A CLINICAL STUDY ON

KALLADAIPPU WITH NILAKUMILAVER KUDINEER

DISSERTATION SUBMITTED TO

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DEPARTMENT OF POTHU MARUTHUVAM

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Documents Filed	1) Protocol 2) Data Collection Forms 3) Patient Information Sheet 4) Consent Form
Clinical / Non Clinical Trial Protocol	Clinical Trial Protocol
Informed Consent Document	Yes
Any other Documents	Case Sheet, Investigation Documents
Date of IEC Approval & its Number	GSMC-II-IEC/2015-Br.-I/01/16.07.2015

We approve the trial to be conducted in its presented form.
The Institutional Ethical Committee expects to be informed about the process report to be submitted to the IEC atleast annually of the study, any changes in the protocol and submission of final report.

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Certificate of Botanical Authenticity

Certified the following plant drugs used in Siddha formulation Nilakumilaverkudineer (Internal) for the management of Kalladaipu (Urolithiasis) is taken up for Post Graduation Dissertation Studies by Dr.K.Annapoorani (Reg No.321311001) PG Dept, ofPothuMaruthuvam are correctly identified and autherticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopical and Taxonomical methods.

S.N	Name	Botanical Name	Family	Parts used	Quantity
1.	Nilakumil	*Gmelina asiatica*	Verbenaceae	Dried Root	350 gms

Station: Palayamkottai

Date: 02/12/15

2/12/15

Authorized Signature

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INTRODUCTION

The siddha medicine is one of the oldest traditional system in the world. The word “Siddha”comes from the word “Siddhi” which means an object to be attained perfection or .Siddha focused to “Astamahasiddhi” that is the eight supranatural power. Those who attained or achieved the above said powers are known as siddhars .It is the first system to emphaise that health is the perfect state of physical, psychological, social and spiritual component of human being.

According to Therayar in his Thaila vargachurukkam, have been explained a physician should have a detailed knowledge about geographical and seasonal variation ,physical and mental state of the patients and deep knowledge to about the diseases.

Faith in the medicine is necessary before taking them .Mental health is necessary for physical well being. Thiruvalluvar says,

“நோய்நாடி நோய் முதல் நாடி அது தணிக்கும்
வாய்நாடி வாய்ப்பச் செயல்”

The system strongly advocates proper food habits.The improper food habits producing disease. The universe and the body are formed by the same elements in different proportions.

“அண்டத்திலுள்ளதே பிண்டம்
பிண்டத்திலுள்ளதே அண்டம்
அண்டமும் பிண்டமும் ஒன்றே
அறிந்து தான் பார்க்கும் போதே”

சட்ட முனி ஞானம்

The first phase in human life is attributed to Vatha, the middle to Pitha, and the late phase to Kapha,

“வாதமாய் படைத்து
பித்த வன்னியாய் காத்து
சேத்தும சீதமாய்துடைத்து”

தேரன் மருத்துவ பாரதம்

The basic theory of siddha system of medicine is “Food is medicine ,medicine is food”. The human body is a compose of three humours such as Vatham, Pitham, kapham. Seven Physical mechanisms Uyir thathukal such as Saaram (plasma), Seneer (blood), Oon (muscle), Kollzuppu (fatty tissue), Enbu (bone), Moolai (brain), Sukkilam (semen).

Siddhars defined 96 principles as the constituents of the human being. They are nothing but the mineralizations of the"Five basic Elements"such Man, Neer, Theyu, Vayu, Agayam

When the normal equilibrium of three humours (Vatham ,Pitham ,kapham) is disturbed, disease is caused. The factors which assumed to affect this equilibrium are environment, seasonal variations, diet, physical activities and stress. Under normal conditions the ratio between these three humours Vatham, Pitham,kapham is 4:2:1.

In diagnosis, examination of eight items is required which is commonly known as "Envagai thervugal"

- Nadi - Pulse
- Sparisam - Touch
- Na - Tongue
- Niram - Colour
- Mozhi - Voice
- Vizhi – Eyes
- Maalam - Stool
- Moothiram - Urine

The drugs used by the siddhars could be classified into 3 groups. Thavara(herbal products),Thathu(organic and inorganic substances)and Sangamam(Animal products).The drugs also classified on the basis of five properties.

Suvai (taste), Gunam (character), Veeriyam (potency), Pirivu (class), Mahimai (action).

The five physical elements combine in various formula to create various types of objects. For example the human body as a whole in general is said to be constituted with Five parts of physical earth, Four parts of physical water, Three parts of physical heat, Two parts of physical air, and One part of physical space. The above said formula is only for the total human organism, and not applicable for the various structures in which it is constituted, such as organs, bone, flesh, nerves, skin.

Tri dosha thathuvam

Vatha : Vathanadi or Chandra nadi with abana vayu recides of the seat of rectum and intestines- situated in Agni mandalam

Pitha **:** Surya nadi with prana vayu is found in the seat of water embracing the liver in Surya mandalam.

Kapham **:** Agni pulse with samana vayu is located in the seat of semen in the cold lunar region –below the top of the head embracing the pineal gland.

Siddhars classified the disease into 4448 types. One among them is KALLADAIPPU NOI, which is assorted under visarkauruppugal noigal KALLADAIPPU NOI is compared to UROLITHIASIS in modern medicine.

According to siddha Noinadal text part I &II Neer noigal classified only quantity of passing urination.

The disease of urinary system is classified into

1. Neerinai arukkal noigal –Acquired or Oliguria state
2. Neerinai perukkal noigal – Polyuria state

In yugi vaidhya chinthamani-800, 2nd part, kalladaippu noi comes under neerinai arukkal noigal. Kidney stones are small, solid masses formed when salts or minerals normally found in urine become solid crystals inside the kidney. Urinary stones consititutes one of the most common diseases in our country and pain due to kidney stone is known worse than that of labour pain.

Kidney stones do not have a single, well defined cause, but are the result of a combination of factors. A stone is created when the urine does not have the correct balance of fluid and a combination of minerals and acids. when the urine contains more crystal-forming substances than the fluid can be dilute, crystals can form. The diagnosis of a kidney stone can be confirmed by radiological studies or ultrasound examination; urine tests and blood tests are also commonly performed. Preventive strategies may include dietary modifications and medication with the goal of reducing excretory load on the kidneys.

Several drugs are available for treating kalladaippu noi in siddha medicine system. It is evident from the siddha literature that therapeutic values of several drugs have been proved. However, clinical trials on the treatment of kalladaippu noi have not yet been undertaken for the medicines like, **NilakumilaVer Kudineer** (internal) mentioned in **Agasthiyar Vaithiya Kaviyam 1500** respectively. Hence, these siddha medicines have been choosen for my dissertation work to evaluate their therapeutic values in the treating kalladaippu noi.

AIM AND OBJECTIVES

Urolithiasis

Urolithiasis (from greek ouron, “urine” + lithos “stone” + iasis) is the formation of urinary calculi (urinary stones), which are calculi formed or located anywhere in the urinary system. It comprises nephrolithiasis (the formation of kidney stones cystolithiasis (The formation of bladder stones)

Urinary stone formation is a common disease with an increasing incidence and prevalence worldwide that appears even more pronounced in industrialized countries such observations seen to underscore the impact of lifestyle and dietary choices as well as access to better medical care for urinary stone formation .

A total of 40 patients of both sex (20 op and 20 ip) were selected and administered with the following trial drug at PG department of pothumaruthuvam, government siddha medical college and hospital, palayamkottai

PRIMARY OBJECTIVE

The aim of my dissertation work is to evaluate the clinical efficacy of the trial drug, **NILAKUMILAVER KUDINEER** .

SPECIFIC OBJECTIVES

1. To recruit patients with KALLADAIPPU NOI for clinical trial, on the basis of etiology and clinical features mentioned in various siddha literatures and modern Text books.

2. To investigate siddha fundamental and modern parameters during and after treatment in all recruited patients.

3. To perform urine analysis, haematological studies and ultrasonography for all patients before and after treatment.

4. To perform stone analysis in selected cases.

5. To undertake biochemical analysis of the clinical trial drug.

6. To evaluate pharmacological activity such as lithotriptic, diuretic and analgesic activities of the trial medicine.(Nilakumilaver Kudineer.)

ABSTRACT

Kalladaippu is the most common any one disease in our country. The disease kalladaippu can correlated in modern science is urolithiasis.. One such drug, "**Nilakumilaver Kudineer"** mentioned in the book **AGATHIYAR VAITHYA KAVIYAM -1500 ,PAGE NO.651 &652)** .Hence I have chosen this medicine as my dissertation work to evaluate its therapeutic efficacy in treating kalladaippu noi.

The disease was diagnosed by following various siddha diagnostic investigation methods. A total of 40 patients of both sex (20 op and 20 ip) were selected and administered with the following trial drug at PG department of pothumaruthuvam, government siddha medical college and hospital, palayamkottai.

Nilakumilaver Kudineer – 50ml , BD morning and evening with . The trial medicine was subjected to biochemical, pharmacological and acute toxicity studies. At the end of the study trial drug is safe and effective in the treatment of **KALLADAIPPU NOI.**

REVIEW OF LITERATURES

SIDDHA ASPECTS

The siddha system of medicine is believed to be more than 10,000 years old and to be considered to be one of the most traditional systems. Among the various diseases, "Kalladaippu noi" is one of the most common diseases in India. Several preventive and curative treatments are found in various siddha literatures for kalladaipu noi.

The ancient siddha literature yugi vaidhya chinthamani 800 is clearly mentioned Kalladaippu noi is under the topic of Neerinai Arukkal Noi.

"நீரிரு வினைக் குணத்தை
நீயறி விரித்துச் சொல்வோம்
நீரினை பெருக்கலொன்றே
நீரினை யருக்க லொன்றே
நீரிழிவுடனே கொல்லும்
நீர்க்கட்டு வினைகளொன்று"

தேரன் கரிசல்

நோய்நாடல் நோய் முதல் நாடல் - 2ம் பாகம், பக்கம் 420

1. வேறுபெயர்கள் (Synonym)

Ashmari Rogam

2. இயல்(Definition)

The kalladaippu noi has been defined differently by various authors.

i. In "Agathiyar Gunavagadam" , kalladaippu noi is defined as below:

"தானென்ற மூத்திரத்தில் நற நறவென்று
தங்கியதோர் பொடியெனும் மணல்தானப்பா
வானென்ற சிறியதொரு கல்லாவதப்பா
வளமாக வந்துவிழும் நோய்க்கு தானே
ஏனென்ற அஸ்மரி ரோகமென்ற பேராம்
எளிதாகக் கல்லுகள்தான் விழுகும்போது
கோனென்ற குண்டிக்காய் மூத்திரக்குழலப்பா
குணமான மூத்திரப்பை நீர்த்தாரைக் கேளே"
கேளடா முன் குறியில் எரிச்சல் கண்டு
கெடியாக வேதனைகள் காட்டுமப்பா
வாளடா சிறியதொரு கற்கள்தானே
வளமான மூத்திரப்பை குழல் வழிப்படியாய்த்

தேளடா வரும்போது திரேகந்தன்னில்
தெரிப்பது போல யிருவேதனை செய்யும்பாரு
நாளடா கற்கள்தா னிறங்கிவிட்டால்
நலமான வேதனைகள்தான் தீரும்பாரே''

அகத்தியர் குணவாகடம்

The above poem written by Agathiyar, defines kalladaippu noi as "Deposition of sand like grains in urine, which results in formation of small, followed by expulsion of stones along with urine". Further, he explained that the stones obstruct in the kidney, ureter, urinary bladder and urethra. When the stones reaching the urethral orifice, intense burning sensation in glans penis occurs. Then the stones get expelled and pain is relieved.

1. In "**T.V. Sambasivam pillai Agarathi**", kalladaippu noi is defined as:
   Large concentration of stones in the bladder or kidney produces calculus or gravel. It causes difficulty in passing urine.
2. In S**iddha maruthuvam**, kalladaippu noi is defined as:
   Sudden obstruction of flow of urine during micturition, pain in the tip of the penis male and clitoris in female, burning sensation in urethral orifice, pain radiating from loin to groin, presence of sand-like stones in the urine are the cardinal symptoms of this disease.
3. **Roga Nirnaya saaram under Roga nithaanam**, kalladaippu noi is defined as:
   Pain in and around the umbilicus, dysuria, urine odours like goat‟ s urine, fever, chills and anorexia are common features of this disease.

Noi varum vazhi (aetiology)

The causes of the disease mentioned in various siddha literature are as follows

01. "அறைகிறேன் விந்தழிந்தால் மேகமாச்சு
அமுது வழிந்து சூடுகொண்டால் வாயு சேரும்
பறைகிறேன் சூலைக் குட்டம் கிரந்தி புற்று
பவுத்திரம் கள் பிளவை போட்டு கண்டமாலை
அறைகிறேன் அரையாப்பு ஒட்டிய புஜ
அருங்கரப்பான் சிரங்கு குன்மம் மாநீர்க்கட்டு
குறையவே நீர் கொண்ட அந்நோய் காசம்
குடிலமாம் பேதியோடு கிராணி பாண்டே

அகத்தியர் வைத்திய வல்லாதி 600

02. ‘‘தெளிந்ததோர் கல்லடைப்பு உற்பத்தி கேளாய்
சிறிதுநாட் டொடங்கியே மேகந் தன்னில்
தளிந்ததோர் சலப்பையி லுதிரத் தோய்ந்து
சந்தசத் தாகவே பருத்துக் கொள்ளும்
வளிந்ததோர் வாதபித்தங் கோபித் தக்கால்
வந்துபெருங் கல்லாய் நீர் வழிய டைத்து
நளிந்தோர் நாலுவிதக் கல்ல டைத்து
நண்பான வரலாறு நாட்டக் கேளே’’

பாடல் 725, பக்கம் 283

03. ‘‘கலங்கினதோர் தண்ணீர்தான் குடித்த பேர்க்குங்
கல்லெலும்பு மயிர்மண்தான் கலந்தன் னத்தில்
அலங்கினதோ ரன்னங்க ளருந்த லாலும்
அழுகலோடு மூத்தபண்ட மருந்த லாலும்
மலங்கினதோர் மாப்பண்ட மருந்த லாலும்
மந்தத்தில் வாய்வான பதார்த்தந் தன்னை
துலங்கினதோர் ருசிதன்னிற் சுவைத்த லாலும்
சுருக்காய்க்கல் லடைப்புவந்து தோன்றுந் தானே’’

யூகி வைத்திய சிந்தாமணி - 800 பாடல் 727, பக்கம் 284

The above mentioned poems reveal that in the patients suffering from chronic mega noi, the semen stagnates in the urinary tract for a long time, leading to the obstruction of the tract. This condition results in the deposition of urine constituents and formation of stones in the urinary tract. In meanwhile, due to the increased vatham and pitham, the small stones become large in size and obstruct the urinary passage. The formation of urinary stones is also attributed to the drinking contaminated, hard water, consuming food adultered with sand and small stones, contaminated food, starchy food and unhealthy food habits etc.

‘‘நீரினைத் தடுத்தல் செய்யின்
நீர்க்கட்டுத் துவாரம் புண்ணாம்
பாரிடுஞ் சந்து சந்தில்
பண்புறு நோவ தாகும்
நேரிலங் கயருங் காமியம்
நிச்சய நோதல் செய்யும்
பாரினி லபான வாயு
பண்புறச் சேரு மன்றே’’

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 212

It is inferred from the above that when urine is ceased deliberately, obstruction of urine flow, ulcer in the urethral orifice, pain in the glans penis, increased abaana vayu in the abdomen will result in.

“சுக்கிலந்தனைய டக்கின்
சுரமுடனீர்க்கட் டாகும்
பக்கமாங்கைகால் சந்து
பார நோய் வழியிறங்கும்
மிக்கமார் நோயுண்டாகும்
மிகுத்திடும் பிரமே கந்தான்
தக்க தோர் போதுமாகின்
தரித்திடும் வாயுக் கூறே”

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் - 212

As per nature of the body of human beings, semen needs to be discharged regularly. In this regard, the above said poem emphasizes that deliberate cessation of seminal discharge results in fever, lumbar pain, oliguria, chest pain and white discharge.

Pothu kuri kunangal (Common symtoms)

- Frequent urination,
- Sudden obstruction of urine flow,
- Excruciating pain in the glans penis and anus,
- Sometimes, when the stone attempts to expel by rolling down, it , may not too able to expel due to obstruction either in urethra or in the urethral orifice. This condition will result in agonizing pain and swelling in the genito-urinary tract.
- When the stones are rough and irregular with sharp projections burning sensation and pain will occur in the lower abdomen and urethral orifice, sometimes it may lead to hematuria.

