A CLINICAL STUDY ON

KALLADAIPPU

(UROLITHIASIS)

WITH THE EVALUATION OF SIDDHA DRUG

KARPOORA SILASATHU PARPAM

The dissertation submitted by

Dr. S.SARATHKUMAR (Reg. No. 321411109)

Under the Guidance of

Prof. Dr. K. KANAKAVALLI, M.D.(S)

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements
For the award of the degree of

SIDDHA MARUTHUVA PERARIGNAR

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I – MARUTHUVAM

POST GRADUATE DEPARTMENT OF MARUTHUVAM

THE GOVERNMENT SIDDHA MEDICAL COLLEGE

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CERTIFICATE

This is to certify that the dissertation entitled "**A CLINICAL STUDY ON KALLADAIPPU"** is a bonafide work done by **Dr.S.SARATHKUMAR.**, Government Siddha Medical College, Chennai – 600 106 in partial fulfillment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2014 – 2017.

Name & Signature of the Guide

Name & Signature of the Head of Department

Name & Signature of the Dean/ Principal

ACKNOWLEDGEMENT

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My humble thanks to the God for giving me the opportunity to do this dissertation work and I also express thanks to siddhars.

I express my cordial and sincere thanks to respected **Prof. Dr. P. Parthibhan M.D(S),** Joint Director ,Indian Medicine and Homoeopathy Chennai – 106, for his valuable guidance, concern suggestion, inspiration and encouragement throughout the course of this study.

I am grateful to thank **Dr. K. Kanakavalli, M.D(S),** Principal, Govt. Siddha Medical College, Chennai – 106.

I feel pleasure to extend my heartful and sincere thanks to respected Guide, **Prof. Dr. K. Kanakavalli, M.D(S),** Govt. Siddha Medical College, Chennai – 106, for her very valuable inputs, supervision and helped as a guide for preclinical and clinical study and submitting this dissertation book right from stage of its formation.

I am particularly indebted to **Prof. Dr. N. Anbu., M.D. (S),** Head of the Department, Post Graduate (Maruthuvam), Government Siddha Medical College, Chennai -106, for his necessary advice at every step of my dissertation work, valuable suggestions, and provided very good command during this study.

I wish to express my thanks to **Dr. U. Chitra, M.D(S),** Asst. Lecturer for her support and encouragement during the course of this study.

I also extend my thanks to **Dr. R. Menaka, M.D(S)**, Asst. Lecturer for her valuable opinions in this dissertation work.

I would like thanks to **Dr. S.M. Chitra, M.D(S)**, Asst. Lecturer for her valuable opinions in this dissertation work.

I am very glad to thank **Dr. R. Sasireka, M.D(S),** Asst. Lecturer for her kind opinions in this dissertation work.

I express my special thanks to **Dr.Vidhya M.B.B.S., D.M.R.D**., Arignar Anna Govt.Hospital of Indian Medicine, Chennai-106 who guided me and cleared my doubts regarding the Ultrasonogram and follicular studies.

I also convey my thanks to **Dr.D, Sivaraman, M.Pharm, PhD, Sathyabama Univercity, Sirucheery, Chennai-97,** for giving **IAEC** approval and helping me tofinish toxicity study and activity study.

I wish to thank **Dr.P.Sathyarajeswaran M.D(s), Clinical Research Scientist** and I also convey my sincere thanks to **R.Shakila,** Bio chemissist CCRS, Chennai, for his encouragement and support during the period of study.

I like to thank **Mr.S.Selvaraj, M.Sc, M.Phil,** Department of Biochemistry, Government Siddha Medical College, Arumbakkam - 106 for my biochemical analysis.

I extend my thanks to **Dr. Manivasagam, B.S.M.S., M.Sc (Epidemiology),** Chennai, for his guidance in Bio – Statistical analysis of my results.

I sincerely thankful to **Dr.R.Ilavarasan, Scientist-3,** asst.Director in-charge, captain srinivasa murti research institute of ayurveda and siddha drug development, aringnar anna hospital campus, arumbakkam, Chennai 106.

I would like to thank all the teaching staffs of PG department, Govt. Siddha Medical College, Chennai – 106 for their timely suggestion and encouragement.

I would like to thank **Mr.L.Dhandabani, M.Com. M.Lis,** Librarian, Dr.Ambedhkar central library, Chennai-106 for his help in literary collection.

I sincerely thank to **all my patients** who have given their consent to record their materials and willingly accepting themselves for this study.

My special thanks goes to my father **Mr.M.Singaravelu**, my mother **Mrs.Banumathi**, my brother **Mr.S.Mohandass** my wife **Mrs.S.Krishnaveni** and my friends for their encouragement and support in completing the dissertation.

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CONTENTS

INTRODUCTION

Siddha system of medicine is the most primitive medical system. This system was formulated and established about more than 5000 years back by the eminent powers called Siddhars and hence the name Siddha Medicine. The medicines were prepared by the various research work done by the Siddhars on herbs, minerals and animals. The father of Siddha Medicine is the primordial Guru, **Agasthiar**. There are also 18 prime Siddhars who are the followers of the primordial Guru, contributed their valuable knowledge and experiences in this field.

Siddha medical system doesn't consider treatment and prevention separately. The main aim of this system is prevention of disease, as it is well said that "Prevention is better than cure"

According to theory of Siddhars, human body is composed of Panchaboothas. The universe is composed of the same. Human body is microsmic and the Universe is macrosmic of the same.

As per siddha aspect, the physiological function of the human body is maintained by three humors called as vatham, pitham and kabam. The normal proportion of naadi is 1:1/2:1/4 respectively, any change in this proportion leads to many diseases.

Food plays vital role in the rhythmic run of three uyirthathus.

Our body doesn't requires medicine, if the food is taken on complete digestion. One of the disease occurred by irregular diet is Kalladaippu.

Siddhars classified 4,448 types of disease. Within that kalladaippu is one of the disease commonly affecting men than women.[1]

Even though many siddhars explain about diseases, **Yug**i in his **"YUGI VAIDHYA CHINTHAMANI 800"** elaborately said the aetiology, pathology, classification, clinical features, and prognosis of kalladaippu. The diseases of urinary system are divided into two types, They are,

"நீரினை அருக்கல் நோய்
நீரினை பெருக்கல் நோய்"

The kalladaippu comes under the classification of "நீரினை அருக்கல் நோய்" which produces low urine output and forms urinary calculi , due to various aetiological factors.

In Greek "ouron" means 'urine', "oros" means 'flow', "Lithos" means 'Stone', from which the term Urolithiosis came.

Urolithiasis or Nephrolithiasis is formation of urinary calculi at any level of the Urinary tract. It is characterized by acute loin pain radiating to groin, and it consists of aggregates of crystals containing small amount of proteins and glycoprotein. Though various kinds of stone have been identified, calcium stones are the most common in human as well as rats[2]

Urinary stone disease is a common disorder estimated to occur in approximately 12% of the world population, with a recurrence rate of 70-81% in males and 47- 60% in females 3.

The peak incidence is observed in 2nd and 3rd decades of life.

According to Siddha system of medicine Kalladaippu occurs due to increase of Pitha.

Now a days, the management of urolithiasis with open renal surgery is unusual and rarely used , since the introduction of extracorporeal shock wave lithotripsy ,which is a standard procedure to remove kidney stones. However it may leave persistent stone fragments and cause acute renal injury, a decrease in renal function and an increase in stone recurrence[4-6]. The procedure is not widely available and very costly to the people in developing countries.

In addition, the standard drugs used to prevent such lithiasis are not effective consistently in all patients, and many of them have adverse effects that compromise their long term use. Hence, the search for anti-lithiatic drugs from natural sources has gained more interest compared to earlier as shown in a recent study[7].)

On this basis, after accurate diagnosis Kalladaippu, Karpoora Silasathu Parpam is selected for clinical study to prove the efficacy of drugs in this disease.

I preferred to select kalladaippu (urolithiasis) as dissertation topic because the prevalence is increasing day by day and many people prefer herbal medicines to remove stones rather than undergoing a painful surgery. Even the patients who were recommended for surgery have also been treated well using herbal medicines with satisfactory results.

So, I have chosen **Kalladaippu (Urolithiasis)** as dissertation topic to find out a complete cure without surgery. For this, I have selected Karpoora Silasathu Parpam to study its effect on Kalladaippu both pharmacologically and clinically.

This study is to look into various factors in the evolution of Kalladaippu and protocol of management with Karpoora Silasathu Parpam.

AIM AND OBJECTIVES

AIM:

The aim of my dissertation work is to evaluate the efficacy of the siddha drug **Karpoora Silasathu Parpam** both clinically and experimentally in the treatment of **kalladaippu**.

OBJECTIVES:

- To collect the authorial measures and literature reviews of Kalladaippu noi in ancient siddha and modern literatures.
- To have an idea of the incidence of the disease with regard to age, sex, occupation, socio economic status, food, climatic conditions and precipitating factors etc.
- To expose the efficacy of Siddhar's diagnostic principles.
- To evaluate
    - ✓ Toxicological screening
        - Acute
        - Sub - acute
    - ✓ Pharmacological screening
        - Anti-Lithiatic activity
- To utilize the possible methods to confirm the diagnosis and prognosis.
- To have clinical trial on patients with Kalladaippu noi with selected siddha medicine.
    - ✓ **Karpoora Silasathu Parpam**
- To find out the statistical analysis of clinical study

REVIEW OF LITERATURE

SIDDHA ASPECT

Theran karisal says,

The diseases of the urinary system are divided into two types. They are

"நீரினை அருக்கல் நோய்

நீரினை பெருக்கல் நோய்"[8]

The disease kalladaippu comes under the classification of "Neerinai Arukkal Noi". In siddha system of medicine, the disease kalladaippu is mentioned by Yugi Munivar in Yugi Vaidhya Chinthamani 800.

"நீரிரு வினைக் குணத்தை நீயறி விரித்துச் சொல்வாம்

நீரினை பெருக்க லொன்றே நீரினை யருக்க லொன்று

நீரிழிவுடனே கொல்லும் நீர்க்கட்டு வினைகளொன்று"[9]

KALLADAIPPU NOI

VERUPEYAR (SYNONYMS)

Achmari

IYAL (DEFINITION)

Sudden obstruction in the flow of urine, pain at the tip of the penis in males and clitoris in females, burning micturition, loin to groin pain, passing of small sand like stones along with urine are the cardinal features of this disease. [10]

Dehydration occurs due to overheat of the body. It leads to solid or crystalline aggregation from the dietary minerals in the kidney. The formed stone cannot expelled by kidnys leads to this diseased condition.[11]

Large concretions of stone in the bladder or kidney are known as calculus or gravel. It is attended with difficulty in passing urine.[12] Sudden obstruction to the flow of urine, pain at the tip of the penis in males and clitoris in females, burning micturition, pain in loin to groin region.[13]

விலகு சிலநேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுமொது கினால் நோகாது
வழுகிய மந்த்தால் வாயுவந்தே புகில்
கழுவி முதிர்ந்திடும் கல்லடைப்பாகும்.[14]

தானான மூத்திரப்பை தன்னிலப்பா
தனியான கல்லிருந்தால் சொல்லக் கேளே
ஊனான மூத்திரந்தா னடிக்கடியே தானும்
உள்ளபடி இறங்குமடா சொன்னேன் பாரு
தேனான கல்லப்பா தாரை மேலே
தொப்பெனவே விழுவதால் தாரை யப்பா
மானாக விழுவதனாலே நின்று போகும்
மக்களுக்கு இதுதீர சலாகை போடே[15.]

"தானென்ற மூத்திரத்தால் நறநறவென்று
தங்கியதோர் பொடியேனும் மணல் தானப்பா
வானென்ற சிறியதொருகல்லா வதப்பா
வளமாக வந்து விழும் நோய்க்குத்தானே
ஏனென்று அச்மரீரோக மென்ற பேராம்
தாக கல்லுகள்தான் விழுகும்போது
கோனென்று குண்டிக்காய் மூத்திரக் குழலப்பா
குணமான மூத்திரப்பை நீர்த்தாரைக்கேளே

கேளடா முங்குறியில் எரிச்சல் கண்டு
கொடியாக வேதனைகள் காட்டுமப்பா
வாளடா சிறியதொரு கற்கள் தானே
வளமான மூத்திரப்பை குழல் வழிப் படியாய்த்
தேளடா வரும்போது திரே கந் தன்னில்

தெரிப்பது போல் யிரு வேதனை செய்யும் பாரு
நாளடா கற்கள் தானிறங்கி விட்டால்
நலமான வேதனைகள் தீரும்பாரே" [15]

If a stone present in the bladder, there will be frequency of Micturition.when the stone descends to urethra there will be obstruction of urine flow. It is cured through catheterization.

NOI VARUM VAZHI (AETIOLOGY)

"தெளிந்ததோர் கல்லடைப்புற் பத்தி கேளாய்
சிறிதுநாட் டுடங்கியே மேகந் தன்னால்
தளிந்ததோர் சலப்பையிலு திரந் தோய்ந்து
சந்து சந்தாகவே பருத்துக் கொள்ளும்
வளிந்ததோர் வாதபித்தங் கோபித் தக்கால்
வந்து பெருங்கல்லாய் நீர்வழி யடைத்து
"நளிந்ததோர் நாலுவிதக் கல்லடைப்பு
நண்பான வரலாறு நாட்டக் கேளே"

கலங்கினதோர் தண்ணீர்தான் குடித்த பேர்க்கும்
கல்லெலும்பு மயிர் மண்தான் கலந்தன் னத்தில்
அலங்கின தோரன்னங்க ளருந்த லாலும்
அழுகலோடு மூத்தபண்ட மருந்த லாலும்
மலங்கினதோர் மாப்பண்ட மருந்தலாலும்
மந்தத்தில் வாய்வானப தார்த்தந் தன்னை
துலங்கின தோருசிதன்னிற் சுவைத்தலாலும்
சுருக்காய் கல்லடைப்புவந் துதோன்றுந் தேனே."[16]

The urine constituents will easily deposit on the urinary tract and form the stone. At that time by vitiation of vatham and pitham these small stone becomes larger in size and block the urinary passage. The semen will stagnate for a long time in the urinary tract, so it will obstruct the urine flow Urinary stone are also formed due to the drinking of contaminated hard water, taking of food mixed with sand and small stones consuming of contaminated food articles, food containing more carbohydrates, unhealthy food habits.

A urinary disease occasionally developed in the urinary bladder which is known as vesical calculus. It is said to be due to the deranged vayu encircling or prevailing in the region of the abdomen arising from any of the following causes.

1) Suppression of seminal discharge during sexual intercourse.
2) Retention of semen in the spermatic region in involuntary discharge during nocturnal emissions due to excessive heat in the body.

The calculus are stone which is formed in the bladder may vary in size from that of the particles of sand or mustard upto things as large as green gram or Bengal gram and sometimes attains the size of a hen's egg even and block the passage of urine. It is accompanied by pain and difficulty in passing urine. [17]

"நீரினைத் தடுத்தல் செய்யின்
நீர்க்கட்டுத் துவாரம் புண்ணாம்
பாறிடு சந்து சந்தில்
பண்பற நோவதாகும்
நேரிலங் கயரும் காமியம்
நிச்சயம் நோதல் செய்யும்
பாரினிலபான வாயு
பண்புறச் சேருமன்றே"[18]

POTHU KURIGUNANGAL (Clinical features)

- Gradual or sudden obstruction of the urine flow.
- Unbearable pain in the penis
- Excruciating pain and swelling is experienced at the tip of penis if the calculus attempts to expel.
- Colicky pain radiating from loin to groin lower abdomen and urethra if the calculus is irregular with sharp projection.
- Burning and scanty micturition and Haematuria.[19]

CLASSIFICATION

Classification according to yugimamunivar,

"தோன்றினதார் நாலினிட நாமங் கேளாய்
சுறுக்கான வாதத்தின் கல்லடைப்பு
பூன்றியதோர் பித்தத்தின் கல்லடைப்பு
புரண்டதோர் சேத்துமத்தின் கல்லடைப்பு
தீன்றியதோர் தொந்தமாங் கல்லடைப்பு
தேகத்திற் பற்றியேசி றிதுகாலம்
தான்றியே சலப்பையில் வந்தி ழிந்து
சருவியே லிங்கத்திற்ற ரிக்குந் தானே" [20]

According to Yugi vaidhya chinthamani, kalladaippu is classified into four types.

1) Vadha kalladaippu
2) Piththa kalladaippu
3) Silethuma kalladaippu
4) Thondha kalladaippu

1) Vadha kalladaippu:

"தரித்து நாபிக்குங்கீழ் சுருக்காய் குற்றில்
சலமலந்தான் விழாமற் றம்ப மாகி
வரித்துமே லிங்கத்தில் வலியுமாகி
மருவியதோர் பொத்தியெலாஞ் சுரந்து கட்டி

திரித்தியே கிடைக்கொடாப் பிரட்டலாகித்
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்தோர் சதைபோல உவர்ப்பு மாகும்
ஓங்கியதோர் வாதக் கல்லடைப்பு தானே."[20]

Acute pricking pain in the lower abdomen, scanty Micturition, obstruction to the flow of urine, pain in the penis, abdominal discomfort, and albuminuria will be present with mucous discharge and black coloured stone will be expelled.

2)Piththa kalladaippu:

"அடைப்பாகிச் சலந்தானு மருவலாகி
அயங்காச்சி சொருகினாற் போலே காணும்
புடைப்பாகிப் பொற்றியெங் கும்பு ழுக்கமாகிப்
பூட்டுபோல் பிகுவாகிப் பிரட்டலாகும்
மடைப்பமாகி உதிரநிற மாய்க்கல் லாகி
வந்திழிந்து லிங்கத்தில் மாட்டிக் கொள்ளும்
குடைப்பாகிக் குற்றலாய்க் கூச்சலாகிக்
குதட்டுமே பித்தக்கல் லடைப்பு தானே." [21]

Obstruction of urine flow, pricking pain and burning sensation in external meatus, expulsion of blood coloured stones.

3)Silethuma kalladaippu:

"தானான தொப்புளிலே வில்லு போலச்
சலியாமற் சுரந்துமே சற்றே குற்றும்
ஏனான காலோடு கைகள் சந்து
இடுப்புதான் குடைசலாயி சிவு காணும்
வேனான லிங்கத்தின் வெண்மை தன்னில்
விறுவிறென் றேகடுப்பாகி வியற்வை யாகும்
தேனான வெளுப்புக்கல் சிறுகல் லாகச்
சிக்கலாய் வந்திறங்குச் சேட்பந் தானே"[21]

Pricking pain in umbilicus, pain in the joints of hands and legs, expulsion of white coloured stone in urine, excessive sweating.

5) Thondha kalladaippu:

"வந்திறங்கும் நீர்தாரை யடியிற் றானும்
மாவருத்த முண்டாகி வலியுமாகி
நொந்திறங்கி நீர்தானு மருவிபாயும்
நொய்தான சிறுமணல் போல் நொறுங்கிக் கல்லான்
சந்திறங்கி நீர் வழியில் வந்து விழும்
தாக்கான சிறங்கைக்கல் தினமொன்றுக்கு
துந்திறங்கித் தினந்தினமு மிழந்து கொல்லும்
தொந்தமாங் கல்லடைப்புச் சூட்டிட் டாயே"[22]

Severe pain in urethra, Dysuria, Oliguria, handful of small sand like stones will expel with severe pain.

According to Dhanvanthri,

"திருந்திய வாதபித்தச் சிலேற்பனம் பிரகோபித்தால்
வகுந்தக மரித்தா நான்கு வகைப்படும் கல்லரிப்பான்
பிரிந்திடுஞ் சிலேற்பனாக மரிபித்தா சுமரி பின்னு
மிருந்திடு சுக்கிலாசு மரி நான்கு மெய்து மென்றே"[23]

Achmari is classified into four types,

1) Kallerippan
2)Silethuma achmari
3)Piththa achmari
4)Sukkilachmari

1) **Kallerippan gunam:**

"சுத்துநீர் நாலிந்தன்னிற் சுக்கிலந் தனிற் சிலேற்பம்
பித்துமீது லர்த்தல் கல்லாய்ப் பீசகிநீ ரடைத்துக் கொள்ளுங்

கொத்து நீரிற்றுவிழுங் கொப்புளநோ குடம்பு காயுஞ்
சித்தமா யருசி யுண்டாஞ் சேர்ந்தகல் லெரிப்பனாமே."[23]

Formations of stones in the urinary tract, oliguria, pricking pain around the umbilicus, fever, anorexia are the symptoms of this type.

2) **Silethuma achmari gunam:**

நீர் வருநாளந் தன்னில் நின்றுநீர் சிறுத்துக் கொண்டு
சோர்தரும் சிவப்பு வெண்மை சுக்கிலம் போலவீழும்
பேர்பெற நாலா மெட்டுப் பின்னமாய்க் கல்லுவீழும்
ஏர்பெறு சிலேற்பனத்தில் அச்சமரி என்னலாமே" [24]

Oliguria due to obstruction of stone in urethral orifice, stones can be expelled out into pieces.

3) **Piththa achmari gunam**:

"பெய்யும் நீர் நாளந் தன்னில் பித்தத்தா லெரிப் பெழுந்து
செய்யுவுஷ் ணத்தால் வெந்து சேங்கொட்டை போற்கல் லுண்டாம்
நய்யவே தனைகள் செய்யும் நவில் குணம் பித்தந்தன்னில்
எய்தசு மரியென்றே முன்னியம் பினரறிவின் மிக்கோர்." [24]

Burning micturition in urethral orifice, formations of stones, severe pain are the symptoms

4)**Sukkilachmari gunam:**

"சுக்கிலம் வருங்காலத்தில் தம்பித்தாற் சுக்கிலந்தான்
மிக்ககல் லாகிவெம்பி விதனமாய் நீர்விடாமற்
சிக்கிநீர் விழாமலங்கே மணல் வீழும் வெளுக்குந்தேகம்
மிக்குணஞ் சுக்கிலாசு மரியசாத் தியமென்றோதே."[25]

Stagnation of semen leads to the formation of stones, oliguria, and expulsion of small sand like stones. It is fatal.

SAATHIYAM, ASAATHIYAM (PROGNOSIS)

As per yugi,

Vatha kalladaippu, piththa kalladaippu, silethuma kalladaippu are curable. Thondha kalladaippu is fatal.

"சூட்டிய சாத்தியத்தைச் சொல்லக் கேளாய்
சுளுக்காகும் வாதத்தின் கல்லடைப்பு
பூட்டிட்ட பித்தத்தின் கல்லடைப்பு
புகழான சேட்டுமத்தின் கல்லடைப்பு
மூட்டிட்ட இதுமூன்று மசாதியமாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்லடைப்புத்
தொடு சுறவே கொல்லுமிது சூட்சந்தானே."[26]

MUKKUTRA VAERUPADUGAL (PATHOGENESIS):

Disease occurs due to the derangement in

- Uyir thathukkal
- Udal thathukkal
- Kalamarupadu (seasonal changes)
- Thinai (living lands) and
- Udal vanmai.

Mukkutra iyal:

The function of the three uyir thathus:

a) Vali – Kattru + Veli
b) Azhal – Thee
c) Iyyam – Neer + Mann

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1:1/2:1/4) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

VATHAM

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations it is classified into ten types. They are tabulated below.

S.No	Vatham	General Features	Changes in Kalladaippu
1	Piranan (Uyirkkaal)	Responsible for respiration and it is necessary for proper digestion.	Normal
2	Abanan (Keel nokkukkaal)	Responsible for all the downward forces such as voiding of urine, stools, semen, menstrual flow.	Affected (scanty Micturition)
3	Viyanan (Paravukaal)	Dwells in the skin and is concerned with the sense of touch, extension and flexion of the parts of the body and distribution of the nutrients to various parts of the body.	Normal
4	Uthanan (Melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc.,	Affected (Nausea, vomiting)
5	Samanan (Nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ.	Affected
6	Nagan	Helps in opening and closing of eyelids.	Normal
7	Koorman	Responsible for vision, lacrimation and yawning.	Normal
8	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing.	Normal
9	Thevathaththan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc.,	Normal
10	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3 days of death, forming a way through the skull[27]	Normal

PITHAM

It is the thermal life force of the body. It is sub divided into five types. They are

S.No	Pitham	General Features	Changes in Kalladaippu
1	Anarpitham	Peps up the appetite and aids in digestion.	Normal
2	Ranjagapitham	Responsible for the colour and contents of blood.	Normal
3	Saathagapitham	Controls the whole body and is held responsible for fulfilling a purpose.	Affected (Dysuria, Oliguria)
4	Pirasagapitham	Dwells in the skin and concerned with the shine, glow, texture and its complexion.	Normal
5	Alosagapitham	Responsible for the perception of vision.[28]	Normal

KABHAM

It is responsible for the stream line functions of the body and maintains body's defence mechanism intact. It is again classified into 5 types.

S.No	KABHAM	GENERAL FEATURES	CHANGES IN KALLADAIPPU
1	Avalambagam	Lies in the respiratory organs, exercises authority over other kabhas and control the heart and circulatory system.	Normal
2	Kilethagam	Found in stomach as it seat, moistens the food, softens and helps to be digested.	Normal
3	Pothagam	Responsible for the perception of taste	Normal
4	Tharpagam	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid.	Normal
5	Santhigam	Necessary for the lubrication and the free movements of joints.[29]	Normal

PARUVAKALAM

S.No	Perum pozhuthugal	Mukkutra marupaadugal
1	Kaar kaalam (Aavani & Purattasi) Mid August to Mid October	VATHAM - Vaetrunilai valarchi PITHAM – Thannilai valarchi
2	Koothir kaalam (Iypasi & Karthigai) Mid October to Mid December	VATHAM – Thannilai adaidhal PITHAM - Vaetrunilai valarchi
3	Munpani kaalam (Margazhi & Thai) Mid December to Mid February	PITHAM – Thannilai adaidhal
4	Pinpani kaalam (Masi & Panguni) Mid February to Mid June	KABHAM – Thannilai valarchi
5	Elavenir kaalam (Chithirai & Vaikaasi) Mid April to Mid June	KABHAM – Vaetrunilai valarchi
6	Mudhuvenir kaalam (Aani & Aadi) Mid June to Mid August	VATHAM – Thannilai valarchi KABHAM – Thannilai adaidhal[30]

THINAI (LAND)

Siddhars classified the lands into five types. They are

1. Kurunji – Mountain range
2. Mullai – Pastoral area of the forest
3. Marudham – The fertile river bed
4. Neidhal – The coastal region
5. Paalai – Arid desert

Kabha diseases will occur in Kurinji land. Pitha diseases occur in Mullai land. Vadha diseases occur in Neidhal land. Staying in Paalai land is not good to health. Marudham land is the fertile area where no disease occurs. So, Marudham land is the best one to stay.The winter season gives good health to the man, early summer and later rainy gives moderate health. Whereas early rainy and later summer are more prone to diseases, that's why siddhars called it as Aanaga kaalam. [31]

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINAIGAL

Mukkutram	**Paruvakaalam (Seasons)**			**Thinai**
	Thannilai valarchi (Accumulation)	**Vaetrunilai valarchi (Aggravation)**	**Thannilai adaidhal (Alleviation)**	
VATHAM	Mudhuvenil kaalam	Kaar kaalam	Koothir kaalam	Vatha disease is more prevalent in Neidhal land
PITHAM	Kaar kaalam	Koothir kaalam	Munpani kaalam	Pitha disease is more prevalent in Mullai land
KABHAM	Pinpani kaalam	Elavenil kaalam	Mudhuvenil kaalam[32]	Kabha disease is more prevalent in Kurunji land[33]

UDAL VANMAI (IMMUNITY)

Siddhars classify udal vanmai into three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

UDAL KATTUGAL

S.No	Udal kattugal	General Features	Changes in Kalladaippu
1	Saaram (Digestive essence)	Responsible for the growth and development. It keeps the individual in good temperament and it enriches the body.	Affected due to pain
2	Senneer (Blood)	Responsible for the color of the blood and for the intellect, nourishment, strength of the body.	Normal
3	Oon (Muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body.	Normal
4	Kozhuppu (Fat)	Lubricates the organs to facilitate frictionless functions.	Normal
5	Enbu (Bones)	Supports and protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body.	Normal
6	Moolai (Bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other system of body.	Normal
7	Sukkilam/Suronitham	Responsible for reproduction.[34]	Normal

PINIYARI MURAIMAI (DIAGNOSIS)

Four steps are followed in diagnosing the disease. They are

1. Poriyaal aridhal
2. Pulanal therdhal
3. Vinaadhal
4. Envagai thervugal

PORIYAAL ARIDHAL:

In this, the physician should carefully observe the changes that occur in the five sensory organs (porigal) of the patient.

PULANAL THERDHAL:

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

VINAADHAL:

The physician should interrogate about the patients name, age, occupation, socio- economic status, food habits, history of past illness, history of present illness, family history, marital status, menstrual history and frequency of pain.

ENVAGAI THERVUGAL:

"நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்."[35]

Nowadays advanced diagnostic tools have been developed by modern bio medical scientists. But siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

Eight fold system of clinical assessments:

Siddhars have given eight diagnostic methodological tools. They are

1. Naadi
2. Sparisam
3. Naa
4. Niram
5. Mozhi
6. Vizhi
7. Malam
8. Moothiram[36]

GENERAL FINDINGS:

NAADI:

Naadi is responsible for the existence of life, can be felt one inch below the wrist on the radial side by means of palpation with tips of index, middle and ring finger, corresponding to vatham, pitham, kabham.

Three humours Vatham, Pitham, and Kabham are in the ratio 1:1/2:1/4 normally. Derangement in these ratio leads to various disease conditions.

Naadi nadai in kalladaippu

When the vatham add with mandham it produces the kalladaippu disease.

“ஏவலாய் குழலாய் பித்தஞ் செய்குணம் விளம்பக் கேளாய்
கோலவேல் விழி சிவந்து குளிர்ந்திடிரிருக்கு மல்லால்
சீலவே நீர்கடுத்து நொந்து சுறுக்கெனச் சிவந்து வீழும்
ஞலமே கிறுகிறென்று நாவுலர்ந் திருக்குங் காணே.”[37]

SPARISAM:

By sparisam, the temperature of skin (thatpam- cold or veppam – heat), smoothness, roughness, sweating, dryness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In Kalladaippu, patient feels tenderness over the lower abdomen, renal angle and lumbar region. Also patient's temperature is increased in lower abdomen, sweating all over the body at the time of colic.

NAA:

Signs and symptoms in the tongue are noted here. Colour, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

- In kalladaippu, the naa is not affected.

NIRAM:

The colour of the skin is noted here.

- In kalladaippu, the Niram may be affected in sukkila achmari.

MOZHI:

Character of the speech is noted, mainly uraththa oli (high pitched), thazhndha oli (low pitched), or resembles the sound of any instrument.

- In kalladaippu, the mozhi will be affected to the patients who have severe pain leading to the thazhndha oli.

VIZHI:

Character of the eye is noted. Colour, warm, burning sensation, irritation, visual perception are generally noted.

- In kalladaippu, the vizhi may be affected. Redness due to renal colic pain.

MALAM:

The stools are examined for quantity, hardening (malakattu), loose motion (bedhi), colour and smell.

- In kalladaippu, the malam will be affected due to either constipation or diarrhoea.

MOOTHIRAM

a) NEERKURI (Urine examination)

Urine examination is good diagnostic method compare to naadi and other Envagai thervugal. Theraiyar mention it as.

"அருந்து மாறி ரதமும் அவிரோதமமாய்
அக்கல் அலர்தல் அகாலவூன் தவிர்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காதுபெய்
தொருமுகூர்த்தக் கலைக்குட்பட்டு நீரின்
நிறக்குறி நெய்குறி நிருமித்தல் கடனே."[38]

The early morning urine sample is collected and sample should be examined within one and hour hours.

SIRUNEERIN POTHU GUNAM:

"வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென
றைந்தியலுளவை யறைகுது முறையே."[38]

The urine is examined for its Niram (colour), Eadai (Specific gravity), Nurai (Froth), Natram (Smell), Enjal (Deposits). In kalladaippu, the moothiram is affected due to scanty Micturition.

NIRAM (COLOUR)

"பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதைங் கொழுமையை யொத்துகு நீரே."[39]

1. Yellow
2. Red
3. Green
4. Black
5. White

KALLADAIPPU NEERIN GUNAM (COLOUR INDICATING URINARY STONES)

The urine colour would look like flesh washing water; this is indicated in kidney diseases. This is mentioned as

"தீப்புலால் கழுநீர்ச் செயலெனிர் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறகப்
பற்பகலாகப் பையப் பதிந்தே."[40]

EADAI (SPECIFIC GRAVITY)

Urine, not thick is considerably healthy. This is mentioned as

"மிகத் தடிப்பும் மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும் மெய்ச் சுபாவ நீர் நன்றே."[41]

NURAI (FROTH)

Urine may be frothy in nature, if it is reduced in vali, azhal and ayyam are said to be deranged. This is mentioned as

"பந்தமெய்ப் பசையிளகப்படும் பருவத்
தந்தர்ப் பூதமாய் அனில மூத்திரத்தில்
சம்பத்தப்படும் ததிநுரைப் புனலே."[42]

NAATRAM (SMELL)

Foul odour with pyuria is observed in patients with urinary lithiasis associated with urinary tract infection and ulcer. This is mentioned as

"ஓதமணத்தோ டவவோத மொத்தி றங்கும்
சீதளத்தாற் கம்மிய தேகிகளுக்கே

காணிதில சீழுற் கலந்திழி மணமுறின்
கருப்பநா பிகளுங் காமநா ளத்துளும்
விரணமுண் டின்றேல் எய்து மாசுமரியல்
திருத்தலே திண்ண மெனமனத் துன்னே."[41]

ENJAL (DEPOSITS)

If urine excretion look like curd water white colour and sand like deposits in urine indicate stones in kidney. This is mentioned as

"நார்த்தி நீர்ப்பால் போல நனவுற்றங் கிழியு மானால்
மாரற்ப முற்ற நீரி லடி மண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய் பற்றிய கல்வி னாலே
சீருற்ற செய்கை யென்று தெரிவுறச் செப்ப லாமே."[43]

NEIKURI

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

The urine kept on the kidney tray in sun light, on non wind condition, should be by dropping a drop of gin oil gently with rod. If oil spread like snake, it indicates vali neer; a ring indicates azhal neer and float like a pearl indicates iyya neer and sinks in urine indicates mukkutram.

