveda	
Pranvaha:- Shape of chest type of respiration position of sternum auscultation percussion DNS/ Polyp/ Dryness	Skin color (varna) auscultation	Ayasenshawasa, shawashkastata
Udakavaha:- Soft palate, Hard palate	Oral cavity salivation , Uvulva, Trushna, Lips	Trishna
Annavaha	Jivha, Agni, Abhyvaran	Annanabhilasha, Chhardi
Rasavaha Heart rate, Blood pressure	Nadi, Twak parishna	Angamarda, Pandu, Asharda, Jawara, Hrullas
Raktavaha Liver, Spleen, Fistulas	Netra, Pitika, Discolouration	Pigmentation, Discoloration
Medovaha Muscle tenderness, Wasting, Muscle tone	Tenderness, Fatigue, Vrukka (Renal angle ten sernons)	
Asthi vaha	Visuvalist d/ Non Visualised, Nails, Hair fall, Areqing of hair	Asthishoola
Majjavaha	Sandhiparishna	Murcha, Bhrama
Purishavaha	Malapravartana, Formed/ Kruchha	Kruchatava
Mutra	Colour/ Pain, Mutrapravartana	Alptava, Ati, Bahu
Sweda	Seasonal exertion sweda pravartana	Asweda, Atisweda
Agni	Abhyvarana	
Raja pravartana	Regular / irregular, No of days.	

Acronyms and Abbreviation:-

DM – Diabetes mellitus
HT- Hypertension
Sr. Cr. – Serum Creatinine
GFR – Glomerular filtration rate
Hb – Hemoglobin
KDOQA – Kidney Disease Outcome Quality Initiative
NKF – National Kidney Foundation
ESRD – End Stage Renal Disease.

Tilak Maharashtra Vidyapeeth, Pune

The Late Vd. P. G. Nanal Dept. of Ayurveda

Patient consent form

Title of the study: The Aetiopathological study of Chronic Renal Failure with Ayurveda Perspective

Name of the Participant: _____

Documentation of the informed consent

I, … … … … … … … … …., have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered.

(1) I have read and understood this consent form and the information provided to me.

(2) I have had the consent document explained to me.

(3) I have been explained about the nature of the study.

(4) My rights and responsibilities have been explained to me by the researcher.

(5) I have informed the researcher of all the treatments I am taking or have taken in the past… …. months including any desi (alternative) treatments.

(6) I hereby give permission to the investigators to release the information obtained from me as result of participation in this study ethics committee. I understand that they may inspect my original records.

(7) My identity will be kept confidential if my data are publicly presented.

(8) I have had my questions answered to my satisfaction.

(9) I have decided to be in the research study.

I am aware, that if I have any questions during this study, I should contact at one of the addresses listed above. By signing this consent from, I attest that the information given in this document

Name and signature / thumb impression of the participant (or legal representative if participant incompetent):

Name :

Address :

Contact no : Email Id :

Signature : Thumb impression

रूग्ण संमतीपत्रक

अभ्यासक्रमाचे शीर्षकः- The Aetiopathological study of Chronic Renal Failure with Ayurveda Perspective

रूग्णाचे नावः-

रूग्ण संमतीपत्रक नियमः-

मी रूग्ण पत्रकातील सर्व माहिती वाचून किंवा मला वाचून दाखविले आहे. मी विचारल्या जाणा-या प्रश्नांची उत्तरे देण्यास संमती दर्शवित आहे.

१. मी रूग्ण पत्रकात नमूद केलेली सर्व माहिती वाचली आहे.

२. मला रूग्ण संमती पत्रक समजवून सांगण्यात आले आहे.

३. मला माझे हक्क अभ्यासकाने माहिती करून दिले आहेत.

४. मला अभ्यासाबद्दल माहिती देण्यात आली आहे.

५. मी सध्या घेत असलेल्या सर्व औषधोपचाराची माहिती अभ्यासकाला देत आहे.

६. माझी ओळख (Identity) माहिती publish करताना गोपनीय ठेवण्यात यावी.

७. मी माझी उत्तरे समाधानकारकरीत्या देत आहे.

८. मी स्वतःअभ्यासाचा एकभाग होण्यास तयारी दर्शवित आहे.