Noi naadal noi mudal naadal,Text vol-II,427

Some disease are associated with kalladaippu noi in Siddha text

“குத்து முத்காரசூலை யின்கு ணந்தான்
கோர்வையாய் விலாவுதனில் முதுகில் நெஞ்சில்
அத்தி யினில் நாபியில பானமாங்கு தத்தில்
அதிக துன்மாங்கிசந்தான் வளர்ந்து மேவிப்

பத்துமணற் படுக்கைபோற் சலத்து வாரப்
பதிநெருக்கி மூத்திரமாங் கிரிச்சி யுண்டாய்த்
தத்துசடங் கடுப்பெடுத்து மதிக லங்கித்
தளர்ச்சி யொடுமயக்கமாய்த் தள்ளுந் தானே''

யூகி வைத்திய சிந்தாமணி – 800, பா 233, பக்கம் 88

This poem reveal that the symptoms of kalladaippu noi include followed stricture of urethral orifice by sand-like crystals, block in the urethral passage. In addition, dysuria, Hematuria, Fever, tiredness and guiddiness also occur.

Classification

There are several types of kidney stones (urolithiasis). In the siddha books, it is evident from various siddha literatures . Some of them given below.

A. As per Maan Murugiyam

கல் தோன்றல்

''நீரகம் நீரியம் அலையம்
மூன்றினுந் கற்கள் தோன்றி
உப்புமுத லுடம்பின் கழிவுப் பொருள்களும்
அனலும் ஐயமும் அடிப்படு மாசும்
திரண்டு சல்லெனச் செறிவது கல்லே''

(நீரகம் - சிறுநீரகம், நீரியம் - சிறுநீர்ப்பை, அலைகம் - கருப்பை)

நீரகக் கல்லடைப்பு

''சுண்ணம் உப்பு முதற்பல பொருள்கள்
சிறுநீ ரகத்தன கலந்து செறிந்து
கற்போல் திரண்டு நீருகவாய்களில்
நிறுநீர்க் குழாய்கள் அடைத்து நிற்பது
நீரகக் கல்லென நகழ்ந்துவர் சான்றோர்

நீரியக் கல்லடைப்பு

''பிசைந்தென நீரியவலிதோன் றிடுதல்
சிறுநீ ரயைடல் எனுமிவை பிறவும்
நீரியக் கல்லடை விளைத்திடு மென்ப''

அனலக் கல்லடைப்பு

''அனலின் கூறுகள்திரண்டு செறிந்து
கற்போல் வடிவாய் அலைக வாயை

அடைப்ப தலைகக் கல்லடைப் பாகும்
காய்தல் வெளிறல் துயர்தல் வலித்தல்
எதிரெடுப் பாதல் தோளில் வலியுறல்
எனுமிவை பிறவும் மவ்வழித் தோன்றும்
கல்லது குடலிற் புகுந்திடு மாயின்
கூறிய குறிகள் மாறிடு மென்ப
அலைகத் தக்கல் புகுந்து செறியின்
காய்தல் நோதல் ஆக்கை வெளிறல்
காமலந் தோன்றல் மெலிதல் பிறவும்
தலைவரு மென்ப நிலையறி நூலோர்
கல்லது மருந்தில் கரையா தாயின்
அறுவையா னீக்கல் மரயென் மொழிப''

தீராதன

''நீரக முகம்புரை படுதல் சீறி
அனல் மிகுதல் உனுமிவை தோன்றின்
சிறுநீர்ப் பிணிகள் தீரா வாகும்''

வகை

''வளிமுதல் மூன்றினுந் தோன்ற லாலும்
கருநீர் தன்னிற் றோன்ற லாலும்
கல்லடை நால்வகைப் படுமென மொழிப''

பொதுக்குறிகுணங்கள்

''வந்தி தன்னில் அதன்கீழ் மருங்கினும்
விரைநரம் பிடத்தும் நோவு தோன்றல்
சிறுநீர் நெறியில் கல்லுறுத் தடுப்பின்
முரிந்து முரிந்து நீர்வீழ்ந் திடும்
கல்லது விலகி நின்றிடின் சிறுநீர்
தெளிந்தின மஞ்சள் நிறுத்தி லொழிதல்
எனுமிவை கல்லடைப் பொதுக்குறி யென்ப
நீரியம் இயக்கும் உழப்பு மிக்கிட்டில்
நீரது குருதி கலந்து வெளிப் படலும்
நோவு தோன்றலு மியல்பென மொழிப''

- Renal colic
- Dysuria
- Testicular pain
- Haematuria

வளிக்கல்டைக் குறிகள்

''கறுத்தும் சிவத்தும் முளைகள் பரந்தும்
வளியின் கல்லது வடிவுறு மென்ப''

அனலக் கல்லடைக் குறிகள்

''சுட்டென நீரியம் மிகவெப் பிடுத்தலும்
நோதலும் அனலக் கல்லடைக் குறியே
சிவந்தும் கறுத்து மஞ்ச ளாகியும்
சேங்குரு வடிவில் கல்லது தோன்றும்''

ஐயக்கல்லலடைக்குறிகள்

''நீரியங் குத்தல் திணித்தல் குளித்தல்
எனுமிவை ஐயக் கல்லடைக்குறியே
வெளுத்தும் தேனிற மாகியு மொளிர்ந்தும்
பெருவடி வுடைத்தாம் ஐயக் கல்லடை''

கருநீர்க்கல்லடைக்குறிகள்

''நீரியம் நோதல் சிறுநீர் தடைப்படல்
விரைவில் கிடுதல் எனுமிவை பிறவும்
கருநீர்க் கல்லடைக் குறியெனமொழிப''

Vali, Azhal, Iyya Kalladaippu commonly stone sited only at Kidney and Ureter, but Veener kalladaippu stone is sited in the Prostate gland(Vesicular stone).

B. In yugi vaidhya chinthamani-800

''தோன்றிடதோர் நாலினிட நாமங்கே ளாய்
சுறுக்கான வாதத்தின் கல்ல டைப்பு
பூன்றியதோர் பித்தத்தின் கல்ல டைப்பு
புரண்டதோர் சிலேட்டுமத்தின் கல்ல டைப்பு
தீன்றியதோர் தொந்தமாங் கல்ல டைப்பு
தேகத்தைப் பற்றியே சிறிது காலம்
தான்றியே சலப்பையில் வந்தி ழிந்து
சருவியே லிங்கத்திற் றரிக்குந் தானே''

பா 728, பக்கம் 284

The above poem mentioned in the yugi vaidhya chinthamani-800, classifies kalladaippu noi in to four types. They are,

1. Vali kalladaippu
2. Azhal kalladaippu
3. Iyya kalladaippu
4. Mukkutra kalladaippu

Vali kalladaippu

"தரித்து நாபிக்கு சுருக்கமாய் குற்றிச்
சலமலந்தான் வீழாமற் றம்ப மாகி
வரித்துமே லிங்கத்தில் வலியு மாகி
மருவியதோர் பொத்தியெலாஞ் சுரந்து கட்டி
திரித்தியே கிடைக்கொடாப் பிரட்டலாகித்
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்ததோர் சதைபோல உவர்ப்பு மாகும்
ஓங்கியதோர் வாதக்கல்ல டைப்பு தானே"

பா 729, பக்கம் 284

This poem clearly states that in vali kalladaippu, patients suffer from acute pricking pain in the lower abdomen, scanty micturition, obstruction of urine flow, pain in the glans penis making them unable to sit. The other symptoms include pain in abdomen, albuminuria, and mucous discharge. Finally stone gets expelled with urine.

Azhal kalladaippu

"அடைப்பாகிச் சலந்தானு மருவ லாகி
அயங்காச்சிச் சொருகினாற் போலே காணும்
புடைப்பாகப் பொற்றியெங் கும்பு ழுக்கமாகிப்
பூட்டுப்போல் பிகுவாகிப் பிரட்ட லாகும்
மடைப்பாகி உதிரநிற மாயக்கல் லாகி
வந்தழிந்து லிங்கத்தில் மாட்டிக கொள்ளும்
குடைப்பாகிக் குற்றலாய்க் கூச்சலாகிக்
குதட்டுமே பித்தக்கல் லடைப்பு தானே" **பா 730, பக்கம் 285**

It is evident from the above poem that the cardinal features of azhal kalladaippu include obstruction of urine flow, burning sensation in external urinary meatus, acute pain in the urethra and excretion of small blood stained stones.

Iyya kalldaippu

"தானா தொப்புளிலே வில்லு போலச்
சலியாமற் சுரந்துமே சற்றே குற்றும்
ஏனான காலோடு கைகள் சந்து
இடுப்புதான் குடைச்சலா யிசிவு காணும்
வேனான லிங்கத்தின் வெண்மை தன்னில்
விறவிறென் றேகடுப்பாகி வியர்வையாகும்
தேனான வெளுப்புக்கல் சிறுகல் லாகிச்
சிக்கலாய் வந்திறங்குஞ் சேட்பந் தானே"

பா 731, பக்கம் 285

This poem clearly mentions that the symptoms of iyya kalldaippu are acute pain around the umbilicus, pain radiating towards thigh, joint pain, Burning micturition, profuse sweating and expulsion of small white stones.

Mukkutra kalladaippu

"வந்திறங்கும் நீர்த்தாரை யடியிற் றானும்
மாவருத்த முண்டாகி வலியு மாகி
நொந்திறங்கி நீர்தானு மருவி பாயும்
நொயதான சிறுமணல் போல் நொறுங்கி கல்லான்
சந்திரங்கி நீர்வழியில் வந்து வீழும்
தாக்கான சிறங்கைக்கல் தினமொன் றுக்கு
துந்திறங்கித் தினந்தினமு மிழந்து கொல்லும்
தொந்தமாங்க லடைப்புச் சூட்டிட் டாயே

பா 732, பக்கம் 286

B. In siddhar aruvai maruthuvam

Kalladaippu noi is classified into four types

1. Vali kalladaippu
2. Azhal kalladaippu
3. Iyya kalladaippu
4. Veneer or manal kalladaippu **Page No - 112**

C. In thanvanthiri vaithyam {IInd part, page no. (9,10)}

"திருத்திய வாத பித்த சிலேற்பனம் பிரகோபித்தால்
வகுத்தசு மரித்தா நான்கு வகைப்படும் கல்லரிப்பான்
பிரித்திடுஞ் சிலேற்பனாசு மரிபித்தா சுமரி பின்னு
மிருத்திடு சுக்கிலாசு மரிநான்கு மெய்துமென்றே"

The alteration of three dhoshas results in occurrence of the following four types of Ashmari

1. Kallarippan
2. Pitha ashmari
3. Silathma ashmari
4. Sukkila ashmari

Kallarippan

"சுத்துநீர் நாளந்தன்னீற் சுக்கிலந்தன்னீற் சிலேற்பனம்
பித்தமீ துலர்த்தல் கல்லாய் பீசகிநீ டைத்துக் கொள்ளுங்
கொத்துநீ ரிற்றுவீழுங் கொப்புள்நோ குடம்பு காயுஞ்
சித்தா யருசி யுண்டாஞ் சேர்ந்தகல் லெரிப்பானாமே"

It is learnt from this poem that when kabam and pitham increase, urine and semen dry up, resulting in the formation of stone which in turn lead to obstruction of urinary tract, dysuria, pricking pain in umbilicus, fever and anorexia.

Slathma ashmari

"நீர் வரு தாளத் தன்னில் நின்றுநீர் சிறுத்துக்கொண்டு
சோர்ந்தரும் சிவப்பு வெண்மை சுக்கிலம் போல வீழும்
பேர்பெற நாலாமெட்டும் பின்னமாய் கல்லு வீழும்
ஏர்பெறு சிலேற்பனத்தில் அச்மரி என்னலாமே"

This poem states that when the calculus arrive the urethral orifice, the volume of urine gets reduced gradually, leading to oliguria. Ultimately, the stone is broken into four to eight pieces and pass through the urine.

Pitha ashmari

"பெய்யும்நீர் நாளத் தன்னில் பித்தத்தா லெரிப் பெழுந்து
செய்யுவுஷ் ணத்தால்வெந்து சேங்கொட்டை போற்கல் லுண்டாம்
நய்யவே தனைகள் செய்யும் நவில் குணம் பித்தந்தன்னில்
எய்தசு மரியென்றெமுன் னியம்பின ரறிவின் மிக்கோர்"

It is evident from this poem that when pitham accumulates in the urethral orifice, severe pain will result in along with burning sensation.

Sukkila ashmari

"சுக்கிலம் வருங்காலத்தில் தம்பித்தாற் சுக்கிலந்தான்
மிக்ககல் லாகிவெம்பி விதனமாய் நீர்விடாமற்
சிக்கிநீர் விழா மலங்கே மணல் விழும் வெளுக்குந்தேகம்
மிக்குணஞ் சுக்கிலாசு மரியசாத் தியமென்றோதே" 18

If semen discharge is ceased it gets concentrated and dried up in the uretero-vesical junction, preventing the flow of urine which results in anuria. Under this condition sand grains like stones pass through the urine and anemia occur.

D. In roga nirnaya saaram under roga nithanam (page.79&80)

Ashmari rogam is classified into five types as given below.

1. Vatha ashmari
2. Pitha ashmari
3. Kapha ashmari
4. Sukkila ashmari
5. Swargara ashmari or kalladaippu rogam

1. Vatha ashmari

Urine turns to black in colour, dribbling, grunting teeth and rubbing of penis.

2. Pitha ashmari

Tip of penis turns to black in colour and urine turning to red, white and yellow in colour

3. Kapha ashmari

Urine becomes white in colour and highly viscous and flows down like honey.

4. Sukkila ashmari

Cessation of seminal fluid discharge during sexual intercourse leads to retention of semen in vas deferens or spermatic cord, which get dripped up by vatham. This condition eventually results in inflammation of testis and lower abdominal pain.

5. Swarkara ashmari or kalladaippu rogam

Semen is retained in the spermatic region and gets dried up. The dried semen turns to stones in the size of sand, green gram, ground nut and get mixed up with urine and pass through the urethral orifice.

In anubhava deva ragasiyam 1 part (page no.131),

Ashmari rogam is classified into five types.

- Vatha ashmari
- Pitha ashmari
- Kapha ashmari
- Sukkila ashmari
- Swarkara ashmari or kalladaipu rogam

Vatha aasmari

Due to increased vatham, rigor, grunting teeth, rubbing of penis and umbilicus, crying, vomiting, forced excretion of faeces, dysuria and dribbling of black colour urine occur.

Pitha ashmari

The symptoms include increased temperature in the lower abdomen, flatulence, Red or high concentrate urine.

Kapha ashmari

The cardinal features are acute pain in the lower abdomen and urine turning to white in colour and highly viscous and flowing down like honey.

Sukkila ashmari

The symptoms are pain and inflammation in the testis, lower abdominal pain, urine taking a long time to get expelled during urination and pain during micturition.

Swarkara ashmari or kalladaippu rogam

The dried semen is crushed by vatham into small stone of various sizes ranging from fine particles of sand or mustard to as large as green gram, or Bengal gram and even dates which block the passage of urine. If vatham is in normal level, stones will be expelled during micturition.

In **sikitcha rathna deepam-vaidhya chinthamani-2nd part (page no.140, 141)** Kalladaippu rogam is classified into following four types.

i. Vatha kalladaippu
ii. Pitha kalladaippu
iii. Kapha kalladaippu
iv. Mukkutra kalladaippu

Vatha kalladaippu

The symptoms include acute pricking pain in the lower abdomen, obstruction of the flow of urine, scanty micturition, pain in the penis, making the patient unable to sit, crying, flatulence, presence of albumin in the urine and mucous discharge with urine.

Pitha kalladaippu

The symptoms are obstruction of the flow of urine, burning sensation in the urethral orifice (the burning sensation refers to melting point of iron put on the external meatus), acute pain in the urethra and expulsion the small blood stained stones.

Silothuma kalladaippu

In this kalladaippu, patient suffers from severe pain in the lower abdomen, pain radiating towards the thigh, joint pain associated with rigor and pain in the tip of glans penis. Finally, small white coloured stones are expelled along with urine.

Mukkutra kalladaippu

Pain in the urethral orifice, pain during micturition, presence of small sand grain like stones in urine are the cardinal features of mukkutra kalladaippu.

Mukkutra verupadugal (pathology)

The three uyir thathukal are formed by combination of

S.NO	**Dasanadikal**	**Dasavayukkal**	**Humours**
01.	Idakalai	Abanan	Vatham
02.	Pinkalai	Praanan	Pitham
03.	Sulumunai	Samaanan	Kapham

Under normal condition, the three factors tend to be in their states of equilibrium (1:1/2:1/4)

‘‘உற்றதோர் உடலின் கூறு
உறுப்புடன் விரவி நின்று
முற்றுமே நோய்களெல்லா
முதிர்பெறத் தோன்றும் போது
பற்றுமே வாதபித்த
சிலேற்பனந் தன்னில்லொன்றை
பற்றியே தோன்றுமென்று
பகர்ந்தனர் முனிவர் தானே’’

அகத்தியர் குருநாடி பக்கம் - 65

As per the siddha principles, the manifestation of any disease is the result of disturbed kutrams (ie, vatham, pitham, kapham).