"அரவென நீண்டினஃதே வாதம்
ஆழி போல் பரவின் அஃதே பித்தம்
முத்தொத்து நிற்கின் மொழிவதென் கபமே".[44]

- Vatha neer – The oil spreads like snake
- Pitha neer – The oil spreads like ring
- Kabha neer – The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis[45]

- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis.[46]

Since kalladaippu is due to the derangement of vatham and pitham, the Neikuri will be vatha or pitha neer.

MARUTHUVAM (LINE OF TREATMENT)

The entire siddha system of medicine consists of three great subdivisions namely,

1) Noyillaneri (preventive) – Kaappu
2) Noineekuneri (curative methods) – Neekkam
3) Uramaakkumurai (strengthening methods) - Niraippu .

Noyillaneri is the special approach of the siddha system where regular dietary habits, early rising, physical and mental discipilinaries are all emphasized. Prevention can mostly save our body and soul, but modernization results in alteration of good health, leads to disease.

Siddha system is playing major role in treating and preventing many chronic diseases. Likewise, herbal medicines have several phyto chemicals which exert their beneficial effect on urolithiasis by multiple mechanisms like,

- Diuretic activity
- Crystallization inhibiting activity
- Anti-Litiatic activity
- Antimicrobial activity
- Analgesic and anti inflammatory activity
- Improving renal function
- Regulates oxalate, calcium mechanisms.

The main object of treatment is to bring down the deranged mukkutrams to natural equilibrium by giving purgatives, which cure derangement of vatham; this is one of the causes for kalladaippu.

In Siddha system, treatment is not only removable of disease but also the prevention and improving the body condition after removal of disease. This is said as kappu, neekkam and niraippu.

Fomentation:

An attack of renal colic may be aborted by the application of heat fomentation (hot water bottle or heater) to the lumbar region. Immediate treatment of loin pain or renal colic is bed rest.

PREVENTION:

1. For prophylactic purpose it is necessary to eliminate all hindrances to a free drainage of urine (constriction, adenoma of the prostrate etc) and to remove foci of infection from the teeth and tonsils.
2. To prevent the formation of urate calculi, a diet of milk and vegetables and mineral water is prescribed.
    a. In the presence of oxalate calculi restrictions are imposed on foods rich in calcium (milk, raw eggs, potatoes) with total abstinence from chocolate, spinach gooseberries and carrots.
3. A patient with phosphorus, carbonate stones is kept on a meat diet and much water to drink.

ADVICE:

1. Patients should drink large amount of water (2 - 3 lit/day)
2. Patient should not suppress the excretion of urine and seminal fluid.
3. Preparation containing Vit. D must be avoided.
4. Regarding prevention Anubhava vaidhya deva ragasiyam states that one should not suppress the excretion of Moothiram (urine) and Sukkilam (Seminal fluid).

NOI KANIPPU VIVADHAM (DIFFERENTIAL DIAGNOSIS)

1) Neerkattu (Anuria)
2) Neerchurukku (Oliguria)
3) Chottu neer (Incontinence)

DO'S AND DONT'S:

DO'S:

1) Drink 2-3 litres of water per day.
2) Dring tender coconut, barley water, lemon juice, raddish juice.
3) The following vegetables can be taken in the diet

   Raddish

   Lady's finger

   Plantain pith

   Mint leaves

   Bottle guard

DONT'S:

1) Avoid cabbage, cauliflower, and tomato seeds, mushroom.
2) Avoid milk and its products.
3) Avoid chicken, fish and other sea foods

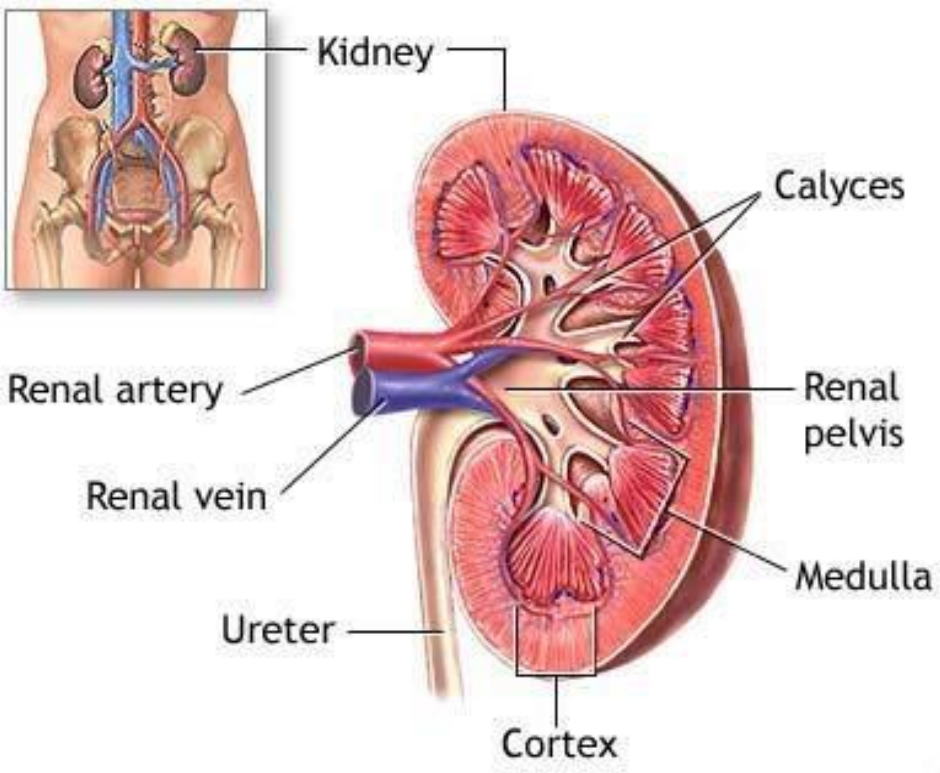
   Avoid drinking fluoride containing water

MODERN ASPECT

ANATOMY AND PHYSIOLOGY OF THE URINARY SYSTEM

KIDNEYS

The kidneys are a pair of excretory organs situated on the posterior abdominal wall, one on each side of the vertebral column, behind the peritoneum. They remove waste products of metabolism and excess of water and salts from the blood, and maintain its pH.



EXTERNAL FEATURES

Each kidney is bean shaped. It has upper and lower poles, medial and lateral borders, and anterior and posterior surfaces.

TWO POLES OF THE KIDNEY

The upper pole is broad and is in close contact with the corresponding suprarenal gland. The lower pole is pointed.

TWO SURFACES

The anterior surface is said to be irregular and the posterior surface flat, but it is often difficult to recognize the anterior and posterior aspects of the kidney by looking at the surfaces. The proper way to do this is to examine the structures present in the hilum as described below.

TWO BORDERS

The lateral border is convex. The medial border is concave. Its middle part shows a depression, the hilus or hilum.

HILUM

The following structures are seen in the hilum from anterior to posterior side: 1) The renal vein 2) the renal artery and 3) the renal pelvis, which is the expanded upper end of the ureter. Examination of these structures enables the anterior and posterior aspects of the kidney to be distinguished from each other. As the pelvis is continuous, inferiorly, with the ureter the superior and inferior poles of the kidney can also be distinguished by examing the hilum. So it is possible to determine the side to which a kidney belongs by examining the structures in the hilum. Commonly, one of the branches of the renal artery enters the hilus behind the renal pelvis, and a tributary of the renal vein may be found in the same plane.

LOCATION

The kidneys occupy the Epigastric, hypochondriac, lumbar and umbilical regions. Vertically they extend from the upper border of twelfth thoracic vertebra to the centre of the body of the third lumbar vertebra.

The right kidney is slightly lower than the left, and the left kidney is a little nearer to the median plane than the right.

SHAPE, SIZE, WEIGHT AND ORIENTATION

Each kidney is about 11cm long, 6 cm broad and 3 cm thick. The left kidney is a little longer and narrower than the right kidney. On an average the kidney weighs 150 g in males and 135 g in females. The kidneys are reddish brown in colour.

The long axis of the kidney is directed downwards and laterally, so that the upper poles are nearer to the median plane than the lower poles. The transverse axis is directed laterally and backwards.

CAPSULES OR COVERINGS OF KIDNEY

1. **The fibrous capsule**: This is a thin membrane which closely invests the kidney and lines the renal sinus.
2. **Perirenal or perinephric fat**: This is a layer of adipose tissue lying outside the fibrous capsule. It is thickest at the borders of the kidney and fills up the extra space in the renal sinus.
3. **Renal fascia:** This is a fibroareolar sheath which surrounds the kidney and the perirenal fat called as the fascia of Gerota. It consists of an anterior layer or fascia of Toldt and a posterior layer or fascia of Zuckerkandl.
4. **Pararenal or paranephric body (fat**): It consists of a variable amount of fat lying outside the renal fascia. It is more abundant posteriorly and towards the lower pole of the kidney. It fills up the paravertebral gutter and forms a cushion for the kidney.

Naked eye examination of a coronal section of the kidney shows: a) an outer, reddish brown cortex; b) an inner, pale medulla; c) a space, the renal sinus.

The renal medulla is made up of about 10 conical masses, called the renal pyramids. Their apices form the renal papillae which indent the minor calices.

The renal cortex is divisible into two parts: a) cortical arches or cortical lobules, which form caps over the bases of the pyramids; and b) renal columns, which dip in between the pyramids. Each pyramid along with the overlying cortical arch forms a lobe of the kidney.

The renal sinus is a space that extends into the kidney from the hilus. It contains a) branches of the renal artery; b) tributaries of the renal vein; and c) the renal pelvis. The pelvis divides into 2 to 3 major calices, and these in their turn divide into 7 to 13 minor calices. Each minor calyx ends in an expansion which is indented by one or three renal papillae.

Histologically, each kidney is composed of one to three million uriniferous tubules. Each tbule consists of two parts which are embryologically distinct from each other. These are as follows.

A) The **secretory part**, called the nephron, which elaborates urine. Nephron is the functional unit of kidney and comprises the renal corpuscle or Malphigian corpuscle and the renal tubule.

B) The **collecting tubule** begins as a junctional tubule from the distal convoluted tubule. Many tubules unite together to form the ducts of Bellini which open into minor calices through the renal papillae.

C) **Juxtaglomerular apparatus** is formed at the vascular pole of glomerulus which is intimately related to its own ascending limb of the Henle's loop near the distal convoluted tubule.

BLOOD SUPPLY

Usually there is one renal artery on each side, arising from the abdominal aorta. Accessory renal arteries are present in 30% of individuals; they arise commonly from the aorta, run parallel to the renal artery, and enter the kidney either at the hilus or at one of its poles.

At or near the hilus the renal artery divides into anterior and posterior divisions. Further branching of these divisions gives rise to segmental arteries each of which

supplies one vascular segment. Five such segments are described. These are apical, upper, middle, lower and posterior. The segmental arteries are end arteries, so that the vascular segments are independent units.

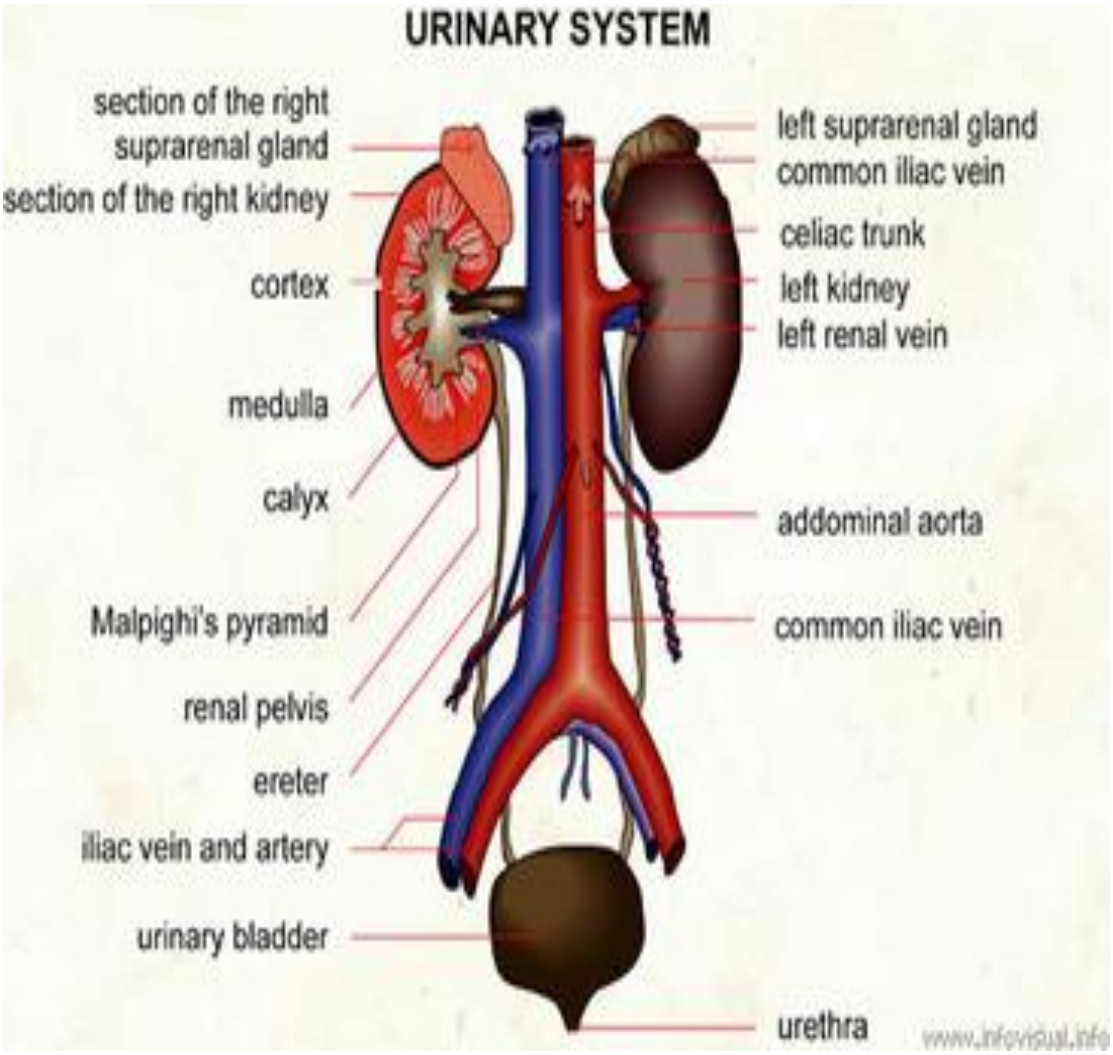
VENOUS DRAINAGE

The venous end of the peritubular capillary plexus gives rise to interlobular veins which run along the corresponding arteries. The interlobular veins drain into the arcuate veins, which in their turn open into the interlobar veins. These emerge at the renal sinus and join to form the renal vein which drains into the inferior vena cava.

The venous end of the capillary plexus along the vasa recta gives rise to veins which drain into the arcuate veins.

LYMPHATIC DRAINAGE

The lymphatics of the kidney drain into the lateral aortic nodes located at the level of origin of the renal arteries.



NERVE SUPPLY

The kidney is supplied by the renal plexus, an offshoot of the celiac plexus. It contains sympathetic (T10 – L1) fibres which are chiefly vasomotor. The afferent nerves of the kidney belong to segments T10 to T12.

FUNCTIONS OF KIDNEYS

Kidneys perform vital functions. By excreting urine, kidneys play principal role in the maintenance of internal environment. In addition, kidneys perform many other functions as described below.

1. ROLE OF HOMEOSTASIS

The primary function of kidneys is homeostasis. It is accomplished by the formation of urine. Kidneys are not only the excretory organs, but are also the regulatory organs because their major role is in homeostasis. During the formation of urine, kidneys regulate various activities in the body, which are concerned with homeostasis.

Excretion of waste products: Removal of wastes help in homeostasis. Kidneys excrete the unwanted waste products which are formed during metabolic activities.

a. Urea – end product of amino acid metabolism
b. Uric acid – end product of nucleic acid metabolism
c. Creatinine – end product of metabolism in muscles
d. Bilirubin – end product of hemoglobin degradation
e. Products of metabolism of other substances.

Kidneys also excrete harmful foreign chemical substances like:

a. Toxins
b. Drugs
c. Heavy metals
d. Pesticides etc.,

Maintenance of water balance: Kidneys maintain the water balance in the body by conserving water when it is decreased and excreting water when it is excess in the body. This is a very important process for homeostasis.

Maintenance of electrolyte balance: Maintenance of electrolyte balance, especially sodium is in relation to water balance. Kidneys retain sodium if the osmolarity of body water decreases and eliminate sodium when osmolarity increases.

Maintenance of acid base balance: The pH of the blood and body fluids should be maintained within narrow range for healthy living. It is achieved by role of kidneys. Body is under constant threat to develop acidosis, because of production

of lot of acids during metabolic activities. However, it is prevented by kidneys, lungs and blood buffers, which eliminate these acids. Among these organs, kidneys play major role in preventing acidosis. In fact, kidneys are the only organs, which are capable of eliminating certain metabolic acids like sulfuric and phosphoric acids.

2. HEMOPOIETIC FUNCTION

Kidneys stimulate the production of erythrocytes by secreting erythropoietin. Erythropoietin is the important stimulating factor for erythropoiesis. Kidney also secretes another factor called thrombopoietin, which stimulates the production of thrombocytes.

3. ENDOCRINE FUNCTION

Kidneys secrete many hormonal substances in addition to erythropoietin and thrombopoietin. The hormones secreted by kidneys are:

a. Erythropoietin
b. Thrombopoietin
c. Renin
d. 1, 25 – Dihydroxycholecalciferol
e. Prostaglandins

4. REGULATION OF BLOOD PRESSURE

Kidneys play an important role in the regulation of arterial blood pressure. Kidneys regulate arterial blood pressure by two ways:

1. By regulating the volume of extracellular fluid
2. Through renin – angiotensin mechanism

5. REGULATION OF BLOOD CALCIUM LEVEL

Kidneys play a role in the regulation of blood calcium level by activating 1, 25 – dihydroxycholecalciferol into vitamin D. Vitamin D is necessary for the absorption of calcium from intestine[44].

MECHANISM OF URINE FORMATION

The process involve in urine formation are,

1. Glomerular filtration
2. Tubular reabsorption
3. Tubular secretion

GLOMERULAR FILTRATION

Glomerular filtrate is protein free plasma. Glomerular filtration is depends upon hydrostatic pressure of the afferent arterioles, glomerular capillary pressure and colloidal osmotic pressure. The glomerular filter contains all the substance present in the plasma except colloids.

Normal amount of urine excreted per day is about 1.5 litres. The glomerular filtrate is alkaline. It contains water, small quantities of urea, glucose, potassium, calcium, bicarbonates and uric acid.

TUBULAR REABSORPTION

When the glomerular filtrate flows through the tubular portion of nephron, both quantitive and qualitative changes occur. Large quantity of water (more than 99%), electrolytes and other substances are reabsorbed by the tubular epithelial cells. The reabsorbed substances move into the interstitial fluid of renal medulla. And, from here, the substances move into the blood in peritubular capillaries.

Since the substances are taken back into the blood from the glomerular filtrate, the entire process is called tubular reabsorption.

TUBULAR SECRETION

In addition to reabsorption from renal tubules, some substances are also secreted into the lumen from the peritubular capillaries through the tubular epithelial cells. It is known as tubular secretion or tubular excretion.

Thus, urine is formed in the nephron by the processes of glomerular filtration, selective reabsorption and tubular secretion[45].

URETERS

The ureters are a pair of narrow, thick-walled muscular tubes which convey urine from the kidneys to the urinary bladder.

They lie deep to the peritoneum, closely applied to the posterior abdominal wall in the upper part, and to the lateral pelvic wall in the lower part.

DIMENSIONS

Each ureter is about 25 cm (10 in.) long, of which the upper half (5 in.) lies in the abdomen, and the lower half (5 in.) in the pelvis. It measures about 3mm in diameter, but it is slightly constricted at three places.

COURSE

The ureter begins within the renal sinus as a funnel shaped dilatation, called the renal pelvis. The pelvis issues from the hilus of the kidney, descends along its median margin, or partly behind it. Gradually it narrows till at the lower end of the kidney it becomes the ureter proper.

The ureter passes downwards and slightly medially on the psoas major muscle, and enters the pelvis by crossing in front of the termination of the common iliac artery. In the lesser or true pelvis the ureter at first runs downwards, and slightly backwards and laterally, following the anterior margin of the greater sciatic notch. Opposite the ischial spine it turns forwards and medially to reach the base of the urinary bladder. The ureter enters the bladder wall obliquely to open into it at the lateral angle of its trigone.

CONSTRICTIONS

The ureter is slightly constricted at three places: 1) at the pelviureteric junction; 2) at the brim of the lesser pelvis; 3) at its passage through the bladder wall. The renal stones tend to get arrested at these places.

BLOOD SUPPLY

Upper part receives branches from renal artery, gonadal or colic vessels, middle part receives branches from aorta, the gonadal or iliac vessels, and pelvic part is supplied by branches from the vesical, middle rectal or uterine vessels.

NERVE SUPPLY

The ureter is supplied by sympathetic from T10 – L1 segments and parasympathetic from S2 – S4 nerves. They reach the ureter though the renal, aortic and hypogastric plexuses. All the nerves appear to be sensory in function[46].

URINARY BLADDER

The urinary bladder is a muscular reservoir of urine, which lies in the anterior part of the pelvic cavity. The detrusor muscle of urinary bladder is arranged in whorls and spirals and is adapted for mass contraction rather than peristalsis.

SIZE, SHAPE AND POSITION

The bladder varies in its size, shape and position according to the amount of urine it contains. When empty it lies entirely within the pelvis; but as it fills it expands and extends upwards into the abdominal cavity, reaching up to the umbilicus or even higher.

EXTERNAL FEATURES

An empty bladder is tetrahedral in shape and has: a) An apex, directed forwards; b) a base or fundus, directed backwards; c) a neck, which is the lowest and most fixed part of the bladder; d) three surfaces, superior and right and left inferolateral; and e) four borders, two lateral, one anterior and one posterior.

A full bladder is ovoid in shape and has: a) An apex, directed upwards towards the umbilicus; b) a neck, directed downwards, and c) two surfaces, anterior and posterior.

INTERNAL SPHINCTER OF THE BLADDER

The bladder wall is made up of longitudinal and circular layers of smooth muscles and they are called detrusor muscle. In the trigone in addition to detrusor muscle, there is trigonal muscle of bell. There is no definite circular muscle fibre at the neck of the bladder stop at the level of neck. Longitudinal fibres from the posterior wall diverge to pass around the urethra on both sides.

CAPACITY OF THE BLADDER

The mean capacity of the bladder in an adult male is 220 ml, varying from 120 to 320 ml. filling beyond 220 ml causes a desire to micturate, and the bladder is

usually emptied when filled to about 250 – 300 ml. filling upto 500 ml may be tolerated, but beyond this it becomes painful. Referred pain is felt in the lower part of the anterior abdominal wall, perineum and penis (T11 to L2; S2 – S4).

BLOOD SUPPLY

Superior vesicle arteries and inferior vesicle arteries supplies the bladder. In addition branches from obturator and inferior gluteal artery are supplied to the bladder.

NERVE SUPPLY

Sympathetic fibers arise from T11 – L2 segment. Parasympathetic fibres branches from S2 – S4. Somatic pudental nerve supplies the sphincter urethrae which is voluntary.

URETHRA

Urethra is a tubular passage extending from the neck of the bladder to the external urethral orifice.

The male urethra extends from the internal urethral orifice at the neck of urinary bladder to the external urethral orifice at the tip of the penis. It is about 20 cm long in flaccid state of the penis; the long axis of urethra shows 2 curvatures and is therefore S shaped. In the erect state it becomes J shaped.

It is divided into 3 parts

1) Prostatic part : Passes through prostate (3 cm long)
2) Membraneous part : Surrounded by sphincter (2 cm long)
3) Spongy part (Penile part) : Passes through the bulb and carpus spongiosum (15 cm long) [48]

SPHINCTER OF THE URETHRA

There are 2 sphincters, in relation with urethra internal and external. The internal sphincter made up of smooth muscle fibre and situated at the neck of the bladder is supplied by sympathetic nerves from lower thoracic segments and upper lumbar segments.

The external sphincter made of light striated muscle fibre surrounds the membraneous part of urethra; it is supplied by prineal branch of the pudental nerve (S2 to S4).

BLOOD SUPPLY

Branches of internal pudental artery supplies urethra[49].

THE FEMALE URETHRA

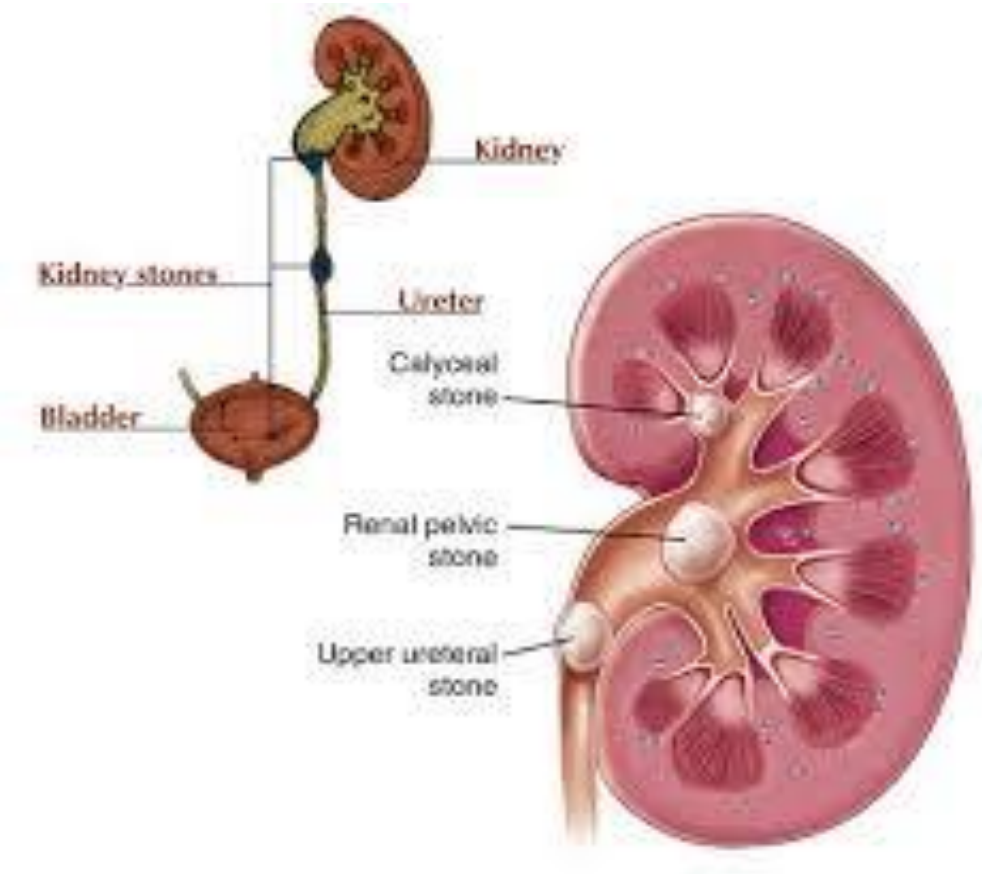
The female urethra is only 4 cm long and 6 mm in diameter. Developmentally, it corresponds to the upper part of the prostatic urethra of the male.

It begins at the internal urethral orifices roughly 5 cm behind the middle of the pubic symphysis. It runs downwards and forwards embedded in the anterior wall of the vagina, traverse the urogenital diaphragm and ends at the lateral urethral orifices in the vestibule.

The mucosa of the urethra is much folded and contains numerous mucous glands and lacunae which open into the urethra. The collections of mucous glands one on each side of the upper part of the urethra is called the paraurethral glands of skene.

The female urethra is dilatable[49].

RENAL CALCULI (UROLITHIASIS)



It refers to calculus formation at any level in the urinary tract but most arise in the kidney.calculi are common in the renal pelvis, calyces and collecting ducts of people in industrialized countries, and is the third most common disorder of the urinary tract[54]

Urolithiasis is a frequent clinical problem, affecting 5 to 10% of Americans in their lifetime. Males are affected more often than females and the peak age at onset is between20 and 30 years. Familial hereditary predisposition to stone formation has long been known. Many of the inborn errors of metabolism, such as gout cystinuria, and primary hyperoxaluria, provide good examples of hereditary disease characterized by excessive production and excretion of stone -forming substances.[55]

In underdeveloped countries nephrolithiasis is rare. In Britain and USA the incidence of renal calculi has become at least 10 times higher in the past 90 years. This increase may be related to changes in diet e.g. increase in protein diet. Current annual

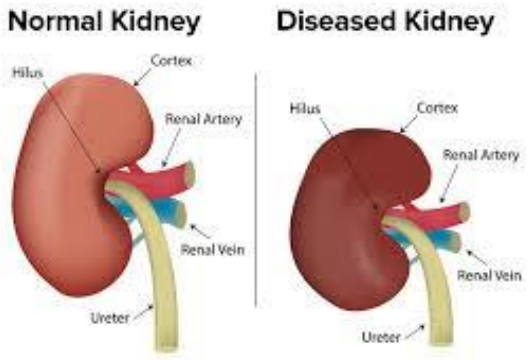
incidence in Britain and the USA ranges from 6.87 to 20.8 per 10,000 of the population. A familial tendency toward stone formation has long been recognized

If stones grow to sufficient size (usually at least 3 millimeters (0.12 in)) they can cause obstruction of the ureter. Ureteral obstruction causes postrenal azotemia and hydronephrosis (distention and dilation of the renal pelvis and calyces), as well as spasm of the ureter. This leads to pain, most commonly felt in the flank (the area between the ribs and hip), lower abdomen, and groin (a condition called renal colic). Renal colic can be associated with nausea, vomiting, fever, blood in the urine, pus in the urine, and painful urination. Renal colic typically comes in waves lasting 20 to 60 minutes, beginning in the flank or lower back and often radiating to the groin or genitals. The diagnosis of kidney stones is made on the basis of information obtained from the history, physical examination, urinalysis and radiographic studies. Ultrasound examination and blood tests may also aid in the diagnosis.[56]

Renal calculi are formed when the urine is supersaturated with salt and minerals such as calcium oxalate, struvite (ammonium magnesium phosphate), uric acid and cystine.60-80% of stones contain calcium[57]. They vary considerably in size from small 'gravel-like' stones, to large staghorn calculi. The calculi may stay in the position in which they are formed, or migrate down the urinary tract, producing symptoms along the way. Studies suggest that the initial factor involved in the formation of a stone may be the presence of nanobacteria that form a calcium phosphate shell[58,59.].

The other factor that leads to stone production is the formation of Randall's plaques. Calcium oxalate precipitates form in the basement membrane of the thin loops of Henle; these eventually accumulate in the subepithelial space of the renal papillae, leading to a Randall's plaque and eventually a calculus.[60]

Pathophysiology



Urinary calculi are consist of aggregates of crystals, usually containing calcium or phosphate in combination with small amounts of proteins and glycoproteins. In developed countries, however, most calculi occur in healthy young men, in whom investigations reveal no clear predisposing cause. Renal stones vary greatly in size. There may be particles like sand anywhere in the urinary tract, or large round stones in the bladder.

In developing countries bladder stones are common, particularly in children. In developed countries, the incidence of childhood bladder stones is low; renal stones in adults are more common. Staghorn calculi fill the whole renal pelvis and branch into the calyces; they are usually associated with infection and composed largely of struvite.

Deposits of calcium may be present throughout the renal parenchyma, giving rise to fine calcification within it (nephrocalcinosis), especially in patients with renal tubular acidosis, hyperparathyroidism, vitamin D intoxication and healed renal tuberculosis. Cortical nephro calcinosis may occur in areas of cortical necrosis, typically after AKI in pregnancy or other severe AKI.

PREVALENCE

Renal stone disease is common , affecting individuals of all countries and ethnic groups. In the UK, the prevalence is about 1.2%, with a lifetime risk of developing a renal stone at age 60-70% of about 7% in men. In some regions the risk is higher, most

notably in countries like Saudi Arabia, where the lifetime risk of developing a renal stone in men aged 60-70 is just over 20%[61].

EPIDEMIOLOGY

- Renal stones are common, being present at some time in one in ten of the population, although a significant proportion will remain asymptomatic.
- The annual incidence is about 1-2 cases of acute renal colic per 1,000 people and the average lifetime risk around 5-10%.
- Men are more commonly affected than women, with a male to female ratio of 3:1. The difference between the sexes is gradually being eroded. This is thought to be due to lifestyle-associated factors, such as obesity and a Western diet.
- The peak age for developing stones is between 30 and 50 and recurrence is common[57].

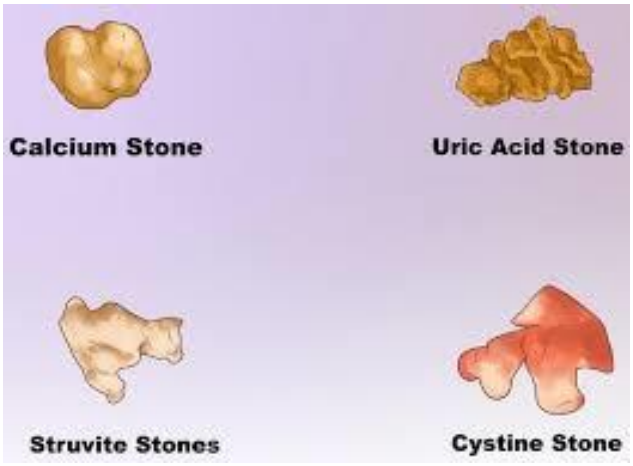
ETIOLOGY AND PATHOGENESIS

The crystals in the stone in the kidney are commonly mixed, although there is usually a preponderance of crystals from one particular solute. 70-75% of renal stones are composed almost entirely of calcium oxalate, mixed with calcium phosphate. 15% of calculi consist predominantly of magnesium ammonium phosphate (struvite). Uric acid stones and cysteine stones together account for about 10% of calculi. Very rarely stones are made up of almost pure xanthine, silica or some chemical foreign to the body.