प्रौढ व्यक्तीसाठी :-

रूग्णाचे नाव सही / अंगठा :-

नावंः-

सहीः- दिनांकः-

Tilak Maharashtra Vidyapeeth, Pune

The Late Vaidya P. G. Nanal Department Of Ayurveda

Case Record Form - For Ph. D. (Ayu)

Title – The Aetiopathological Study of Chronic Renal Failure with Ayurvedic Perspective

Name of the scholar – Dr. Swaroopa Bhujbal

Name of the Guide – Dr. S.P.Sardeshmukh

Case Record Form

Sr. No.:- **Date of admission:-**

Case No:- **Tel. No.** **Religion :- H/M/O**

Name:- **Date:-** **Occupation:-**

Address:- **Age:-** **Gender:- M/F**

Chief complaints **प्रमुख तक्रारी**	Duration (vyadhi udagama)	Intensity
History of Present illness Month/year-		
History of Past illness		

Investigations

Date	Place/ Hospital	Hb/CBC/ESR	Blood urea	Sr. creatinine	Urine

History

Dialysis-Cycle started **Frequency**

	Pre – Dialysis	**During Dialysis**	**Post Dialysis**
Symptom			
Duration			
Intensity			
Upashaya- Anupashaya			

Treatment History

	Years	**Months**	**Days**
Antihypertensive			
Analgesic			
Renal Antibiotic			
D. M,			

Ayurvedic Drugs			
Oral Contraceptives			
OTC Drugs			
Panchakarma vyapad			

Addiction History

	Per day	**Per week**	**Per month**
Tobacco			
Alcohol			
Drug			
Other			

Family History:

Maternal	**Paternal**
Essential Hypertension	
DM	
Inherited kidney disease	

Polycystic kidney disease	

Dietary History

	Pravar	**Madham**	**Avar**
Madhur			
Amla			
Lavan			
Katu			
Tikta			
Kashay			
Viruudha			
Ati / Ana/Adhya/Vishamashan			
Paryusheet			
Abhishyandi			
Vishtambhi			
Mansa sevan			

Navadhnya/Bakery /freeze water			

Vihar evaluation:

Vyayam parikshana

Daily (Time):- Present / Absent

Occasional:- Present / Absent

Yoga:- Present / Absent

Aerobics / walking :- Present / Absent

Nidra - 6hrs/more than 6hrs

Sound - frequent /occasionally

Disturbed-Frequent/occasionally

Wake Up Time -4am/6am/duties schedule

Divaswap- Daily(15min/30min/2hr)

Jagaran-upto 11pm ( )/ more than 12am onwards ( )

Aatap : Present / Absent

Rajasevan: Present / Absent

Upawas: week /month /year

Vyavay:Present /absent

Bhashana: Daily ( )/Occasionally

Asyasukham:Present/absent

Vegavarodha:

Name of vega and lakhana	Kayaic/Dharniya	Vachic /Adharniya

Manoguna Parikshana	Pre – Diseaese(Pravara)/Avara	With disease

Manasa Hetu:

Name of vega	Mansic (Present / Absent)
Kama	
Krodha	
Bhaya	
Shoka	
Others	

Quality of life: Joyful/ Enthusiastic/ Lethargy/ Burdensome

Viruddha Parishana:

Viruddha Name	Present	Absent	Specific
Desha			
Kala			
Agni			
Matra			
Satmya			

doshadi			
Sansakara			
Vriya			
Kostha			
Avatsha			
Krama			
Parihara			
Upchra			
Paka			
Sanyog			
Hrud			
Vidhi			
Sampat			

Clinical Examination: (Darshana ,Sparshana and Prashne)-

Darshan-

Pranavahasrotas-

Shape of chest: Pigeon/ Barrel/ Normal

Type of respiration: Thoracoabdominal/ Abdominothoracic/ Other

Position of stenum/ribs: Normal/ Abnormal-

Skin colour: Normal/ Abnormal

Any other finding:

Ascultation: Normal/ Abnormal-

Percussion: Typanic/ Dull/ Other

Nose examination:DNS/ Polyp/ Dryness/ Any other

Shira parishana:

Udakavahasrotas –

Talu parishana:

Soft Palate: Normal/ Congestion/ Ulceration/ Dryness

Hard Palate: Normal/ Congestion

Oral Cavity: Salivation-Normal/ Less/ Excess/ Fibrosis

Uulva: Normal/ Abnormal/ Elongated

Trushna: Prakrut/ Aprakrut

Lips: Normal/ Dry/ Scaly/ Bleeds/ Any Other

Annavahasrotas:

Jivha: Sama/ Niram/ Other/ Pattern Of Samata

Agni: Sama/ Vishama/ Manda/ Tikhsna

Abhyavarnashakti: Prakrut( )/ Aprakrut( )

Rasavahastrotas-

Heart rate:

Heart Sound:

Nadi/ pluse- Bala- Gati- Dosha- Sankhya-

Blood pressure-

Stana Parishana:

Tawaka Parishana: Normal/ Dry/ Scaly/ Scratch mark/ pale

Scracthining/ itchining details: day/ night/ specific part/ general

Raktavahastrotas-

Eyes: Pallor/ Nonpallor

Liver: Palpable ( ) / Non palpable/ Tenderness/ Non tender

Spleen: Palpable ( )/ Non palpable/ Tenderness/ Non tender

Sirajala Abdominal: Present/ Absent

Pitika: Present/Absent

Fistulas; Present/ Absent

Discoloration: Grey/ Black/ Tanned/ Pale

Pigmentation: Present ( )/ Absent

Icterus: Present/Absent

Mansvahasrotas-

Tenderness: Present/ Absent

Fatigue: Present/ Absent

Wasting: Present/ Absent

Muscle Examination: Thorax/ Abdomin/ Upeerextrimities/ Lowerextrimites

Muscle Tone: Present/ Absent

Refexes:

Mala: Nose- Present/ Absent Ear: Absent/ Present Eyes: Absent/ Present

Medovahastrotas-

Vrukka parishana:

Gala : Stula/ Krusha Udra : Stula/ Krusha

Sphika : Stula/ Krusha Sthna: Stula/ Krusha

Asthivahasrotas-

Asthisandhi Parishana : Visuvalised/ Nonvisuvalised

Nails: Normal/ Pale/ Clubbing/ Kolinychia

Kesashmashru- Growth: Present/ Absent

Hairfall:P/A

Greying :P/A

Majjavahastrotas-

Sandhiparisha- Kriyahani/ Kriyaksahatva/ Sparshasahatva

Refelexes:

Shukravahasrotas-

Testicular Examination: If Necessary:

Menses: Normal/ Regular/ Dysmenorrhoea / metrorrhagia/ Menopause

Purishavahasrotas-

Guda parishana If necessary:

Malapravartana: Normal/abnormal

Mutravahastrotas-

Basti: Tenderness/ Non tender

Mutrapravartana: Normal/ Abnormal/ Incontinanace/ Dribbling

Mutraprashina: Gandha:

Varna: Matra Frquency

Swedovahastrotas-

Swedapravartana: Present/ Absent

Romkup: Normal/ Abnormal/ Dryness

Sweda: Gandha: Vistra/ Normal

Manovahastrotas-

Panchadynendriya- Ear / Twak / Netra / Jivaha / Nasa

Panchakarmendriy - Hasta Pada / Payu / Upastha / Vani

Agni Parikshana:

Abhyavaranashakti-:

Jaranshakati-

Mala Pravrutti

Daily	**Time**	**Mala Formed**	**Semisolid**	**Liquid**	**Kruchha**	**other**

Mutra Pravrutti

During Day (Frequency)	**During Night (Frequency)**	**Colour**	**Pain / Burning**	**Fluid Input Urine Output**

Sweda Pravrutti

All Season	**Specific Season**	**Exertion**	**Odour**	**Odourless**	**Stain**	**Other**

Rajpravrutti

Monthly	Regular	Irregular	No of days	No Of Pads	Pain

Dosha

Dushya

Mala

Hetu

Ahara	Vihara	Mansic	Other

Samprapti

Sign of Researcher **Sign of guide**

Dr.Mrs. Swarupa Bhujbal **Dr. S. P. Sardeshmukh**

Annexure I

Diet Detail

Limbu	Per Day	Per Week	Per Month
Chincha			
Tomato			
Kokama			
Sauces Sour			
Lavan Rasa			
Sendhav			
Baking Powder			
Soda			
Yavakshar			
Katurasa			
Chilli			
Spicy Food			
Sheet Sevan			
Freeze Water			
Icecream			
Viruddha Ahar			

Fermentaed Product			
Vishatambha			
Abhishyandi			
Virudha Ahar			
Mamsa			
Navadhnya			