Mukuttra vaerupaadugal

‘‘வாயுவினால் மலசலங்கட்டிடும்
பிரித்திடும் பித்தம் பேராஞ் சலத்தினாலே

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 154

The above poem mentioned in siddha maruthuvanga surukkam emphasizes that alteration in diet, salt and water intake will lead to alteration, mainly increase in vatha and pitha kutram in the body, which in turn result in sedimentation of urine in the kidney. Under this condition, if abaanan is favorable, these deposits do not get expelled with urine. Instead, they get deposited in the urinary tract, which pave way for the occurrence of this disease.

Functions of mukkutram

Vatham	Pain in the body, twitching pricking pain, inflammation, reddish complexion, dryness of skin, hardness of limbs, astringent sense of taste in the mouth, unpalatable taste, sweating during sleep, traumatic pain, constipation, **oliguria, blackish discoloration of skin, stool and urine and muddy conjunctiva.**

Pitham	Hyperacidity, burning sensation in the stomach, **yellowish discoloration of skin, eye**, urine, sense of defecation, **profuse sweating**, dizziness.
Kapham	Fair complexion, itching, dullness, cold, heaviness, loss of sensation, sweetness in mouth, indigestion.

Roles of mukkutram

Abnormal change in these three mukkutram lead to abnormal changes in structure, function and behavior of various body organs. These changes are listed below.

	Increase	**Decrease**
Vatham	Tremors, distended, abdomen, constipation, weakness, insomnia,	Body pain, feeble voice, diminished competence of intellectual functions, syncope etc...
Pitham	Yellowish discoloration of eyes, skin, urine and motion polyphagia, polydipsia, burning sensation all over the body, sleeplessness.	Decreased appetite, cold, pallor, symptoms associated with defective growth of kapham.
Kapham	Loss of appetite, excessive salivation, Heaviness, excessive musculature, Dyspnoea, excessive sleepiness.	Prominence of bony edges, dry cough, lightness, profuse sweating, Palpitation.

Vatham

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations, It is classified into the following ten types. They are

S No	Vatham	General features	Changes in kalladaippu
1.	Piranan (uyirkaal)	Responsible for respiration and it is necessary for proper digestion	Normal
2.	Abanan (keezh nokku kaal)	Responsible for all downward forces such as voiding of urine, stools, semen, menstrual flow	Affected (scanty micturition, haematuria (due to sharp edged stones) and dusuria.
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.	Udhanan (melnokkuka al)	Responsible for all kinds of upward motion such as nausea, vomiting .	Affected(nausea, vomiting)
5.	Samanan (nadukkaaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ.	Normal
6.	Naagan	Helps in opening & closing of eyelids.	Normal
7.	Koormam	Responsible for vision , lacrimation and yawning	Normal
8.	Kirugaran	Induces appetite, salivation , all secretions in the body including nasal secretion and sneezing.	Normal
9.	Thevathathan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep	Normal

10.	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3days of death, forming a way through the skul	Normal

Pitham

It is the terminal life force of the body. It is subdivided into five types. They are,

S.No	Pitham	Normal features	Changes in Kalladaippu
1.	Analapitham	Promotes appetite and 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 complexions.	Normal
5.	Alosagapitham	Responsible for the perception of vision.	Normal

Kapham

It is responsible for the streamlined functions of the body and maintains body's defence mechanism intact. It is classified into five types as below

S.no	Kabham	General features	Changes in Kalladaippu
1.	Avalambagam	Lies in the respiratory organs, exercise authority over other kapas and controls the heart and circulatory system.	Normal
2.	Kilethagam	Found in stomach as its seat, moistens the food, softens and helps to be digested.	Normal

3.	Pothagam	Hold responsible for the sensory 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.	Patient will not able to walk and move the joints (hip and knee joints) freely.

Udal thathukkal

When the functional elements (vatham, pitham, kapham) are upset repercussions are felt immediately over the components due to alteration in the nature of somatic components.

Saaram (digestive enzyme)

It is responsible for the growth and development. It keeps the individual in good temperament and enriches the blood.

In kalladippu noi, saaram is affected by change in our food habits, which results in increase in level of vitamin D and become hypervitaminosis. Increased vitamin D is a main cause for kalladaippu noi (renal calculi).

1. Senneer (blood)

It is responsible for the colour of blood and for the intellect, nourishment, strength, vigour and valour of the body.

2. Oon (muscle)

It gives suitable contour to the body, required for the physical activity. It feed the fat next day and gives a sort of plumpness to the body.

In kalladaippu noi, oon is affected. When there is a change in the level of vitamins due to change in our food habits. The decrease in vitamin A level may lead to destruction and shrinkage of the mucous membrane in the urinary tract. Sometimes, damaged mucous membrane get mixedup with urine and expelled.

3. Kozhuppu (fat)

It lubricates the organs to facilitate frictionless function.

4. Enbu (bones)

It supports and protects the vital organs, structure to the body and is responsible for the posture and movement of the body.

5. Moolai (bone marrow, brain)

It nourishes the bone (marrow) and the brain which is the centre that controls all systems of the body.

6. Sukkilam or suronitham (sperm & ovum)

It is responsible for reproduction.

In kalladaippu noi, sukkilam is affected by the increased body heat and hence semen in the spermatic region get dried up and shrunken and become small vesicle stones.

Udal thathukkal	**Increased features**	**Decreased features**
i. Saaram	Leads to a disease identical to increase in kabam like loss of appetite, saliva secretion, depression .	Loss of weight, lassitude, dryness of the skin and dimished activity of the sense organs.
ii. Senneer	Increased blood pressure, reddish eye and skin, jaundice, haematuria.	Tiredness, lassitude and anaemia.
iii. Oon	Excessive muscle growth around the neck, cheek, abdomen, thigh, external genitalia .	Muscle wasting
iv. Kozhuppu	Identical features of increased oon associated with dyspnoea on exertion	Hip pain, liver enlargement and emaciation.
v. Enbu	Excessive ossification and dentition	Joint pain, teeth falling, crack in nails and hair falling.

vi. Moolai	Weariness of the body and eye, swollen interphalangeal joints, oliguria and rarely healing wound.	Osteoporosis and sunken eyes.
vii. Sukkilam or suronitham	Increased sexual activity, urinary calculi .	Pricking pain in genitalia and Impotence or infertility.

Udal Vanmai(Immunity)

1. Iyarkai vanmai(Natural immunity)

The natural immunity of the body is present even at the time of the birth .This is a nonspecific immunity due to the passing of maternal antibodies to the foetus.

2. Seyarkai vanmai(Acquired immunity)

Improving the health by nutritious food,medicines,karpams,vaccines all fall into this category of increasing the immunity by human measures.There are both specific special and non specific special karpams comes under the category specific immunity or seyarkai vanmai since such karpams are used against acquired diseases both as
prevention and treatment for them.

3.Kala vanmai

Developing the immunity and stamina according to the age of the person, seasons and environmental factors.

Noi kanippu murai (diagnosis and prognosis)

In piniyari muraimai , the following principles are followed in siddha system. they are,

1. Poriyaal arithal - Physical examination ,Perception
2. Pulanaal arithal - Clinical examination
3. Vinaathal - Medical history

The maruthuvar (physician) should observe the patient, palpate and interrogate the patient thoroughly.

Poriyaal arithal and pulannal arithal

Poriyaal arithal means understanding by the five organs of perception, whereas pulannal arithal is the understanding by sensing the objects. They are,

- Mei - ooru (somatic ense)
- Vai - suvai (taste)
- Kan - Oli (vision)
- Mookku - natram (smell
- Sevi - osai (sound)

Pulanal arithal

- Kai - All manoeuvres
- Kal - Walking
- Vai - Speaking
- Eruvai - Defecation
- Karuvai - Reproduction

Vinaathal (interrogation)

The first and foremost step in diagnosing a disease is to get to know the personal history of the patient through interrogation. By vinaathal, the physician should ask the patients about their native place, socio economic status, food habits, personal habits, complaints, and duration of illness poriyal arithal, pulanaal arithal and vinaathal are appliled through eight tools of investigation that are “envagai thervugal”.

Mukkutra kaalam (age distribution)

The period of human life of 100 years is divided into three stages.

Stage	**Year**	**Dominant humor**
First stage	01 -33 years	Vatha period
Second stage	34- 66 years	Pitha period
Third stage	67 -100 years	Kaba period

Thinai (land or place)

The study of dwelling places of patients is essential as the prevalence of endemic disease is very common in certain areas.

Generally, the nilam is classified into five. They are

Kurinji nilam	mountain and its surrounding
Mullai nilam	forest and its surroundings
Marutha nilam	fertile plains and their surroundings
Neithal nilam	seashore and their surroundings
Palai nilam	deserts and their surroundings

Marutha nilam people are more prone to disease than other land people, as fertile lands of Maruthanilam is used for industrial purpose which have way for urbanization and settlement of huge population in smaller area.

Paruvakaalam (season)

In siddha system of medicine, siddhars classified a year into five seasons, each having two months.

S.No	Kaalam	Kutram	State of kutram
1.	Kar kaalam (Avani & Purattasi) (Aug. 16-Oct .15)	Vaatham Pitham	Vetrunilai valarchi Thannilai valarchi
2.	Koothir kaalam (iypasi & karthigai) (Oct.16-Dec.15)	Vaatham Pitham	Thannilai valarchi Vetrunilai valarchi
3.	Munpani kaalam (Margazhi & thai) (Dec.16-Feb.15)	Pitham	Thannilai adithal
4.	Pinpani kaalam (masi & panguni) (Feb.16-Apr. 15)	Kapam	Thannilai valarchi
5.	Elavenir kaalam (chithirai & vaigasi) (Apr.16-june 15)	Kapam	Vetrunilai valrchi
6.	Mudhuvenir kaalam (Aani & Aadi) (June. 16- Aug.15)	Vaatham	Thannilai valarchi

Envagai thervugal

"அன்பான சாத்திரங்கள் அறிய வேண்டும்
அன்பான நாடிதனை பிடிக்கவேண்டும்
குன்றான மலைபோன்ற நாடியெல்லாம்
குறிப்புடன் அசாத்தியமுஞ் சாத்தியமுங்கண்டு
தன்றான அட்டவித பரீட்சை கண்டு
தக்கான குணங்குறிகள் யாவுந் தேர்ந்து
வன்றான வாகடத்தின் நுணுக்கம் பார்த்து
வளமாக பிணியதனைத் தீர்ப்போர் தாமே

யூகி வைத்திய சிந்தாமணி

"நாடி ஸ்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்

அகத்தியர் நாடி

"மெயக்குறி நிறந்தொனி விழிநாவிருமலம் கைக்குறி"

தேரையர்

The above mentioned poems list out the Envagai thervugal as below,

1. Naadi (Pulse)
2. Sparisam (Palpation)
3. Naa (Tongue examination)
4. Niram (Colour of the body)
5. Mozhi (Speech)
6. Vizhi (Eye examination)
7. Malam (Motion examination)
8. Moothiram(Urine examination)

Gunavagada naadi emphasises about the Envagai thervugal as the following

"தரணியுள்ள வியாதி தன்னை யட்டாங்கத்தால்
தானியறிய வேண்டுவது யேதோ வென்னில்
திணியதோர் நாடிகண்கள் சத்தத்தோடு
தேகத்தினது பரிசம் வருணம் நாக்கு
யிரணமல மூத்தரமாவிவைக ளெட்டும்
யிதம்படவேதான் பார்த்து குறிப்புங்கண்டு
பரணருளால் பெரியோர்கள் பாதம் போற்றி
பண்பு தவறாமல் பண்டிதஞ் செய்வீரே"

குணவாகட நாடி

நோய் நாடல் நோய் முதல் நாடல் திரட்டு – முதல் பாகம், பக்கம் - 129

The above poem that it is imperative to apply the Envagai thervugal for diagnosis of the diseases.

"தொகுக்கலுற்ற அட்டவித பரீட்சை தன்னை
துலக்கமுறும் பண்டிதரே தெளிவதாகப்
படுக்கரிய நாடியை நீ பிடித்துப்பாரு
பகர்கின்ற வார்த்தையைப் பார் நாவை பாரு
வகுக்கரிய தேகமென தொட்டுப் பாரு
வளமான சரீரத்தின் நிறத்தைப் பாரு
சகிக்கரிய மலத்தைப் பாரு சலத்தைப் பாரு
சார்ந்த விழிதனைப் பார்த்து தெளிவாய் காணே
நீடிய விழியினாலும் நின்ற நாக்குறிப்பினாலும்
வாடிய மேனியினாலும் மலமொடு நீரினாலும்
சூடிய வியாதி தன்னைச் சுகம்பெற அறிந்து சொல்லே"

அகத்தியர் வைத்திய வல்லாதி

Naadi nadai in kalladaippu

"விழுகும் சிலநேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுவும் ஒதுங்கினால் நோகாது
வழுகிய மந்தத்தால் வாயுவந்தே புகில்
கழுமி முதிர்ந்திடும் கல்லெரிப்பு ஆகுமே"

திருமூலர் கருக்கிடை வைத்தியம் 600

When the vatham and mantham combined together, kalladaippu noi may occur.

"அறைந்தோம்வாத ரோகியுடல்
அழறக்ண்முகமும் பல்மலமும்
நிறைந்த விழியில் நீர் வடியும்
நீண்ட நாவு கறுத்திடவும்
திறைந்தமுள்ளாய் தானிருக்குஞ்
சிறு நீர் பொருமி கடுத்து வரும்
உறைந்த நீருங்கருகருத்து
முறையாய் ரோகமு முண்டாமே"

அகத்தியர் நாடி

நோய் நாடல் நோய் முதல் நாடல் திரட்டு – முதல் பாகம், பக்கம் - 165

"மேவியவாதஞ் செய்யும்
குணந்தனை விரும்பிக்கேளு
தாவிய வயிறு மந்தஞ்

சந்துகால் பொருத்து நோவாம்
சேவிய தாது நாசஞ்
சிறுத்துடன் சிறு நீர்வீழும்
காவியங் கண்ணினாளே
மலமது கருகிக் காணும்''

இரத்தின சுருக்க நாடி

Agaththiyar Naadi and Raththina churukka naadi describes that aggravation of vaatham produces symptoms of kalladaippu noi.

''பொருளான வாதத்தால் பித்தஞ் சேர்ந்து
பொருத்து குணங்களா முஷண வாயுசத்தி
செரியாமை புளித்தேப்பம் பொருமல் நீரிற்
சிவப்பு மலம் பிடித்தலுருந்தாது நட்டம்
கருவான தேகமதிலுளைச்சல் சோம்பல்
கை கால் தறிப்பு நாக்கசக்கு மன்னம்
பரிவான வூண் குறைதல் ருசி கேடாதல்
பல நோயும் வருத்திவைக்கும் பாங்கு தானே''

சதக நாடி

நோய் நாடல் நோய் முதல் நாடல் திரட்டு – முதல் பாகம், பக்கம் - 171

''எண்ணிய வாதமொன்றும் பித்தமிரண் டெழுந்தாகில்
புண்ணென யுடம்பு நோவாம் புகையெழ யெரியும் நெஞ்சு
திண்ணமாய் நாவரண்டு சிறுத்துநீர்க் கடுத்து விழும்
அண்ணவார் உரைந்தவுண்மை யாயுரு தேவன்தானே''

தன்வந்திரி வைத்தியம் - முதல் பாகம், பக்கம் 11

Sathaganaadi and Dhanvanthiri vaidhyam describes, when vatham and pitham combined together which may result kalladaippu noi.

ஸ்பரிசம் (touch)

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.

On examination in kalladaippu patients, tenderness over the lower abdomen, renal angle and lumbar region can be felt in kalladaippu noi. In vali kalladaippu, swelling can be felt (may be due to hydronephrosis).

In pitha kalladaippu, body temperature increase in lower abdomen. In iyya kalladaippu, sweating occurs all over the body at the time of colic.

நா (**Tongue**)

The physician is expected to observe the features of the tongue and its associated structures that include colour, size, shape, coating, moisture, movement, ulcer, fissure, crust and condition of teeth and gums. In kalladaippu noi, if the patient suffers from constipation (valikalladaippu), the tongue would seem to be coated.