Uric acid crystals are commoner in some parts of the Middle East, India and North Africa than in Europe and the USA.

The formation of stones within the kidney is not a specific disease, it is potential complication of many different disorders. The cause is obscure. The most important is almost certainly an increased urine concentration of the some constiuents[62].

TYPES OF RENAL CALCULI



There are 4 main tyoes of renal calculi- calcium containing, mixed(struvite), uric acid and cysteine stones, a few rare types.

1) CALCIUM STONES

Calcium stones are the most common comprising about 75% of all urinary calculi. They may be pure stones of calcium oxalate (50%), or calcium phosphate (5%), or mixture of calcium oxalate and calcium phosphate (45%).

Etiology

Etiology of calcium stones are variable.

About 50% of patients with calcium stones have *idiopathic hypercalciuria without hypercalcaemia.*

Approximately 10 % cases are associated with *hypercalcaemia and hypercalciuria*, the most commonly due to hyperparathyroidism, or a defect in the bowel (i.e. absorptive hypercalciuria), or in the kidney (i.e. renal hypercalciuria).

About 15% of patients with calcium stones have *hyperuricosuria with a normal blood uric acid level* and without any abnormality of calcium metabolism.

In about 25% of patients with clcium stones, the cause is known as there is no abnormality in urinary excretion of calcium, uric acid or oxalate and is referred to as *idiopathic calcium stone disease*.

Pathogenesis

The mechanism of calcium stone formation is explained on the basis of imbalance between the degree of supersaturation of the ions forming the stone and the concentration of inhibitors in the urine. Most likely site where the crystals of calcium oxalate and / or calcium phosphate are precipitated is the tubular lining or around some fragment of debris in the tubule acting as nidus of the stone. The predisposing factors contributing to formation of calcium stones are alkaline pH, decreased urinary volume and increased excretion of oxalate and uric acid

Morphology

Calcium stones are usually small (less than a centimeter), ovaid, hard, with granular rough surface. They are dark brown due to old blood pigment deposited in them as a result of repeated trauma caused to the urinary tract by these sharp – edged stones.

2.MIXED STONES

About 15% of urinary calculi are made of magnesium-ammonium-calcium phosphate, often called struvite; hence mixed stones are also called as *struvite stones* or triple phosphate stones.

Etiology

Struvite stones are formed as a result of infection of the urinary tract with urea-splitting organisms that produce urease such as by specious of Proteus, and occasionally klebsiella, pseudomonas, and Enterobacter. these are, therefore, also known as infection – induced stones. However, E.coli does not form urease.

Morphology

Struvite stones are yellow- white or grey. They tend to be soft and irregular in shape. Staghorn stone which is large, solitary stone that takes the shape of the renal pelvis where it is often formed is an example of struvite stone[63]

3).URIC ACID STONES

Approximately 6% of urinary calculi are made of uric acid. Uric acid calculi are *radiolucent* unlike radio- opaque calcium stones.

Etiology

Uric acid stones are frequently formed in cases with hyperuricaemia and hyperuricosuria such as due to primary gout or secondary gout due to myeloproliferative disorders (e.g. in leukaemias), especially those on chemotherapy, and administration of uricosuric drugs (e.g. salicylates, probenacid).

Other factors contributing to their formation are acidic urinary pH (below 6) and low urine volume.

Pathogenesis

The solubility of uric acid at pH of 7 is 200 mg/ dl while at pH of 5 is 15 mg /dl. Thus as the urine becomes more acidic, the solubility of uric acid in urine decreases and precipitation of uric acid crystals increases favoring the formation of uric acid stones. Hyperuricosuria is the most important factor in the production of uric acid stones, while hyperuricaemia is found in about half the cases.

Morphology

Uric acid stones are smooth, yellowish- brown, hard and often multiple. On cut section, they show laminated structure.

4).CYSTINE STONES

Cysteine stones comprise less than 2% of urinary calculi.

Etiology

Cysteine are associated with cystinuria due to a genitically- determined defect in the transport of cysteine and other amino acids across the cell membrane of the renal tubules and the small intestinal mucosa.

Pathogenesis

The resultant excessive excretion of cysteine which is least soluble of the naturally- occurring amino acids leads to formation of crystals and eventually cysteine calculi.

Morphology

cysteine stones are small, rounded, smooth and multiple. They are yellowish and waxy.

5.OTHER CALCULI

Less than 2% of urinary calculi consist of other rare types such as due to inherited abnormality of enzyme metabolism e.g. hereditary xanthinuria developing xanthine stones[64].

CLINICAL COURSE

Stones are of importance when they obstruct urinary flow or produce ulceration and bleeding. They may be present without producing any symptoms or significant renal damage. In general, smaller stones are most hazardous, as they may pass into the ureters, producing pain referred to as colic (one of the most intense forms of pain) as well as ureteral obstruction. Larger stones cannot enter the ureters and are more likely to remain silent within the renal pelvis. Commonly, these larger stones first manifest themselves by hematuria. Stones also predispose to superimposed infection, both by their obstructive nature and by the trauma they produce[65]**.**

MORPHOLOGY OF KIDNEY

In 80% of patients, stones are unilateral. Common sites are renal pelvis, calyces and the bladder. Calcium oxalate crystals are mostly unilateral and solitary. They are usually either yellow-brown or dark from altered blood, and hard. Predominantly oxalate calculi may be nodular with smaller blunt spikes; mixed oxalate and phosphate stones may be fairly smooth. If small they are triangular in section. The nodular form may be called mulberry type in Britain or the jackstone type in the USA. Occasionally progressive accretion of salts leads to the development of

branching structures known as staghorn calculi, which create a cast of the renal pelvic and calyceal system. These massive stones are usually composed of magnesium ammonium phosphates[62].

CLINICAL FEATURES

The clinical presentation is highly variable. Most patients with renal stone disease are asymptomatic, whereas others present with pain, hematuria, UTI, or urinary tract infection.

A common presentation is with acute loin pain radiating to the anterior abdominal wall, together with hematuria; a symptom complex termed renal or ureteric calculi. This most commonly caused by a calculus but the same symptoms can occur in association with a sloughed renal papilla, tumour or blood clot.

The patient is suddenly aware of pain in the loin, which radiates round the flank to the groin and often into the testis or labium, in the sensory distribution of the first lumbar nerve. The pain steadily increase in intensity to reach a peak in a few minutes.

The patient is restless and generally tries unsuccessfully to obtain relief by changing position or packing the room. There is pallor, sweating, and often vomiting. Frequency, dysuria, and hematuria may occur. The intense pain usually subsides within 2 hours but may continue unabated for hours or days[61].

PRESENTATION

- Many stones are asymptomatic and discovered during investigations for other conditions.
- The classical features of renal colic (or ureteric colic) are sudden severe pain. It is usually caused by stones in the kidney, renal pelvis or ureter, causing dilatation, stretching and spasm of the ureter. In most cases no cause is found:

- Pain starts in the loin about the level of the costovertebral angle (but sometimes lower) and moves to the groin, with tenderness of the loin or renal angle, sometimes with haematuria.

- If the stone is high and distends the renal capsule then pain will be in the flank but as it moves down pain will move anteriorly and down towards the groin.
- A stone that is moving is often more painful than a stone that is static.
- The pain radiates down to the testis, scrotum, labia or anterior thigh.
- Whereas the pain of biliary or intestinal colic is intermittent, the pain of renal colic is more constant but there are often periods of relief or just a dull ache before it returns. The pain may change as the stone moves. The patient is often able to point to the place of maximal pain and this has a good correlation with the current site of the stone.

Other symptoms which may be present include:

- ❖ Rigors and fever.
- ❖ Dysuria.
- ❖ Haematuria.
- ❖ Urinary retention.
- ❖ Nausea and vomiting[57].

PREDISPOSING FACTORS FOR KIDNEY STONES

Environmental and dietary:

- Low urine volumes, high ambient temperatures, low fluid intake
- Diet: high protein intake, high sodium, low calcium
- High sodium excretion
- High urate excretion
- High oxalate excretion
- Low citrate excretion

Acquired causes:

- Hypercalcemia of any cause
- Ileal disease or resection (leads to increased oxalate absorption and urinary excretion)
- Renal tubular acidosis type I

Congenital and inherited causes:

- Familial hypercalciuria
- Renal tubular acidosis type I (distal)
- Medullary sponge kidney
- Cystinuria
- Primary hyperoxaluria

The majority of stones pass spontaneously within 48 hours. However, some stones may not. There are several factors which influence the ability to pass a stone. These include the size of the person, prior stone passage, prostate enlargement, pregnancy, and the size of the stone. A 4 mm stone has an 80% chance of passage while a 5 mm stone has a 20% chance. If a stone does not pass, certain procedures (usually by a urology specialist doctor) may be needed[66].

RISK FACTORS

Several risk factors are recognised to increase the potential of a susceptible individual to develop stones. These include:

- Anatomical anomalies in the kidneys and/or urinary tract - eg, horseshoe kidney, ureteral stricture.
- Family history of renal stones.
- Hypertension.
- Gout.
- Hyperparathyroidism.
- Immobilisation.
- Relative dehydration.
- Metabolic disorders which increase excretion of solutes - eg, chronic metabolic acidosis, hypercalciuria, hyperuricosuria.
- Deficiency of citrate in the urine.
- Cystinuria (an autosomal-recessive aminoaciduria).
- Drugs - eg, diuretics such as triamterene and calcium/vitamin D supplements.
- More common occurrence in hot climates.

- Increased risk of stones in higher socio-economic groups.
- Contamination - as demonstrated by a spate of melamine-contaminated infant milk formula[67].

EXAMINATION

- The patient with colic of any sort writhes around in agony. This is in contrast to the patient with peritoneal irritation who lies still.
- The patient is apyrexial in uncomplicated renal colic (pyrexia suggests infection and the body temperature is usually very high with pyelonephritis).
- Examination of the abdomen can sometimes reveal tenderness over the affected loin. Bowel sounds may be reduced. This is common with any severe pain.
- There may be severe pain in the testis but the testis should not be tender.
- Blood pressure may be low.
- Full and thorough abdominal examination is essential to check for other possible diagnoses - eg, acute appendicitis, ectopic pregnancy, aortic aneurysm[57.]

DIFFERENTIAL DIAGNOSIS

This depends upon the position of the pain and the presence or absence of pyrexia and includes:

- Biliary colic.
- Pyelonephritis: very high temperature. Pain is unlikely to radiate to the groin.
- Acute pancreatitis
- Acute appendicitis
- Perforated peptic ulcer.
- Epididymo – orchitis or torsion of the testis: very tender testis.
- Sinister causes of back pain: usually tender over vertebrae.
- Dissection of an aortic aneurysm: the patient who presents with features of renal colic for the first time over the age of 60. This may be dissection of aortic aneurysm leading to ruptured aortic aneurysm.

- Drug addiction: there are reports of people with fictitious stories of renal colic, designed to obtain an injection of pethidine. These patients tend to be abusive when offered anything other than pethidine[68.]

INVESTIGATIONS

Basic analysis should include:

- Blood for FBC, CRP, renal function, electrolytes, calcium, phosphate and urate, creatinine.
- Midstream specimen of urine for microscopy (pyuria suggests infection), culture and sensitivities.
- Prothrombin time and international normalised ratio if intervention is planned.
- Stick testing of urine for red cells (suggestive of urolithiasis), white cells and nitrites (both suggestive of infection) and pH (pH above 7 suggests urea-splitting organisms such as *Proteus* spp. whilst a pH below 5 suggests uric acid stones).

- Intravenous pyelogram (IVP)[69] .
- Computed tomography (CT)
- Ultrasound scanning may be helpful to differentiate radio-opaque from radiolucent stones and in detecting evidence of obstruction.
- Plain X-rays of the kidney, ureter and bladder (KUB) are useful in watching the passage of radio-opaque stones (around 75% of stones are of calcium and so will be radio-opaque).
- The European Association of Urology's guidelines on urolithiasis recommend stone analysis for:

- All first-time stone formers.
- All patients with recurrent stones who are on pharmacological preventing therapy.
- Patients who have had early recurrence after complete stone clearance.
- Late recurrence after a long stone-free period (stone composition may change)[70].

COMPLICATIONS

The complications of calcium oxalate and hydroxyapatite renal calculi are acute and chronic pyelonephritis, hydronephrosis and obstructive nephropathy.

The stone fragments may obstruct the ureter. This occurs in 5-15% of cases. 8% of patients develop hypertension or exacerbation of pre- existing hypertension within 1 year. thirdly there is a risk of renal damage.

HYDRONEPHROSIS

In bilateral complete obstruction, patient present with anuria. When the obstruction is below the bladder the dominant symptoms are bladder distention. Unilateral hydronephrosis may remain completely silent for long periods of time. Enlargement of kidney is made out on physical examination. Sometimes the obstructing cause e.g. calculi can produce symptoms.

Early removal of the cause of obstruction can return the full function of the kidney. In long standing cases the changes become irreversible.

Two metabolic disorders need to be mentioned here which are associated with precipitation of the crystalline material causing obstruction to urine flow.

1.HYPERURICAEMIA

Uric acid stones are formed In 22% of patients with gout. The urate crystals get deposited in distal collecting tubules. *Collecting ducts as well in the interstitium, forming gouty tophus.* Uric acid crystal deposition takes place also following chemotherapy to the patients of leukemia and lymphoma. this is due to the breaking down of nucleic acid.

2.HYPERCALCAEMIA

Hyperparathyroidism, end stage kidney disease, vit. D intoxication, excessive calcium intake, osteolytic disease of bones, milk- alkali syndrome are some of the conditions giving rise to hypercalcaemia. This induces deposition of calcium in renal tubules called nephrocalcinosis. Sometimes, the deposition can also form renal stones[62].

Complete blockage of the urinary flow from a kidney decreases glomerular filtration rate (GFR) and, if it persists for more than 48 hours, may cause irreversible renal damage.

If ureteric stones cause symptoms after four weeks, there is a 20% risk of complications, including deterioration of renal function, sepsis and ureteric stricture.

Infection can be life-threatening.

Persisting obstruction predisposes to pyelonephritis[70].

PROGNOSIS

The prognosis will depend upon the underlying condition causing the renal stones. Calcium oxalate and hydroxyapatite stones per se rarely lead to renal failure. If at the time of diagnosis there is renal damage due to the calculi then this may cause renal dysfunction and hypertension. The main problem is recurrence unless the causative condition can be treated ; the recurrence rate is high, approaching 70% by 10 years after spontaneous passage or surgical removal of a calculus[71].

- Most symptomatic renal stones are small (less than 5 mm in diameter) and pass spontaneously.
- Stones less than 5 mm in diameter pass spontaneously in up to 80% of people.
- Stones between 5 mm and 10 mm in diameter pass spontaneously in about 50% of people.
- Stones larger than 1 cm in diameter usually require intervention (urgent intervention is required if complete obstruction or infection is present).
- Two thirds of stones that pass spontaneously will do so within four weeks of onset of symptoms.
- A stone that has not passed within 1-2 months is unlikely to pass spontaneously.
- The following features predispose to recurrent stone formation:
- First attack before 25 years of age.
- Single functioning kidney.
- A disease that predisposes to stone formation.
- Abnormalities of the renal tract.

PREVENTION

Recurrence of renal stones is common and therefore patients who have had a renal stone should be advised to adapt and adopt several lifestyle measures which will help to prevent or delay recurrence:

- Increase fluid intake to maintain urine output at 2-3 litres per day.
- Reduce salt intake.
- Reduce the amount of meat and animal protein eaten.
- Reduce oxalate intake (foods rich in oxalate include chocolate, rhubarb, nuts) and urate-rich foods (eg, certain fish).
- Drink regular cranberry juice: increases citrate excretion and reduces oxalate and phosphate excretion.

CALCIUM

- Maintain good calcium intake(calcium forms an insoluble salt with dietary oxalate, lowering oxalate absorption and excretion).
- Avoid calcium supplements separate from meals (increase calcium excretion without reducing oxalate excretion).

OXALATE

Depending on the composition of the stone, medication to prevent further stone formation is sometimes given - eg, thiazide diuretics (for calcium stones),allopurinol (for uric acid stones) and calcium citrate (for oxalate stones)

TRIAL DRUG

கற்பூர சிலாசத்து பற்பம்

ஆதாரம் - அகஸ்தியர் செந்தூரம் 300 பக்க எண் 23

கேளப்பா கற்பூர சலாசத் துத்தான்
கெடியாக பலமொன்று பொரிகா ரந்தான்
நாளப்பா பலமொன்று ரெண்டுங் கூட்டி
நலமான பருத்தி நீறு துத்தி வேர்த்தோல்
நீளப்பா பாங்காகக் கஷாயம் செய்து
நிசமாக விட்டாட்டு ஒரு நாள் மட்டும்
மாளப்பா அஞ்செருவிற் புடத்தைப் போடு
ஆறியெடு கல்வத்தி லாட்டு தானே.
ஆட்டத்தூளாகுமடா வெண்கருவை விட்டு
அரைத்துவில்லை செய்துரவி யிலுர்த்திக் கொண்டு
மாட்டவே அஞ்செருவிற் புடத்தைப்போடு
வாகாக அரைத்து அஞ்சுதரமு மானால்
நாட்டவே வெண்பொடியாம் பதனம் பண்ணு
நலமான பெருவயிறும் மகோதரங்கள்
ஊட்டவே நீர்ச்செரிப்பு கல்லடைப்பு
ஓடு மப்பா வெடிய்ப்பு சுன்ன நீரே...

INGREDIENTS

- Karpoora silasathu (Gypsum)
- Venkaram (Borax)
- Paruththi samoola champal (*Gossypium herbaceum*)
- Thuththi ver (*Abutilon indicum*)

ACTIONS OF TRIAL DRUGS

S.No	Drugs	Botanical name	Actions
1	Karpoora silasathu	(Gypsum)	Diuratic Lithontriptic Astringent[72]
2	Venkaram	(Borax)	Diuratic Lithontriptic Refrigerant[73]
3	Paruththi samoola champal	*Gossypium herbaceum*	Demulcent Astringent Diuratic[74]
4	Thuththi ver	*Abutilon indicum*	Tonic Diuratic Laxative[75]

STANDARD OPERATIVE PROCEDURE

Take karpoora silasathu and venkaaram (35 gms each) and grind them with decoction of paruthi chedi saambal and thuththi ver thol for 24 hours, make it paste and dry and calcinate with five cow dungs.

Then grind it again with white egg yolk, make it into paste and dry, calcinate with 5 cow dungs. Repeat the process for five times.[76]

DOSAGE

130 mg, bid after food

ADJUVANT

Ilaneer

DURATION

1 Mandalam (48 days)

INGREDIENTS OF TRIAL DRUG

Before purification

KARPOORA SILASATHU

VENKARAM

AFTER PURIFICATION

KARPOORA SILASATHU

VENKARAM

PARUTHI CHEDI

THUTHI VER

TRIAL MEDICINE

KARPOORA SILASATHU PARPAM

MATERIALS AND METHODS

PROTOCOL

Study Design

A clinical study on kalladaippu was carried out in the Post graduate Department of Maruthuvam in Govt.Siddha Medical College attached to Arignar Anna Hospital of Indian Medicine, Chennai – 106 during the period of 2015 – 2017.

The study was approved by **Instititional Ethics Committee (IEC)** and the approval number is **GSMC-CH-ME-4/2015/010**. It was registered in **Clinical Trials Registry – India (CTRI)** and the register number is **CTRI/2017/04/008435**.

Sample size

The study is conducted in 40 selected kalladaippu patients of both genders between age groups of 18 to 60 years.

Selection Criteria

The patients having following parameters are selected for the study.

- Pain in the flank
- Burning Micturition
- Oliguria
- Dysuria
- Nausea
- Vomiting
- Haematuria
- Fever
- History of Urolithiasis (with USG-Whole Abdomen reports)

Exclusion Criteria

- Age group less than 18
- Stag horn calculus
- Pyonephrosis
- Calculi associated with elevated serum creatinine level
- Calculi in pregnancy

Proforma

The case sheet proforma for kalladaippu was prepared based on Siddha Diagnostic methodology with necessary modern techniques.

History taking

For better treatments and results a detailed clinical history was taken regarding present illness, past illness, family history, menstrual history, occupational history, socio economic status, residential area, etc.,

Investigation

All patients were screened by the following investigations. This was carried out regularly before and after treatment.

- **Blood for biochemical examination**

The blood was tested for sugar, urea, serum creatinine to know the renal function and its excretion.

- **Urine Examination**

Albumin, Sugar, Deposits.

- **Ultra sonogram**

Ultra sonogram of complete abdomen including KUB was done in cases to know the location, size and number of calculi.

Drug and dose schedule

Karpoora silasathu parpam – 130 mg, bid after food with Ilaneer for 48 days.

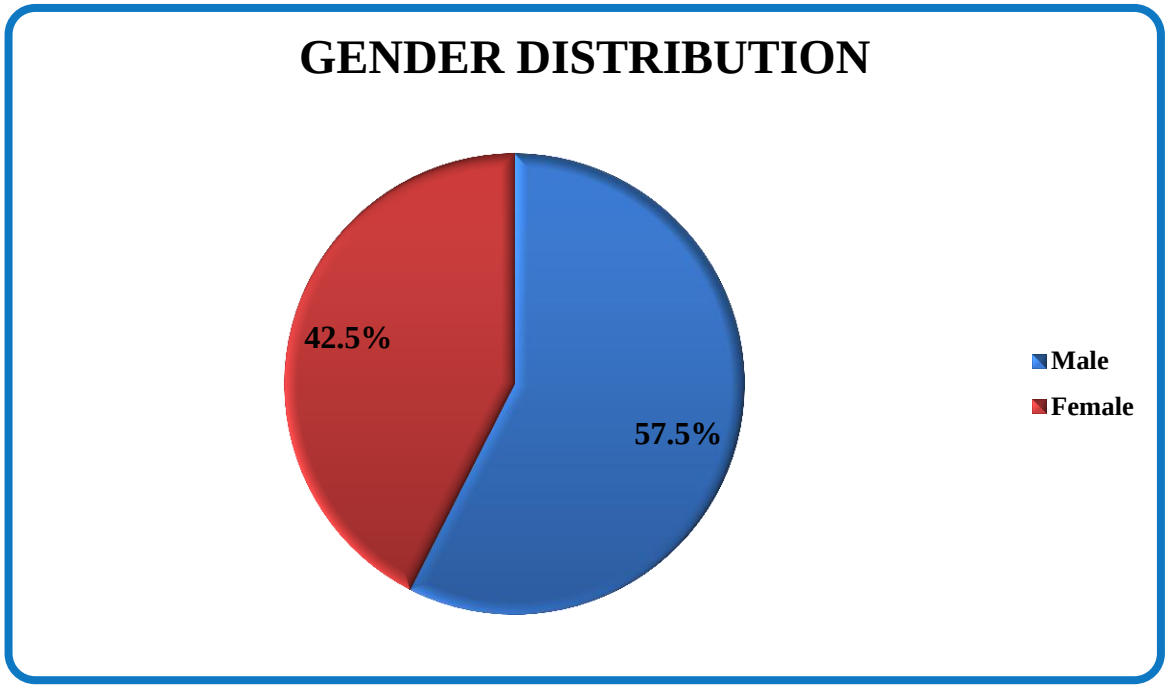
RESULTS AND OBSERVATION

40 cases having Kalladaippu were selected and treated in OPD of PG Maruthuvam Department attached to AAGHIM, Chennai – 106 during the year 2015 – 2017. The result and observation during that clinical study are as follows.

- Gender distribution
- Age distribution
- Occupation
- Socio- economic status
- Dietary habits
- Seasonal occurence
- Distribution of thinai
- Distribution of mukkutram – vatham
- Distribution of mukkutram – pitham
- Distribution of mukkutram – kabham
- Ezhu udal thathukkal
- En vagai thervugal
- Naadi
- Neikuri
- Clinical features
- Clinical prognosis
- Distribution of calculi based on location
- Grading of results

GENDER DISTRIBUTION

S.No	GENDER	NUMBER OF CASES	PERCENTAGE (%)
1	Male	23	57.50%
2	Female	17	42.50%



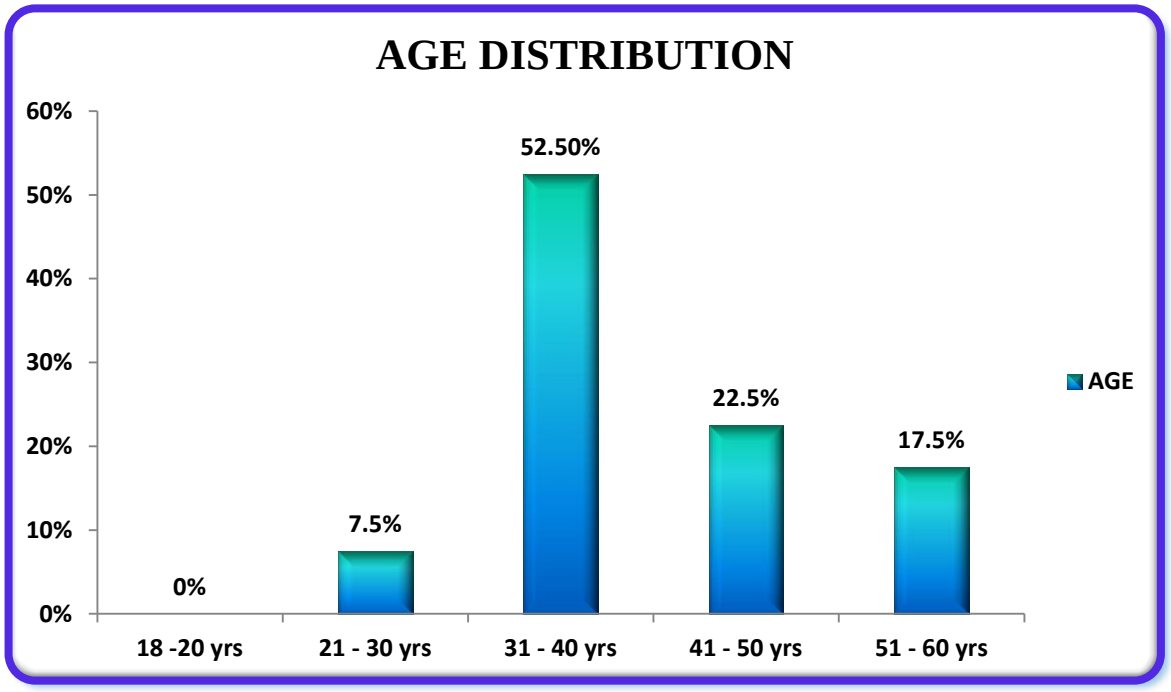
INFERENCE

About 57.5% were males and 42.5% were females

Literature: according to literature males are more prone to renal calculi

AGE DISTRIBUTION

S.No	AGE IN YEARS	NUMBER OF CASES	PERCENTAGE (%)
1	18 – 20 yrs	0	0%
2	21 - 30 yrs	3	7.5%
3	31 - 40 yrs	21	52.5%
4	41 - 50 yrs	9	22.5%
5	51 - 60 yrs	7	17.5%



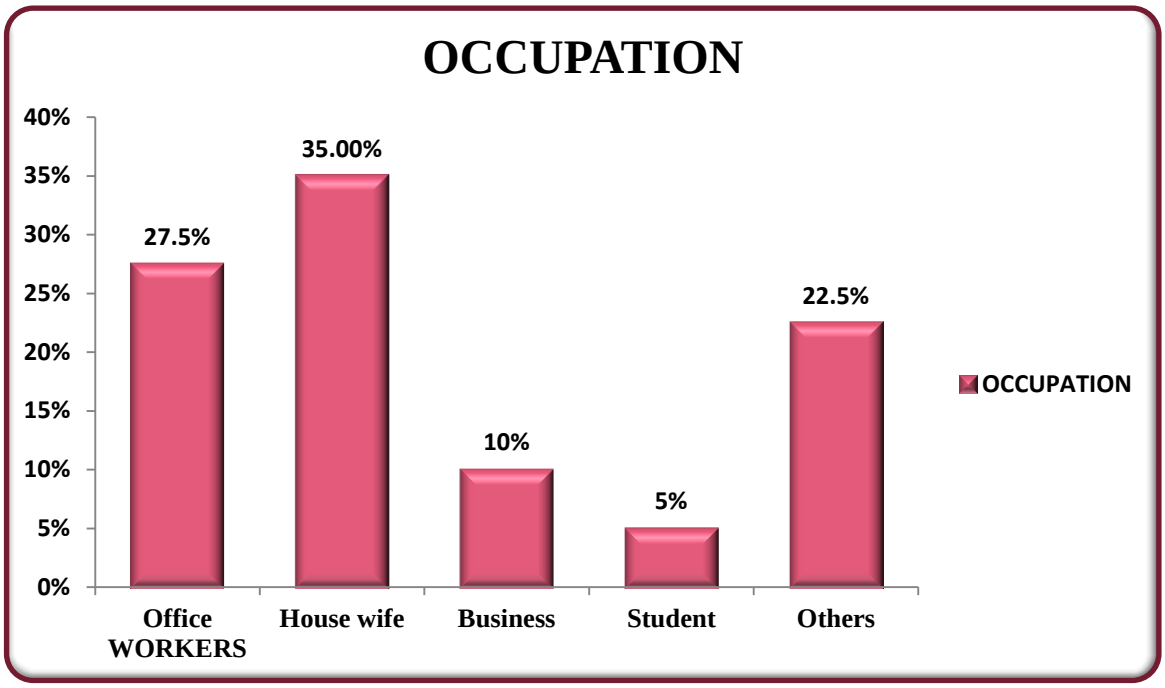
INFERENCE

Majority of the case that is 52.5% were in the 3rd decade, 22.5% were in the 4th decade, 7.5% were in the2nd decade, 17.5% were in the 5th d

ecade.

OCCUPATION

S.No	OCCUPATION	NUMBER OF CASES	PERCENTAGE (%)
1	Office worker	11	27.5%
2	House Wife	14	35%
3	Business	4	10%
4	Student	2	5%
5	Others(Coolies)	9	22.5%

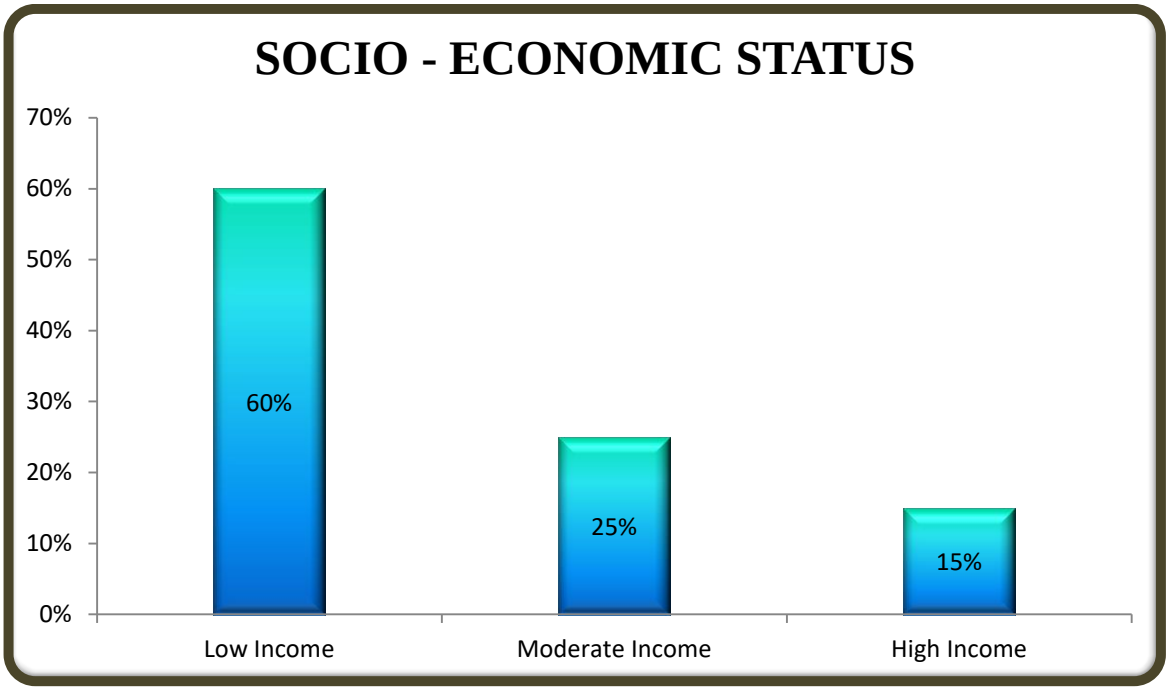


INFERENCE

0ut of 40 patients (100%), 35% were house wife, 27.5% were office worker,10% were business and student 5%, 22.5% were in other occupation.