நிறம் (Colour)

The physician is required to observe the colour of skin, conjunctiva, tongue, nail bed hair etc and make a note of any abnormal colour changes. Normally,

- Vali udal - Black colour
- Azhal udal - Yellow or red colour
- Iyya udal - white or yellow colour

In kalladaippu noi, body complexion depends upon the body constitution. Pallor of the body is observed in sukkila ashmari.

"சிக்கிநீர் விழா மலங்கே மணல்விழும் வெளுக்குந்தேகம்
மிக்குணஞ் சுக்கிலாசு மரியசாத் தியமென்றோதோ"

தன்வந்திரி வைத்தியம் - 2 பாகம், பக்கம் 10

மொழி (Speech)

By examining mozhi (speech), characters, hoarseness, slurring speech, various disorders of speech such as dysarthria can be noted. In kalladaippu noi, there is low pitch voice due to agonizing pain in lower abdomen and burning micturition.

விழி (Eye)

The colour of conjunctiva is observed in kalladaippu noi, patients, the conjunctiva is pallor due to haematuria. Because haematuria may cause secondary anaemia.

மலம் (Stools)

It is necessary to examine the nature, colour and quantity of stool and presence of blood or pus. In vali kalladaippu, proctalgia and oliguria occur due to renal colic.

“தரித்து நாபிக்குங்கீழ் சுருக்காய் குற்றிச்
சலமலந்தான் வீழாமற் றம்பமாகி”

யூகி வைத்திய சிந்தாமணி 800, பக்கம் - 284

Urinary examination is a good diagnostic method compare to naadi and other Envagai thervugal. Theraiyar mentioned about urine examination as below.

நீர்க்குறி சிறப்பு

“தர்க்கசாத்தி திரிக ளானோர்
தங்களிற் றேர்ந்து நாடி
வர்க்கமாம் நாடி தன்னில்
வருவது மயக்கம் மென்றே
உற்றநீர்ப் பரீஷை பாய்ந்தே
யுரைத்தன ரிதற்கு நேராய்
மற்றொரு விதிநூ லில்லை
மருத்துவ கலைவல்லோர்க்கே”

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 568

Siruneer pothugunam

“வந்தநீருக் கரியெடை மணம் நுரை எஞ்சலேன்
றைந்திய லுளுவை யறைகுது முறையே

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 510

The above poems states that the five parameters should be examined in each urine sample.

1. Niram (Colour)
2. Edai (Specific gravity)
3. Nurai (Froth)
4. Natram (Smell)
5. Enjal (Deposits)

NIRAM (colour)

Nira thogai

“பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதங் கொழுமையை யொத்துகு நீரே

சித்த மருத்துவாங்கச் சுருக்கம் பக்கம் 510

The above poem reveals that urine colour may be any one of the followings,

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

கல்லடைப்பு நீரின் குணம் (colour indicating urinary stones)

''தீப்புலால் கழுநீர்ச் செயலெனிற் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறப்
பற்பக லாகப் பையைப் பதிந்தே''

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 520

The urine colour resembling the colour of flesh washed water, indicate the occurrence of kidney diseases. (crystaluria)

Edai (specific gravity)

''மிகத்தடிப் புமமிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும்மெய்ச் சுபாவநீர் நன்றே''

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 528

urine which is not thick, is considered healthy

Nurai(Froth)

''பந்தமெய்ப் பசையிள கப்படும் பருவத்
தந்தர்ப் பூதமாய நிலமுத் திரத்தில்
சம்பந்தப் படும் ததி நுரைப் புனலே''

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 528

Urine is frothy in nature under normal condition. When there is a change in vali, Iyyam there will be a change in the frothiness of urine too.

Naatram (Smell)

மணவிலக்கணம்

''காணிதில் சீழும் கலந்திழி மணமுறின்
கருப்பநா பிகளுளுங் காமநாளத்துளும்
விரணமுன் டின்றேன் எய்துமஸ் மரியல
திருத்தலே திண்ண மெனமனத் துன்னே''

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 519

The above states that foul odour with pyuria is observed in patients with urolithiasis associated with secondary urinary tract infection and urethral ulcer.

Enjal (Deposits)

"நார்த்தி நீர்பால் போல
நவையுற்றங் கிழியு மானால்
மாரற்ப முற்ற நீரி
லடிமண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய்
பற்றிய கல்லினாலே
சீருற்ற செய்கை யென்று
தெரிவுற செப்ப லாமே

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 575

In siddha maruthuvanga surukkam the following clinical features have been mentioned with regard to kalladaippu noi. They are,

Pyuria - Curd like micturition (cloudy)

Crystaluria - Sand like deposits

Neikuri

"அருந்துமா றிரதமும் அவிரோ தமதாய்
அ.்.கல் அலர்தல் அகாலவூண் தவிர்ந்தழற்
குற்றள வருந்தி உறங்கி வைகறை
ஆடிக் கலசத் தாவியெ காதுபெய்
தொருமுகூர்த் தக்கலைக் குட்படு நீரின்
நிறக்குறி நெய்குறி நிருமித்தல் கடனே"

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 509

Preparation of Patient

Prior to the day of urine examination for Neikuri, the patient should be advised to take balanced diet and the quantity of food must be proportionate to his appetite, In addition, he/she should have a good sleep.

Method

After waking up in the early morning, urine should be collected in a glass container and must be examined within 11/2 hours. Then a drop of gingelly oil should be added through the side of the container without any disturbance. The nature of neikuri should be noted under direct sunlight.

Observation

Vaatha neer

“அரவென நீண்டின் அ.ஂதே வாதம்”

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 532

If the drop of oil lengthening like a snake, it indicate vatha neer.

Pitha neer

“ஆழிபோற் பரவின் அ.ஂதேபித்தஞ்”

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 533

If the drop of oil spreading like a ring, it indicates pitha neer.

Kapha neer

“முத்தொத்து நிற்கின் மொழி வதென் கபமே”

சித்த மருத்துவாங்கச் சுருக்கம், பக்கம் 534

If the drop of oil remaining as that of pearl, it indicates kapha neer.

Thontha neer

“அரவிலாலியும் ஆழியில் அரவும்
அரவின்முத்தும் ஆழியில் முத்தும்
தோற்றில் தொந்த தோடங்களாமே”

The thontha neer appears as a combination of the above patterns.

Mukkutra neer

The drop of oil immersing in to the urine, indicates mukutra neer.

NOI KANIPPU VIVAADHAM

(Differential diagnosis of Kalladaippu)

Neeradaippu

It is characterized by decreased urinary output along with burning sensation of urinary bladder (Neerpai) and urethra (Neerpuzhai) with distend abdomen.

Eventually it causes unbearable pain in voiding of urine with fresh blood after/before micturiation. It results in difficulty in breathing, swelling of face, back pain, inflammation of urethra and abdominal pain.

Neer churukku

"நீரினை யருக்க லென்னும் நீர்க்கட்டின் குணத்தைக் கேட்டு
நீதமில் லாமற்கோச நீர்ப்புழை நெருப்பு போலாம்
நீபனா யுதத்தாற் பட்ட நீலவம் பராமாங் குக்கி"

Theraiyar karisal, theraiyar stated that the disease is charecterized by reduction in the formation of urine, micturition burning sensation, pain in the neer puzhai and facial odema may result.

It also symptoms like dribbing of urine. During micturation urinary out put is less in amount, volume, red in colour, abnoxious/fish smell in urine, long time micturation , burning sensation, dribbing of urine and painful urethra.

The disease may also cause with out above signs and symptoms. While passing urine sudden blockage of urine outflow, pain in urethra, burning sensation, pain in flanks and back side of the hip may results sand like stone may be observed in urine.

சாத்தியம், அசாத்தியம் (Prognosis)

"சூட்டிட்ட சாத்தியத்தை சொல்லக் கேளாய்
சுளுக்காகும் வாத்தின் கல்லடைப்பு
பூட்டிட்ட பித்தத்தின் கல்ல டைப்புப்
புகழான சேட்டுமத்தின் கல்ல டைப்பு
மூட்டிட்ட இதுமூன்று மசாத்ய மாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்ல டைப்புத்
தொடுசுறவே கொல்லுமிது சூட்சந் தானே"

யூகி வைத்திய சிந்தாமணி 800

According to Yugi vaidhya chintaamani and siktcharathna deepam, vatha, pitha and kapha kalladaippu noi are curable and preventable. Mukkuta kalladaippu is not curable.

According to Roga nirnaya saaram under Roga Nithanam, symptoms of Kalladaippu noi like scrotal swelling and anuria are not curable.

மருத்துவம் (**Line of Treatment)**

"வைத்தியர் செயல் வைத்தியமே"

திருமூலர் 800

The main objective of treatment is to bring down the deranged mukkutrams to natural eqilibrium by giving purgatives, which cure derangement of vatham, which is one of the causes for kalladaippu noi.

"பேதியால் வாதம் தாழும்
வாந்தியால் பித்தம் தாழும்
நசிய அஞ்சனத்தால் கபம் தாழும்"

As per the above mentioned poem purgation should be given to all patients as per their body condition. In this present study,

NILAKUMILAVER KUDINEER - 50 ml BD Morning and Evening were given to the selected patients which showed good response and in all kalladaippu noi patients.

In siddha system, treatment is not only for curing the disease but also for prevention and improving the body condition after curing the disease. This is said as kappu, neekkam and thiraippu.

The mode of preparation and properties and constituents of ingredient of the drug are given in Annexure – I.

Diet

Diet for kalladaippu patient

குருவை அல்லது மணக்கத்தை அரிசியாலாக்கிய சோறு முள்ளங்கி (Raphanus sativus) கீரைத்தண்டு, சிறுகீரை (Amaranthus gangeticus) வாழைத்தண்டு Plantain stem), அவரை (Dolichos lablab), வெண்டை (Hibiscus esculentas), வசலை (Portulacca oleracea) பசலைக்கீரை (Portulacea pudrifida) காசினிக்கீரை (Hibiscus kannabinus) பார்லி அரிசிக் கஞ்சி (Parley water) கொடுக்கவும்.

Diet restriction for kalladaippu

தக்காளி (Tomato), முட்டைக்கோஸ் (cabbage), காலி.ப்ளவர் (Cauliflower) நீக்கவும். உப்பு நிறைந்த நீர் குடிக்கக்கூடாது.

Advice

1. Patients should drink large quantity of water (4 lit/day)

2. Patient should not suppress the excretion of urine and seminal fluid.

3. Regarding prevention **Anubhava vaidhya deva ragasiyam** states the one should not suppress the excretion of Moothiram (Urine) and sukkilam (seminal fluid) is most predisposing cause for kalladaippu noi.

MODERN ASPECTS

KIDNEY

The kidney: structure and functions

Kidneys are a pair of excreatory organs situated obliquely in the retro peritoneum on either side of the vertebral column.

The kidneys are well protected, tucked in on either side of the spine. In front of the liver, behind there is the lung, constantly moving up and down.

In anatomical terms the posterior relations of the kidney are the 12th rib, diaphragm, pleura and lung and below them the quadratus lumborum and psoas muscles. The iliolinguinal and hypogastric nerves cross the quadratus lumborum muscle. Since the kidney moves up and down in respiration, at any on time it may be anywhere between the level of the 2nd and 3rd transverse processes of the lumbar vertebrae.

They remove waste products of metabolism and excess of water and salts from the blood and maintain its PH.

Location

The kidney is occupying the epigastric ,hypochondrium, lumbar and umblical region. Vertically they extends from the upper border of T12 to the centre of the body of L3 vertebra.

Size , Shape ,Measurement

Each kidney is Bean shaped

It is 11cm length

6cm breadth

3cm thickness

Weight of the kidney - 150gm in Males

135gm in Females

The left kidney is little longer and narrower than the right kidney because the long axis of kidney is directed towards and laterally .So that the upper poles are nearer to the median plane.

External Features

Kidneys are reddish brown in colour .It has following features.

Two poles

The upper pole – broad

The lower pole – pointed

Two surfaces

Anterior surface - Irregular

Posterior surface- flat

Two borders

The lateral border –Convex (close contact with the corresponding suprarenal gland)

The Medial border – concave

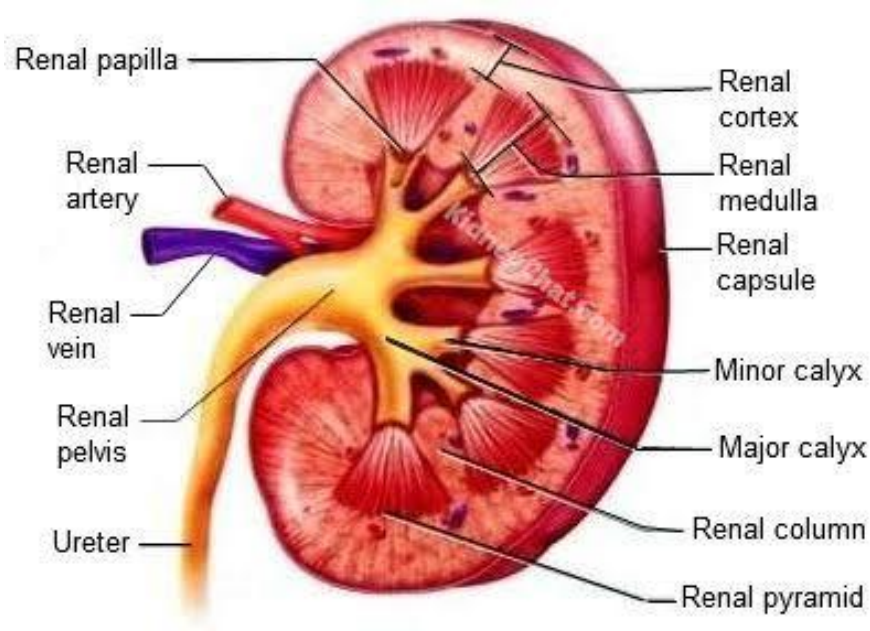
Hilum

The following structures are seen in the hilum from anterior to posterior side.

1. The renal vein.
2. The renal artery
3. The renal pelvis

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.

In the foetus the kidney is lobulated and is made up of about 12 lobules. After birth the lobules gradually fuse. So that in adults the kidney is uniformly smooth. However, the evidence of foetal lobulation may persist.



Renal system includes:

1. Pair of kidneys
2. Ureters
3. Urinary bladder
4. Urethra

Capsules or coverings of kidney

The fibrous capsule

Perirenal or perinephric fat

Renal fascia

It cosists of an anterior layer of fascia of toldt and a posterior layer of fascia of zuckerkandl.

Histologically, each kidney is composed of one to three million urineferous tubles. Each tubule consists of two parts which are embryologically distinct from each other.

Left kidney

- The tail of the pancreas and the spleen lie in front of the left kidney;
- Anteriorly the left kidney is related to stomach, jejenum, pancreas, spleen and descending colon.
- The left kidneys rises to as high as the 11^{th} thoracic ribs posteriarly, extending from T11-L3 resting on diaphragm.

Right kidney

- The right kidney is 2-8cm lower than the left kidney, because of the large liver which sits superior to it.
- Anteriorly the right kidney is related to the liver, duodenum and hepatic flexure of ascending colon.
- Right kidney is related to the 12^{th} rib posteriorly resting on diapharagm.

Renal pyramid

The basic unit of the mammalian kidney is the pyramid. Each pyramid is like a bunch of flowers in a vase, the blooms are glomeruli, the stems the collecting duct and the whole bunch, the papilla, sits in vase, the calyx.

Nephron

Nephron is defined as the structural and functional unit of kidney. Each kidney consists of 1 – 1. 3 millions of nephrons. The number of nephrons starts decreasing after about 45 to years of age at the rate of 0. 8 to 1% every year.

Each nephron is formed by two parts

1. A blind end called renal or Malpighian corpuscle
2. A tubular partion called renal tubule.

Renal corpuscles

The renal carpuscle is otherwise known as malpighian carpulse. It is a spheroidal and slightly flattened structure with a diameter of about 200 μ .

The function of renal corpuscle is the filtration of blood which forms the first phase of urine formation.

Situation of Renal corpuscles – types of nephron

Cortical nephrons or superficial nephrons (outer cortex)

Juxtamedullary nephrons (Inner medulla).

URINE FORMATION

The urine formation includes the following three processes.

- Glomerular filtration
- Tubular reabsorption and
- Tubular secretion

Kidneys excrete the unwanted substances including metabolic and products and those substances, which are present in excessive quantities in the body, through urine. Normally, about 1 to 1. 5 liters of urine is formed every day. Filtration is the function of the glomerulus or renal corpuscle or nephron and, reabsorption and secretion are the functions of tubular portion of the nephron.