SOCIO – ECONOMIC STATUS

S.No	SOCIO – ECONOMIC STATUS	NUMBER OF CASES	PERCENTAGE (%)
1	Low Income (below 200,000 per annum)	24	60%
2	Moderate Income (200,000 – 500,000 per annum)	10	25%
3	High Income (Above 500,000 per annum)	6	15%

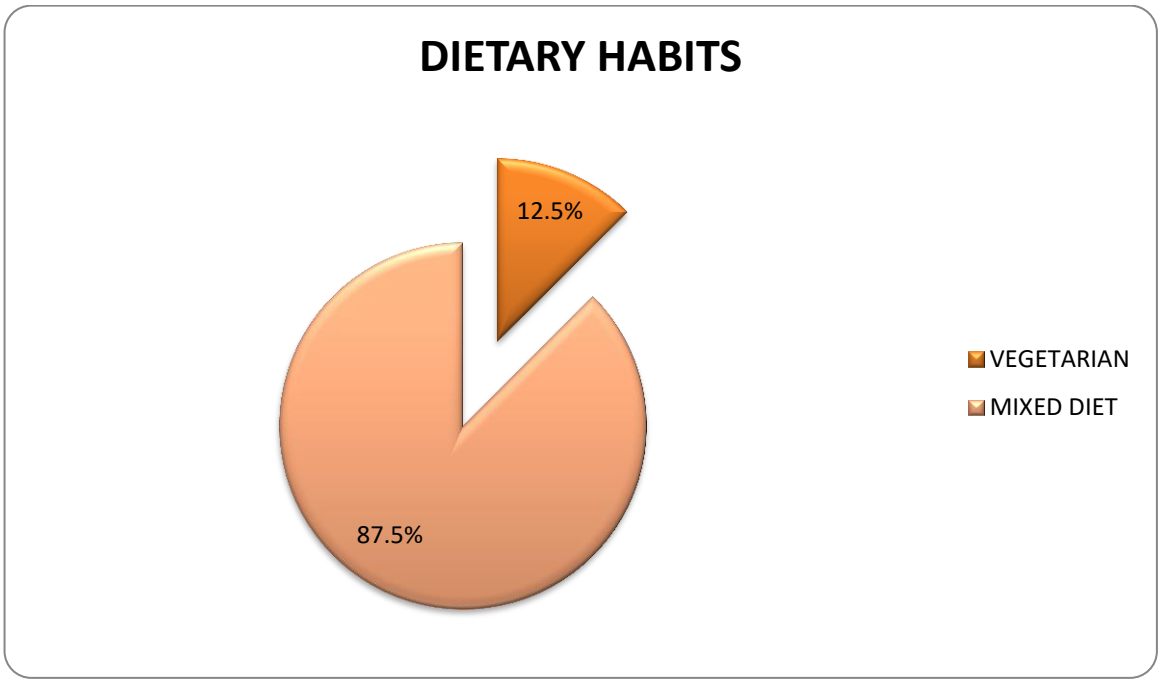


INFERENCE:

Among 40 cases 60% comes under low economic status, 25% of them under moderate status and 15% of them under high income status.

DIETARY HABITS

S.No	DIET	NUMBER OF CASES	PERCENTAGE (%)
1	Vegetarian	5	12.5%
2	Mixed diet	35	87.5%

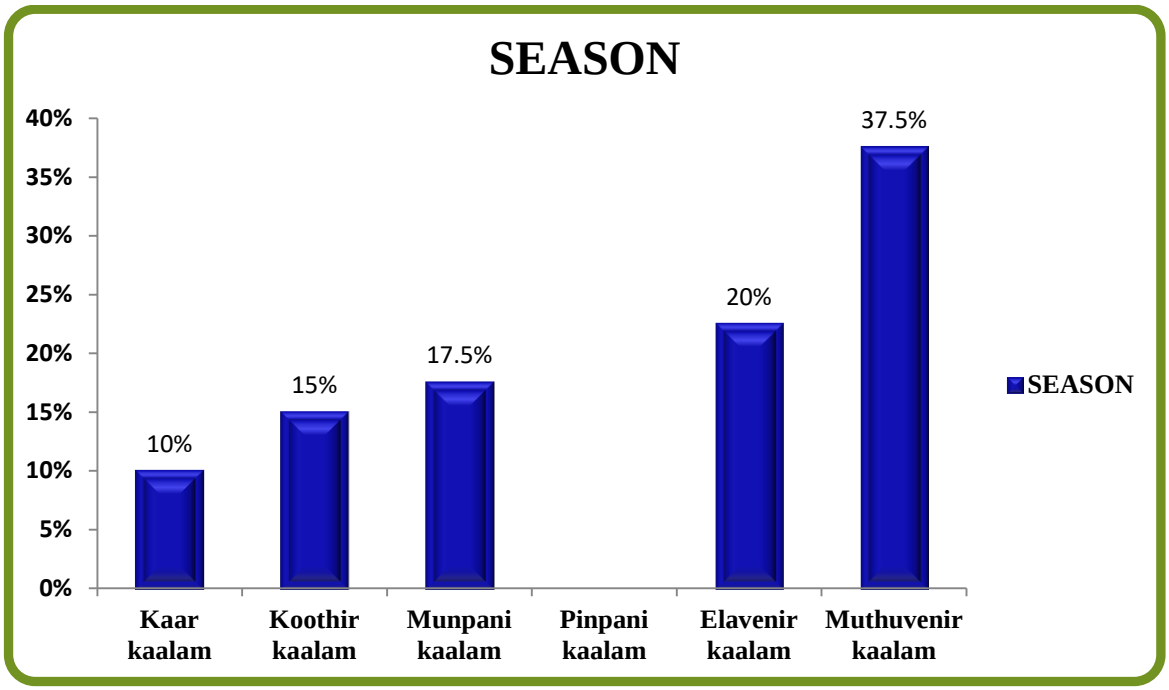


INFERENCE

Among 40 patients, five patients (12.5%) were taking vegetarian food and 35 patients were taking mixed diet.

SEASONAL OCCURENCE

S.No	KAALAM (Season)	NUMBER OF CASES	PERCENTAGE (%)
1	Kaar kaalam (Mid Aug – Mid Oct)	4	10%
2	koothir Kaalam (Mid Oct – Mid Dec)	6	15%
3	Munpani kaalam (Mid Dec – Mid Feb)	7	17.5%
4	Pinpani kaalam (Mid Feb – Mid Apr)	0	0%
5	Elavenir kaalam (Mid Apr – Mid Jun)	8	20%
6	Muthuvenir kaalam (Mid Jun – Mid Aug)	15	37.5%

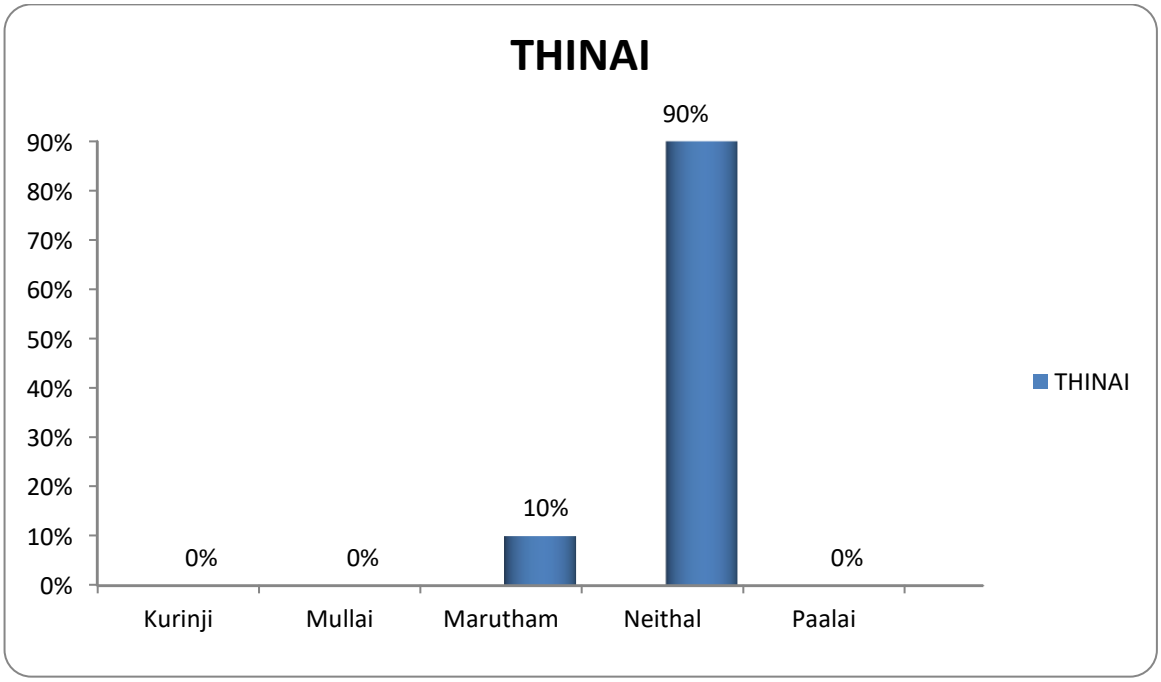


INFERENCE

According to paruvakaalam highest incident of 15 cases (37.5%) were noted in muthuvenir kaalam and 8 cases (20%) were noted in elavenir kaalam, 6 cases (15%) were noted in koothir kaalam, 4 cases (10%) were noted in kaar kaalam .

DISTRIBUTION OF THINAI

S.No	THINAI	NUMBER OF CASES	PERCENTAGE (%)
1	Kurinji	0	0%
2	Mullai	0	0%
3	Maruthuvam	4	10%
4	Neithal	36	90%
5	Paalai	0	0%

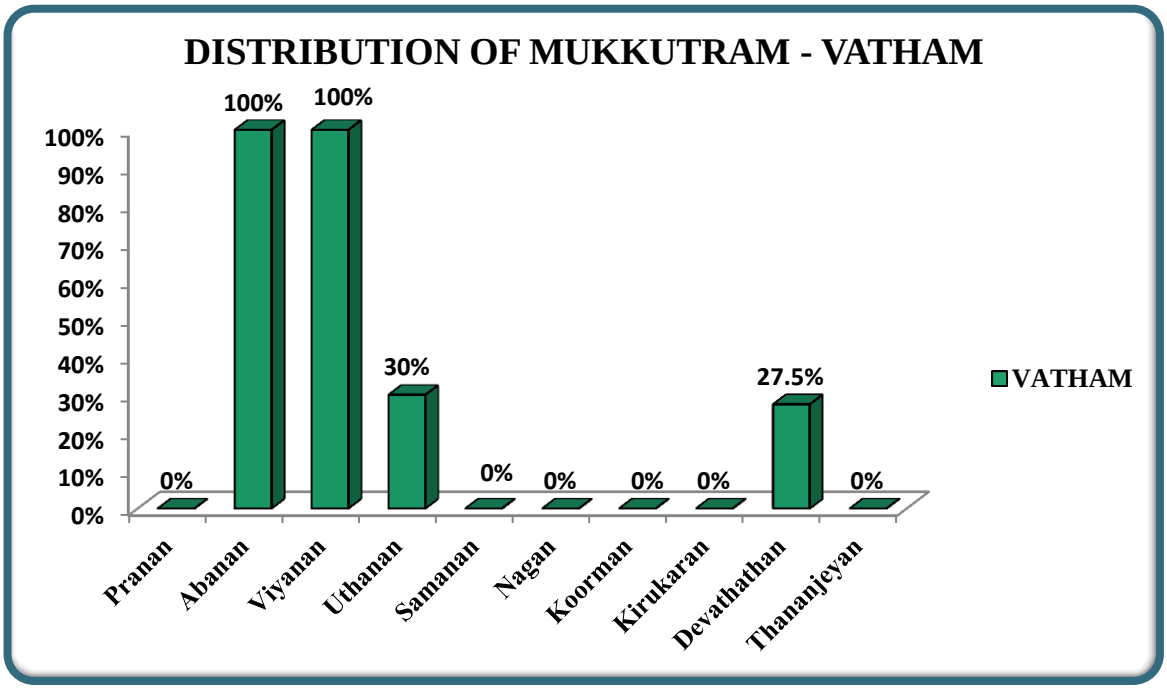


INFERENCE

According to thinai the highest distribution 90 % was noted in neithal, 10% in marutham .

DISTRIBUTION OF MUKKUTRAM – VATHAM

S.No	VATHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Pranan	0	0%
2	Abanan	40	100%
3	Viyanan	40	100%
4	Uthanan	12	30%
5	Samanan	0	0%
6	Nagan	0	0%
7	Koorman	0	0%
8	Kirukaran	0	0%
9	Devathathan	11	27.5%
10	Thananjeyan	0	0%

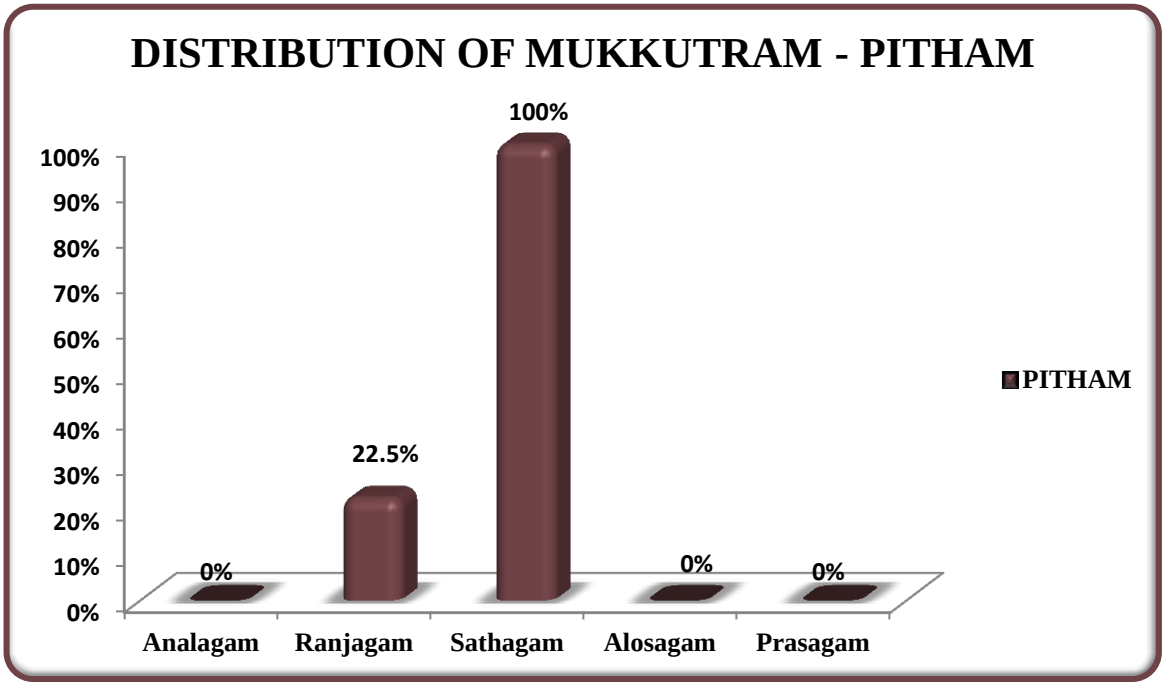


INFERENCE

Out of 40 patients Abanan was affected in 40 patients (100%), Viyanan was affected in 40 pateints (100%), Uthanan was affected in 12 patients (30%), and Devathathan was affected in 11 patients(27.5).

DISTRIBUTION OF MUKKUTRAM – PITHAM

S.No	PITHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Analagam	0	0%
2	Ranjagam	9	22.5%
3	Saathagam	40	100%
4	Alosagam	0	0%
5	Pirasagam	0	0%

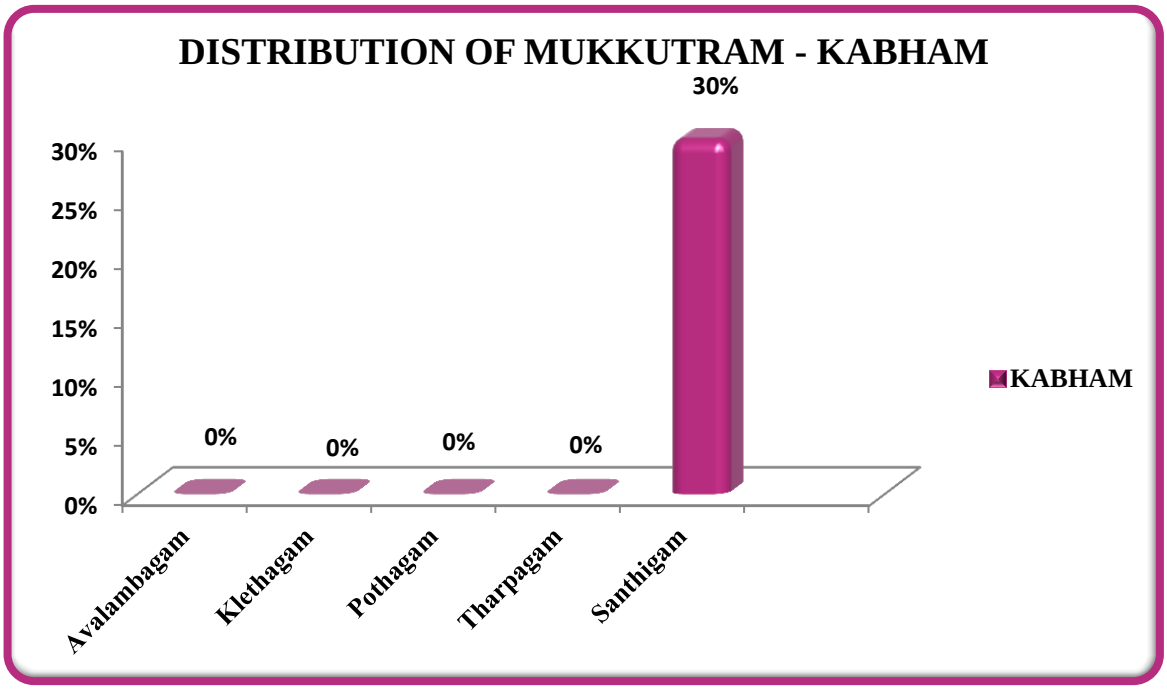


INFERENCE

Out of 40 patients Ranjagam was affected in 9 patients (22.5%),Sathagam was affected in 40 patients (100%).

DISTRIBUTION OF MUKKUTRAM – KABHAM

S.No	KABHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Avalambagam	0	0%
2	Klethagam	0	0%
3	Pothagam	0	0%
4	Tharpagam	0	0%
5	Santhigam	12	30%

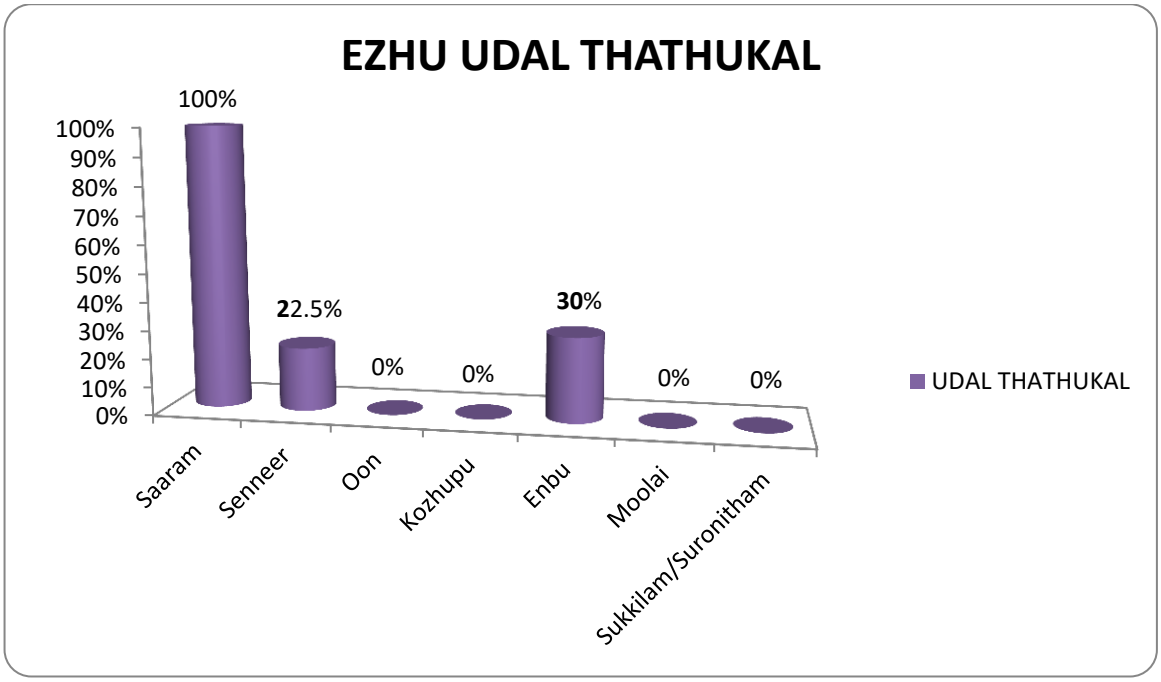


INFERENCE

Out of 40 patients, Santhigam was affected in12 patients (30%).

EZHU UDAL THATHUKAL

S.No	EZHU UDAL THATHUKAL	NUMBER OF CASES	PERCENTAGE (%)
1	Saaram	40	100%
2	Senneer	9	22.5%
3	Oon	0	0%
4	Kozhupu	0	0%
5	Enbu	12	30%
6	Moolai	0	0%
7	Sukkilam /Suronitham	0	0%

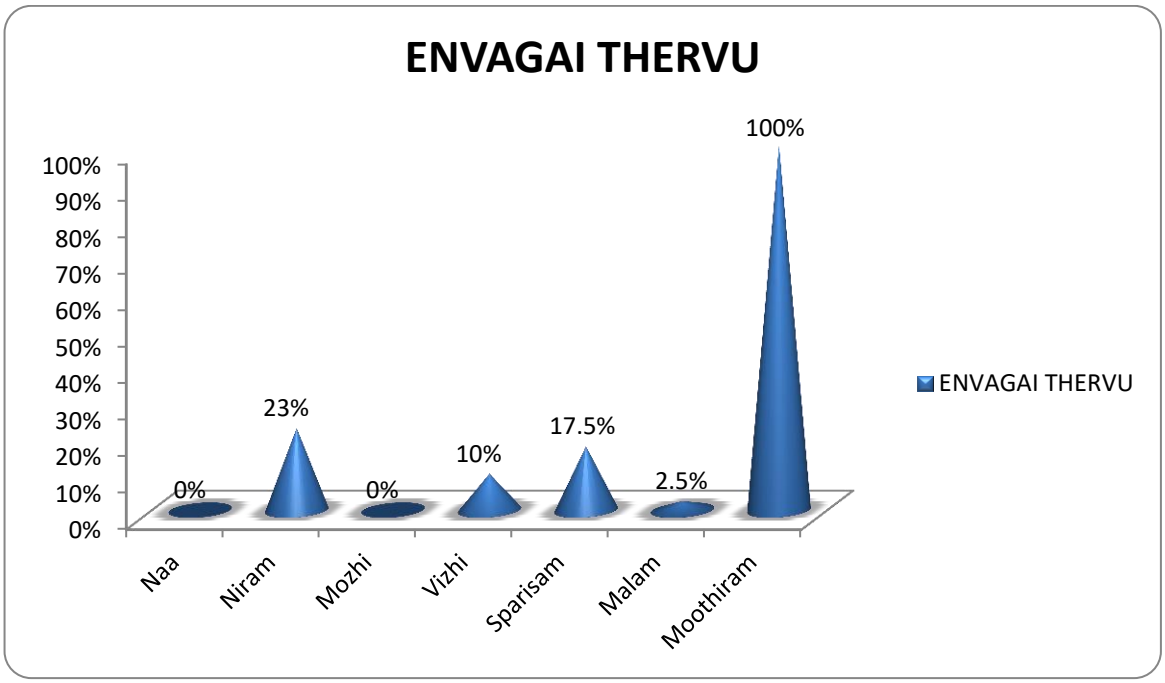


INFERENCE

Out of 40 patients, Saaram was affected in 40 patients (100%), Senneer was affected in 9 patients (22.5%), Enbu was affected in 12 patients (30%).

EN VAGAI THERVUGAL

S.No	EN VAGAI THERVUGAL	NUMBER OF CASES	PERCENTAGE (%)
1	Naa	0	0%
2	Niram	9	22.5%
3	Mozhi	0	0%
4	Vizhi	4	10%
5	Sparisam	7	17.5%
6	Malam	1	2.5%
7	Moothiram	40	100%

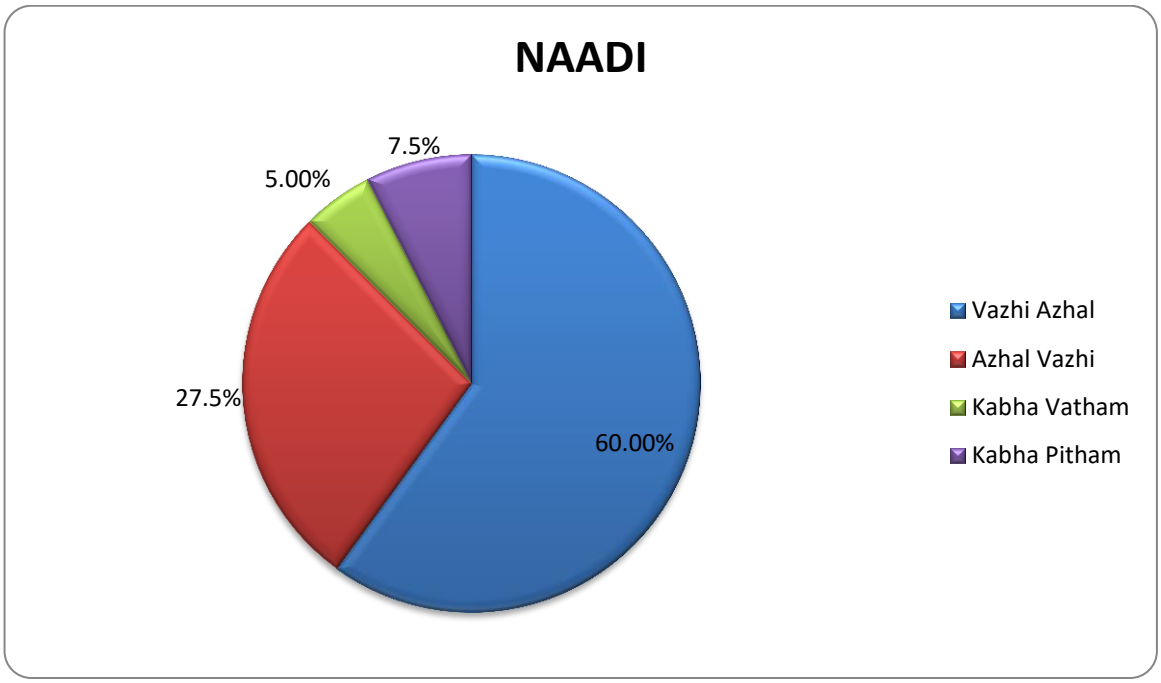


INFERENCE

In Envagai thervu , Niram was affected in 9 patients (22.5), Vizhi was affected in 4 patients (10%), Sparisam was affected in 7 patients (17.5%), Malam was affected in 1 patient (2.5%) and Moothiram was affected in 40 patients (100%).

NAADI

S.No	NAADI	NUMBER OF CASES	PERCENTAGE (%)
1	Vali Azhal	24	60%
2	Azhal Vali	11	27.5%
3	Kabha Vatham	2	5%
4	Kabha Pitham	3	7.50%

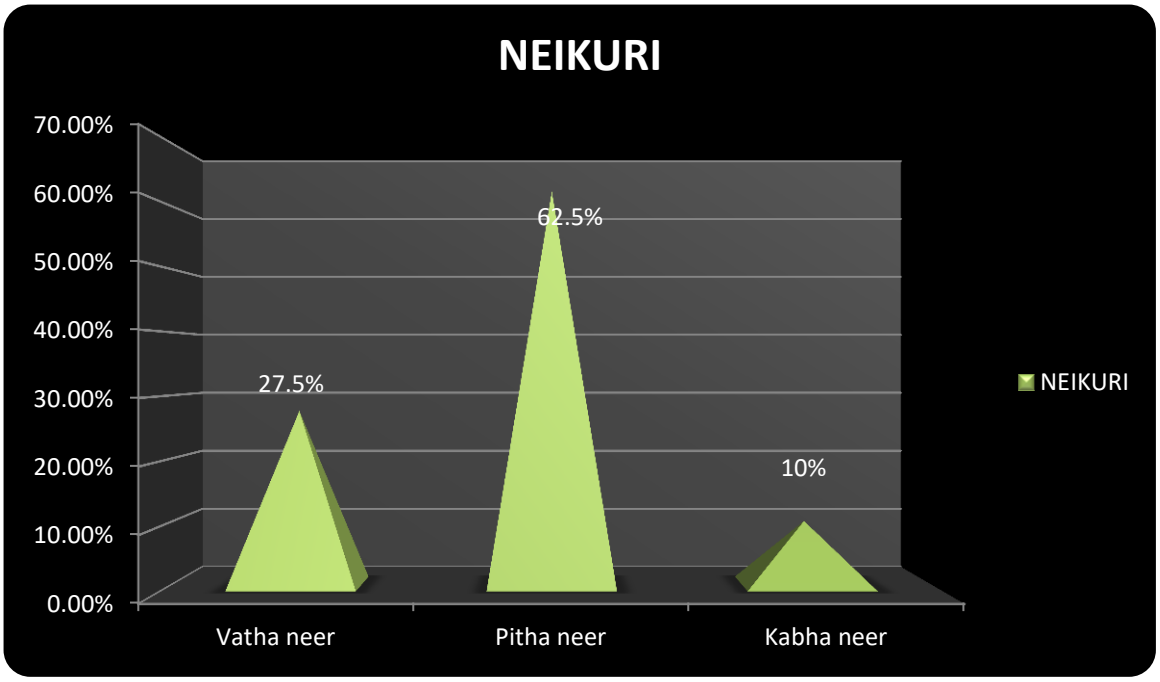


INFERENCE

24 patients (60%) had Vali azhal naadi, 11 patients (27.5%) had Azhal vali naadi, 2 patients (5%) had Kabha vatha naadi and 3 patients (7.5%) had Kabha pitha naadi.

NEIKURI

S.No	THATHU	NEIKURI	NUMBER OF CASES	PERCENTAGE (%)
1	Vatha neer	Spread like snake	11	27.5%
2	Pitha neer	Spread like ring	25	62.5%
3	Kabha neer	Spread like pearl	4	10%

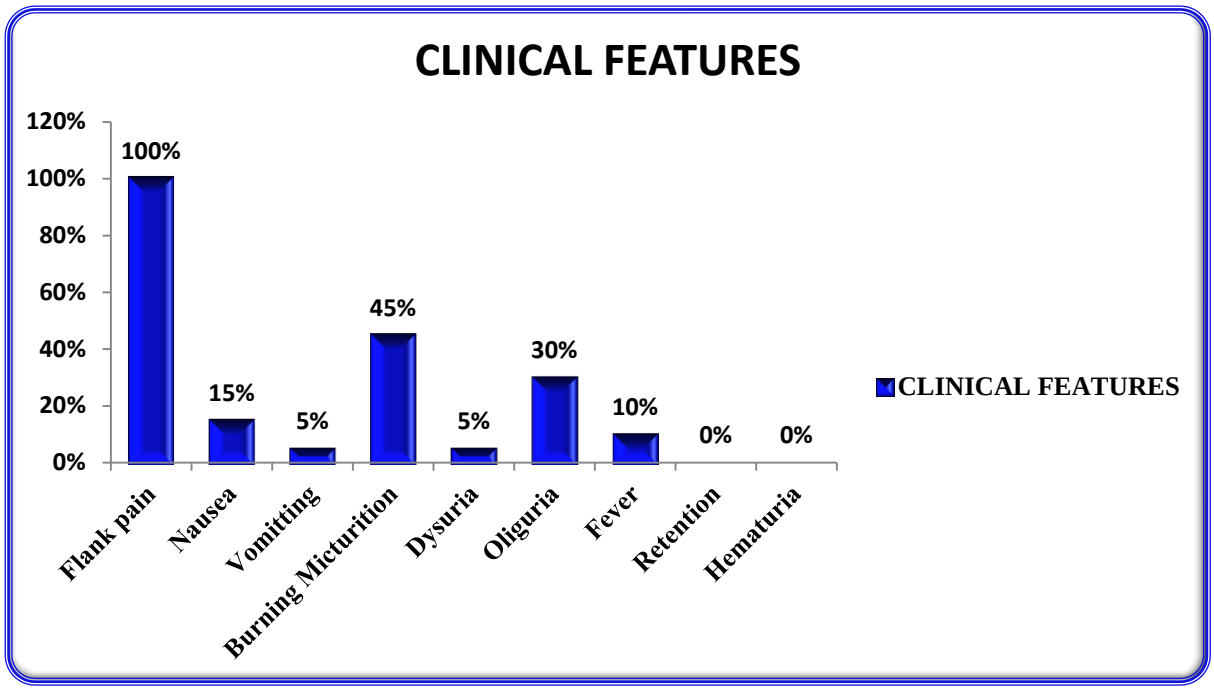


INFERENCE

25 patients (62.5%) had Pitha neer, 11 patients (27.5%) had vatha neer, and 5 patients (10%) had Kabha neer.

CLINICAL FEATURES

S.No	SIGNS & SYMPTOMS	NUMBER OF CASES	PERCENTAGE (%)
1	Flank pain	40	100%
2	Nausea	6	15%
3	Vomitting	2	5%
4	Burning Micturition	18	45%
5	Dysuria	2	5%
6	Oliguria	12	30%
7	Fever	4	10%
8	Retention	0	0%
9	Hematuria	0	0%



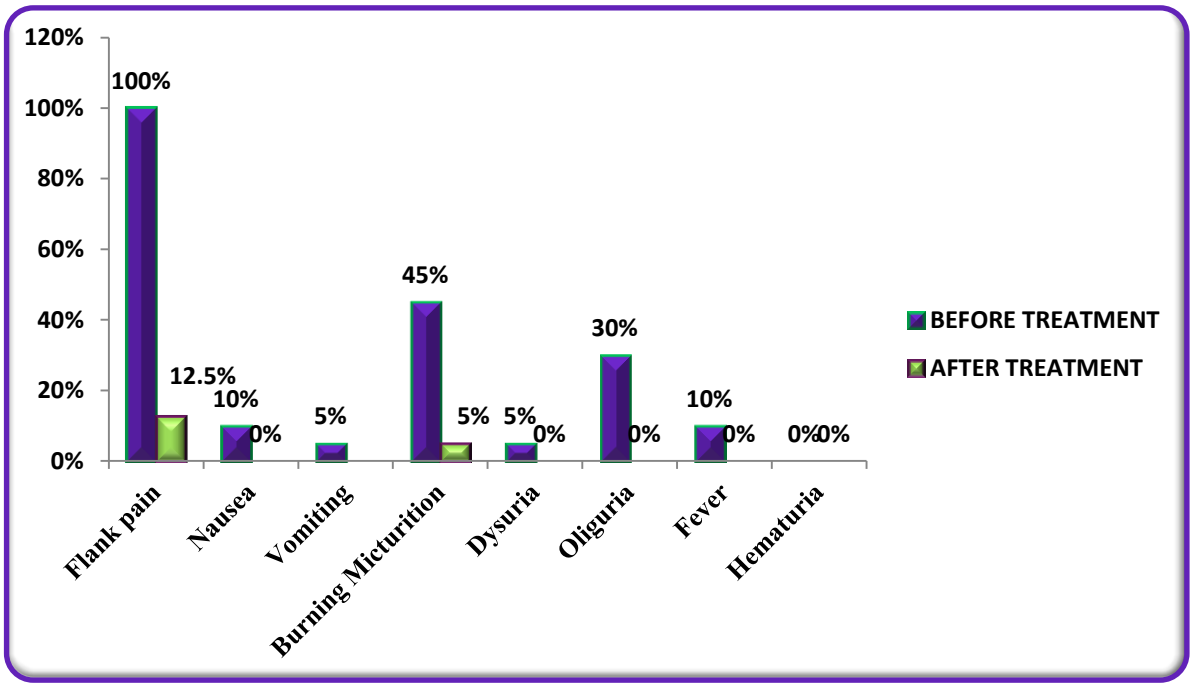
INFERENCE

Out of 40 patients, 40 patients (100%) had Flank pain, 6 patients (10%) had Nausea, 2 patients (5%) had vomiting ,18 patients (45%) had Burning Micturition, 2 patients (5%) had Dysuria, 12 patients (30) had Oliguria, 4 patients (10%) had Fever, and No patients had Hematuria.