Glomerular Filtration

When the blood passes through the glomerular capillaries and the plasma is filtered into the bowman's cpsule. All the substances of plasma are filtered except the plasma proteins. The filtrate is called glomerular filtrate. The glomerular filtration is called ultrafiltration because, even the minute particles are filtered. But, the plasma proteins are not filtered due to their large molecular size. The normal glomerular filtration rate is 125 ml/minute or about 180 liters

The glomerular filterate depends upon glomerualar capillary pressure colloidal osmotic pressure in the glomeruli and the hydrostatic pressure in the Bowman's capsule. Among these pressure, the glomerular capillary pressure favours filtration and the colloidal osmatic pressure and hydrostatic pressure oppose the filtration.

Tubular reabsorption

When the glomerular filtrate passes through the tubular portion of nephron, both quantitative and qualitative changes occur. Large quantity of water (more than 99%), electrolytes and other substances are reabsorbed by the tubular epithelial cells. The substances which are reabsorbed pass into the interstitial fluid of renal medulla. And, from here, the substances move into the blood in peritubular capillaries. As the substances are taken back into the blood, the entire process is called tubular reabsorption.

Selective reabsorption

The tubular cells of kidney selectively reabsorb the substances present in the glomerular filtrate. According to the needs of the body, degree of reabsorption is classified into three categories.

I. **HIGH THRESHOLD SUBSTANCES**

II. **LOW THESHOLD SUBSTANCES**

III**. NON-THRESHOLD SUBSTANCES**

Mechanism of reabsorption

The mechanisms involved in tubular reabsorption are of two types:

(i) **Active reabsorption**

The substances reabsorbed actively from the renal tubule are sodium, calcium, pottasium, phoshates, sulphates, bicarbonates, glucose, amino acids, ascorbic acid, uric acid and ketone bodies.

(ii) Passive reabsorption

The substances reabsorbed by passive transport are chloride, urea and water.

Site of reabsorption

- Proximal convoluted tubule
- Loop of henle
- Distal tubule
- Collecting tubule

Blood supply of the kidney

The upper part receives branches from renal artery, whereas middle part receives from aorta, the gonadal and iliac vessels. The pelvic part is supplied by the internal iliac vessel. The middle rectal or uterine vessels.

Renal veins

The left renal vein is about 5cm long, close to inferior vena cava, another reason why the left kidney is prefered live-donor transplantation.

Venous drainage

The interlobular veins drain into arcuate veins, which in their turn open into the interlobar veins.

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

Lymphatic drainage

The lymphatic 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 coeliac plexus. It contains sympathetic (T10 – L1) fibres which are chiefly vasometer. The afferent nerves of the kidney belong to segments T10 – T12.

Function of kidneys

Kidney 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 in homeostasis**

Kidneys are not only the excretary organs, but are also the regulatory organs because their major role is in homeostasis.

- **Excretion of waste products**

  Removal of wastes helps in homeostasis. Kidney excrete the unwanted waste products which are formed during metabolic activitis.

  Urea - end product of amino acid metabolism.

  Uric acid - end product of nucleic acid metabolism

  Creatinine - end product of metabolism in muscles.

  Bilirubin - end product of hemoglobin degradation**.**

  Products of metabolism of other substances.

Kidneys also excrete harmful foreign chemical substances like

- Toxins
- Drugs
- Heavy metals
- Pesticides

- **Maintenance of water balance**
- **Maintenance of electrolyte balance**
- **Maintenance of acid base balance**

2. **Hemopoietic function**

Kidneys stimulate the production of erythroocytes by secretery 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**

The hormones secreted by kidneys are

- Erythropoietin
- Thrombopoietin,
- Renin
- 1,25-di-hydroxycholecalciferol
- Prostaglandins

4. **Regulation of blood pressure**

Kidneys regulate arterial blood pressure by two ways

- By regulating the volume of extracellular fluid
- Through rennin-angiotensin mechanism.
- Regulation of blood calcium level

Kidneys play a role in the regulation of blood calcium level by activating 1,25-di-hydroxycholecalciferol into Vitamin –D. Vitamin –D is necessory for the absorbtion of calcium from intestine.

The ureter

The ureters are a pair of narrow, thick walled muscular tubes which convey urine from kidneys to the urinary bladder. They lie deep to the peritoneum, closely applied to the posteior abdominal wall in the upper part and to the lateral pelvic wall in the lower part.

Dimentions

Each ureter is about 25cm (10 inches) long of which the upper half (5 inch) lies in the abdomen and the lower half (5 inch) 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 dilation called the renal pelvis. The pelvis issues from the hilus of the kidney, descends along its medial margin, or partly behind it.

Normal constrictions

The ureter is slightly constricted at three places

- At the pelvic ureteric juction
- At the brim of the lesser pelvis
- At the passage through the bladder wall.

The ureters are muscular tubes that transport urine from the kidney to the bladder. They are continuous superiorly with the renal pelvis, which is a funnel shaped structure in the renal sinus. The renal pelvis is formed from a condensation of two or three major calices.

At the three following points along their course, the ureters are constricted.

- At the ureteropelvic junction, just inferior to the kidney.
- The ureters cross the common iliae vessels at the pelvic brim.
- The ureters enter the wall of the bladder.

Urinary bladder

The urinary bladder is a muscular sac in the pelvis, just above and behind the pubic bone. When empty, the bladder is about the size and shap of a pear.

Urine is made in the kidneys and travels down two tubes called ureters to the bladder. The bladder stones urine allowing urination to be infrequent and voluntary. The ladder is lined by layers of muscle tisue that stretch to accommodate urine. The normal capacity of the bladder is 400-600ml.

During urination, the bladder muscles contract and two sphincters (valves) open to allow urine to flow out. Urine exits the bladder into the urethra which carries urine out of the body. Because it passes through the penis, the urethra is longer in men (8 inches) than in women (1.5 inches)

Urethra

In both genders, the urethra works as a tube connecting the urinary bladder to the genitals. The bladder collects and stores urine until it is ready to be discharged through the urethra.

While the function remains the same for both genders, slight differences exist due to differences between male and female genitals.The female urethra is quite a bit shorter than its male counterpart and its opening is situated between a women's clitoris and vagina.For males, this tube travels the length of the penis and serves a dual purpose; it is a conduit for both urine and semen ejaculation.

Infection is more common in the female urethra. The most common condition is called urethritis, which involves inflammation and painful urinatio causes vary and can include viral or bacterial infections. symptoms may include frequent, urgent urination and purulent or pus-like excretions and discharges.

Treatments depend on the exact causes and symptoms, but most involve differing types of prescription medications.

UROLITHIASIS

The process of forming stones in the kidney, bladder, and/or urethra. Kindey stones are a common cause of blood in the urine and pain in the abdomen, flank, or groin. Kidney stones occur in 1-20 people at some time in their life. The development of the stone is related to decreased urine volume or increased excretion of stone – forming components such as calcium, oxalate, urate, cysteine, xanthine, and phosphate. The stones form in the urine collecting area (the pelvis) of the kidney and may range in size from tiny to staghorn stones the size of the renal pelvis itself.

Epidemiological factors like age, sex, hereditary, occupation, seasonal variations, socioeconomic status and dietary habits may determine the geographical preponderance.

In India approximately 5-7 million patients suffer from kalladaippu and least 1/1000 of Indian population needs hospitalization due to this disease. About 80%of those with kidney stones are men. Men most commonly experience their first episode between 20-30 years of age, while for women it is comparatively later.

12% of men were suffered with kalladaippu by the age 40-50 years 5% of women suffer from this disease by the age 50-60 years. The ratio is 3:1. 50% have recurrence within 5-10 years.

About 15% contain magnesium ammonium phosphate (struvite stone) and small numbers of cysteine or uric acid stones are found. The most common type of stones is formed from uric acid. About 10-15% of kalladaippu consists of struvite stones.

In developing countries, bladder stones are common, particularly in children. In developed countries the incidence of childhood bladder stone is slow and renal stones in adult are common, and this urolithiasis is worse than that of labour pain. The pathogenesis of calcium oxalate , which accounts for >80% of all urinary stones. Between 120 and 140 per 1000,000 will develop urinary stones each year with a male/female ratio of 3:1. (update march 2011) , for more than 40years the urolithiasis laboratory has perfected and perform done test, calculus analysis. We analyze approximately 50,000 stones annually, servicing more than 10,000 clients throughout the United States and internationally. The annual incidence of urolithiasis in 2008 was 1.7%. The male and female ratio was 1:1 in

participants with urolithiasis. A family history of urolithiasis found In 28.5% of the first-degree relatives of the stone- free participants (p=0.01). (urological research- august 2011, volume 39, issue4, pp 309-314)

AGE : Urinary stones disease is commonly seen in the age group of 20-40 years.

SEX : Ninety percent of the stone patients are males in India. This differs in European countries, where the incidence in females is much higher.

HEREDITY :

Heredity plays a significant role in the etiopathogenesis. 15% of patients give a family history of stone disease.

OCCUPATION :

The disease is more common among sedentary workers .

GEOGRAPHICAL INFLUENCE :

There is wide variation in the pattern of the disease in different parts of the world, thus projecting the significance of the stone belt areas.

SEASONAL FACTORS :

Higher incidence of the disease and aggravation of symptoms are noted in summer.

SOCIOECONOMIC STATUS :

It is more a disease of the middle and upper classes of people.

DIET :

High oxalate, high calcium and high uric acid diets may promote stones disease but the role of these alone as causative factors is debatable. Vitamin A, pyridoxine and magnesium are thought to inhibit stone disease.

PREDISPOSING FACTORS FOR URINARY STONES ENVIRONMENTAL AND DIETARY

Low urine volumes:

- High body temperatures, low fluid intake

Diet:

- High protein, sodium, low calcium diet.

High sodium excretion

- High oxalate excretion
- Low citrate excretion

ACQUIRED CAUSES

- Hypercalcemia of any cause
- Heal disease or resection(increases oxalate absorption and urinary excretion)
- Renal tubular acidosis type I

CONGENITAL AND INHERITED CAUSES

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

TYPES OF URINARY STONES

There are 4 types of urinary calculi-calcium containing, mixed (struvite), uric acid and cystine stones, and a few rare types.

l.CALCIUM STONES:

Most kidney stones are made up of calcium compounds , especially calcium oxalate.

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 a mixture of calcium oxalate and calcium phosphate (45%).

Calcium stones are usually small (4-5mm), ovoid, hard, with granular rough surface. They are dark brown due to old blood pigment deposited in them and as a result of repeated trauma caused to the urinary tract by these sharp-edged stones. Conditions that cause high calcium levels in the body, such as hyperparathyroidism, increase the risk of calcium stones. High levels of oxalate also increase in the risk for calcium stones.

2. MIXED (STRUVITE) 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.

Struvite stones are yellow-white or grey. They tend to be soft and friable 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.

They can also be called infection stones if they occur with kidney or urinary tract infections. These types of kidney stones sometimes are also called staghorn calculi if they grow large enough.

3. URIC ACID STONES:

Approximately 6% of urinary calculi are made of uric acid. Uric acid stones are adiolucent unlike radio-opaque calcium stones. Uric acid stones are smooth, yellowish-brown, hard and often multiple. On cut section, they show laminated structure.

You are more likely to have uric acid stones if u have:

- Low urine output.
- A diet high in animal protein, such as red meat.
- An increase in how much alcohol you drink .
- Gout
- Inflamattory bowel disease.

4.CYSTINE STONES:

Cystine stones comprise less than 2% of urinary calculi. Cystine stones are small, rounded, smooth and often multiple. They are yellowish and waxy. They occur in both men and women who have the genetic disorder cystinuria.

5. OTHER CALCULI:

Less than 2% of urinary calculi consist of other rare types such as xanthine stones.

AETIOLOGY

l.CALCIUM STONES:

Aetiology of calcium stones is variable.

1. About 50% of patients with calcium stones have idiopathic hypercalciuria without hyper calcaemia.
2. Approximately 10% cases are associated with *hyper calcaemia* and *hypercalciuria,* most commonly due to hyperparathyroidism, or a defect in the bowel (i.e.,absorptive hypercalciuria), or in the kidney (i.e., renal hypercalciuria)
3. About 15% of the patients with calcium stones have *hyperuricosuria with a* normal blood uric acid level and without any abnormality of calcium metabolism.
4. In about 25% of patients with calcium stones, the cause is unknown as there is no abnormality in urinary excretion of calcium, uric acid or oxalate and is referred to as 'idiopathic calcium stone disease'.

2. MIXED (STRUVITE) STONES:

Stuvite stones arre formed as a result of infection of the urinary tract with urea-splitting organisms that produce urease such as by species of Proteus, and occasionally Klebsiella,Pseudomonas and Enterobacter. These are, therefore, also known as infection-induced stones.However, E.coli does not form urease.

3. URIC ACID STONES:

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 urine pH (below 6) and low urinary volume.

4. CYSTINE STONES:

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

5. OTHER CALCULI

Are caused due to inherited abnormality of enzyme metabolism.

This tabular column represented with prevalence of various stones and its colours

Kidney stone type	Population	Circumstances	Color	sensitivity	Details
Calcium oxalate	80%	When urine is acidic (decreases PH)	Black / dark brown	Radio-opaque	Some of the oxalate in urine is produced by the body. Calcium and oxalate in the diet play a part but are not the only factors that affect theformation of calcium
Calcium phosphate	5-10%	When urine is alkaline (high PH)	Dirty white	Radio-opaque	Tends to grow in alkaline urine especially whenproteus are present.
Uric acid	5-10%	When urine is persistently acidic.	Yellow reddish brown	Radioulcent	Diets rich in animal proteins and purines: substances found naturally in all food but especially in organ meats, fish, and shellfish.
Struvite	10-15%	Infections in the kidney	Dirty white	Radio-opaque	Preventing struvite stones depends on staying infection-free. Diet has not been shown to affect struvite stone formation.`
Cystine	1-2%	rare genetic disorder	Pink/ yellow	Radio-opaque	Cysteine, an amino acid (one of the building blocks of protein), leaks through the kidneys and into the urine to form crystals.
Xanthine		Extremely rare	Brick red	Radio-opaque	

PATHOGENESIS OF STONES

Urinary stones usually arise because of the breakdown of a delicate balance between solubility and precipitation of salts. The kidneys must conserve water, but they must excrete materials that have a low solubility. These two opposing requirements must be balanced during adaptation to diet, climate, and activity. The problem is mitigated to some extent by the fact that urine contains substances that inhibit crystallization. These protective mechanisms are less than perfect. When the urine becomes supersaturated with insoluble materials, because excretion rates are excessive and/or because water conservation is extreme, crystals form and may grow and aggregate to form a stone.

Supersaturation

A solution in equilibrium with crystals of calcium oxalate is said to be saturated with respect to calcium oxalate. If crystals are removed, and if either calcium or oxalate ions are added to the solution, the chemical activities increase, but no new crystals form. Such a solution is *metastably supersaturated.* If calcium oxalate crystals are now added, they will grow in size. Ultimately, as calcium or oxalate is added to the solution, supersaturation reaches a critical value at which a solid phase begins to develop spontaneously. This value is called the *upper limit of metastability.* Kidney stone growth requires a urine that, on average, is supersaturated. Excessive supersaturation is common in stone formation.

Calcium, oxalate, and phosphate form many soluble complexes among themselves and with other substances in urine, such as citrate. As a result, their free ion activities are below their chemical concentrations. Reduction in ligands such as citrate can increase ion activity and, therefore, supersaturation. Urine supersaturation can be increased by dehydration or by overexcretion of calcium, oxalate, phosphate, cystine, or uric acid. Urine pH is also important; phosphate and uric acid are acids that dissociate readily over the physiologic range of urine pH. Alkaline urine contains more dibasic phosphate, favoring deposits of brushite and apatite. Below a urine pH of 5.5, uric acid crystals *(pK* 5.47) predominate, whereas phosphate crystals are rare. The solubility of calcium oxalate is not influenced by changes in urine pH. Measurements of supersaturation in a 24-h urine sample probably underestimate the risk of precipitation. Transient, dehydration, variation of urine pH, and postprandial bursts of overexcretion may cause values considerably above average.

Crystallization

When urine supersaturation exceeds the upper limit of metastability, crystals begin to nucleate. Cell debris and other crystals present in the urinary tract can serve as templates for crystal formation, a process known as *heterogeneous nucleation.* Heterogeneous nucleation lowers the level of supersaturation required for crystal formation. Once formed, crystal nuclei will grow in size if urine is supersaturated with respect to that crystal phase. Multiple crystals can then aggregate to form a kidney stone.

In order for a kidney stone to form, crystals must be retained in the renal pelvis long enough to grow and aggregate to a clinically significant size. The mechanism of crystal retention has been a matter of much debate. Recent studies have shown that common calcium oxalate kidney stones form as overgrowths on apatite plaques in the renal papillae. These plaques, called Randall's plaques, provide an excellent surface for heterogeneous nucleation of calcium oxalate salts. The Randall's plaques begin in the deep medulla in the basement membrane of the thin limb of the loop of Henle and then spread through the interstitium to the basement membrane of the papillary urothelium. If the urothelium becomes damaged, the plaque is exposed to the urine, and calcium oxalate crystallization and stone formation begins.

Inhibitors of Crystal Formation

Urine contains potent inhibitors of nucleation, growth, and aggregation for calcium salts. Inorganic pyrophosphate is a potent inhibitor that appears to affect formation of calcium phosphate more than calcium oxalate crystals. Citrate inhibits crystal growth and nucleation, although most of the stone inhibitory activity of citrate is due to lowering urine supersaturation via complexation of calcium. Other urine components such as glycoproteins inhibit calcium oxalate crystallization.

1. CALCIUM STONES:

The mechanism of calcium stone formation is explained on the basis of imbalance between the degree of super saturation 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 stone grows, as more and more crystals are deposited around the nidus. A number of other predisposing factors contributing to formation of calcium stones are alkaline urinary pH, decreased urinary volume and increased excretion of oxalate and uric acid.

2. MIXED (STRUVITE) STONES:

Struvite stones are formed as a result of infection of the urinary tract with urea-splitting organisms that produce urease.

3. URIC ACID STONES:

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 favouring 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.

4. CYSTINE STONES:

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

CLINICAL SYMPTOMS

- Acute renal or ureteral pain(Renal colic)
- Hematuria (microscopic or gross blood in urine)
- Recurrent Urinary tract infection
- Vague abdominal and flank pain

Small non obstructing stones or 'silent stones' located in the calyces of the kidney are sometimes found incidentally on x-rays or may be present with asymptomatic hematuria. Such stones often pass without causing pain or discomfort.

KIDNEY STONE SYMPTOMS:

Stones in the kidneys can become lodged at the junction of kidney and ureter (uretro pelvic junction), resulting in

- Acute ureteral obstruction with severe colicky flank pain. Pain can be localized at the costo-vertebral angle.
- Hematuria may be present intermittently or persistently and it may be microscopic or gross.

URETERAL STONE SYMPTOMS:

Stones that can pass into the ureter may produce

- Ureteral colic which is an acute, sharp, spasm like pain located in the flank.
- Hematuria may be present.
- Stones moving down the ureter to the pelvic brim and iliac vessels will produce.
- Spasms with intermittent, sharp, colicky pain radiating to the lateral flank and around the umbilical region.
- As a stone passes through the distal ureter near the bladder,
- The pain remains sharp but with a waxing and waning quality. Relief is offered when the spasm subsides or the pain may intensify and radiate to the groin, testicles and labia.
- Nausea
- Vomiting
- Diaphoresis
- Tachycardia
- Tachypnoea may be present and patients are typically uncomfortable.

BLADDER STONE SYMPTOMS:

Once a stone enters the bladder,

- Dysuria and Urgency and frequency of micturition may be the only symptoms experienced.
- Immediate relief of symptoms occurs once the stone passes out of bladder.

Staghorn Calculi

Struvite, cystine, and uric acid stones often grow too large to enter the ureter. They gradually fill the renal pelvis and may extend outward through the infundibulum to the calyces themselves. Very large staghorn stones can have surprisingly few symptoms and may lead to the eventual loss of kidney function.

Nephrocalcinosis

Calcium stones grow on the papillae. Most break loose and cause colic, but they may remain in place so that multiple papillary calcifications are found by x-ray, a condition termed *nephrocalcinosis.* Papillary nephrocalcinosis is common in

hereditary distal renal tubular acidosis (RTA) and in other types of severe hypercalciuria. In medullary sponge kidney disease, calcification may occur in dilated distal collecting ducts.

Infection

Although urinary tract infection is not a direct consequence of stone disease, it can occur after instrumentation and surgery of the urinary tract, which are frequent in the treatment of stone disease. Stone disease and urinary tract infection can enhance their respective seriousness and interfere with treatment. Obstruction of an infected kidney by a stone may lead to sepsis and extensive damage of renal tissue, since it converts the urinary tract proximal to the obstruction into a closed, or partially closed, space that can become an abscess. Stones may harbor bacteria in the stone matrix, leading to recurrent urinary tract infection. On the other hand, infection due to bacteria that possess the enzyme urease can cause stones composed of struvite.

Activity of Stone Disease

Active disease means that new stones are forming or that preformed stones are growing. Sequential radiographs are needed to document the growth or appearance of new stones and to ensure that passed stones are actually newly formed, not preexistent.

DIAGNOSIS

It has been standard practice to diagnose acute renal colic by intravenous pyelography; however, helical CT scan without radiocontrast enhancement is now the preferred procedure. The advantages of CT include detection of uric acid stones in addition to the traditional radiopaque stones, no exposure to the risk of radiocontrast agents, and possible diagnosis of other causes of abdominal pain in a patient suspected of having renal colic from stones. Ultrasound is not as sensitive as CT in detecting renal or ureteral stones.

Standard abdominal x-rays may be used to monitor patients for formation and growth of kidney stones, as they are less expensive and provide less radiation exposure than CT scans. Calcium, cystine, and struvite stones are all radiopaque on standard x-rays, whereas uric acid stones are radiolucent.

DIFFERENTIAL DIAGNOSIS

- Appendicitis
- Cholecystitis and cholelithiasis
- Chron's disease
- Ectopic pregnancy and pelvic inflammatory diseases
- Pancreatitis
- Gastric and duodenal ulcers
- Pyelonephritis
- Dissecting aneurysm
- Diverticulis

Evaluation and Treatment of Patients with Urolithiasis

Most patients with nephrolithiasis have remediable metabolic disorders that cause stones and can be detected by chemical analyses of serum and urine. Adults with recurrent kidney stones and children with even a single kidney stone should be evaluated. A practical outpatient evaluation consists of two 24-h urine collections, with a corresponding blood sample; measurements of serum and urine calcium, uric acid, electrolytes, and creatinine, and urine pH, volume, oxalate, and citrate should be made. Since stone risks vary with diet, activity, and environment, at least one urine collection should be made on a weekend when the patient is at home and another on a work day. When possible, the composition of kidney stones should be determined because treatment depends on stone type. No matter what disorders are found, every patient should be counseled to avoid dehydration and to drink copious amounts of water. The efficacy of high fluid intake was confirmed in a prospective study of first-time stone formers. Increasing urine volume to 2.5 L per day resulted in a 50% reduction of stone recurrence compared to the control group.

Urolithiasis: Treatment

The management of stones already present in the kidneys or urinary tract requires a combined medical and surgical approach. The specific treatment depends on the location of the stone, the extent of obstruction, the nature of the stone, the function of the affected and unaffected kidney, the presence or absence of urinary tract infection, the progress of stone passage, and the risks of operation or anesthesia given the clinical state of the patient. Medical therapy can enhance passage of ureteral stones. In general, severe obstruction, infection, intractable pain, and serious bleeding are indications for removal of a stone.

Calcium Stones Hypercalciuria: Treatment

For many years the standard therapy for hypercalciuria was dietary calcium restriction. However, recent studies have shown that low-calcium diets increase the risk of incident stone formation. Low-calcium diets may lead to stone formation by reducing the amount of calcium to bind oxalate in the intestine, thereby increasing urine oxalate levels. However, the mechanism by which a low-calcium diet increases stone risk has not been clearly defined. In addition, hypercalciuric stone formers have reduced bone mineral density and an increased risk of fracture compared to the non-stone-forming population. Low calcium intake likely contributes to the low bone mineral density. A 5-year prospective trial compared the efficacy of a low-calcium diet to a low-protein, low-sodium, normal-calcium diet in preventing stone recurrence in male calcium stone formers. The group on the low-calcium diet had a significantly greater rate of stone relapse. Low-calcium diets are of unknown efficacy in preventing stone formation and carry a long-term risk of bone disease in the stone-forming population. Low-sodium and low-protein diets are a superior option in stone formers. If diet therapy is not sufficient to prevent stones, then thiazide diuretics may be used. Thiazide diuretics lower urine calcium and are effective in preventing the formation of stones. Three 3-year randomized trials have shown a 50% decrease in stone formation in the thiazi de-treated groups as compared to the placebo-treated controls. The drug effect requires slight contraction of the extracellular fluid volume, and high dietary NaCl intake reduces its therapeutic effect. Thiazide-induced hypokalemia should be aggressively treated since hypokalemia will reduce urine citrate, an important inhibitor of calcium crystallization.

Hyperuricosuria-Treatment

About 20% of calcium oxalate stone formers are hyperuricosuric, primarily because of an excessive intake of purine from meat, fish, and poultry. The mechanism of stone formation is probably due to salting out calcium oxalate by urate. A low-purine diet is desirable but difficult for many patients to achieve. The alternative is allopurinol, which has been shown to be effective in a randomized, controlled trial. A dose of 100 mg bid is usually sufficient.

Primary Hyperparathyroidism

The diagnosis of this condition is established by documenting that hypercalcemia that cannot be otherwise explained is accompanied by inappropriately elevated serum concentrations of parathyroid hormone. Hypercalciuria, usually

present, raises the urine supersaturation of calcium phosphate and/or calcium oxalate. Prompt diagnosis is important because parathyroidectomy should be carried out before renal damage or bone disease occurs.

Hyperoxaluria: Treatment

Patients with mild to moderate hyperoxaluria should be treated with a diet low in oxalate and with a normal intake of calcium and magnesium to reduce oxalate absorption. Enteric hyperoxaluria can be treated with a low-fat, low-oxalate diet and calcium supplements, given with meals, to bind oxalate in the gut lumen. The oxalate-binding resin cholestyramine at a dose of 8-16 g/d, provides an additional form of therapy. Treatment for primary hyperoxaluria includes a high fluid intake, neutral phosphate, and pyridoxine (25-200 mg/d). Citrate supplementation may also have some benefit. Even with aggressive therapy, irreversible renal failure may occur. Liver transplantation, to correct the enzyme defect, combined with a kidney transplantation has been successfully utilized in patients with primary hyperoxaluria.

Hypocitraturia: Treatment

Treatment is with alkali, which increases urine citrate excretion; generally bicarbonate or citrate salts are used. Potassium salts are preferred as sodium loading increases urinary excretion of calcium, reducing the effectiveness of treatment. Two randomized, placebo-controlled trials have demonstrated the effectiveness of citrate supplements in calcium oxalate stone formers.

Idiopathic Calcium Lithiasis

Some patients have no metabolic cause for stones despite a thorough metabolic evaluation. The best treatment appears to be high fluid intake so that the urine specific gravity remains at 1.005 throughout the day and night. Thiazide diuretics, allopurinol, and citrate therapy may help reduce crystallization of calcium salts, but there are no prospective trials in this patient population. Oral phosphate at a dose of 2 g phosphorus daily may lower urine calcium and increase urine pyrophosphate and thereby reduce the rate of recurrence. Orthophosphate causes mild nausea and diarrhea, but tolerance may improve with continued intake.

Uric Acid Lithiasis: Treatment

The two goals of treatment are to raise urine pH and to lower excessive urine uric acid excretion to <1 g/d. Supplemental alkali, 1-3 mmol/kg of body weight per day, should be given in three or four divided doses, one of which should be given at bedtime. The goal of treatment should be a urine pH between 6.0 and 6.5 in a 24-h

urine collection. Increasing urine pH above 6.5 will not provide additional benefit in preventing uric acid crystallization but does increase the risk of calcium phosphate stone formation. The form of the alkali may be important. Potassium citrate may reduce the risk of calcium salts crystallizing when urine pH is increased, whereas sodium citrate or sodium bicarbonate may increase the risk. A low-purine diet should be instituted in uric acid stone formers with hyperuricosuria. Patients who continue to form uric acid stones despite treatment with fluids, alkali, and a low-purine diet should have allopurinol added to their regimen.

Cystinuria and Cystine Stones: Treatment

High fluid intake, even at night, is the cornerstone of therapy. Daily urine volume should exceed 3 L. Raising urine pH with alkali is helpful, provided the urine pH exceeds 7.5. A low-salt diet (100 mmol/d) can reduce cystine excretion up to 40%. Because side effects are frequent, drugs such as penicillamine and tiopronin, which form the mixed soluble disulfide cysteine-drug complexes, should be used only when fluid loading, salt reduction, and alkali therapy are ineffective. Low-methionine diets have not proved to be practical for clinical use, but patients should avoid protein gluttony.

Struvite Stones: Treatment

Complete removal of the stone with subsequent sterilization of the urinary tract is the treatment of choice for patients who can tolerate the procedures. Percutaneous nephrolithotomy is the preferred surgical approach for most patients. At times, extracorporeal lithotripsy may be used in combination with a percutaneous approach. Open surgery is rarely required. Irrigation of the renal pelvis and calyces with hemiacidrin, a solution that dissolves struvite, can reduce recurrence after surgery. Stone-free rates of 50-90% have been reported after surgical intervention. Antimicrobial treatment is best reserved for dealing with acute infection and for maintenance of a sterile urine after surgery. Urine cultures and culture of stone fragments removed at surgery should guide the choice of antibiotic. For patients who are not candidates for surgical removal of stone, acetohydroxamic acid, an inhibitor of urease, can be used. Unfortunately, acetohydroxamic acid has many side effects, such as headache, tremor, and thrombophlebitis, that limit its use.

COMPLICATIONS

Ocassionally, stones can injure the kidneys by causing infection, resulting in fever, chills, loss of appetite or urinary obstruction. If an UTI accompanies the urinary obstruction, pyelonephritis or urosepsis can occur. If stones are bilateral,they can cause renal scarring and damage, resulting in acute or chronic renal failure.

DIET FOR URINARY STONES

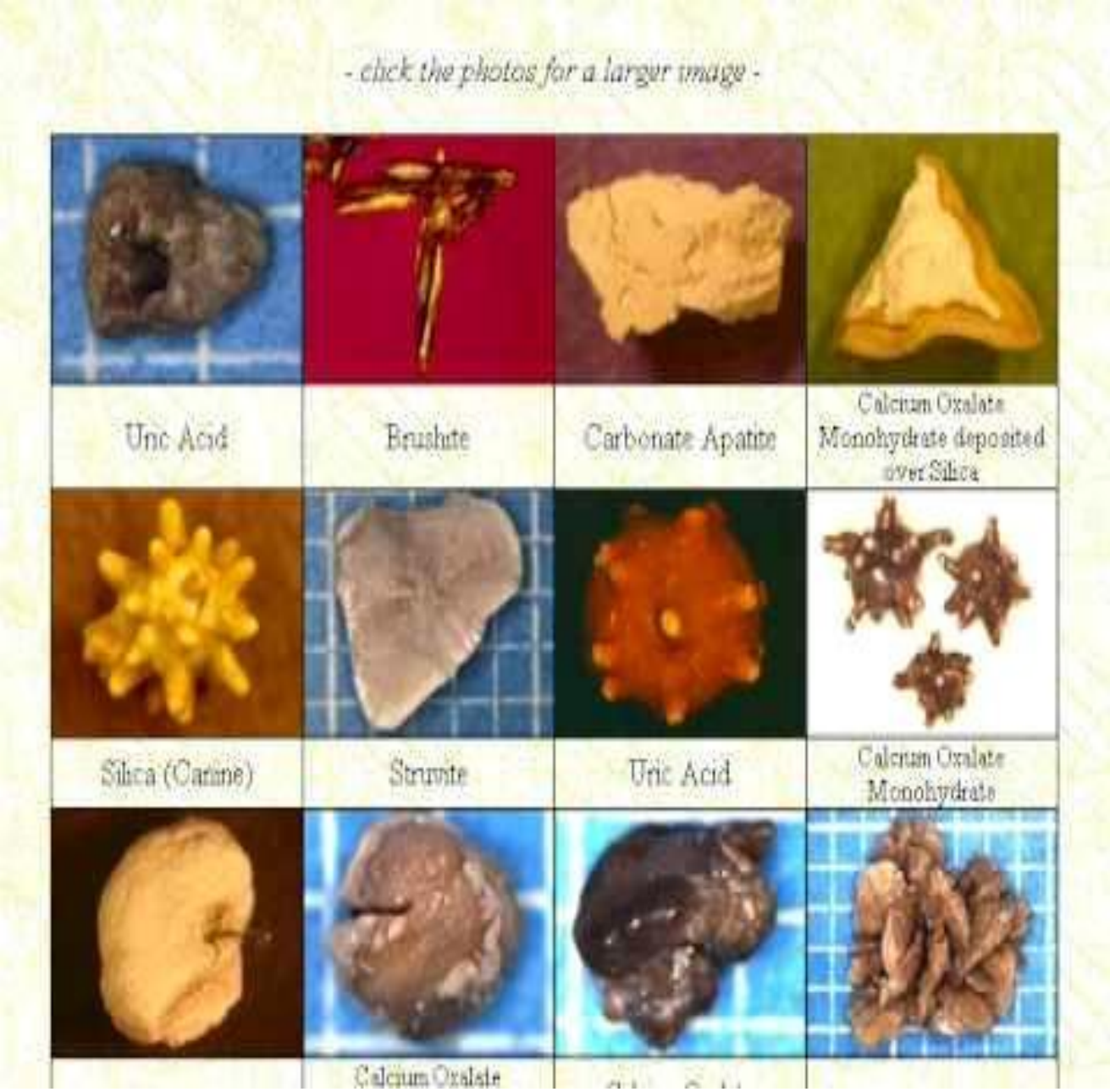
A diet for Calcium stones should limit substances that increase blood and urine levels of calcium, such as caffeine and sodium. It is better to avoid foods and beverages such as coffee, chocolate and fast foods. Food that reduce excretion of calcium into the urine such as cranberries can be included. Mineral supplements that are reported to reduce urinary calcium are magnesium and potassium. Vitamin B6 is also reported to reduce urinary calcium.Oxalate rich foods like spinach, beet, black tea and nuts should be avoided. Also avoid vitamin C rich food as vitamin C converts to oxalate in urine.