CLINICAL PROGNOSIS

S.No	SIGNS& SYMPTOMS	BEFORE TREATMENT		AFTER TREATMENT	
		NO.OF CASES	PERCENTAGE (%)	NO.OF CASES	PERCENTAGE (%)
1	Flank pain	40	100%	5	12.5%
2	Nausea	6	15%	0	0%
3	Vomiting	2	5%	0	0%
3	Burning Micturition	18	45%	2	5%
4	Dysuria	2	5%	0	0%
5	Fever	4	10%	0	0%
6	Oliguria	12	30%	0	0%

CLINICAL PROGNOSIS



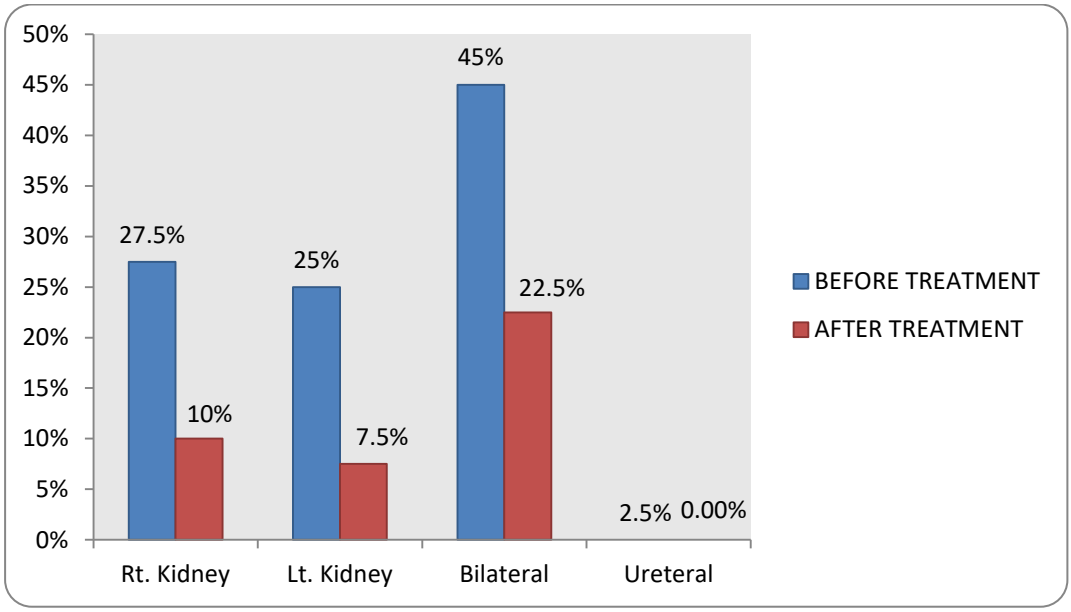
INFERENCE

After treatment Flank pain present in 5 patients (12.5%), Burning Micturition present in 2 patients (5%). No patients have experienced Nausea, Vomiting, Dysuria, Fever and Hematuria

DISTRIBUTION OF CALCULI BASED ON LOCATION

SIDE	BEFORE TREATMENT		AFTER TREATMENT	
	NO.OF	PERCENTAGE	NO.OF	PERCENTAGE
Rt – Kidney	11	27.5%	4	10%
Lt – Kidney	10	25%	3	7.5%
Bilateral	18	45%	9	22.5%
Ureteral	1	2.5%	0	0%

DISTRIBUTION OF CALCULI BASED ON LOCATION



INFERENCE

Before treatment:

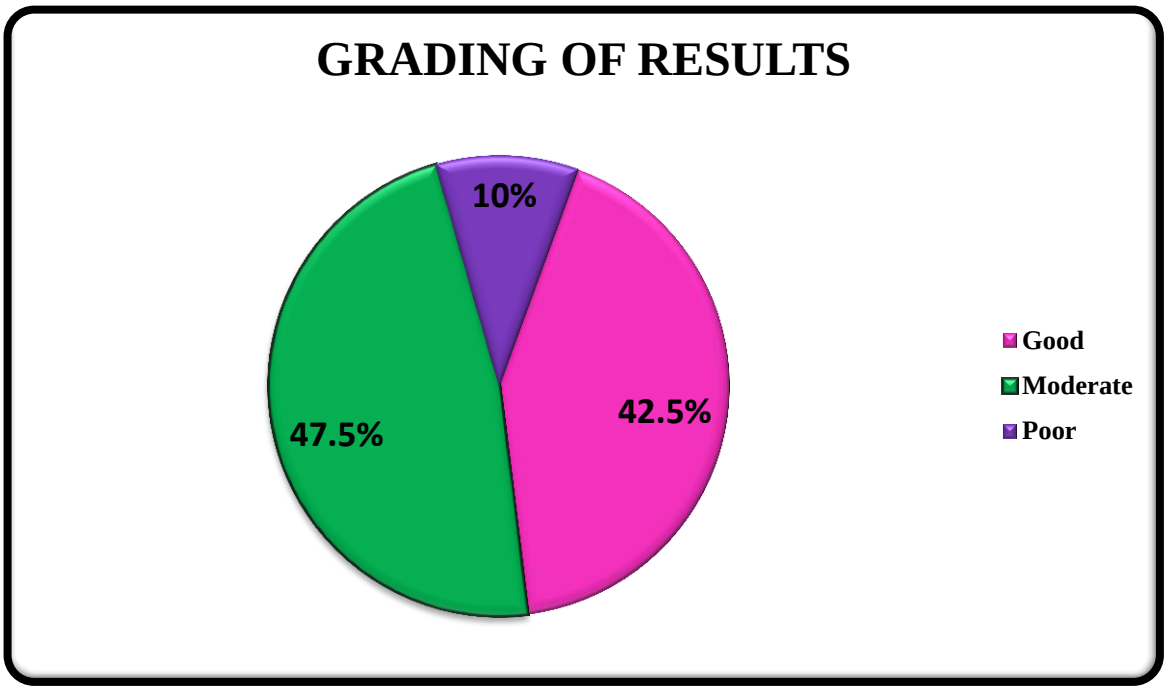
Out of 40 patients, 18 patients (45%) were having bilateral renal calculi, 11 patients 27%) were having right renal calculi, and 10 patients (25%) were having left renal calculi,1 patient (2.5%) having Ureteral calculi.

After treatment:

Out of 40 patients, 9 patients (22.5%) were having bilateral renal calculi, 4 patients (10%) having right renal calculi and 3 patients (7.5%) having left renal calculi.

GRADING OF RESULTS

S.No	GRADING	NUMBER OF CASES	PERCENTAGE (%)
1	Good	17	42.5%
2	Moderate	19	47.5%
3	Poor	4	10%



INFERENCE

Out of 40 patients, 17 cases (42.5%) shows good result, 19 cases (47.5%) shows moderate result, 4 cases (10%) shows poor result.

LIST OF PATIENTS

S.no	Op. no	Patient's name	Age/Sex	Occupation	Date of Medicine
1	6126	Mr.kumar	35/M	IT Profession	20-05-16
2	6238	Mrs.Rajeswari	36/F	House wife	22-05-16
3	6243	Mr. Ilango	37/M	IT Profession	22-05-16
4	2516	Mrs.Lalitha	31/F	House wife	25-05-16
5	5986	Mr.saravanan	32/M	Watchman	27-05-16
6	2093	Mr. karuppaiya	40/M	Clerk	29-05-16
7	8914	Mr.venkat	27/M	Student	31-05-16
8	8923	Mr.saravanan	42/M	Business	04-06-16
9	7981	Mr.chandran	46/M	Bank cashier	17-06-16
10	8123	Mrs.Mahalakshmi	40/F	House wife	18-06-16
11	6823	Mr .John	42//M	Electrician	13-06-16
12	8123	Mrs.suganya	36/F	House wife	19-06-16
13	7289	Mr.jabhar	31/M	Farmer	20-06-16
14	8855	Mr.mahalingam	40/M	Painter	27-06-16
15	9900	Mrs.vimala	37/F	House wife	05-07-16
16	8110	Mr. mahendran	33/M	Mechanic	13-07-16
17	7263	Mr.sundar	38/M	Painter	21-07-16
18	7464	Mr. karuppasamy	35/M	Office work	30-07-16
19	7632	Mrs , kokila	35/F	House wife	01-08-16
20	7704	Mrs.Rani	55/F	House wife	01-08-16
21	8002	Mr .manikandan	36/M	Camera service	03-08-16
22	8223	Mr.tamilarasan	38/M	Electrician	07-08-16
23	8863	Mrs .jayabharathi	55/F	House wife	10-08-16
24	1452	Mr.karthikeyan	34/M	Business	19-09-16
25	4084	Mrs. Dhanalakshmi	42/F	House wife	28-09-16
26	6318	Mrs.Geethapandian	44/F	House wife	06-10-16
27	7954	Mrs. Lalitha	48/F	House wife	13-10-16
28	2669	Mrs. Lalitha	31/F	Office work	01-11-16

29	629	Mr.mankandan	42/M	IT profession	30-11-16
30	1613	Mr .arunkumar	31/M	Business	05-12-16
31	2109	Mrs.valarmathi	28/F	House wife	07-12-16
32	1947	Mr. sreeramalu	48/M	Farmer	07-12-16
33	3451	Mrs. Gowri	50/F	House wife	15-12-16
34	8163	Mr.gaanam	24/M	Student	26-12-16
35	6430	Mr. Sathishbabu	37/M	Auto driver	27-12-16
36	8298	Mr.kumar	53/M	Coolie	03-01-17
37	5597	Mr.venkatesan	58/M	Hostel warden	02-02-17
38	6782	Mrs.tamilkodi	57/F	Business	07-02-17
39	7074	Mrs.valli	52/F	Clerk	08-02-17
40	7337	Mrs. Usha	58/F	House wife	09-02-16

RESULTS OF PATIENTS BEFORE AND AFTER TREATMENT
GOVT.SIDDHA MEDICAL COLLEGE
ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE, CHENNAI

S.No	Name of the patient Age / Sex		OP.No	Date of Treatment Started	Duration of medicine taken	Size of stone BT	Prognosis	Remarks
1	Mr.kumar	35/M	6126	20-05-16	7- Weeks	LT 7-8 mm	USG Normal	Completed
2	Mrs.Rajeswari	36/F	6238	22-05-16	7- Weeks	Bilateral 5mm,4mm	USG Normal	Completed
3	Mr. Ilango	37/M	6243	22-05-16	7- Weeks	Bilateral RT 5 Stones (4mm, 5mm), LT 7 (4mm)	Bilateral RT 3 Stones (4mm, 5mm), LT(4.5mm)	Referred to urologist
4	Mrs.Lalitha	31/F	2516	25-05-16	7- Weeks	LT 2(8mm, 3mm)	LT (6mm)	Symptoms reduced
5	Mr .Saravanan	32/M	5986	27-05-16	7- Weeks	RT 3 (3.5 mm)	USG Normal	Completed
6	Mr. karuppaiya	40/M	2093	29-05-16	5- Weeks	Bilateral RT 2 (8mm, 4mm) LT 3 (8mm, 6mm, 3mm)	One stone expelled, RT 5mm, LT 3 (4mm,3mm)	Advice to continue medicine
7	Mr.karthikeyan	34/M	1452	31-05-16	7- Weeks	Bilateral RT 5mm, LT 7.mm	LT 3.5mm	Symptoms relieved
8	Mr.saravanan	42/M	8923	04-06-16	7- Weeks	LT 3 (4.5mm, 4mm, 3.5mm)	LT 3mm	Symptoms relieved
9	Mr.chandran	46/M	7981	17-06-16	7- Weeks	Bilateral RT 3 (4mm), LT 2 (3.5mm, 3mm)	Bilateral RT2 (4mm),LT 2(3.5mm)	Poor prognosis
10	Mrs.Mahalakshmi	40/F	8123	18-06-16	7- Weeks	Bilateral RT 5mm , LT 8mm,9mm	RT (6mm)	Symptoms reduced

11	Mr .John	42//M	6823	13-06-16	7- Weeks	Bilateral LT 12mm, RT 2 (3mm)	LT 12mm, RT 3mm	Poor prognosis
12	Mrs.suganya	36/F	8123	19-06-16	7- Weeks	Bilateral RT 3(4, 4.5,3mm), LT 4 (3mm)	RT 2 (4, 3.5mm), LT 3 (3mm)	Symptoms relieved
13	Mr.jaffar	31/M	7289	20-06-16	7- Weeks	RT 5.3mm,	USG Normal	Completed
14	Mr.mahalingam	40/M	8855	27-06-16	7- Weeks	Bilateral RT 5 (4mm), LT 3.5mm	RT 2(3,4mm)	Symptoms relieved
15	Mrs.vimala	37/F	9900	05-07-16	7- Weeks	LT 2(4mm)	USG Normal	Completed
16	Mr. mahendran	33/M	8110	13-07-16	7- Weeks	RT 3 (4.5mm)	USG Normal	Completed
17	Mr.sundar	38/M	7263	21-07-16	7- Weeks	LT 3 (3.5,4mm)	LT 4mm	Symptoms reduced
18	Mr. karuppasamy	35/M	7464	30-07-16	7- Weeks	Bilateral RT 5 (4.5, 3.5mm), LT 6 (3.5mm)	RT 2 (3mm), LT 3 (3.5mm)	Symptoms relieved
19	Mrs , kokila	35/F	7632	01-08-16	7- Weeks	Bilateral RT 4 (6, 5, 4.5, 4mm), LT 2 (8, 6mm)	RT 5(4, 5mm), LT 2 (7, 4mm)	Symptoms reduced
20	Mrs.Rani	55/F	7704	01-08-16	7- Weeks	RT 5 (4.5mm)	USG Normal	Completed
21	Mr .manikandan	36/M	8002	03-08-16	7- Weeks	RT 4 (5, 3.5mm)	RT 2 (5, 3.5mm)	Symptoms relieved
22	Mr.tamilarasan	38/M	8223	07-08-16	7- Weeks	LT 3 (3mm)	USG Normal	Completed
23	Mrs .jayabharathi	55/F	8863	10-08-16	7- Weeks	RT 4 (3.5mm)	RT 2 (4mm)	Symptoms relieved
24	Mr.venkat	27/M	8914	19-09-16	7- Weeks	Bilateral RT 5mm, LT 7.5mm	LT 3mm	Symptoms relieved
25	Mrs. Dhanalakshmi	42/F	4084	28-09-16	7- Weeks	RT 4 (3.5, 4mm),	USG Normal	Completed

26	Mrs.Geethapandian	44/F	6318	06-10-16	7- Weeks	LT 4mm	USG Normal	Completed
27	Mrs. Lalitha	48/F	7954	13-10-16	7- Weeks	RT 3 (3.5-4mm)	RT 2 (3mm),	Symptoms relieved
28	Mrs. Lalitha	31/F	2669	01-11-16	7- Weeks	RT 3 (3-4mm)	USG Normal	Completed
29	Mr.mankandan	42/M	629	30-11-16	7- Weeks	Bilateral RT 3 (3.5, 4mm), LT 4(3mm)	USG Normal	Completed
30	Mr .arunkumar	31/M	1613	05-12-16	7- Weeks	Bilateral RT 5 (3.5mm), LT 6 (3mm)	RT 5(3mm), LT 4 (3mm)	Symptoms reduced
31	Mrs.valarmathi	28/F	2109	07-12-16	7- Weeks	Bilateral RT 4mm,4mm , LT 4 mm	USG Normal	Completed
32	Mr. sreeramalu	48/M	1947	07-12-16	7- Weeks	Upper ureteric calculus 1.1cm	USG Normal	Completed
33	Mrs. Gowri	50/F	3451	15-12-16	7- Weeks	LT 2 (3mm)	USG Normal	Completed
34	Mr.gaanam	24/M	8163	26-12-16	7- Weeks	LT VUJN 6mm,RT 5mm	LT 4mm	Symptoms reduced
35	Mr. Sathishbabu	37/M	6430	27-12-16	7- Weeks	RT 6mm	USG Normal	Completed
36	Mr.kumar	53/M	8298	03-01-17	8- Weeks	LT 5mm	USG Normal	Completed
37	Mr.venkatesan	58/M	5597	02-02-17	7- Weeks	RT 10mm	RT (10mm)	Poor prognosis
38	Mrs.tamilkodi	57/F	6782	07-02-17	7- Weeks	Bilateral RT 2(6mm,9mm),LT 2(3mm,4mm)	RT 4mm,5mm	Advice to continue medicine
39	Mrs.valli	52/F	7074	08-02-17	7- Weeks	Bilateral 5 (4, 3.5mm)	Bilateral 2 (3.5mm)	Symptoms reduced
40	Mrs. Usha	58/F	7337	09-02-17	7- Weeks	LT 2 (3-4mm)	USG Normal	Completed

LABORATORY INVESTIGATION REPORT

BEFORE TREATMENT

S.No	OP. No	Name	Age/Sex	Haematological report							RFT		Urine Analysis		
				TC cells/ cu.mm	DC %			ESR		Hb gms %	Urea mg/ dl	Creatinine mg/dl	Alb	Sug	Dep
					P	L	E	½ hr	1 hr						
1	6126	Mr.kumar	35/M	8500	63	34	3	22	59	13.0	32	1.3	Nil	++	Opc
2	6238	Mrs.Rajeswari	36/F	9100	64	34	2	4	19	7.8	34	0.8	Nil	Nil	Opc
3	6243	Mr. Ilango	37/M	8100	58	39	3	23	57	12.6	23	0.8	+	Nil	Nil
4	2516	Mrs.Lalitha	31/F	8500	63	34	3	18	43	10.7	24	1.0	Nil	Nil	Oec
5	5986	Mr.saravann	32/M	9000	58	36	6	16	37	14.9	27	0.8	Nil	Nil	Nil
6	2093	Mr. karuppaiya	40/M	8700	60	36	4	2	9	8.9	37	1.0	Nil	Nil	Opc
7	8914	Mr.venkat	27/M	9200	56	39	5	28	54	14.2	34	1.3	Nil	Nil	Opc
8	8923	Mr.saravanan	42/M	9000	58	40	2	13	43	12.9	30	0.8	Nil	Nil	Opc
9	7981	Mr.chandran	46/M	7900	56	42	2	6	21	14.9	35	0.9	Nil	+	Oec
10	8123	Mrs.priya	38/F	6300	63	32	5	16	29	15	26	0.7	Nil	Nil	Opc
11	6823	Mr .John	42//M	8700	62	35	3	12	28	8.5	25	0.7	Nil	Nil	Nil
12	8123	Mrs.suganya	36/F	9700	55	42	3	7	15	13	25	0.9	Nil	Nil	Nil
13	7289	Mr.jabhar	31/M	9500	62	34	4	2	5	10.8	27	0.7	Nil	Nil	Nil
14	8855	Mr.mahalingam	40/M	8900	61	36	3	5	12	9.8	26	0.7	Nil	Nil	Nil
15	9900	Mrs.vimala	37/F	8400	52	44	4	15	20	10.1	25	0.8	Nil	Nil	Nil
16	8110	Mr. mahendran	33/M	8500	60	37	3	4	7	13.2	20	0.7	Nil	Nil	Nil
17	7263	Mr.sundar	38/M	8900	55	42	3	9	15	13	24	0.9	Nil	Nil	Oec
18	7464	Mr. karuppasamy	35/M	8800	61	34	5	8	12	11.2	25	0.8	Nil	Nil	Nil
19	7632	Mrs , kokila	35/F	8400	60	37	3	8	16	13	25	0.8	Nil	Nil	Opc
20	7704	Mrs.Rani	55/F	9200	59	37	4	5	10	13	30	0.9	Nil	Nil	Nil

21	8002	Mr .manikandan	36/M	8900	58	39	3	8	12	13.5	31	1.1	Nil	Nil	Opc
22	8223	Mr.tamilarasan	38/M	8900	61	36	3	5	9	12.8	24	0.7	Nil	Nil	Nil
23	8863	Mrs .jayabharathi	55/F	9400	54	42	4	2	6	12.1	22	0.8	Nil	Nil	Nil
24	1452	Mr.karthikeyan	34/M	8700	60	37	3	6	15	10.8	26	1.1	Nil	Nil	Nil
25	4084	Mrs. Dhanalakshmi	42/F	10300	61	36	3	5	12	14.2	28	0.7	Nil	Nil	Nil
26	6318	Mrs.Geethapandian	44/F	8300	58	39	3	5	13	13.2	22	0.8	Nil	Nil	Nil
27	7954	Mrs. Lalitha	48/F	8600	59	37	4	7	16	11.2	24	0.8	Nil	Nil	Nil
28	2669	Mrs. Lalitha	31/F	9700	56	40	4	2	4	12.5	28	0.9	Nil	Nil	Nil
29	629	Mr.mankandan	42/M	8000	58	39	3	4	8	13.1	25	0.7	Nil	Nil	Nil
30	1613	Mr .arunkumar	31/M	9700	56	41	3	5	10	12	26	0.7	Nil	Nil	Nil
31	2109	Mrs.valarmathi	28/F	9700	63	33	4	5	10	11.6	29	0.8	Nil	Nil	Nil
32	1947	Mr. kuppusamy	48/M	8400	50	46	4	4	8	10.9	24	0.9	Nil	++	Nil
33	3451	Mrs. Gowri	50/F	8200	59	38	3	6	15	14.6	24	0.7	Nil	Nil	Nil
34	8163	Mrs .gaanam	24/M	8500	62	35	3	10	20	14	26	0.8	Nil	Nil	Nil
35	6430	Mr. Sathishbabu	37/M	8300	62	34	4	5	10	10.6	26	0.7	Nil	Nil	Nil
36	8298	Mr.kumar	53/M	9600	55	42	3	3	7	10.6	30	0.9	Nil	Nil	Nil
37	5597	Mr.venkatesan	58/M	8700	61	35	4	10	25	11.8	26	0.7	Nil	Nil	Nil
38	6782	Mrs.tamilkodi	57/F	8500	65	32	3	2	5	11.2	26	0.7	Nil	Nil	Nil
39	7074	Mrs.valli	52/F	7700	63	31	6	5	16	9.4	25	0.8	Nil	Nil	Nil
40	7337	Mrs. Usha	58/F	8100	49	46	5	12	30	14.8	23	0.7	Nil	Nil	Nil

TC – Total count, DC – Differential count, P – Polymorphs, L – Lymphocyte, E – Eosinophil, ESR – Erythrocyte Sedimentation Rate, Oec – Occasional epithelial cells, Opc – Occasional pus cells, Alb – Albumin, Sug – Sugar, Dep – Deposits

LABORATORY INVESTIGATION REPORT

AFTER TREATMENT

S.No	OP. No	Name	Age/Sex	Haematological report							RFT		Urine Analysis		
				TC cells/ cu.mm	DC %			ESR mm		Hb gms %	Urea mg/dl	Creatinine mg/dl	Alb	Sug	Dep
					P	L	E	½ hr	1 hr						
1	6126	Mr.kumar	35/M	9400	57	39	4	7	13	11	32	0.6	Nil	Nil	Opc
2	6238	Mrs.Rajeswari	36/F	8400	54	42	4	6	15	10.2	31	1.0	Nil	Nil	Oec
3	6243	Mr. Ilango	37/M	8000	57	39	4	5	15	12.6	34	0.9	Nil	Nil	Opc
4	2516	Mrs.Lalitha	31/F	9300	52	45	3	12	25	11	29	1.0	+	Nil	Opc
5	5986	Mr.Saravanan	32/M	8900	60	37	3	7	12	10.2	29	0.8	Nil	Nil	Oec
6	2093	Mr. karuppaiya	40/M	9200	57	41	2	10	27	9.6	31	0.6	Nil	Nil	Nil
7	8914	Mr.venkat	27/M	9200	60	36	4	10	26	11.9	29	0.9	Nil	Nil	Nil
8	8923	Mr.saravanan	42/M	8800	61	35	4	6	15	10.2	24	1.0	Nil	Nil	Nil
9	7981	Mr.chandran	46/M	8400	53	41	6	15	26	13.4	26	0.5	Nil	Nil	Opc
10	8123	Mrs.Mahalakshmi	38/F	8900	51	43	6	17	30	12.8	28	0.7	Nil	Nil	Opc
11	6823	Mr .John	42//M	8500	62	35	3	12	28	8.5	20	0.8	Nil	Nil	Opc
12	8123	Mrs.suganya	36/F	9400	55	42	3	7	15	13	23	1.1	Nil	Nil	Oec
13	7289	Mr.jabhar	31/M	9200	63	33	4	2	5	10.8	26	0.7	Nil	Nil	Oec
14	8855	Mr.mahalingam	40/M	8500	58	37	5	5	12	9.6	29	0.7	Nil	Nil	Nil
15	9900	Mrs.vimala	37/F	8700	49	46	5	22	40	10.2	28	0.9	Nil	Nil	Opc
16	8110	Mr. mahendran	33/M	8000	58	39	3	4	10	13	21	0.7	Nil	Nil	Oec
17	7263	Mr.sundar	38/M	8200	53	44	3	14	25	13	24	0.9	Nil	Nil	Oec
18	7464	Mr. karuppasamy	35/M	8900	61	34	5	8	15	11.2	26	0.8	Nil	Nil	Oec
19	7632	Mrs , kokila	35/F	8000	61	37	2	10	22	13	27	0.9	Nil	Nil	Opc
20	7704	Mrs.Rani	55/F	9000	57	39	4	5	11	13	32	1.0	Nil	Nil	Opc

21	8002	Mr .manikandan	36/M	8600	58	39	3	10	15	13.6	36	1.1	Nil	Nil	Opc
22	8223	Mr.tamilarasan	38/M	8700	62	35	3	6	12	12.8	24	0.8	Nil	Nil	Oec
23	8863	Mrs .jayabharathi	55/F	9000	54	42	4	2	8	12	24	0.9	Nil	Nil	Nil
24	1452	Mr.karthikeyan	34/M	8200	59	39	2	6	15	10.8	25	1.1	Nil	Nil	Opc
25	4084	Mrs.Dhanalakshmi	42/F	10000	57	38	5	5	12	14.2	28	0.6	Nil	Nil	Nil
26	6318	MrsGeethapandian	44/F	8600	65	32	3	2	5	13.4	22	0.7	Nil	Nil	Nil
27	7954	Mrs. Lalitha	48/F	8700	57	40	3	10	20	11.2	21	0.6	Nil	Nil	Nil
28	2669	Mrs. Lalitha	31/F	9400	55	41	4	2	4	12.5	28	0.9	Nil	Nil	Opc
29	629	Mr.mankandan	42/M	7600	55	42	3	3	7	13	25	0.7	Nil	+	Oec
30	1613	Mr .arunkumar	31/M	9700	57	38	5	5	12	12	27	0.7	Nil	Nil	Oec
31	2109	Mrs.valarmathi	28/F	8200	50	47	3	2	7	10.8	26	1.0	Nil	+++	Opc
32	1947	Mr. sreeramalu	48/M	8200	57	40	3	7	15	14.6	24	0.7	Nil	Nil	Oec
33	3451	Mrs. Gowri	50/F	8300	60	37	3	15	35	14	28	0.9	+	Nil	Opc
34	8163	Mr.gaanam	24/M	9900	71	25	4	5	12	13.7	-	-	Nil	Nil	Opc
35	6430	Mr. Sathishbabu	37/M	9600	55	42	3	3	7	10.6	30	0.9	Nil	Nil	Oec
36	8298	Mr.kumar	53/M	8600	61	35	4	10	25	11.8	25	0.6	Nil	Nil	Nil
37	5597	Mr.venkatesan	58/M	8600	65	32	3	2	5	11.2	28	0.7	Nil	Nil	Nil
38	6782	Mrs.tamilkodi	57/F	8400	66	29	5	22	40	12.2	-	-	Nil	Nil	Nil
39	7074	Mrs.valli	52/F	7900	49	46	5	12	30	14.8	21	0.7	Nil	Nil	Opc
40	7337	Mrs. Usha	58/F	9600	59	37	4	5	15	11.6	30	0.8	Nil	Nil	Opc

TC – Total count, DC – Differential count, P – Polymorphs, L – Lymphocyte, E – Eosinophil, ESR – Erythrocyte Sedimentation Rate, Oec – Occasional epithelial cells, Opc – Occasional pus cells, Alb – Albumin, Sug – Sugar, Dep – Deposit

Karuppaiya /40yrs /M/ 2093

BEFORE TREATMENT

MEDALL

Name	**MR.VENKATRAMAN B**	**ID**	**KLP79816**
Age & Gender	**27Y/MALE**	**Visit Date**	**08/08/2016**
Ref Doctor	**C/O. MERRILL TECHNOLOGY**		

SONOGRAM REPORT

WHOLE ABDOMEN

The liver is normal in size and shows uniform echotexture with no focal abnormality.

The gall bladder is normal sized and smooth walled and contains no calculus.

There is no intra or extra hepatic biliary ductal dilatation.

The pancreas shows a normal configuration and echotexture.

The pancreatic duct is normal.

The portal vein and IVC are normal.

The spleen is normal.

There is no free or loculated peritoneal fluid.

No para aortic lymphadenopathy is seen.

No abnormality is seen in the region of the adrenal glands.

The right kidney measures 10.2 x 4.9 cm.

The left kidney measures 10.3 x 4.8 cm.

Cortical echoes are normal bilaterally.

Name	MR.VENKATRAMAN B	ID	KLP79816
Age & Gender	27Y/MALE	Visit Date	08/08/2016
Ref Doctor	C/O. MERRILL TECHNOLOGY		

A calculus of 5 mm is seen in the interpolar calyx of the right kidney.

A calculus of 7.5 mm is seen in the upper pole calyx of the left kidney.

The ureters are not dilated.

The bladder is smooth walled and uniformly transonic.

There is no intravesical mass or calculus.

The prostate measures 3.2 x 2.6 x 3.6 cm (16.7 cc) and is normal sized.

The echotexture is homogeneous.

The seminal vesicles are normal.

Iliac fossae are normal.

IMPRESSION : = Bilateral renal calculi.

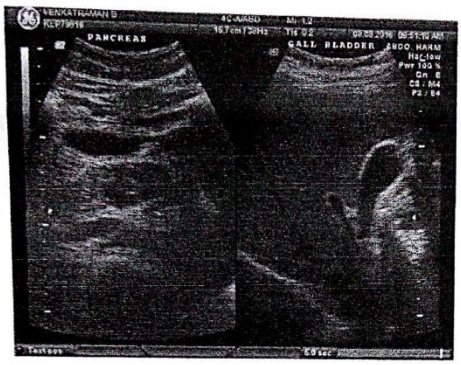
= Other organs are normal.

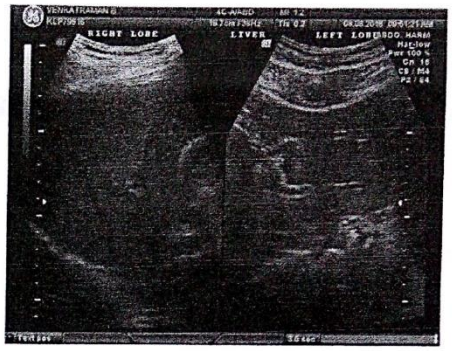
Dr. RAMYA RAMESHWARAN
SONOLOGIST

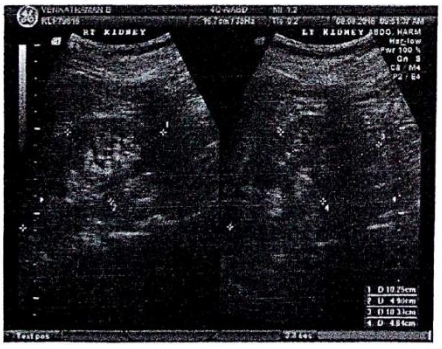
SI

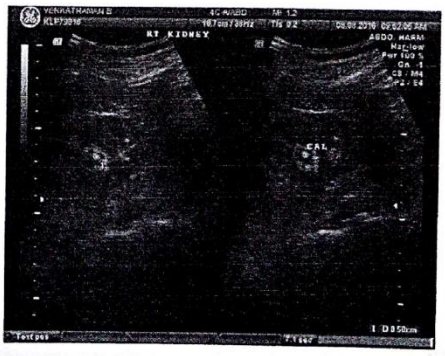
Precision Diagnostics,
Kilpauk

Name	**MR.VENKATRAMAN B**	**ID**	**KLP79816**
Age & Gender	**27Y/MALE**	**Visit Date**	**08/08/2016**
Ref Doctor	**C/O. MERRILL TECHNOLOGY**		









AFTER TREATMENT

SWAMI VIVEKANANDA DIAGNOSTIC CENTRE

Lions Edifice for Trust Complex, Inside D.G.Vaishunav College
Chennai-600106.PH:044-236307521 / 23637604
Registration No.(Under PC & PNDT Act , 1994) PNA 860/2001

Patient name	MR.VENKATRAMAN	Age/Sex	27 Years/ Male
Patient ID	18_10_2016_12_04_03	Visit No	1
Referred by	DR.SARATHKUMAR	Visit Date	18/10/2016

Abdomen and KUB Scan Report

Real time B-mode Ultrasonography of Abdomen and KUB done

Abdomen

Liver filled with homogeneous parenchymal echoes.No abscess or mass lesion in the liver
Gall bladder appeared normal.no calculi seen in gall ballader
Commonduct appeared normal.no calculi seen in commonduct
Pancreas appeared normal
Spleen measured 10.4cms
Spleen appeared normal
Aorta appeared normal. No para aortic nodes seen
Peritoneal cavity appeared normal

KUB

Right kidney measured 9.6 X 5.0 cms
Cortex and collecting system of the right kidney appeared normal. No calculi seen
Left kidney measured 10.3 X 4.8 cms
Cortex and collecting system of the left kidney appeared normal
Bladder appeared normal
Prostate measured 3.7 X 2.6 X 3.3 cms. (Weight =13.28gms)
Prostate appeared normal. No intra vesical enlargement of prostate glands seen.