Uric acid stone former should avoid animal sources of protein and red wine and increase their daily fluid intake. Even vegetable sources of protein like pulses and grains should be restricted.

Struvite stone formers may modify their diet to increase the acidity of urine to inhibit bacterial growth. Urine acidifiers include specific tablets, animal proteins and citrus fruits.

Cystine stone formers should avoid fish and grains containing protein that are rich in methionine.As excessively acidic urine encourages cystine stone formation foodwhich help to raise the pH of the urine such as vegetable juices and fruits can be taken.

TYPES OF KIDNEY STONES



MATERIALS AND METHODS

A clinical study on "The evaluation of efficacy of the trial drug Nilakumilaver Kudineer **(Internal)** in treating **Kalladaippu noi**", was carried out at the Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai.

For the study, a total of 40 patients of both sex (20 Out-Patients and 20 In-Patients), suffering from Kalladaippu noi were selected and administered the fore-said trial drug for the purpose of treating the disease.

The patients, both in-patients and out-patients were enrolled for the study after diagnosing the Kalladaippu noi, through clinical, pathological, biochemical and microscopic examination. In addition, Ultra-Sonogram was also done for all the patients who were enrolled for the study to confirm the occurrence of Kalladiappu noi.

After discharging the inpatients from the IP ward, treatment with the trial medicine was continued in the OP Department as follow-up cases.

Preparation of trial medicine

The trial medicine, **Nilakumilaver Kudineer** (Internal) is selected based on their medicinal values in treating **Kalladaippu noi**, mentioned in the Siddha Literature, **AGATHIYAR VAITHYA KAVIYAM – 1500**

Pg- 651,652

The ingredient was collected, purified and the medicine was prepared, stored and given to the patients based on the reference cited above. References are enclosed in **Annexure – I**.

Biochemical analysis of the trial medicine

Biochemical analyses of the trial medicine was performed at the Biochemistry Unit of Government Siddha Medical College, Palayamkottai. Experiments were conducted by the unit by following the standard procedures to know the presence of minerals like Calcium, Sulphate, Chloride, Carbonate, Starch, Ferric Iron, Ferrous Iron, Phosphate, Albumin, Tannic Acid, Unsaturated Compound, Reducing sugar, Amino Acid and Zinc. The results of the biochemical analysis and inferences are given in **Annexure – II**.

Acute toxicity studies of the trial medicine

Acute toxicity studies were conducted in Pharmacology laboratory, at Periyar College of Pharmaceutical Sciences Trichy Experiments were conducted with albino rats by following the standard procedures. The procedures, results and inferences are given in detail in **Annexure – III.**

Pharmacological analysis of the trial medicine

Pharmacological actions of the trial medicine were studied at Periyar College of Pharmaceutical Sciences Trichy. Experiments were conducted with albino rats by following the standard procedures to determine the pharmacological actions like lithotriptic ,diuretic and analgesic effect of the trial medicine. The procedures, results and inferences are given in detail in **Annexure – IV.**

Criteria for the selection of patients

For the present study, patients were selected based on the following criteria.

- Ultra-Sonogram report, indicating the presence of Renal Calculi.
- Urine analysis report, indicating the presence of crystals, albumin and
- RBC depositions in the urine.
- Past history of Renal Calculi.

Clinical history of patients with the following symptoms.

- Renal colicky pain
- Burning micturation
- Obstruction during micturation
- Oliguria
- Haematuria
- Nausea
- Vomiting
- Sweating

Collection and maintenance of Patients' database

A proforma was prepared to collect details about the patients personal and family history, present symptoms, history of recent and past illness, laboratory investigation (including urine and blood analysis), Ultra-Sonogram, method adopted for the purpose of treatment and management of the disease and follow-up procedures. The details collected were recorded in proforma for each individual patient and database was maintained for all the patients.

Investigation of Kalladaippu noi patients

The patients were subjected to the following investigations to establish the diagnosis. The investigations were carried out regularly before and after the treatment.

i) Urine examination

The urine of patients was subjected to microscopic investigations with regard to the colour, specific gravity, sugar, albumin, RBCs, cast, pus cells, epithelial cells, crystals and other pathological constituents.

ii) Biochemical examination of blood

The blood of patients was tested biochemically to estimate the blood urea and creatinine level to know the present functional status of kidney.

iii) Ultra-Sonogram (USG Abdomen KUB)

All the patients were subjected to USG Abdomen KUB before and after the treatment. The USG reports of few patients are enclosed in **Annexure - V.**

iv)Analysis of kidney stones

Knowing the chemical composition of the stone helps the doctor identify why the patient is prone to stone formation. The kind of stone a person's body makes determines what dietary changes may be needed.For example, limiting oxalate in the diet may help prevent calcium oxalate stones but will do nothing to prevent uric acid stones. Some dietary recommendations may apply to more than one type of stone.

Most notably, drinking enough water helps prevent all kinds of kidney stones. In the present study, the urinary tract stones expelled along with urine in some of the patients during the course of treatment were analysed in a well reputed medical laboratory to determine the types of stones. The stone analysis reports are given in **Annexure – VI**.

Criteria for measuring the positive outcome of the study

The positive outcomes of the study was measured using the following Urolithiasis Symptom Score. The score of each case at the time of enrolment and at the end of treatment were compared and on its basis the patient was informed that he or she was symptoms free and cured.

Urolithiasis Symptom Score

(Circle relevant number on each line)

1. Pain/colic	0- No pain	1- Mild pain	2 -Moderate pain	3- Severe pain
2. Haematuria	0 -No haematuria	1- Microscopic	2- Persistent	3 -Gross
3. Dysuria	0 -No dysuria	1- Mild dysuria	2- Moderate dysuria	3- Severe
4. Stone		1- Single stone	2- Multiple stone	
5. Size of stones	0- Gravel < 03mm	1-3 mm to < 4 mm	2-4 mm to < 5 mm	3-5 mm and above
6. Position of stone in kidney	0- no stone in kidney	1-Pelvic ureteric junction	2- Pelvis of kidney	3 Calyces of kidney
7. Position of stone in ureter	0- no stone in ureter	1- Lower part of ureter	2- Middle of ureter	3- Upper part of Ureter
8. Position of stone in bladder	0 -no stone in bladder	1 -Base of bladder	2- Intramural ureter	

Total scoring – 22, 1-7 mild, 8-14 moderate, 15-22 severe. Symptoms score – (Some of 8 circled numbers)

RESULTS AND OBSERVATION

The results obtained from the presence study were recorded in the proforma with respect to the following parameters for eacch patient both out patients and in patients. They were analysed statistically inorder to arrive at the percentage composition.

1. Age
2. Sex
3. Religion
4. Occupation
5. Socio-economic stattus
6. Food habits
7. Personal habits
8. Aetiological factors
9. Thinai
10. Kaalangal
11. Paruvakaalam
12. Thegi (Constitution of body)
13. Mukkutram

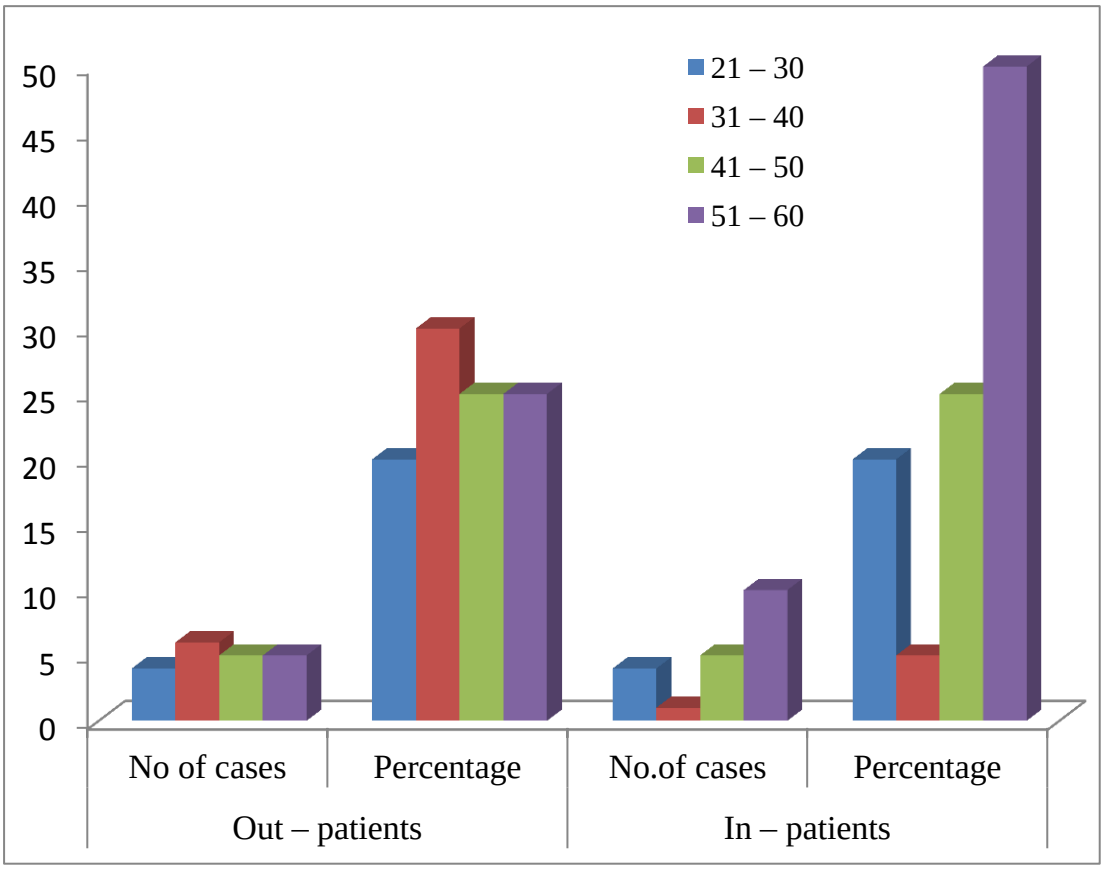
    Derangement of Vatham

    a. Derangement of Pitham
    b. Derangement of kabam
14. Udal Thathukkal
15. Envagai Thervugal
16. Neerkuri
17. Neikuri
18. Duration of illness
19. Types of pain
20. Types of Dysuria
21. Numbeer of Stones
22. Size of stones
23. Position of stone
24. Urolithiasis symptom score
25. Grading outcomes of the study

TABLE –I

AGE DISTRIBUTION

S.No	Age (in years)	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	21 – 30	04	20	04	20
02	31 – 40	06	30	01	05
03	41 – 50	05	25	05	25
04	51 – 60	05	25	10	50

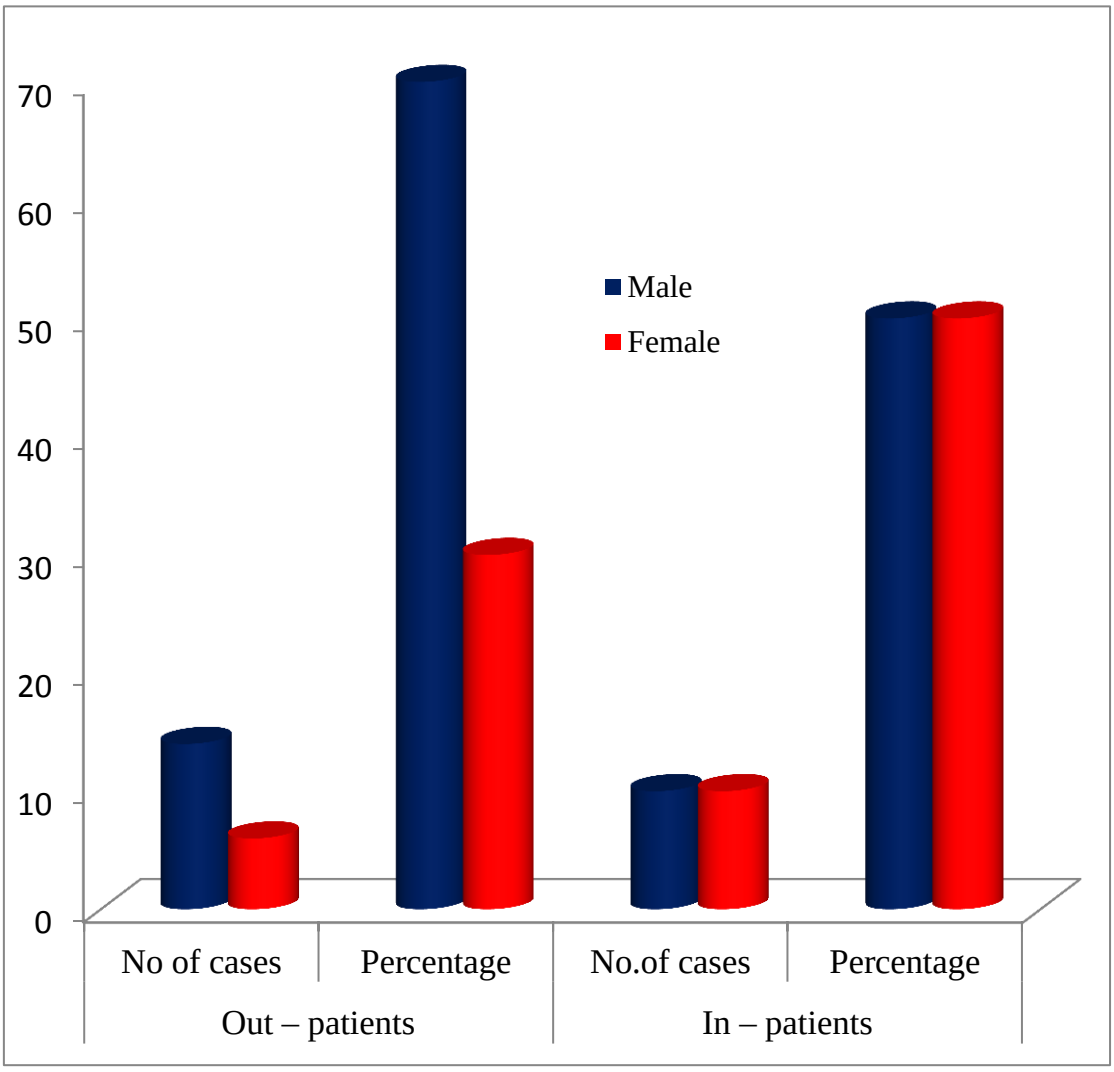


In the patients study among the Outpatients, Kalladaippu Noi was found to be most common in the age group of 41 – 50 years. Among In Patients it was predominent in the age group of 51 – 60 years.