Impression

Normal study

DR.R.KANAGASABAI.,MD.,DMRD
CONSULTANT RADIOLOGIST

SWAMI VIVEKANANDA DIAGNOSTIC CENTRE

Lions Edifice for Trust Complex, Inside D.G.Vaishunav College
Chennai-600106.PH:044-236307521 / 23637604
Registration No.(Under PC & PNDT Act , 1994) PNA 860/2001

Patient name	MR.VENKATRAMAN	Age/Sex	27 Years/ Male
Patient ID	18_10_2016_12_04_06	Visit No	1
Referred by	DR.SARATHKUMAR	Visit Date	18/10/2016

DISCUSSION

KALLADAIPPU is a common disease pertaining to the kidney. Large populations are suffering from this disease. But they are not completely relieved from their symptoms by other systems of medicine. Hence with the help of trial medicine from Siddha system, results and observations are noted for this study.

The patients were examined base on Siddha and as well as modern aspects. All the necessary investigations were made during the study. The results obtained from their studies were discussed below for better conclusion.

Trial medicine administered was Karpoora silasathu parpam – 130 mg 2 times a day with Ilaneer after food for 48 days.

40 cases were selected and admitted in the In- patient ward of Arignar Anna Government Hospital of Indian Medicine attached to Government Siddha Medical College, Arumbakkam, Chennai – 106 during the period of 2014 – 2017.All necessary investications were carried out to all patients an

Trail drug were given. Daily follow up were done. All the patients were strictly advised to follow diet restriction and peaceful lifestyle to normalize the immune mechanism.

My trial drug was justified for Kalladaippu through various process Drug Authentication, Trial drug, IAEC, Toxicity study, Pharmacological Activity, Biochemical Analysis, Phsiochemical Analysis, IEC, CTRI, Clinical study, Biostatistic.

DRUG AUTHETICATION:

I have got a drug authentication of minerals like Karpoora silasathu, venkaram from Department of Pharmacology and freshly specimens of *Gossypium herbaceum ,Abutilon indicum* were collected from their nativity and got authentication from the botanist Dept, of Medicinal Botany, Govt. Siddha Medical College, Arumbakkam, Chennai – 106.

PRE CLINICAL SCREENINGS:

PHSIOCHEMICAL ANALYSIS:

Loss on Drying(at 105^0C) was 5.37%, The total ash value of Karpoora silasathu parpam was 73.16%, The water soluble ash value was 32.19% The acid soluble ash value was 10.23%, pH value was 8.56%.

IAEC:

IAEC NO: SU/CLATR/IEAC/VII/050/2016.

TOXICITY STUDY:

ACUTE TOXICITY:

Acute and sub acute toxicity studies were conducted on experimental rats at Sathyabama University, Chennai, Tamilnadu.

Acute toxicity study of the drug Karpoora silasathu parpam with Ilaneer was carried out as the OECD guideline - 423 (Organisation to Econamic Co-operation and Development).

The acute toxicity study of my trial drug was studied and the drug was proved safer for long term administration, as it did not exhibit any significant toxicity at 2000 mg / kg body weight.

SUB ACUTE TOXICITY:

Sub acute toxicity study as per the guideline of – 407. Under the dosage of trial drug 200mg / kg (Low dose), 400mg / kg (High dose) it did not exhibit any significant.

HISTO PATHOLOGY:

At the end of toxicity studies the animal were sacrified and they were subjected to hematological parameters (TC, DC & Hb) chemical parameters (LFT, RFT) and histopathology of vital organs like Liver, Kidney, Spleen, Lungs were

carried out. The studied did not exhibit the evidence of remarkable pathological lesions in the tissues.

PHARMACOLOGICAL ACTIVITY:

The pharmacology studies of trial medicine *Karpoora silasathu parpam* showed significant Anti Urolithiaticc action in wistar rats.

The Anti Urolithiaticc activity of Karpoora silasathu parpam was carried out in wistar rats through Ethylene Glycol-induced urolithiasis method. Then trial drug was administrated shows a potent Anti Urolithiatic activity during the studies.

The result of preclinical screening, the result of chemical analysis, Toxicological studies, Pharmacological studies were shown in anexures.

BIOCHEMICAL ANALYSIS:

Karpoora silasathu parpam contains, Calcium, Potassium, Chlorides, Phosphate.

IEC, CTRI:

Study Design

The study was approved by Institutional Ethics Committee (IEC) and the approval number is **GSMC-CH-ME-4/2015/010**.It was registered in **Clinical Trials Registry – India (CTRI)** and the reference number is **CTRI/2017/04/008435**.

Population and sample :

The population consists of all patients satisfying the inclusion and exclusion criteria mentioned below. Sample consists of KALLADAIPPU patients who were attending the OPD of Arignar Anna Hospital, Arumbakkam, Chennai – 106.

Sample Size :

The trial size will be 40 patients.

CLINICAL STUDY:

All the necessary investigation were carried out to all patients and trial drug were given. Weekly once follow up were done. Total duration of treatment ranges

from 48 days. All the patients were strictly advised to follow diet restriction and peaceful life style to normalize the immune mechanism.

GENDER DISTRIBUTION:

From selected 40 cases of 57.5% were males and 42.5% were females Urolithiasis is most commonly affected in male.

AGE DISTRIBUTION:

Out of 40 cases 21 patients (52.5%) were between 31 – 40 years, 9 patients (22.5%) were between 41 – 50 years, 7 patients (17.5%) were between 51 – 60 years and 3 patients (7.5%) were between 21-30 years.

High incidences of cases were noted in age ranging of 31 – 40 years during the studies. The disease is more common in 3^{rd} and 4^{th} decade.

SEASONAL INCIDENCE:

According to Paruva kaalam highest incidence of 37.5% were noted in Muthuvenir kaalam and 20% cases were noted in Ilavenir kaalam and 17.5% comes under Munpani kaalam , 15% of cases were noted in Koothir kaalam and 10% of cases were noted in Karaalam.

When clinical trial of 40 cases were enquired about the seasonal link, most of the cases were in Muthuvenir Kaalam due to seasonal variation.

OCCUPATIONAL STATUS:

From selected 40 cases, 14 patients (35%) were house wife, 11 patients (27.5%) were office workers,9 patients (22.5%) were coolies, 4 patients (25%) were business and 2 patients were (5%) students.

Mixed catagories of people are affected from housewife, workers.

SOCIO ECONOMIC STATUS

Recording Socio Economic Status 24 patients (60%) were low income and 10 cases (25%) from middle income and 6 cases (15%) from high income.

The people living in poor Socio Economic Status were more affected because of life style and environmental factors.

DIET REFERENCE:

Out of 40 cases, most of the cases 35 (87.5%) were taken mixed diet and 5 cases (12.5%) had vegetarian diet only.

THINAI DISTRIBUTION:

According to the study, nearly 36 cases (90%) were from Neithal thinai,10% cases from marutham.

CLINICAL MANIFESTATION:

In respect of the patients with Kalladaippu, the clinical manifestation of Flank Pain were present in 40 cases (100%), Burning micturation had present in 18 cases (45%), Oliguria were present in 12 cases (30%), Nausea were present in 6 cases (15%), Fever in 4 patients (10%) and Vomitting and Dysuria in 2 cases (5%).

MUKKUTRAM:

DISTRIBUTION OF VATHAM:

According to classification of Vatham, derangement of Abanan, Viyanan,Uthhanan and Devathathan. 40 patients (100%) was affected with Abanan, 40 patients (100%) was affected with Viyanan,12 patients (30%) was affected withUthanan,11 patients (22.5%) was affected with Devathathan and none affected with Pranan, Samanan , kirukaran, Naagan ,korman and Thananjeyan.

Affected Abanan produced Burning micturition ,constipation and hematuria.

Affected Viyanan produced (pain) tenderness from loin to groin.

Affected Uthanan produced nausea and vomiting.

Affected Devathathan produced insomnia.

DISTRIBUTION OF PITHAM:

According to Pitham 40 cases (100%) were affected Saathgam , 9 cases (22.5%) was affected with Ranjagam.

All the cases were unable to carryout regular works properly. Sathagam indicates this one. So 100% were affected in Sathaga pitham.

Affected Ranjagam produced pallor of skin, eye and reduced hemoglobin.

DISTTRIBUTION OF KABAM:

According to the study, 12 cases (30%) affected by Santhigam,

Santhigam iyam gives stability, lubrication and movements of joints.

Affected Santhigam produced low back pain.

EZHU UDAL KATTUGAL:

From the above chart, we observe that Saaram, were affected in all the patients (100%),Senneer was affected in 9cases (22.5%), Enbu was affected in 12 cases (30%), None affected with Oon,Kozhupu,Moolai and Sukkilam / Suronitham.

ENVAGAI THERVUGAL:

According to Envagai thervugal,Niram was affected in 9 patients (22.5), Vizhi was affected in 4 patients (10%), Sparisam was affected in 7 patients (17.5%), Malam was affected in 1 patient (2.5%), Naadi was affected in for all the 40 patients.

Niram were affected due to anaemia (pale colour).

In Vizhi were affected had dullness of vision.

Mozhi were affected low pitched sound.

Sparisam were affected due to tenderness pain.

In Malam were affected due to constipation.

Naadi was affected in all patients.

NAADI

Out of 40 patients, 24 patients (60%) had Vatha Azhal, 11 patients (27.5%) had Azhal Vali, 3 patient (7.5%) Kabha Pitham, 2 patient (5%) Kabha Vatham.

NEIKURI :

Out of 40 patients (62.5%) had Pitha Neer, 11 patients (27.5%) had Vatha Neer and 4 patients (10%)had Kabha Neer.

CLINICAL PROGNOSIS:

The clinical signs and symptoms were improved after treatment,showing only 5 cases (12.5%) had Flank pain, 2 cases (5%) had Burning micturation.

IMPROVEMENT:

Among the total 40 patients all were improved both subjectively and objectively.

Clinical symptoms before and after treatment were noted. To obtain prognosis of each clinical symptom, the following formulae was used

$$\left.\frac{\text{No. Of cases after treatment}}{\text{No. of cases before treatment}}\right\} \times 100$$

Thus the clinical trial study showed significant clinical improvement in certain clinical manifestation of Kalladaippu such as Flank pain were present in all cases. Burning micturation were present in 18 cases (45%), Oliguria had present in 12 cases (30%), Nausea were present in 6 cases (10%), Fever were present in 4 cases (10%), Vomiting in 2 patients (5%) and Dysuria in 2 cases (5%).

INVESTICATION:

In Blood tests, TC, DC, ESR, Hb% serum creatinine, blood urea were investigated.

URINE:

Albumin, Sugar, Deposit were investigated.

SPECIAL INVESTICATION:

USG- abdomen and pelvis is advised for all the patients to confirm the diagnosis.

After confirming the diagnosis, the patients were given the trial medicine and instructed to follow the diet and other restrictions based on Siddha system.

BIO STATISTICAL STUDY:

Since the p value C.I: 95%, *P<0.05; **P<0.01 is significant in all clinical manifestations. So there is significant reducing of clinical manifestations among the patients for the treatment of Kalladaippu. Hence it is concluded that the treatment was effective and **significant.**

DISTRIBUTION OF CALCULI BASED ON LOCATION :

Since the p value C.I: 95%, *P<0.05; **P<0.01 is significant in all sides. So there is significant changes grades of pain among the patients for the treatment of Kalladaippu. Hence it is concluded that the treatment was effective and **significant.**

OVER ALL RESULT:

Out of 40 patients, 17 cases (42.5%) shows good result, 19 cases (47.5%) shows moderate result, 4 cases (10%) shows poor result.

SUMMARY

The clinical study on **Kalladaippu** was carried out in Post graduate department of Pothu Maruthuvam, Government Siddha Medical College, Aringar Anna Hospital, Chennai – 106 during the period of 2015-2017.

A total of 40 patients were treated in the Outpatient department. The clinical and pathological assessment was carried out on the basis of Siddha and Modern aspects.

All the patients were treated with **Karpoora silasathu parpam** with Ilaneer 130 mg b.d daily, after food for duration of 48days.

- males were mostly affected (57.5%).
- Most of the patients were in the age group between 31-40 years (52.5%)
- Most of the patients were from Neithal Thinai (82.5%).
- In this study, most of the cases (35%) were house wives.
- Most of the patients were affected in Muthuvenirkaalam (37.5%).
- In Vali, Abanan (100%), Viyanan (100%), Uthanan (30%), and Devadhathan (27.5%) was affected.
- In Azhal, Saathagam (100%), Ranjagam (22.5%) was affected.
- In Iyyam, Santhigam (30%) was affected.
- In Ezhu udal kattugal, Saaram (100%), Seneer (22.5%) and Enbu (30%) was affected.
- Regarding naadi, (60%) had Vatha Azhal was the most common naadi observed.
- The Toxicological studies of the trial medicine reveal no toxicity.
- The Pharmacological studies reveal that, the trial drug has Anti Urolithiatic activity.
- Bio- statistical analysis of the clinical trial reveals significant p value < 0.05 and < 0.01 and concluded that the treatment is effective and significant.

Regarding grading of the result, 17 cases (42.5%) shows good improvement, 19 cases (47.5%) shows moderate improvement, 4 cases (10%) shows poor improvement.

CONCLUSION

CONCLUSION

- Kalladaippu is a common disorder of pitha kutram. The dearranged pitham is settled down by the ingredients of trial medicine having astringent taste thereby the medicine acts as ethirurai maruthuvam to cure the disease.
- Most of the cases noted in muthuvenir kalam and elavenir kalam in my clinical trial. So, people should take all preventive measures during this period and take enough water.
- Toxicological study shows no acute and sub - acute toxicity.
- Pharmacological study reveals that the trial medicines possess Anti Urolithiatic activity.
- During clinical trial, no adverse reactions or complications were observed.
- The trial medicine Karpoora silasathu parpam showed good results with relieving symptoms in almost 90% patients.
- Once again Siddha medicine proves itself as a great boon to mankind.

The Tamil Nadu Dr.M.G.R. Medical University

#69, Anna salai, Guindy, Chennai-600 032.

This certificate is awarded to

Dr./Mr./Ms. ...S. SARATH KUMAR.....

for participating as ~~Resource Person~~ / Delegate in the First Workshop on

"Pre-clinical Studies in Research"

for Faculties & PG students of ASU Systems

Organised by the Department of Siddha,

The Tamil Nadu Dr. M.G.R. Medical University on 16.12.2014

Dr. N. KABILAN M.D. (Siddha)
Reader; Dept. of Siddha

Dr. JHANSI CHARLES, M.D.
Registrar

Prof. Dr. D. SHANTHARAM, M.D., D.Diab.,
Vice-Chancellor

Government Siddha Medical College
Department of Medicinal Botany

Dr.S.Sankaranarayanan M.Sc., M.Phil., Ph.D.,
Asst. Professor
Head of the Department

6, Anna Arch Rd,
NSK Nagar,
Arumbakkam, Chennai,
Tamil Nadu 600106.

AUTHENTICATION CERTIFICATE

Based upon the organoleptic/macroscopic/microscopic examination of fresh/market sample, it is certified that the specimen given by Dr. S. Sarath Kumar BSMS studying MD (S), Government Siddha Medical College, Arumbakkam, Chennai is identified below

Binomial name	Family	Regional names
Abutilon indicum Don	Malvaceae	Thuthi
Gossypium herbaceum	Malvaceae	Paruthi

13/6/2016

Dr. S. Sankaranarayanan M.Sc., M.Phil., Ph.D.,

Dr. S. SANKARANARAYANAN, M.Sc., M.Phil., Ph.D.,
Assistant Professor
Dept. of Maruthuva Thavaraiyal
(Medicinal Botany and Pharmacognosy)
Govt. Siddha Medical College,
Arumbakkam, Chennai-600 106.

GSMC/MB-11/2016

Date:13.06.2016

POST GRADUATE DEPARTMENT OF GUNAPADAM (PHARMACOLOGY)

GOVERNMENT SIDDHA MEDICAL COLLEGE, CHENNAI-106

IDENTIFICATION AND AUTHENTICATION CERTIFICATE

Name of the Student	:	S. SARATHKUMAR
Department	:	MARUTHUVAM
Batch year	:	2014 - 2017
Name of the sample	:	KARPOORA SILASATHU AND VENKARAM
Sample description	:	**Dried whole plant / metal /mineral** ✓
Date of the receipt	:	03 - 06 - 2016

REPORT

This sample has been critically studied with macroscopic and organoleptic characters along with relevant literature, I declared that this plant/metal/mineral material is correctly identified as SELENITE AND SODIUM BIBORATE and I hereby authenticate that the sample given by Dr. S. SARATHKUMAR.

This certificate issued at his/her request and is given only for dissertation purpose.

Date: 06 - 06 - 2016

Place: CHENNAI.

Signature with Seal

Dr. VIVEKANANDAN, M.D(s), Ph.D,
H.O.D - Department of Gunapadam,
Govt. Siddha Medical College,
Chennai - 600 106.

CERTIFICATE

This is to certify that the project entitled "**SAFETY EVALUATION OF *KARPOORASILASATHU PARPAM* BY ACUTE TOXICITY -OECD 423 AND SUB-ACUTE REPEATED DOSE ORAL TOXICITY STUDY- OECD 407 IN RATS**" has been approved by the IAEC of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/IV/014/2016**
Animal Sanctioned: *Rattus norvegicus* / Wistar albino rats
Male: 9; Female: 15; Total: 24 (Twenty Four)
Date: 5.3.2016

DR.B.SHEELA RANI
Chair Person

DR.R.ILAVARASAN
CPCSEA Main Nominee

LABORATORY ANIMAL TECHNOLOGY AND RESEARCH (CLATR) ★ CENTRE FOR
SATHYABAMA UNIVERSITY
Chennai - 600 119

ACUTE AND REPEATED 28 DAYS ORAL TOXICITY STUDY ON

KARPOORA SILASATHU PARPAM

ACUTE TOXICITY STUDY

Acute toxicity study of the study drug *Karpoora Silasathu Parpam* was carried out as per OECD guideline (Organization for Economic Co-operation and Development) Guideline-423.

Animal

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^0 C and relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

Acute toxicity Study

Acute toxicity study will be carried out in accordance with OECD guideline 423 [77]. The animals were fasted overnight with free access to water. The study was conducted with single oral dose administration of *Karpoora Silasathu Parpam*.

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Animal Grouping

One group consist of 6 female rats were used for this study . The dose utilized for evaluation of acute toxicity study is about 2000 mg/kg higher than that of the therapeutic dose.

GROUP I : Animals received Test drug 2000 mg/kg (p.o)

The animals were fasted overnight (12- 16 hrs) with free access to water. The study was conducted with single oral administration of study drug *Karpoora Silasathu Parpam* 2000mg/kg (p.o). The animals were observed continuously for first 72 h and

then 14 days for emerging signs of behavioral changes, body weight changes and for mortality.

Occurrence of toxicity in animals were observed continuously for the first 4 to 24 h and observed periodically for the next 14 days. Observation includes the change in skin, fur, eyes and mucus membrane. Appearance of C.N.S,C.V.S and A.N.S related toxicity such as tremors, convulsions, sedation, steric behavior, respiratory distress, cardiovascular collapse, response to sensory stimuli, salivation, diarrhea, lethargy, sleep, coma and mortality were observed with special attention.

Body weight was recorded periodically. At the end of the experiment all animals were subjected for gross necropsy and observed for pathological changes.

SUB-ACUTE TOXICITY STUDY

Sub-acute toxicity study was carried out as per OECD guidelines Guideline-407 [78].

Animals

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} C and relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

IAEC SU/CLATR/IAEC/IV/014/2016

Animal Grouping

Animals were divided into three groups of 06 animals each consist of 3 male and 3 female rats.

GROUP I **:** Animals received saline 5 ml/kg b.w (p.o)

GROUP II **:** Animals received low dose of test drug 200 mg/kg (p.o)

GROUP III **:** Animals received high dose of test drug 400 mg/kg (p.o)

The animals were randomly divided into control group and drug treated groups

for two different doses viz. low dose (200 mg/kg b.w) and high dose (400 mg/kg b.w).

The animals were administrated with the study drug once daily for 28 days. The animals in group I (control group) received normal saline 5 ml/kg b.w. The animals in group II received low dose of *Karpoora Silasathu Parpam* 200 mg/kg b.w (p.o) and group III received high dose of *Karpoora Silasathu Parpam* 400 mg/kg b.w (p.o).

The rats were weighed periodically and observed for signs of toxicity pertains to C.N.S, C.V.S, A.N.S including behavioral changes, food - water intake and morphological changes. At the end of 28th day, the animals were fasted for overnight with free access to water. On 29th day the animals were sacrificed with excess anesthesia. Blood samples were collected from aorta and stored in EDTA (ethylenediamine –tetra actate) for Hematological analysis and for serum generation for biochemical analysis.

The vital organs including heart, brain, lungs, spleen, kidneys, liver, stomach, testes, and ovary were harvested and carefully examined for gross lesions. The organs were preserved in 10% formalin for histopathological assessment and interpretation.

Hematological analysis

Blood samples were analyzed using established procedures and automated Bayer Hematology analyzer. Parameters evaluated include Packed Cell Volume (PCV), Red Blood Cells (RBC) count, White blood cell count (WBC), Platelet Count, Hemoglobin (Hb), Mean cell Haemoglobin Concentration (MCHC), Mean Red Cell Volume (MCV), Mean Cell Hemoglobin (MCH), Mean platelet volume (MPV), Neutrophils, Eosinophil's, Basophils, Lymphocytes and Monocytes.

Biochemical analysis [79]

Serum samples were analyzed for High Density Lipoprotein (HDL), Low density Lipoprotein (LDL) , Very low density Lipoprotein (VLDL) , Triglycerides (TGL), Total Cholesterol , Blood urea nitrogen (BUN), Creatinine, Albumin, Total Protein, Glucose, Uric acid, Aspartate Transaminase (AST), Alanine amino Transaminase (ALT) and Alkaline Phosphatase (ALP) using Mind ray auto analyzer model BS 120.

Histopathological evaluation [80]

Organs included of heart, brain, lungs, spleen, kidneys, liver, stomach, testes and ovary. Histological slides of organs were made and observed under the microscope. The pathological observations of cross section of these organs were performed on gross and microscopic bases. Histological examinations were performed on the preserved tissues with particular emphasis on those which showed gross pathological changes.

Statistical analysis

The statistical analysis was carried by one way ANOVA (GRAPH PAD PRISM 5 computer program). Results were expressed as mean ± standard error .A statistical comparison was carried out using the Dunnet's test for the control and treatment group.

Fecal Pellet Analysis

Methodology

Rats of control and treatment group were allowed to explore to open field on clean and sterile Stainless steel tray. The collected pellets were analyzed for consistency, color, Shape, Presence of blood cells etc

Acute Toxicity Study

Analysis	**Group I**
Consistency	Soft
Shape	Pointed Head
Colour	Greenish
Mucous Shedding	Absence
Blood Cells	Absent
Signs of Infection	None Observed

Sub-Acute Toxicity Study			
Analysis	**Group I**	**Group II**	**Group III**
Consistency	Soft	Soft	Soft
Shape	Oblong	Pointed Head	Pointed Head
Colour	Brownish green	Greenish	Greenish
Mucous Shedding	Absence	Absence	Absence
Blood Cells	Absent	Absent	Absent
Signs of Infection	None Observed	None Observed	None Observed

Muscle Grip Strength Analysis

The grip strength test is a simple non-invasive method designed to evaluate rat muscle force in vivo. Rats of control and drug treated group was allowed to hold the pull bar with both the hind limbs firmly then the animal was gently pulled back with the tail until the animal lost the grip toward the bar. The procedure was repeated to get the average value. Muscle grip ness of the drug treated group was compared to that of the control rat to ensure the change in coordination.

Metabolic Cage for Urine Collection

Rat of control and treatment group was placed individually in metabolic cage with free access to feed and water. Urine dropping from the animal was collected using specialized wire mesh system fixed at the base of the cage having provision to trap the fecal pellet mixed with urine sample. The collected urine sample was subjected to analysis with respect to colour, pH, glucose, ketone bodies, pus and blood cells.

RESULTS

Assessment of clinical signs in rats treated with *Karpoora Silasathu Parpam* on Acute toxicity study

Parameter	**Group I**
Clinical Signs Parameters for the duration of 14 days	Test Drug 2000 mg/ Kg
Number of animals observed	6 Female
Lacrimation	Absence
Salivation	Absence
Animal appearance	Normal
Tonic Movement	Absence
Clonic Movement	Absence
Laxative action	Absence
Touch Response	Normal
Response to Sound	Normal Response
Response to Light	Normal Response
Mobility	Normal Response
Respiratory Distress	Nil
Skin Color	Normal
Stereotype behavior	Absence
Piloerection	Absence
Limb Paralysis	Absence
Posture	Normal
Open field behavior	Normal
Gait Balancing	Normal
Freezing Behaviour	Absent
Sings of Stress and Anxiety	None Observed
Muscular coordination	Normal

Muscle grip	Normal
Sedation	Absence
Social Behavior	Normal
Urine Analysis	No Abnormality
Urine Colour	Yellowish
Urine pH	6
Urine -Glucose	Absence
Urine -Ketones	Absence
Urine- Bilirubin	Absence
Urine-Blood Cells	Negative
Urine - Pus cells	Negative
Mortality	Nil

Quantitative data on the body weight of rats treated with *Karpoora Silasathu Parpam* in Acute toxicity study

Group I	**Before Treatment**	**After Treatment**
Mean	189.8	193
Std. Deviation	4.309	3.578
Std. Error	1.759	1.461

Values are mean ± S.D (n = 6 per group). Control and treatment group were compared statistically using one way ANOVA followed by Dunnett's test.

Assessment of clinical signs in rats treated with *Karpoora Silasathu Parpam* on Sub-Acute toxicity study

Parameter	**Group I**	**Group II**	**Group III**
Clinical Signs Parameters for the duration of 28 days	Control	Test Drug 200mg/ Kg	Test Drug 400mg/ Kg
Number of animals observed	3 Male and 3 Female	3 Male and 3 Female	3 Male and 3 Female
Lacrimation	Absence	Absence	Absence
Salivation	Absence	Absence	Absence
Animal appearance	Normal	Normal	Normal
Tonic Movement	Absence	Absence	Absence
Clonic Movement	Absence	Absence	Absence

Absence	Absence	Absence	Absence
Touch Response	Normal	Normal	Normal
Response to Sound	Normal Response	Normal Response	Normal Response
Response to Light	Normal Response	Normal Response	Normal Response
Mobility	Normal	Normal	Normal
Respiratory Distress	Nil	Nil	Nil
Skin Color	Normal	Normal	Normal
Stereotype behavior	Absence	Absence	Absence
Piloerection	Absence	Absence	Absence
Limb Paralysis	Absence	Absence	Absence
Posture	Normal	Normal	Normal
Open field behavior	Normal	Normal	Normal
Gait Balancing	Normal	Normal	Normal
Freezing Behavior	Absent	Absent	Absent
Sings of Stress and Anxiety	None Observed	None Observed	None Observed
Muscular coordination	Normal	Normal	Normal
Muscle grip	Normal	Normal	Normal
Sedation	Absence	Absence	Absence
Social Behavior	Normal	Normal	Normal
Urine Analysis	No Abnormality	No Abnormality	No Abnormality
Urine Colour	Yellowish	Yellowish	Yellowish
Urine pH	7	7	7
Urine - Glucose	Absence	Absence	Absence
Urine - Ketones	Absence	Absence	Absence
Urine- Bilirubin	Absence	Absence	Absence
Urine-Blood Cells	Negative	Negative	Negative
Urine - Pus cells	Negative	Negative	Negative
Mortality	Nil	Nil	Nil

Effect of *Karpoora Silasathu Parpam* on Body weight of Rats in Sub-acute toxicity study

Group I	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	184.2	186.5

Std. Deviation	5.981	5.891
Std. Error	2.442	2.405
Group II	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	179.3	187.2
Std. Deviation	8.066	7.885
Std. Error	3.293	3.219
Group III	**Before Treatment**	**After Treatment Weight in Gms**
Mean	180.7	191.2
Std. Deviation	3.67	4.167
Std. Error	1.498	1.701

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Quantitative data on the food and water intake of rats treated with *Karpoora Silasathu Parpam* for 28 days in Sub-acute toxicity study

Group I	**Food intake**	**Water intake**
Mean	16.08	20.42
Std. Deviation	2.47	5.984
Std. Error	1.235	2.992
Group II	Food intake	Water intake
Mean	15.25	28.42
Std. Deviation	1.549	0.3191
Std. Error	0.7743	0.1596
Group III	Food intake	Water intake
Mean	13.67	27.75
Std. Deviation	1.333	1.424
Std. Error	0.6667	0.712

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Karpoora Silasathu Parpam* on Haematology profile of rats in sub-acute toxicity study.

Group I	**WBC count (×103 µl)**	**RBC (×10 6 µl)**	**PLT (×10 3 µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	7.983	5.683	506.2	56.35	17.98	31.78	12.04

Std. Deviation	2.408	0.9347	311.2	4.615	2.771	2.477	2.252
Std. Error	0.983	0.3816	127	1.884	1.131	1.011	0.919
Group II	WBC count (×103 µl)	RBC (×10 6 µl)	PLT (×10 3 µl)	MCV (fl)	MCH (pg)	MCHC (g/dl)	HGB (g/dl)
Mean	11.08	7.05	925.7	60.5	17.32	30.9	13.24
Std. Deviation	3.293	1.046	233.9	6.637	2.022	1.462	2.314
Std. Error	1.344	0.4272	95.47	2.71	0.8256	0.5967	0.945
Group III	WBC count (×103 µl)	RBC (×10 6 µl)	PLT (×10 3 µl)	MCV (fl)	MCH (pg)	MCHC (g/dl)	HGB (g/dl)
Mean	9.567	6.833	672.2	60.17	18.92	31.43	12.45
Std. Deviation	1.527	1.305	302.1	6.156	3.484	1.48	2.109
Std. Error	0.6233	0.5327	123.3	2.513	1.423	0.6042	0.861

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Karpoora Silasathu Parpam* on Haematology profile of rats in sub-acute toxicity study.

Group I	**Lymph (%)**	**Mon (%)**	**Neutrophil ($X10^3$/mm3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV(fl)**
Mean	75.23	2.4	2.1	1.217	0.3333	5.2
Std. Deviation	10.23	1.255	0.4	0.3061	0.5164	1.664
Std. Error	4.175	0.5125	0.1633	0.1249	0.2108	0.6792
Group II	**Lymph (%)**	**Mon (%)**	**Neutrophils103/mm3**	**Eosinophils (%)**	**Basophils (%)**	**MPV(fl)**
Mean	74.65	4.1	2.267	1.35	0.5	5.733
Std. Deviation	7.701	0.8899	0.6314	0.2739	0.5477	1.129
Std. Error	3.144	0.3633	0.2578	0.1118	0.2236	0.4609
Group III	**Lymph (%)**	**Mon (%)**	**Neutrophils103/mm3**	**Eosinophils (%)**	**Basophils (%)**	**MPV(fl)**
Mean	72	2.25	2.333	1.267	0.3333	5.283
Std. Deviation	9.595	1.203	0.4131	0.2805	0.5164	1.367
Std. Error	3.917	0.4911	0.1687	0.1145	0.2108	0.5582

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Karpoora Silasathu Parpam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

Group I	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	67.17	14.33	0.4333	110.3	79.5	49.17	35.5	12.98
Std. Deviation	7.167	4.457	0.08165	22.6	16.26	7.96	17.42	3.277
Std. Error	2.926	1.82	0.03333	9.226	6.637	3.25	7.112	1.338
Group II	Blood sugar ® (mg/dl)	BUN (mg/dl)	Serum creatinine (mg/dl)	Serum total cholesterol (mg/dl)	Serum triglycerides level (mg/dl)	Serum HDL cholesterol (mg/dl)	Serum LDL cholesterol (mg/dl)	Serum VLDL cholesterol (mg/dl)
Mean	90.83	15.67	0.6167	125	71.33	54.33	36.83	19.13
Std. Deviation	9.867	1.966	0.2563	7.266	10.48	13.57	12.69	3.267
Std. Error	4.028	0.8028	0.1046	2.966	4.279	5.542	5.18	1.334
Group III	Blood sugar ® (mg/dl)	BUN (mg/dl)	Serum creatinine (mg/dl)	Serum total cholesterol (mg/dl)	Serum triglycerides level (mg/dl)	Serum HDL cholesterol (mg/dl)	Serum LDL cholesterol (mg/dl)	Serum VLDL cholesterol (mg/dl)
Mean	74.33	15.67	0.6667	103.3	86.83	62.17	42.67	18.63
Std. Deviation	12.77	5.046	0.2582	18.39	9.786	11.02	12.82	2.67
Std. Error	5.213	2.06	0.1054	7.509	3.995	4.498	5.232	1.09

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Karpoora Silasathu Parpam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

Group I	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	3.633	2.917	131.2	31.83	155.3
Std. Deviation	0.9668	0.5345	8.256	9.02	74.81
Std. Error	0.3947	0.2182	3.371	3.683	30.54

Group II	Serum total protein (g/dl)	Serum albumin (g/dl)	(AST) (IU/ml)	(ALT) (IU/L)	(ALP) (IU/L)
Mean	5.9	3.75	108	37.33	135.2
Std. Deviation	1.11	0.5753	28.59	7.866	14.66
Std. Error	0.4531	0.2349	11.67	3.211	5.986
Group III	Serum total protein (g/dl)	Serum albumin (g/dl)	(AST) (IU/ml)	(ALT) (IU/L)	(ALP) (IU/L)
Mean	5.067	2.967	117.3	29	114
Std. Deviation	1.451	0.9288	24.94	6.325	24.09
Std. Error	0.5925	0.3792	10.18	2.582	9.835

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Quantitative data on absolute organ weight of rats treated with *Karpoora Silasathu Parpam* **for 28 days in Sub-acute toxicity study.**

Group I	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**Uterus and Ovary (gms)**
Mean	0.54	4.672	1.283	0.55	1.383	1.317	1.233	2.7	1.333
Std. Deviation	0.1534	0.7039	0.1236	0.2588	0.2229	0.1941	0.2251	0.6083	0.1528
Std. Error	0.06261	0.2874	0.05044	0.1057	0.09098	0.07923	0.09189	0.3512	0.08819
Group II	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**Uterus and Ovary (gms)**
Mean	0.5917	5.843	1.51	0.5167	1.483	1.517	1.4	4.133	1.167
Std. Deviation	0.1439	1.002	0.2059	0.1722	0.2137	0.2994	0.4147	0.3786	0.3512
Std. Error	0.05873	0.4091	0.08406	0.07032	0.08724	0.1222	0.1693	0.2186	0.2028
Group III	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**Uterus and Ovary (gms)**
Mean	0.7883	5.653	1.348	0.6167	1.517	1.583	1.467	2.1	1.067
Std. Deviation	0.1401	0.5721	0.205	0.2137	0.1835	0.1472	0.4885	0.7	0.3215
Std. Error	0.05718	0.2335	0.08368	0.08724	0.07491	0.06009	0.1994	0.4041	0.1856

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females) for Heart, Liver, Kidney, Brain, Spleen, Lung, Stomach. Values are mean ± S.D (n = 3 per group per sex) for testes , ovary and uterus for Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

HISTOPATHOLOGY REPORT

BRAIN

Histology of brain revealed the presence of normal cortex showing neurons, glial cells and capillaries. Section of cerebellum shows distinct molecular and granular layer. Neuronal architecture appears normal with sufficient numbers. No signs of ischemia or lesion were observed in sample belongs to group I,II and III.

HEART

Appearance of fibrils and cross striations are equidistant. Sarcoplasmic region of myocardium appears normal. Appearance of cardiomyocyte was normal with dark nuclear region. The nuclei of muscle fibers appear oval arrangement were observed in samples belongs to group I, II and III.

LUNG

Perivascular region appears normal, Alveolar septa and wall appeared widen and normal. No signs of airway secretion and bronchial secretion. Bronchial blood vessels and connective tissue appears normal with no sings of pulmonary edema in control and treatment group rats.

LIVER 2

The walls of the lumen appears normal with no evidence of ischemic changes .No evidence of infiltration were observed in sample belongs to group I, II and III.

STOMACH

Microscopic analysis of stomach sample reveals normal anatomy of muscular stomach with epithelial layer keratinized stratified squamous epithelium, Lamina propria and Sub-mucosa were observed in sample belongs to group I, II and III.

KIDNEY

Appearance of glomerular basement membrane was normal. Lumen of distal convolutes tubule and collecting duct was normal in sample belongs to group I,II and III.

SPLEEN

Appearance of central artery and marginal sinus are normal. Erythropoietic cells (EP) are scattered throughout the red pulp of both the samples. No abnormalities found in lymph nodes were observed in sample belongs to group I, II and III.

TESTES

Presence of mature somatic cells project the perfect histomorphology of testicular cells in this group. Primary spermatocytes with large centered nucleus and dense chromatin were observed in sample belongs to group I,II and III.

UTERUS

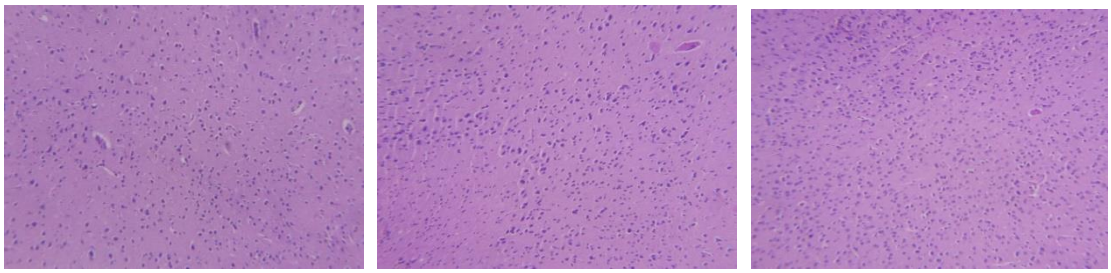
Appearance of endometrium, myometrium and uterine glands was normal. Arrangement of stratum basale, functionale and surface epithelium seems normal in sampls belongs to group I,II and III.

OVARY

Histopathological analysis of ovary showing normal corpus luteum (CL) and Primordial follicles with few mature ovarian follicles with no signs of abnormality. Appearance of antral follicle, primary oocyte and secondary follicles are normal in sample belong to group I,II and III.

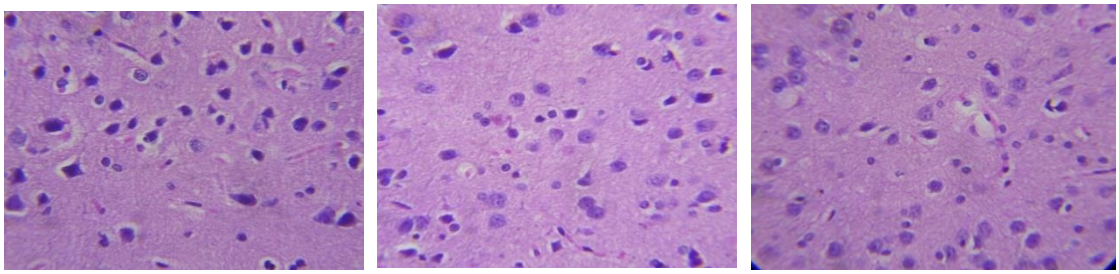
Histopathology of Brain (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

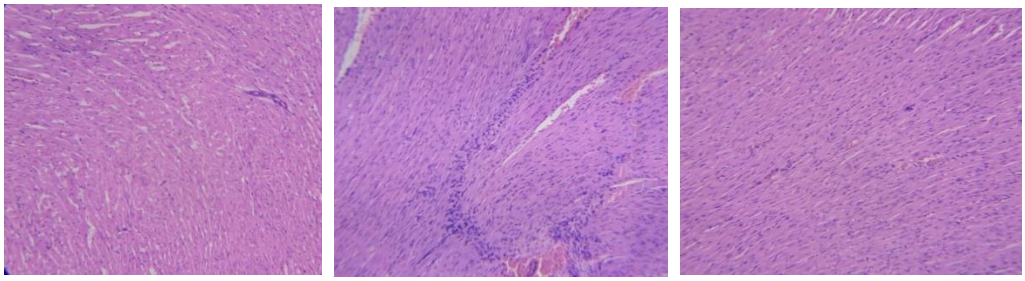
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

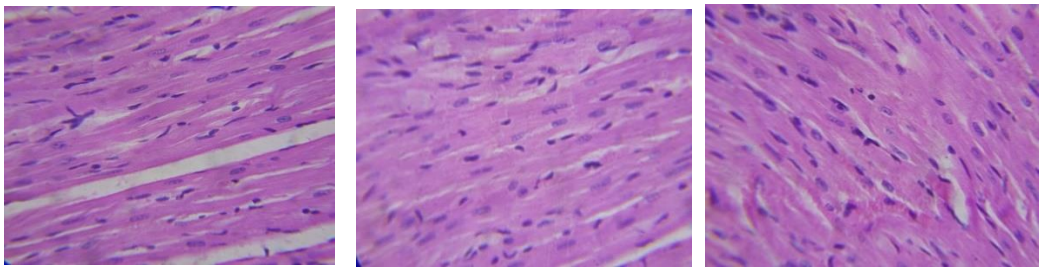
Histopathology of Heart (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

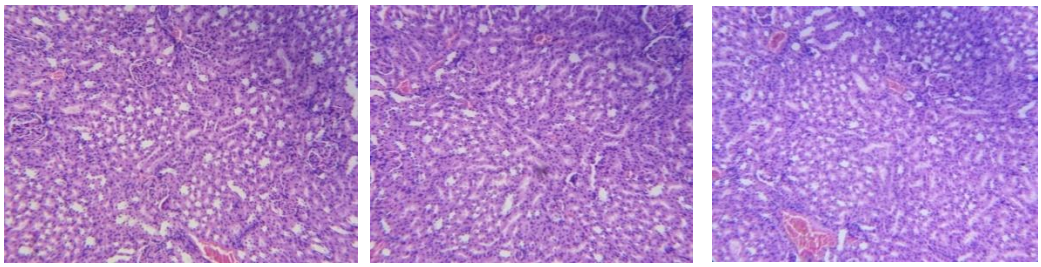
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

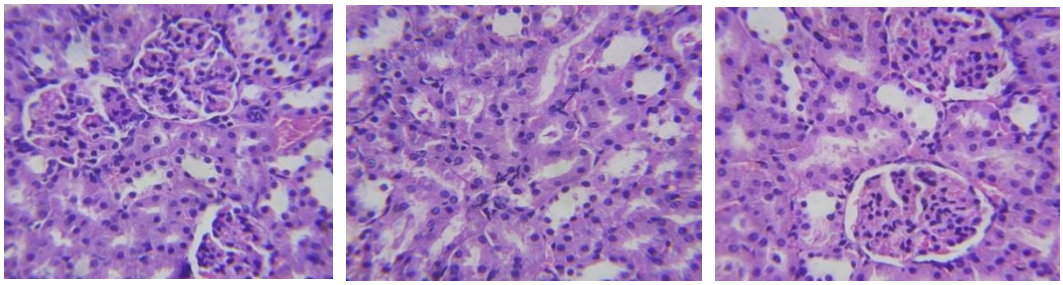
Histopathology of Kidney (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

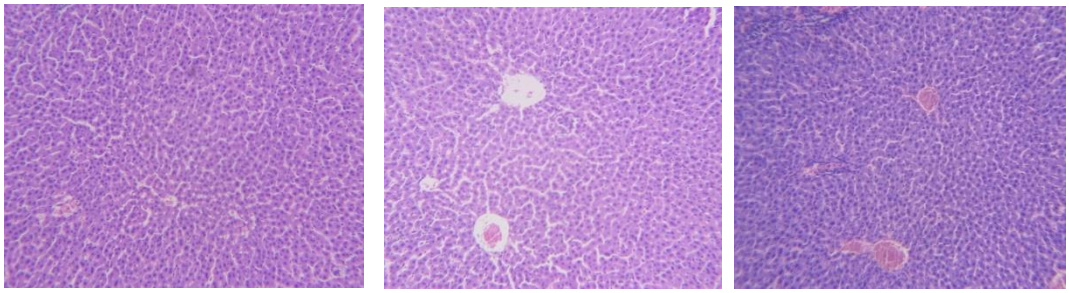
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

Histopathology of Liver (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



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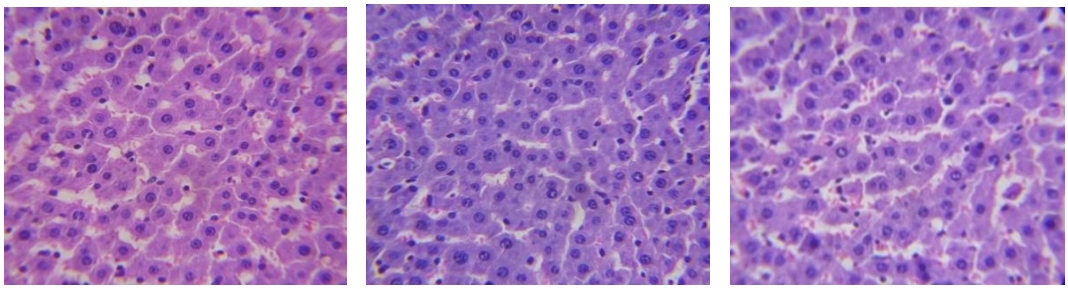
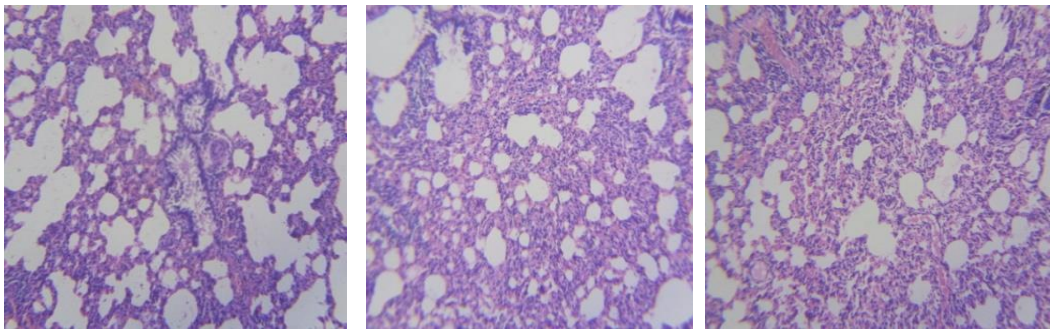


GROUP I **GROUP II** **GROUP III**

Histopathology of Lung (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



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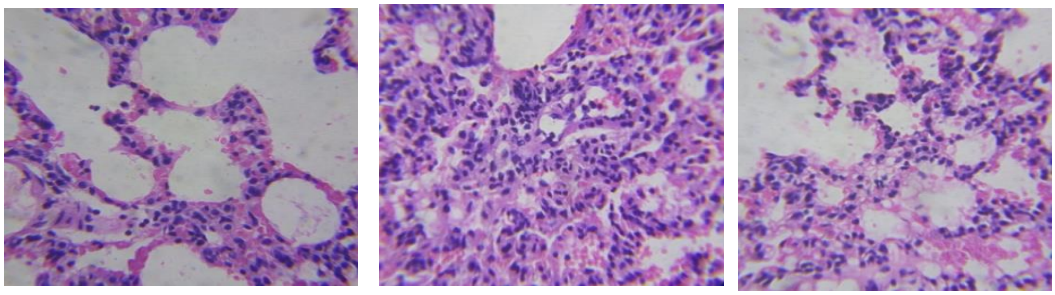
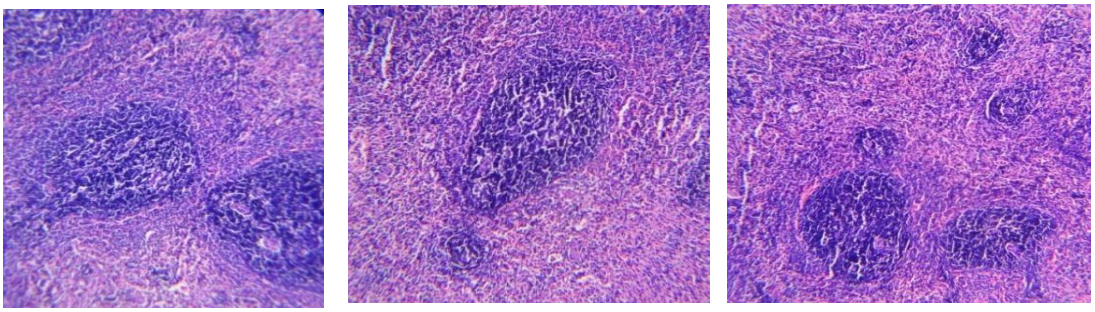


GROUP I ` **GROUP II** **GROUP III**

Histopathology of Spleen (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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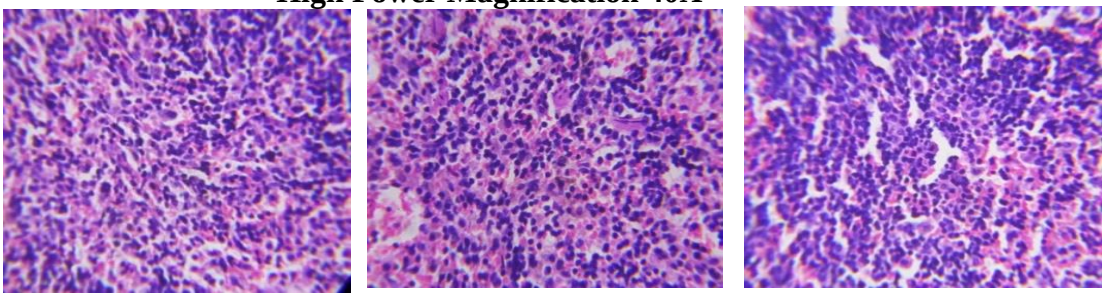
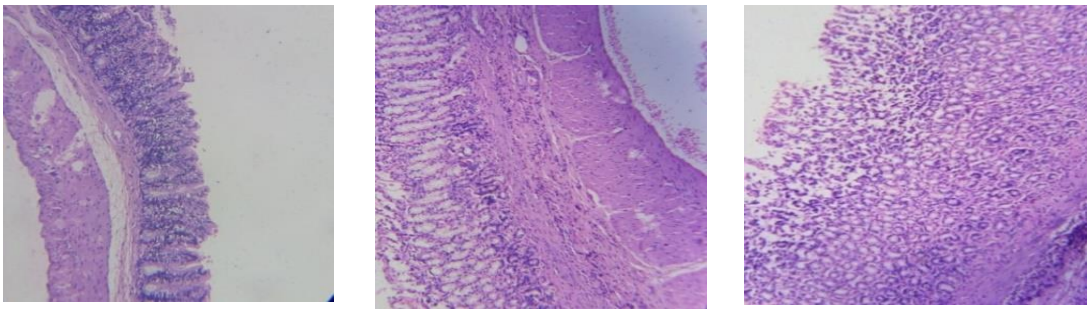


GROUP I **GROUP II** **GROUP III**

Histopathology of Stomach (Male Rat) in Sub-acute toxicity Study



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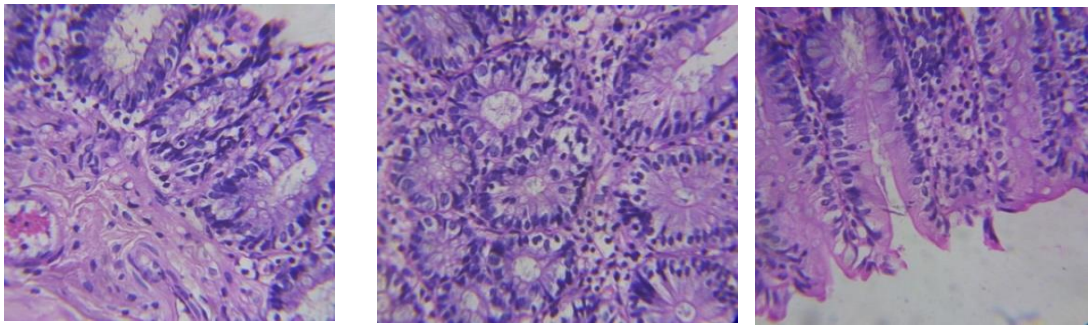
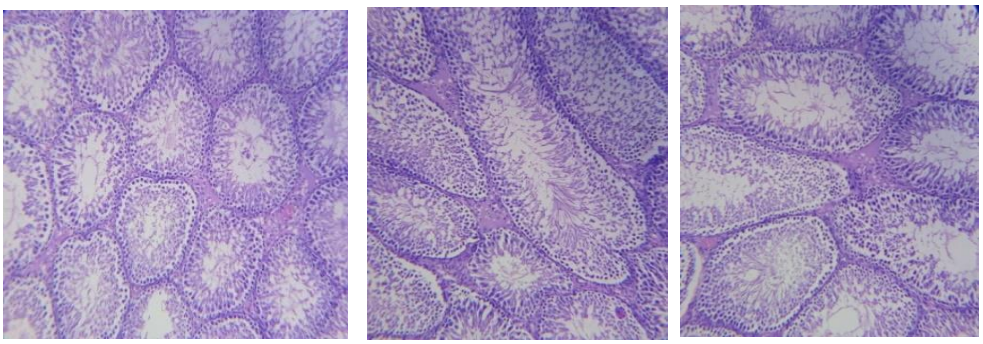


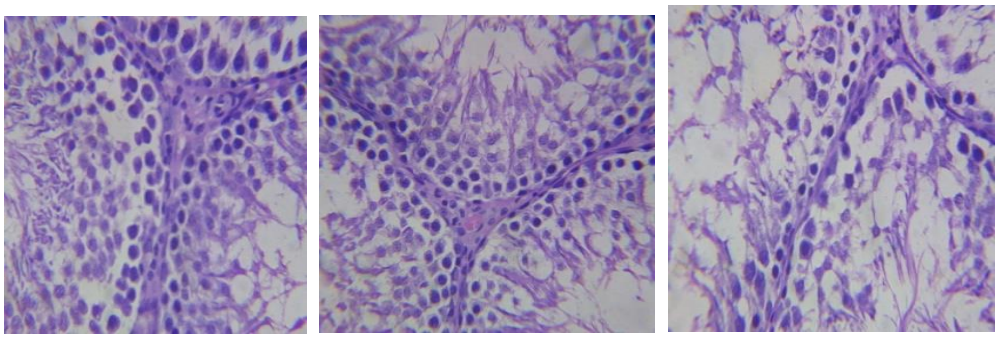
GROUP I **GROUP II** **GROUP III**

Histopathology of Testes (Male Rat) in Sub-acute toxicity Study



GROUP I **GROUP II** **GROUP III**

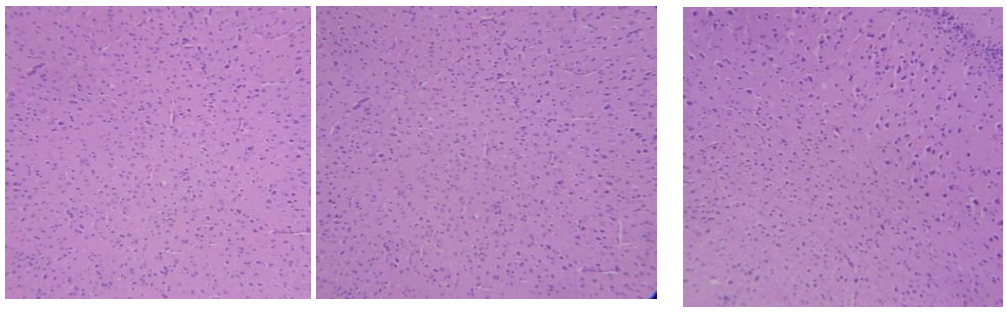
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

Histopathology of Brain (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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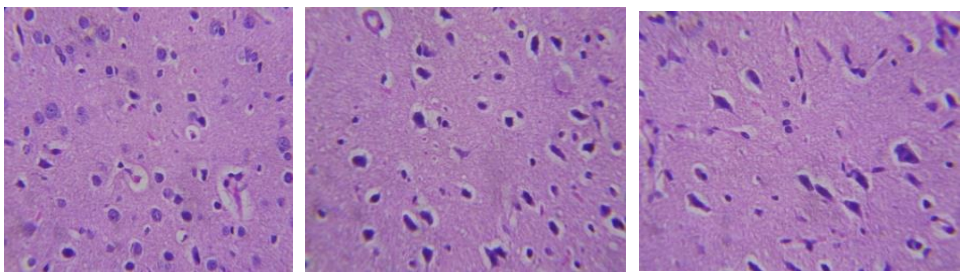
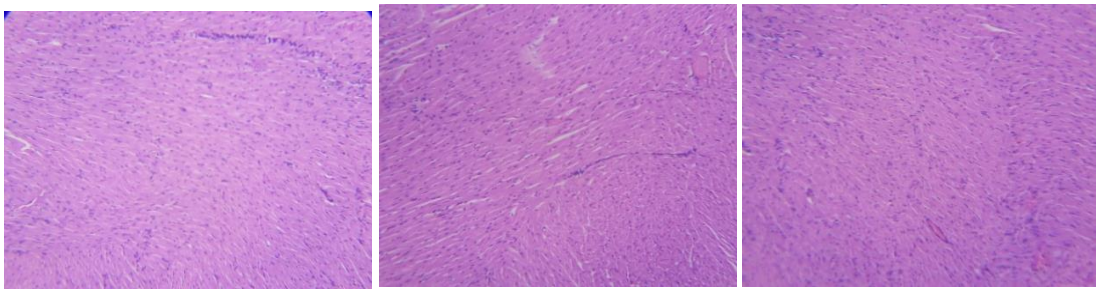


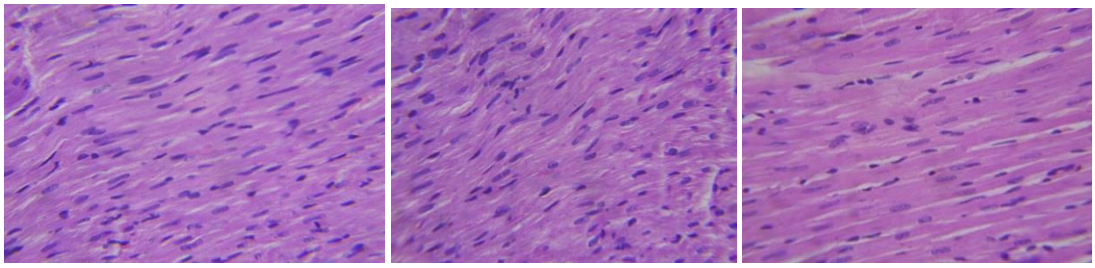
GROUP I **GROUP II** **GROUP III**

Histopathology of Heart (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

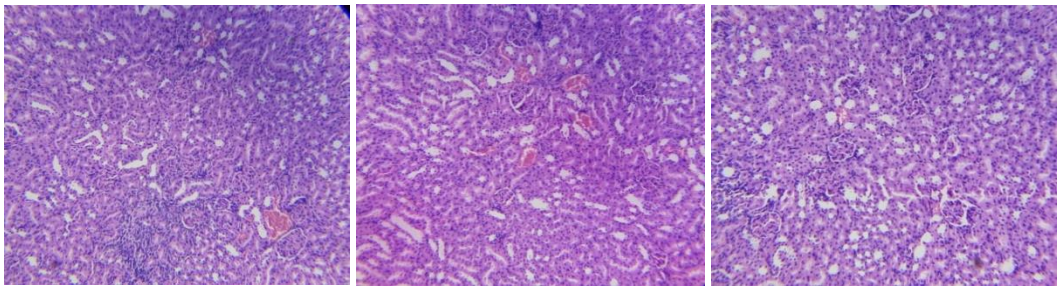
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

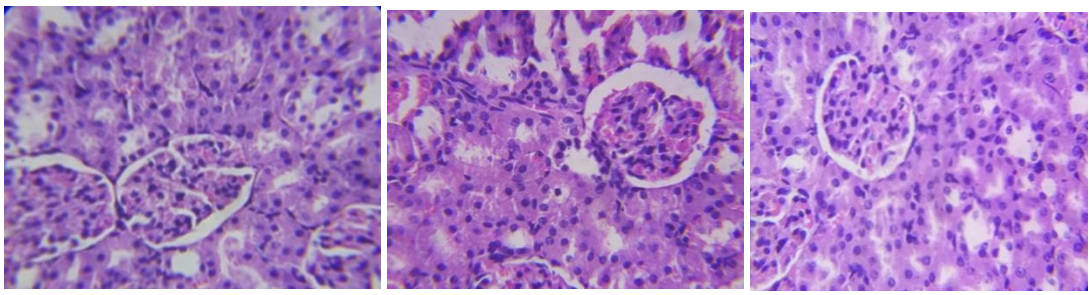
Histopathology of Kidney (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

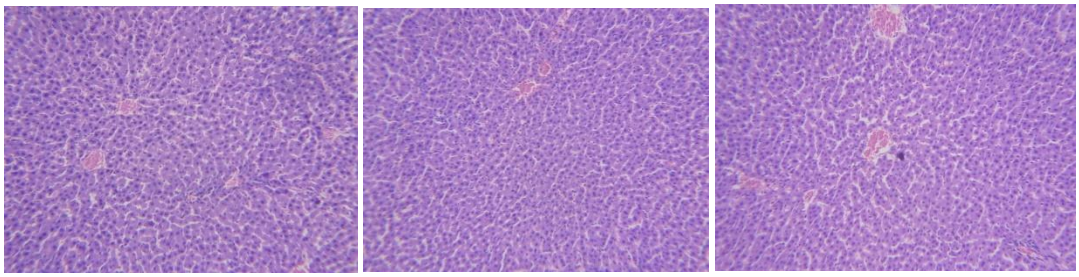
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

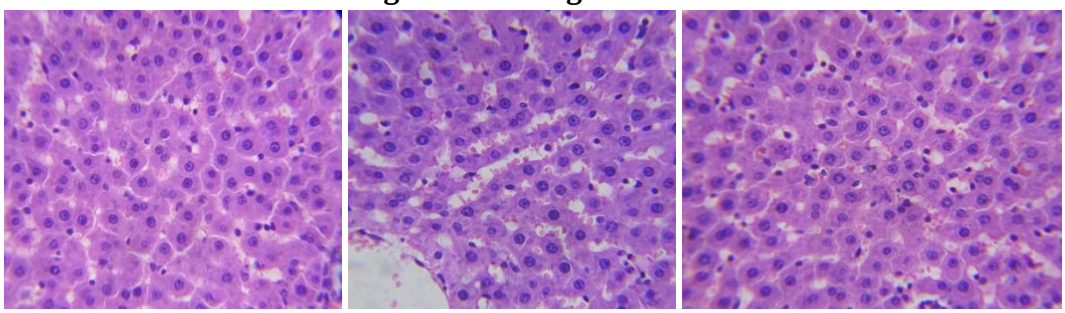
Histopathology of Liver (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

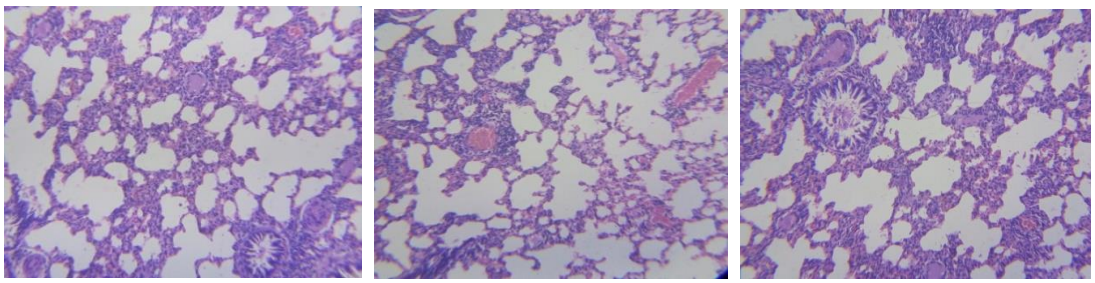
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

Histopathology of Lung (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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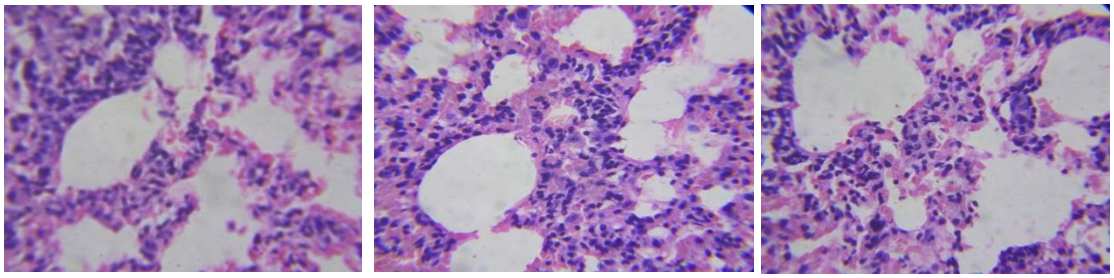
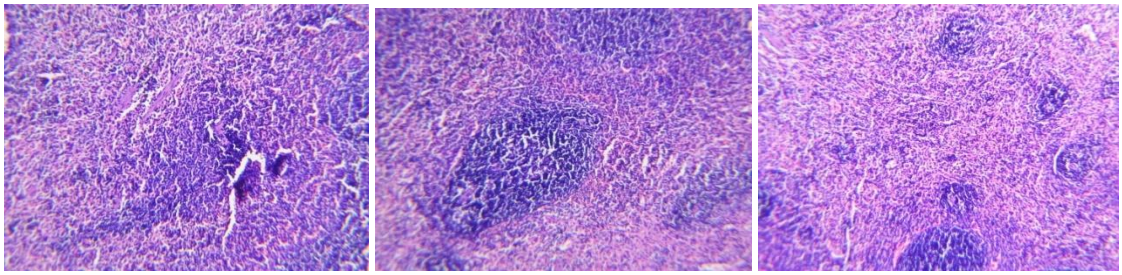


GROUP I **GROUP II** **GROUP III**

Histopathology of Spleen (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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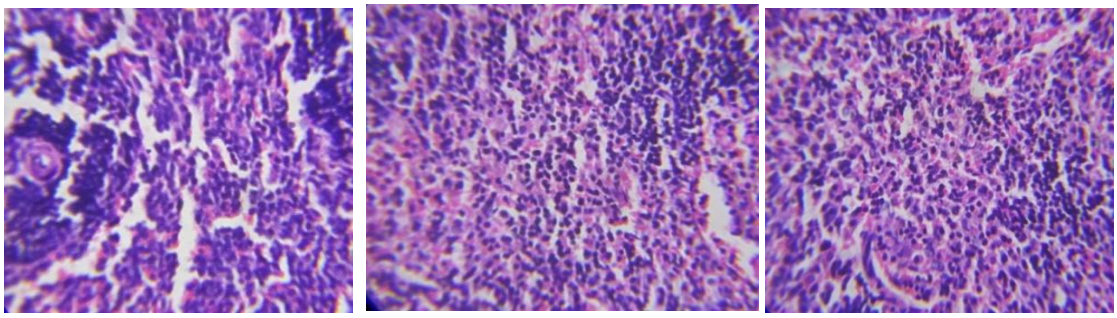
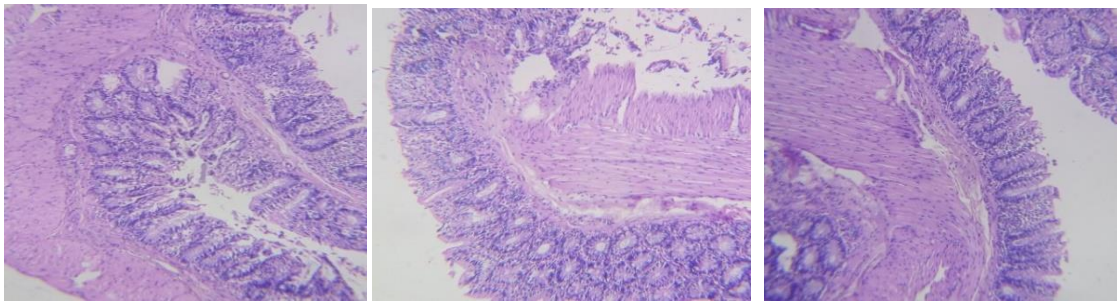


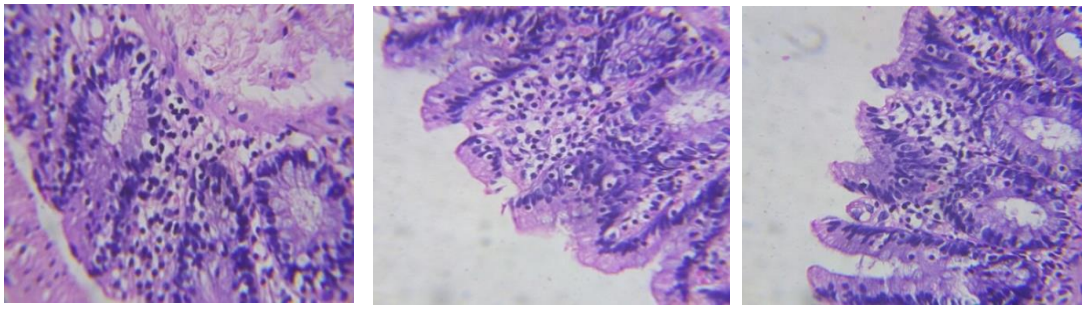
GROUP I **GROUP II** **GROUP III**

Histopathology of Stomach (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



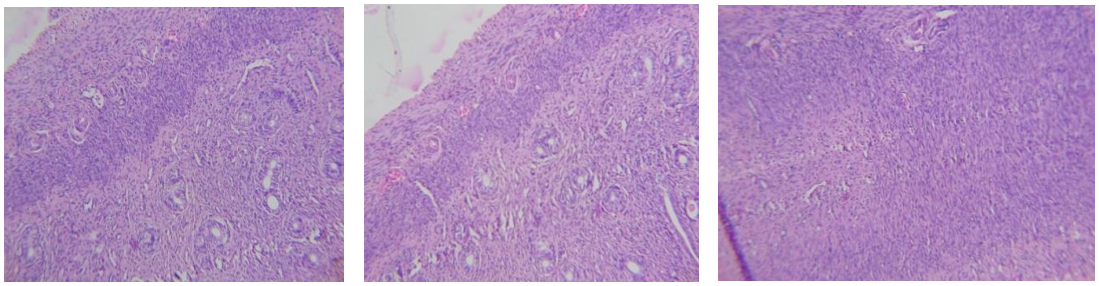
GROUP I **GROUP II** **GROUP III**



GROUP I **GROUP II** **GROUP III**

Histopathology of Uterus (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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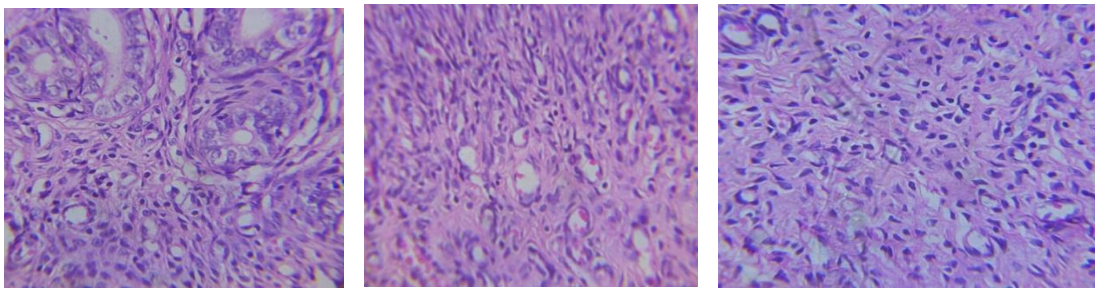
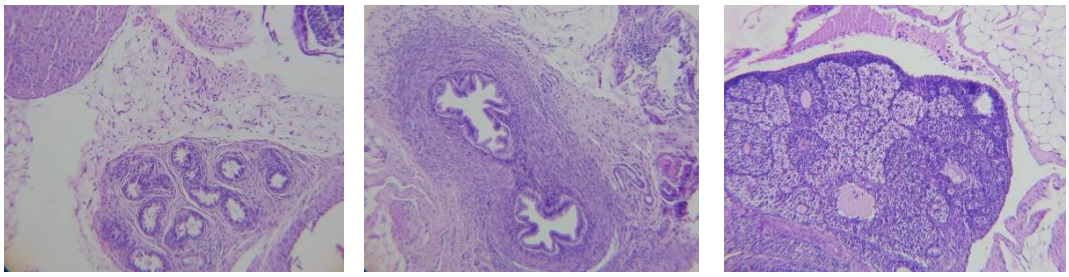


GROUP I **GROUP II** **GROUP III**

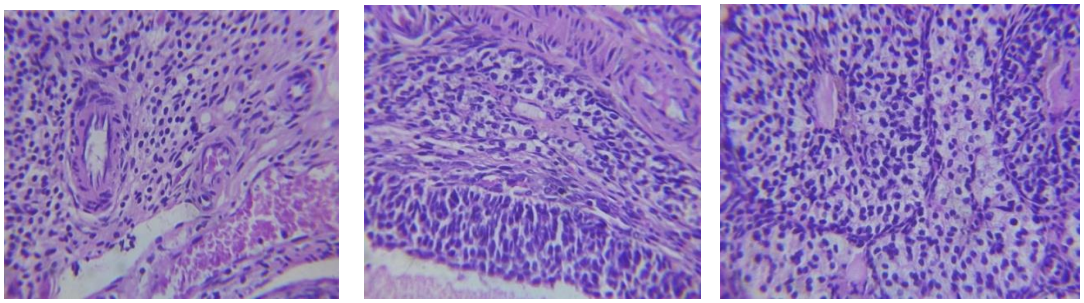
Histopathology of Ovary (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

CERTIFICATE

This is to certify that the project entitled "**PHARMACOLOGICAL EVALUATION OF ANTI-UROLITHIATIC POTENTIAL OF KARPOORA SILASATHU PARPAM IN ETHYLENE GLYCOL INDUCED IN RATS**" has been approved by the Institutional Animal Ethics Committee of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/VII/050/2016**
Principal Investigator: Dr. S. Sarath kumar
Animal Sanctioned: *Rattus norvegicus* / Wistar Albino rats
Male: 24; Total: 24 (Twenty Four)
Date: 05.10.2016

B. Sheela Rani

DR. B. SHEELA RANI
Chairperson

DR. R. ILAVARASAN
CPCSEA Nominee

CENTRE FOR LABORATORY ANIMAL TECHNOLOGY AND RESEARCH
SATHYABAMA UNIVERSITY
Chennai - 600 119

ANTI UROLITHIATIC ACTIVITY OF KARPOORA SILASATHU PARPAM (KSP) IN ETHYLENE GLYCOL INDUCED LITHIATIC RATS

Animals

Healthy adult Wistar albino male rats weighing between 200-220 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit . A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} C and relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.
IAEC**:** SU/CLATR/IEAC/VII/050/2016

Experimental Methodology

The animals were grouped into four groups of 6 animals each. Group I (Control group) -received normal saline, Group II – Urolithiatic control received EG (0.75% w/v, p.o.) in drinking water for 28 days ad libitum (Day1 and Day 28). Group III - Received EG (0.75% w/v, p.o.) in drinking water and treated with 200mg/kg of *Karpoora Silasathu Parpam* for the period of 28 days. Group IV Received EG (0.75% w/v, p.o.) in drinking water and treated with 400mg/kg of *Karpoora Silasathu Parpam* for the period of 28 days.

Sample Collection

At the end of the study, before sacrifice, the animals were fasted for overnight with free access to water. Animals were sacrificed with excess anesthesia. Blood samples were collected from retro orbital sinus puncture and stored in EDTA (ethylenediamine –tetra acetate) test tubes for Hematological analysis and in clot activator coated test tubes for serum biochemical analysis. Kidney sample were harvested and carefully investigated for gross lesions. The organ (kidney) were preserved in 10% formalin for histopathological assessment.

Urine Sample Analysis

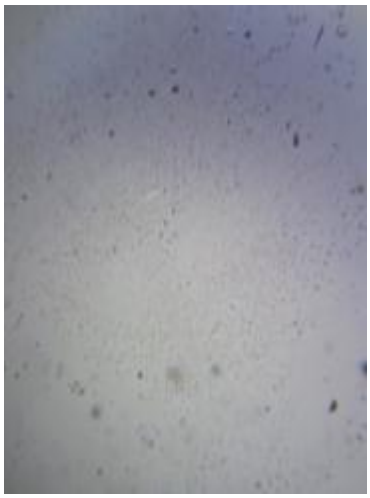
Urine samples (24 h) will be collected on the 28th day by keeping the animals in an individual metabolic cage. The animal had free access to drinking water during urine collection period.

Parameters

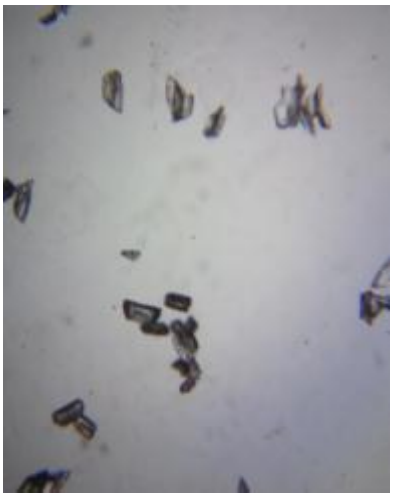
The parameters such as serum magnesium, calcium, Phosphate, uric acid and urine biochemistry such as BUN, pH, uric acid and Creatinine was estimated.

The CaOX crystals viewed in light microscope in urine

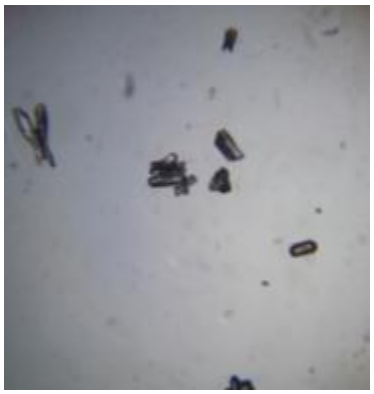
Control Group



Ethylene Glycol-induced



KSP 200 mg/kg



KSP400mg/kg

Effect of KSP on Urine output and pH of EG Induced urolithiatic rats

GROUP I	**Urine Out put (ml/ 24 hr)**	**Ph**
Mean	7.633	6.117
Std. Deviation	0.4033	0.1602
Std. Error	0.1647	0.0654
GROUP II	**Urine Out put (ml/ 24 hr)**	**pH**
Mean	11	7.433
Std. Deviation	1.045	0.3011
Std. Error	0.4266	0.1229
GROUP III	**Urine Out put (ml/ 24 hr)**	**pH**
Mean	15.27	6.4
Std. Deviation	0.3882	0.2898
Std. Error	0.1585	0.1183
GROUP IV	**Urine Out put (ml/ 24 hr)**	**pH**
Mean	20.07	6.333
Std. Deviation	1.181	0.2944
Std. Error	0.4821	0.1202

Values are mean ± S.D / S.E (n = 6 per group)

Effect of KSP on kidney weight of EG Induced urolithiatic rats

GROUP I	**Kidney weight (gms)**
Mean	1.3
Std. Deviation	0.1265
Std. Error	0.05164
GROUP II	**Kidney weight (gms)**
Mean	2.433
Std. Deviation	0.1366
Std. Error	0.05578
GROUP III	**Kidney weight (gms)**
Mean	1.7
Std. Deviation	0.08944
Std. Error	0.03651
GROUP IV	**Kidney weight (gms)**
Mean	1.55
Std. Deviation	0.1378

Std. Error	0.05627

Values are mean ± S.D / S.E (n = 6 per group)

Effect of KSP on Serum biochemistry of EG Induced urolithiatic rats

GROUP I	**Blood urea nitrogen (BUN)**	**Creatinine (mg/dl)**	**Uric acid (mg/dl)**
Mean	37.17	0.5833	3.833
Std. Deviation	3.971	0.2137	0.5164
Std. Error	1.621	0.08724	0.2108
GROUP II	**Blood urea nitrogen (BUN)**	**Creatinine (mg/dl)**	**Uric acid (mg/dl)**
Mean	64	1.283	8.667
Std. Deviation	3.521	0.2229	0.4633
Std. Error	1.438	0.09098	0.1892
GROUP III	**Blood urea nitrogen (BUN)**	**Creatinine (mg/dl)**	**Uric acid (mg/dl)**
Mean	53.33	0.8333	4.85
Std. Deviation	2.503	0.1966	0.295
Std. Error	1.022	0.08028	0.1204
GROUP IV	**Blood urea nitrogen (BUN)**	**Creatinine (mg/dl)**	**Uric acid (mg/dl)**
Mean	45.17	0.4833	4
Std. Deviation	3.189	0.07528	0.2608
Std. Error	1.302	0.03073	0.1065

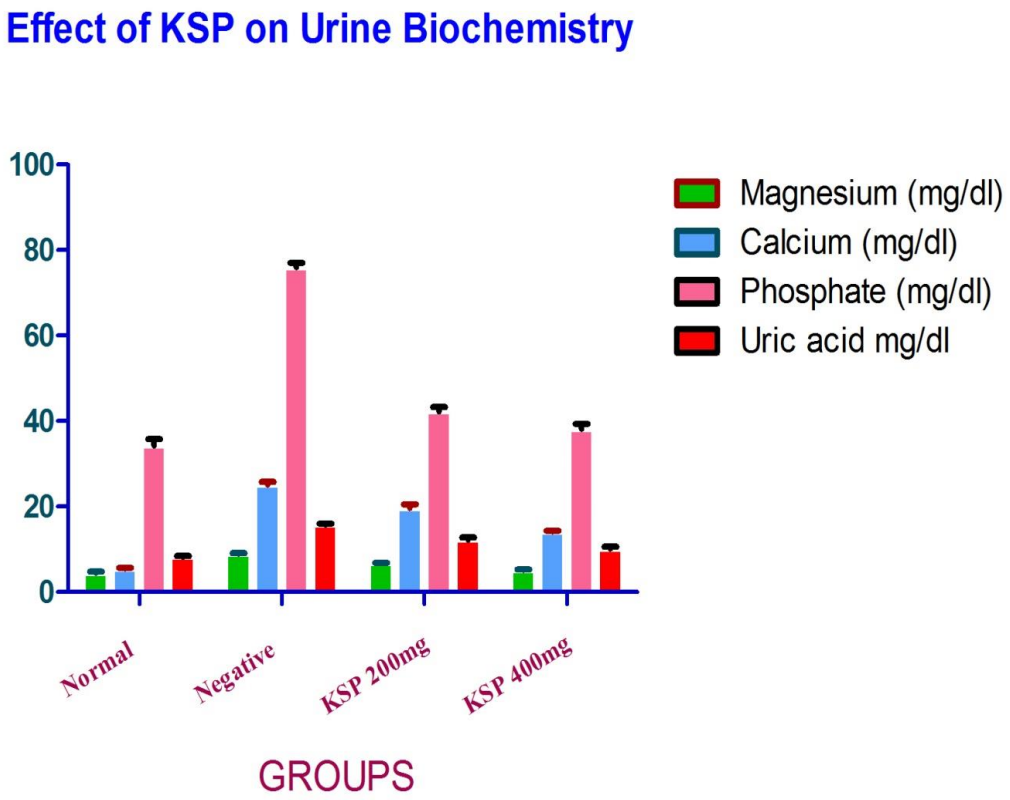
Values are mean ± S.D / S.E (n = 6 per group)

Effect of KSP on Urine Biochemistry of EG Induced urolithiatic rats

GROUP I	**Magnesium (mg/dl/24hr)**	**Calcium (mg/dl/24hr)**	**Phosphate (mg/dl/24hr)**	**Uric acid (mg/dl/24hr)**
Mean	4.567	5.5	34.33	8.283
Std. Deviation	0.5279	0.429	3.67	0.3371
Std. Error	0.2155	0.1751	1.498	0.1376
GROUP II	**Magnesium (mg/dl/24hr)**	**Calcium (mg/dl/24hr)**	**Phosphate (mg/dl/24hr)**	**Uric acid (mg/dl/24hr)**
Mean	9.065	25.18	76	15.83
Std. Deviation	0.05244	1.466	2.53	0.5164
Std. Error	0.02141	0.5986	1.033	0.2108
GROUP III	**Magnesium (mg/dl/24hr)**	**Calcium (mg/dl/24hr)**	**Phosphate (mg/dl/24hr)**	**Uric acid (mg/dl/24hr)**
Mean	6.783	19.73	42.33	12.33
Std. Deviation	0.2317	2.088	2.422	1.033

Std. Error	0.09458	0.8523	0.9888	0.4216
GROUP IV	**Magnesium (mg/dl/24hr)**	**Calcium (mg/dl/24hr)**	**Phosphate (mg/dl/24hr)**	**Uric acid (mg/dl/24hr)**
Mean	5.2	14.23	38.17	10.17
Std. Deviation	0.2366	0.3724	2.927	1.169
Std. Error	0.09661	0.152	1.195	0.4773

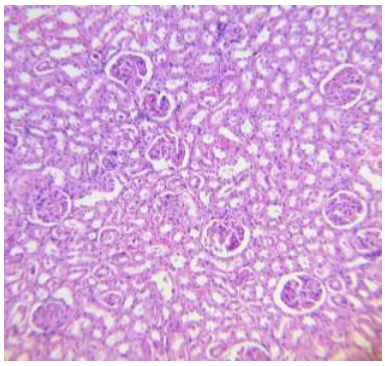
Values are mean ± S.D / S.E (n = 6 per group)



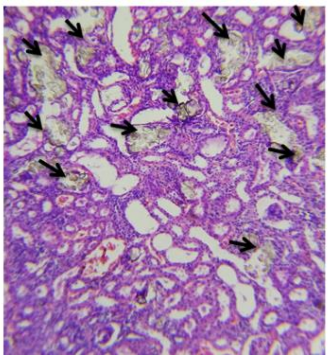
Histopathology of Rat Kidney (H&E) Staining

Low Power Magnification 10 X

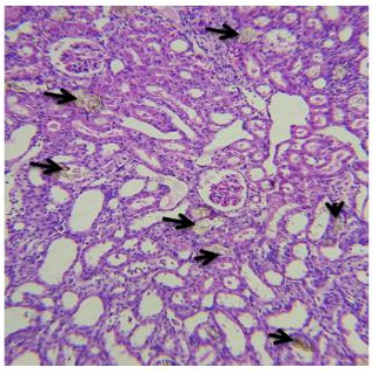
Control Group



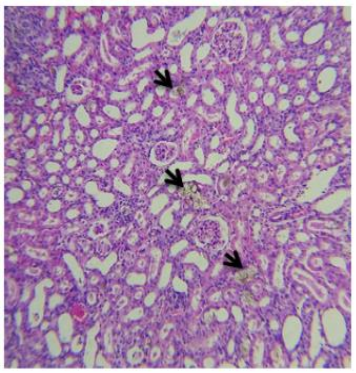
EG treated Rat



KSP 200mg/kg



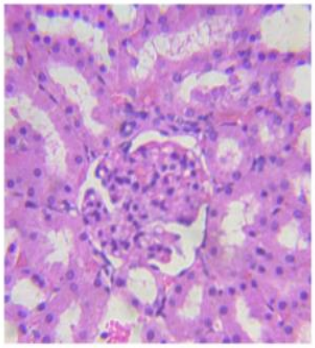
KSP 400mg/kg



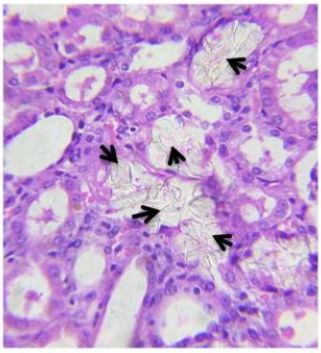
Histopathology of Rat Kidney (H&E) Staining

High Power Magnification 40 X

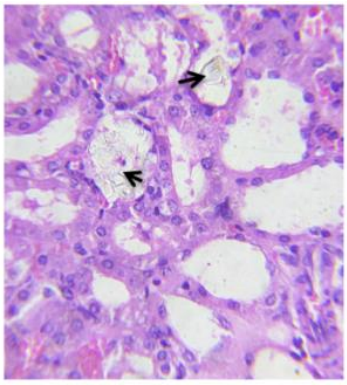
Control Group



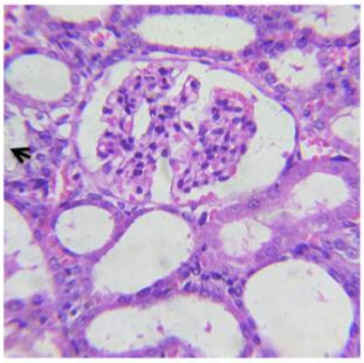
EG treated Rat



KSP 200mg/kg



KSP 400mg/kg



Pathology report

- Normal glomerulus (G) surrounded by a narrow capsular space and the parietal layer of Bowman's capsule. Epithelial lining on proximal convoluted tubule appears normal. Lumen of distal convolutes tubule and collecting duct was normal
- Ethyl glycol treated group reveals marked tubular dilatation, glomerular degeneration and increased crystal deposition.
- Mild derangement in mesenchymal density along with reduced crystal deposition was observed in sample belongs to KSP 200mg/kg and 400 mg/kg treated rats. Accumulation of calcium oxalate deposits inside the tubules was much controlled in treatment group when compare to EG alone treated group.

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், சென்னை - 600 106
सिद्ध केंद्रीय अनुसन्धान संस्थान,
अण्णा सरकारी अस्पताल परिसर, अरुम्बाक्कम, चेन्नई – 600 106
SIDDHA CENTRAL RESEARCH INSTITUTE
(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)
Anna Govt. Hospital Campus, Arumbakkam, Chennai – 600106
Phone: 044-2621 4925, Fax: 044-2621 4809

01.3.17

CERTIFICATE

Name of the student: Dr. S. Sarathkumar, III year PG Student, Department of Maruthuvam Government Siddha Medical College, Arumbakkam, Chennai-600 106.

Name of the sample: Karpara Silasathu Parpam

Name of the Experiment		Mean
Loss on drying(at 105°C)	:	5.37%
Total ash	:	73.16%
Water soluble ash	:	32.19%
Acid insoluble ash	:	10.23%
pH value (10%)	:	8.56
Particle size	:	Passes through 200 mesh

(R. Shakila)
Research Officer (Chemistry) & Head,
Department of Chemistry

(Dr. P. Sathiyarajeswaran)
Assistant Director (Siddha) I/c

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
B	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	white precipitate not obtained	Absent
3	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	presence of Yellow precipitate	Present
4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white Precipitate	Absent
5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hcl.	Absence of Rotten egg smelling	Absent
6	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and	Absence of reddish brown gas.	Absent

	concentrated sulphuric acid and viewed the test tube vertically down.		
7a	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white Precipitate	Absent
B	5 drops of clear solution is added with 2ml of diluted sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	KMNO4 solution Discolourisation not obtained	Absent
8	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops of Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent
9	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Presence of Green tinged flame	Present
II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution.	Absence of Yellow Precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absence of Bluish green coloured flame.	Absent
B	2ml of the extract is added with excess of Ammonia solution	Absence of deep Blue	Absent
12	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess	Absence of White Precipitate.	Absent
13a	**Test for Iron**	Absence of Blood	Absent

	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate Solution is added.	red colour	
B	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Blood red colour not obtained	Absent
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
15	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	presence of White precipitate.	Present
16	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown Precipitate	Absent
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Presence of Yellow precipitate	Present
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow Precipitate	Absent

21	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution.	Absence of Yellow precipitate	Absent
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Absence of Green colour	Absent
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium lodide solution.	Absence of Red colour	Absent
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH, mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

The given sample Karpoora silasathu parpam contains,

Calcium, Potassium, Borate, Phosphate.

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethics Committee (IEC)

IEC No: GSMC-CH-ME-4/2015/010

Protocol title: A CLINICALSTUDYONKALLADAIPPU (UROLITHIASIS) WITH THE EVALUATION OF SIDDHA DRUG KARPOORA SILASATHU PARPAM
Principal Investigator: DR.S. SARATH KUMAR
Name & Address of Institution : Government siddha medical college, Arumbakkam, Chennai-106
☑ New Review ☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY): 26-03-2015 Date Of Previous Review, If Revised Application :
Decision of the IEC ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions / Reasons / Remarks : 1. In renal function test, add uric acid level.
Recommended for a period of 1 year from date of completion of preclinical studies:

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

26/3/15.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V. Banumathi
Member Secretary

INSTITUTIONAL ETHICS COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref:Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
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MR.P.SARAVANAN., Puplic Person	✓	

26/3/15

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V.Banumathi
Member Secretary

BIO STATISTICAL ANALYSIS

CLINICAL PROGNOSIS

Treatment for Kalladaippu:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs&Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Flank pain	40(100)	5(12.5)**
2.	Nausea	6(15)	0(0)*
3.	Vomiting	2(5)	0(0)*
4.	Burning Micturition	18(45)	2(5)**
5.	Dysuria	2(5)	0(0)*
6.	Fever	4(10)	0(0)*
7.	Oliguria	12(30)	0(0)*

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in all symptoms. So there is significant reducing of symptoms among the patients for the treatment of Kalladaippu. Hence it is concluded that the treatment was effective and **significant.**

Treatment for Kalladaippu:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

DISTRIBUTION OF CALCULI BASED ON LOCATION

S. No	Side	Before Treatment	After Treatment
		n%	n%
1.	Rt – Kidney	11(27.5)	4(10)*
2.	Lt – Kidney	10(25)	3(7.5)*
3.	Bilateral	18(45)	9(22.5)*
4.	Ureteral	1(2.5)	0(0)*

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in sides and ureteral there is a significant changes in distribution of calculi based on location. Hence it is concluded that the treatment was effective and **significant.**

GOVERNMENT SIDDHA MEDICAL COLLEGE

ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE

CHENNAI – 600 106

CLINICAL STUDY ON **"KARPOORA SILASATHU PARPAM"** IN THE TREATMENT OF

"KALLADAIPPU" (UROLITHIASIS)

INFORMED CONSENT FORM

"I have read the foregoing information. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.

I consent voluntarily to participate in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care".

"I have received a copy of the information sheet/consent form".

Date:

Station:

Signature of participant:

Signature of the Guide: Signature of the Investigator:

அரசு சித்த மருத்துவக் கல்லூரி, சென்னை-106

அறிஞர் அண்ணா மருத்துவமனை, சென்னை

கல்லடைப்பு நோய்க்கான சித்த மருந்தின் **(கற்பூர சிலாசத்து பற்பம்)**

பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

ஒப்புதல் படிவம்

ஆய்வாளரால் சான்றளிக்கப்பட்டது

நான் இந்த ஆய்வு குறித்த அனைத்து விபரங்களையும் நோயாளிக்கு புரியும் வகையில் எடுத்துரைத்தேன் என உறுதியளிக்கிறேன்.

தேதி: **கையொப்பம்:**

இடம்: **பெயர் :**

நோயாளியின் ஒப்புதல்

என்னிடம் இந்த மருத்துவ ஆய்வின் காரணத்தையும், மருந்தின் தன்மை மற்றும் மருத்துவ வழிமுறை பற்றியும், தொடர்ந்து எனது உடல் இயக்கத்தை கண்காணிக்கவும், அதனை பாதுகாக்கவும் பயன்படும் மருத்துவ ஆய்வுக்கூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் இந்த மருத்துவ ஆய்வின் போது, காரணம் எதுவும் கூறாமல், எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்து கொள்ளும் உரிமையை தெரிந்திருக்கின்றேன். நான் என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு கல்லடைப்பு நோய்க்கான கற்பூர சிலாசத்து பற்பம் மருந்தின் பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

தேதி : **கையொப்பம் :**

இடம் : **பெயர் :**

தேதி : **சாட்சிக்காரர் கையொப்பம் :**

இடம் : **பெயர் :**

உறவுமுறை :

துறைத்தலைவர் கையொப்பம் : ஆராய்ச்சியாளர்
கையொப்பம்:

CASE SHEET PROFORMA FOR KALLADAIPPU

GOVT.SIDDHA MEDICAL COLLEGE&HOSPITAL, CHENNAI-106

POST GRADUATE DEPARTMENT BRANCH –I MARUTHUVAM

Duration: 2015-2017

OP No / IP No	:	Occupation	:
Ward No	:	Income	:
Bed No	:	Nationality	:
Name	:	Religion	:
Age	:	D.O.A	:
Sex	:	D.O.D	:
Address	:	Diagnosis	:

1. Complaints and duration :

2. History of present illness :

3. History of past illness :

4. Personal history :

5. Occupational history :

6. Menstrual history :

7. Personal Habits : Veg/non veg/smoker/Alcoholic/Tobacco chewer

8. Family History :

GENERAL EXAMINATION

Patient consciousness :

Body Built :

Nourishment :

Anemia :

Jaundice :

Cyanosis :

Clubbing :

JVP :

Tracheal deviation :

Pedal oedema :

Lymph adenopathy :

VITAL SIGNS

Body Temp :

Pulse :

Respiratory rate :

Blood Pressure :

Weight :

SIDDHA ASPECT

NILAM

Kurinchi :

Mullai :

Marutham :

Neithal :

Palai :

PARUVA KALAM

Kaar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Muduvenil :

YAAKKAI(Udal)

Vaatham :

Pittham :

Kabam :

Kalappu :

GUNAM

Satthuvam :

Rajotham :

Thamasam

PULANGAL (SENSORY ORGANS)

Mei –Sensation :

Vaai – Taste :

Kan – Vision :

Mooku - Smell :

Sevi – Hearing :

KANMENTHRIYAM/KANNMA VIDAYAM [MOTOR ORGANS]

Kai- Dhaanam :

Kaal-Kamanam :

Vaai-Vasanam :

Eruvaai- Visarkkam :

Karuvaai-Aanantham :

UTHKAAYA ATHAKAAYAM

Puyam[forearm] :

Sayam[arm] :

Kaal[leg] :

Paaatham[feet] :

UYIR THATHUKKAL

A.VATHAM

Piranan :

Abanan :

Viyanan :

Udanan :

Samanan :

Nagan :

Koorman :

Kirukaran :

Devathathan :

Thananjeyan :

B.PITHAM

Anar pitham :

Ranjaga pitham :

Saathaga pitham :

Pirrasaga pitham :

Alosaga pitham :

C.KABAM

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

UDALTHAATHUKKAL

Saaram :

Senner :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam/Suronitham :

ENVAGAI THERVUGAL

1.Naa :

2.Niram :

3.Mozhi :

4.Vizhi :

5.Sparisam :

6.Malam :

7.Moothiram

a)Neer Kuri :

b)Nei Kuri :

8.Naadi :

MALAM

Niram :

Edai :

Irugal :

Elagal :

MOOTHIRAM

1.Neerkuri

Niram :

Manam :

Edai :

Nurai :

Enjal :

2.Neikuri

MODERN ASPECT

Systemic Examination

Inspection :

Palpation :

Renal angle :

Tenderness : Present/Absent

Radiation :

Percussion :

Auscultation :

Others Systems

Cardio Vascular System :

Respiratory system :

Central nervous system :

CLINICAL SIGN AND SYMPTOMS OF KALLADAIPPU

S.No	Symptoms	Before Treatment	After Treatment						
			7th day	14th day	21st day	28th day	35th day	42nd day	49th day
1	Pain ✓ Site ✓ Radiation ✓ Character								
2	Nausea								
3	Vomiting								
4	Burning Micturition								
5	Dysuria								
6	Oliguria								
7	Haematuria								
8	Retention								
9	Fever								
10	Frequency of Micturition								

INVESTIGATION

1. BLOOD
   - TC
   - DC
   - ESR
   - Bleeding time
   - Clotting time
   - Blood sugar
   - Blood urea
   - Serum cholesterol
   - Serum calcium
   - VDRL

2. URINE
   - Colour
   - Turbidity
   - Albumin
   - Sugar
   - Deposits

- Epithelial cells
- RBC's
- Pus cells

Casts
Specific gravity
Urine culture and sensitivity

3. USG Abdomen and Pelvis

4. X-Ray KUB

CASE SUMMARY

DIAGNOSIS

KALLADAIPPU (UROLITHIASIS)

TRIAL DRUG: KARPOORA SILASATHU PARPAM

Dose: 130 mg

Anubanam : Ilaneer

Duration of Treatment: 48 days

Pathiam (Do's and Don'ts)

DO'S:

1) Drink 4-5 litres of water per day.
2) Dring tender coconut, barley water, lemon juice, raddish juice.
3) The following vegetables can be taken in the diet

Raddish

Lady's finger

Plantain pith

Mint leaves

Bottle guard

DONT'S:

1) Avoid cabbage, cauliflower, tomato seeds, mushroom.
2) Avoid milk and its products.
3) Avoid chicken, fish and other sea foods
4) Avoid drinking fluoride containing water.

Prognosis

Medical Officer Signature: **HOD**

DATE	DAILY REPORT	MEDICINE

ADVICE:

MEDICAL OFFICER:

H.O.D/Guide

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