TABLE – 2

SEX DISTRIBUTION

S.No	Sex	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Male	14	70	10	50
02	Female	06	30	10	50

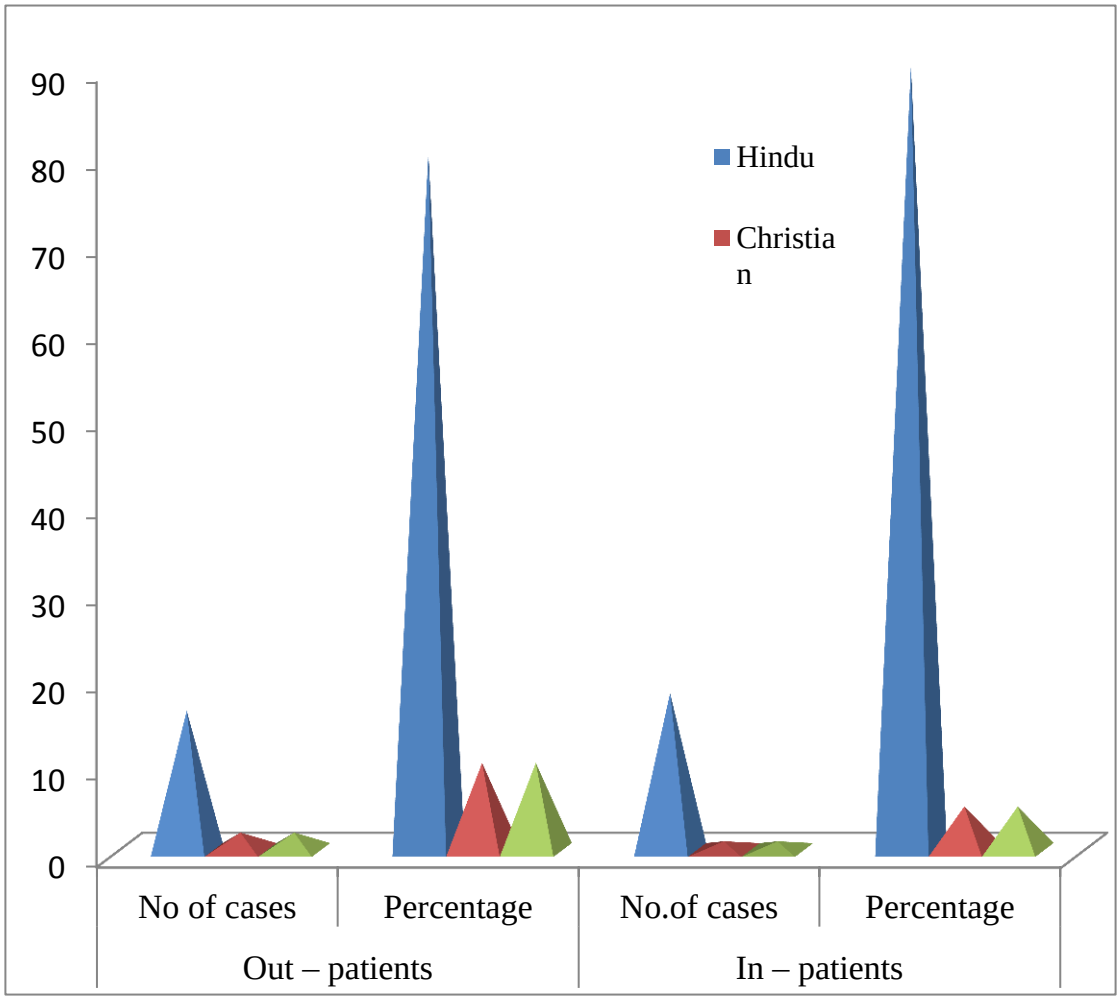


From the above table male outpatients were most affected by Kalladaippu Noi and In patients were equally afffected.

TABLE –3

RELIGION DISTRIBUTION

S.No	Religion	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Hindu	16	80	18	90
02	Christian	02	10	01	05
03	Muslim	02	10	01	05

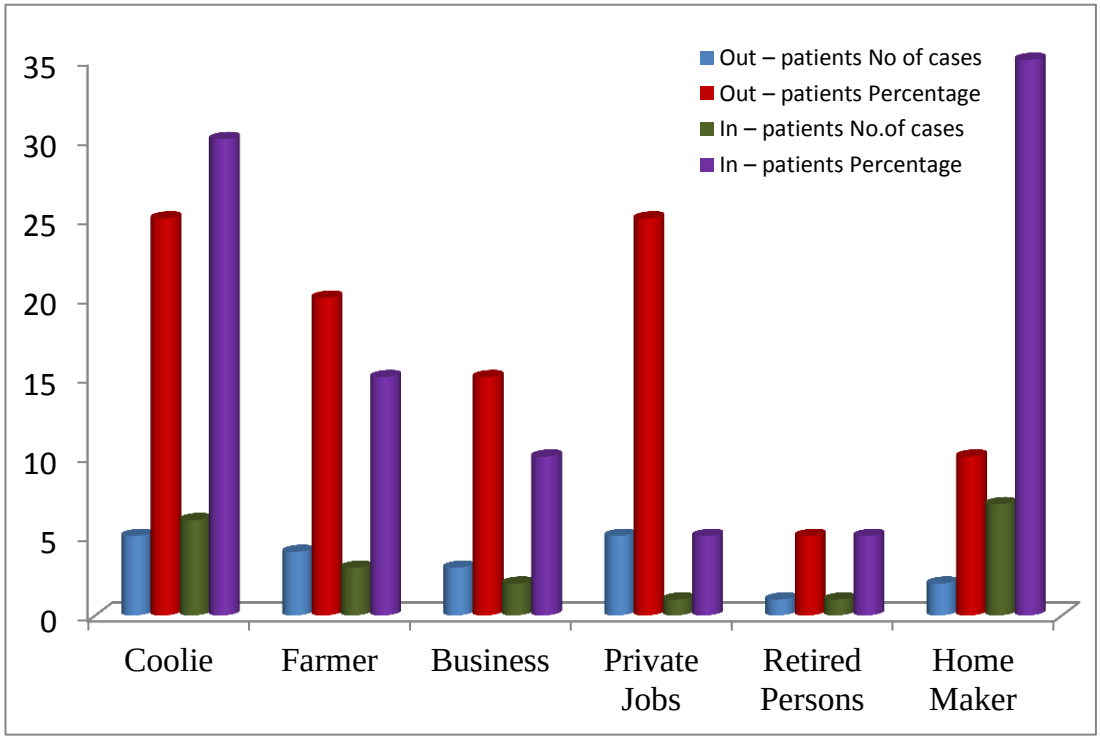


It is observed that hindus were most affected by kalladaippu noi and Muslims were minimally affected

TABLE –4

OCCUPATION DISTRIBUTION

S.No	Occupation	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Coolie	05	25	06	30
02	Farmer	04	20	03	15
03	Business	03	15	02	10
04	Private Jobs	05	25	01	05
05	Retired Persons	01	05	01	05
06	Home Maker	02	10	07	35

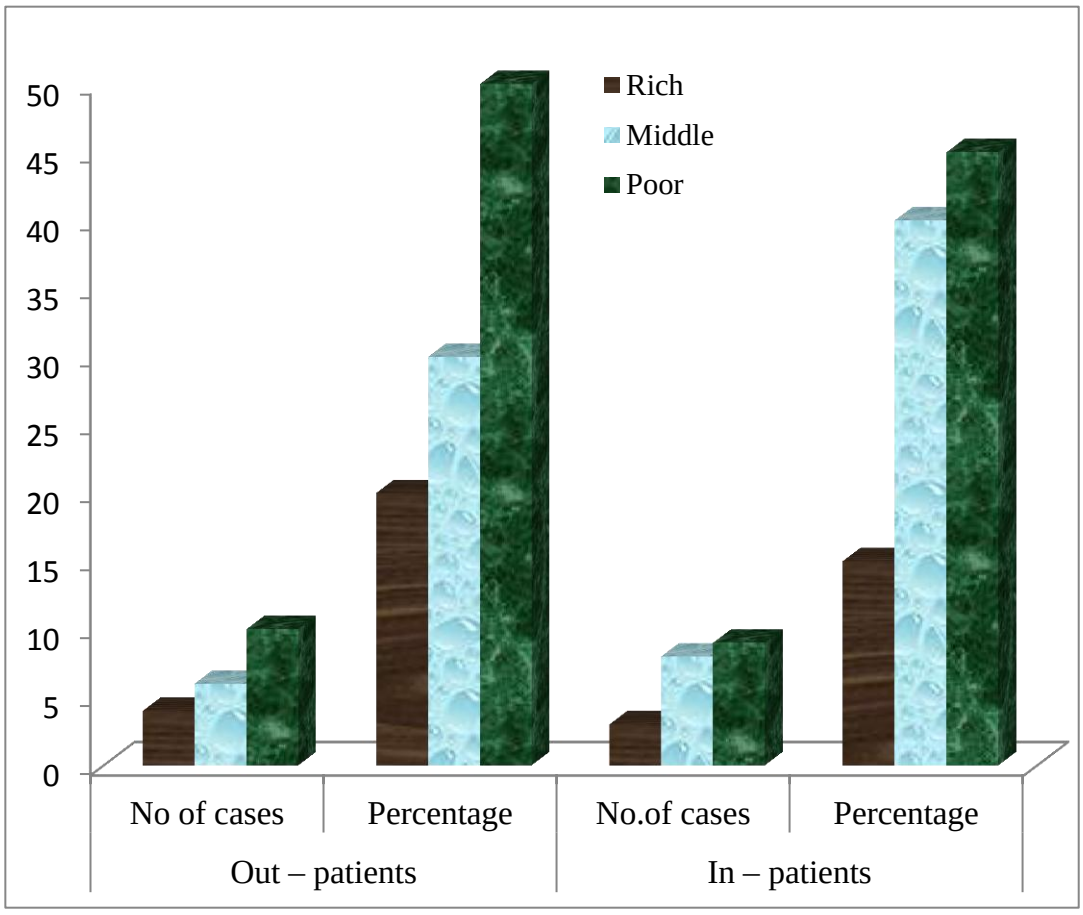


From the above table shows that 30% of the out patients with kalladaippu noi were in the private jobs and majority of the inpatients were home maker.

TABLE –5

SOCIO ECONOMIC STATUS

S.No	Socio Economic Status	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Rich	04	20	03	15
02	Middle	06	30	08	40
03	Poor	10	50	09	45

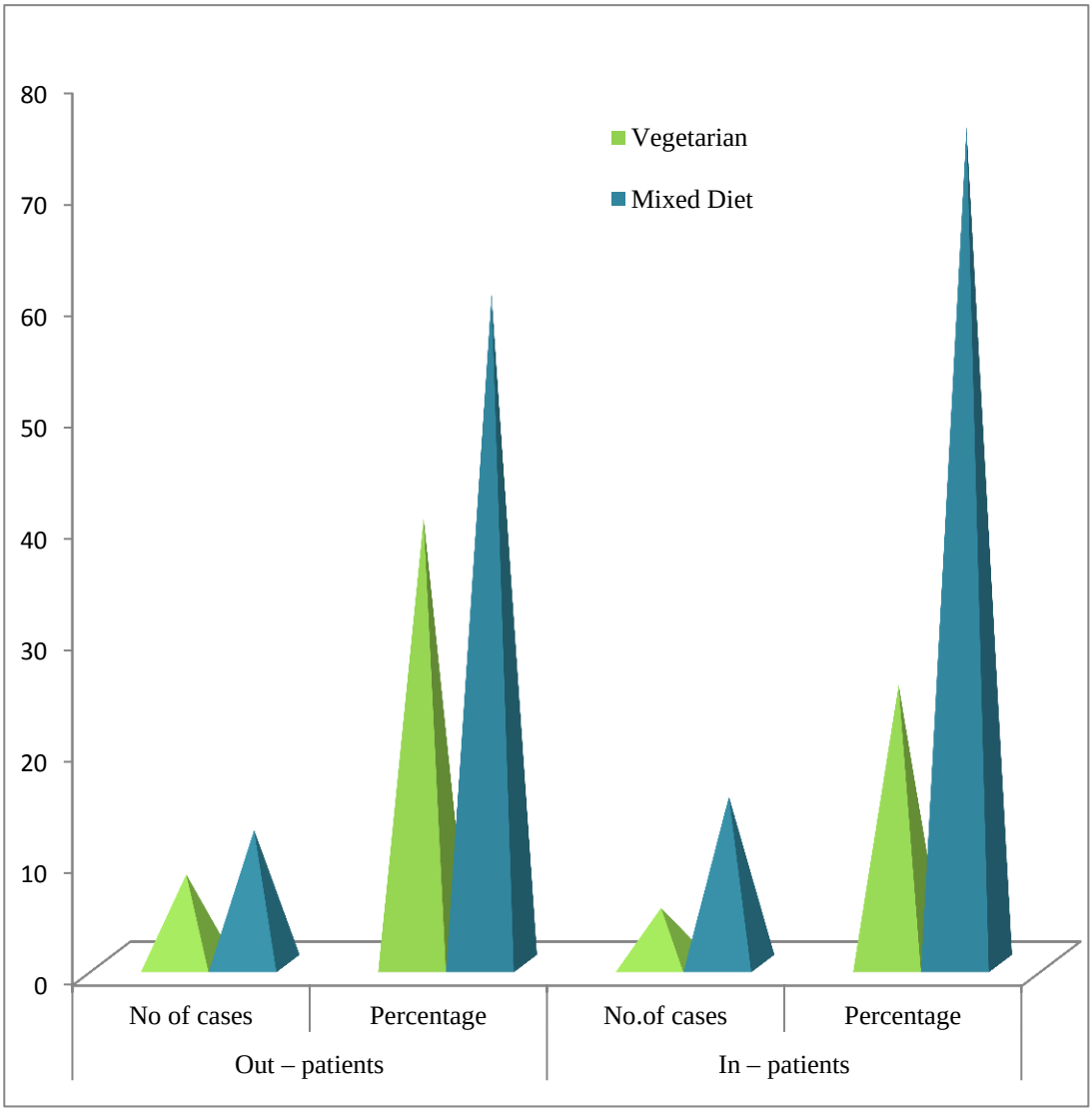


It is determine from the above data that the majority of the In patients affected by the Kalladaippu Noi were from the poor class. In IN Patients Middle and Poor class were equally affected.

TABLE – 6

DIET

S.No	Diet	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Vegetarian	08	40	05	25
02	Mixed Diet	12	60	15	75

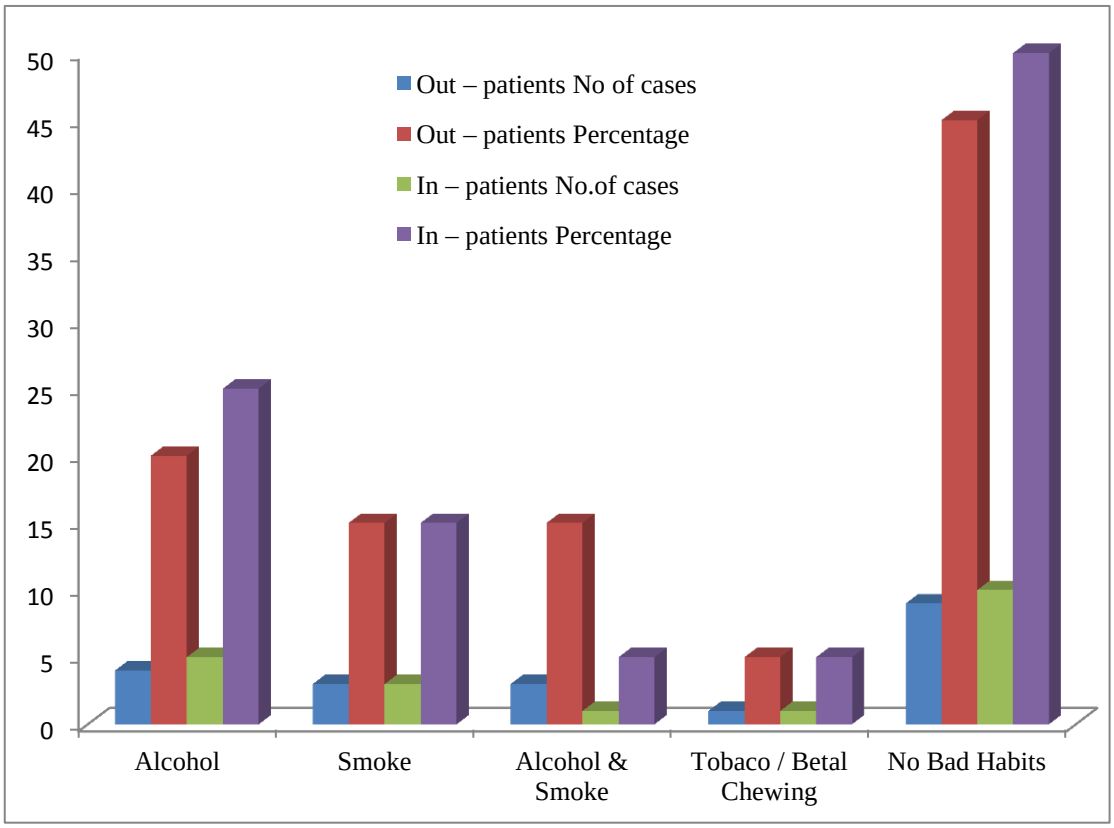


It is noticed that most of the patients affected were taking mixed diet comparing of both vegetarian and mixed food.

TABLE –7

PERSONAL HABITS

S.No	Personal Habits	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Alcohol	04	20	05	25
02	Smoke	03	15	03	15
03	Alcohol & Smoke	03	15	01	05
04	Tobaco / Betal Chewing	01	05	01	05
05	No Bad Habits	09	45	10	50

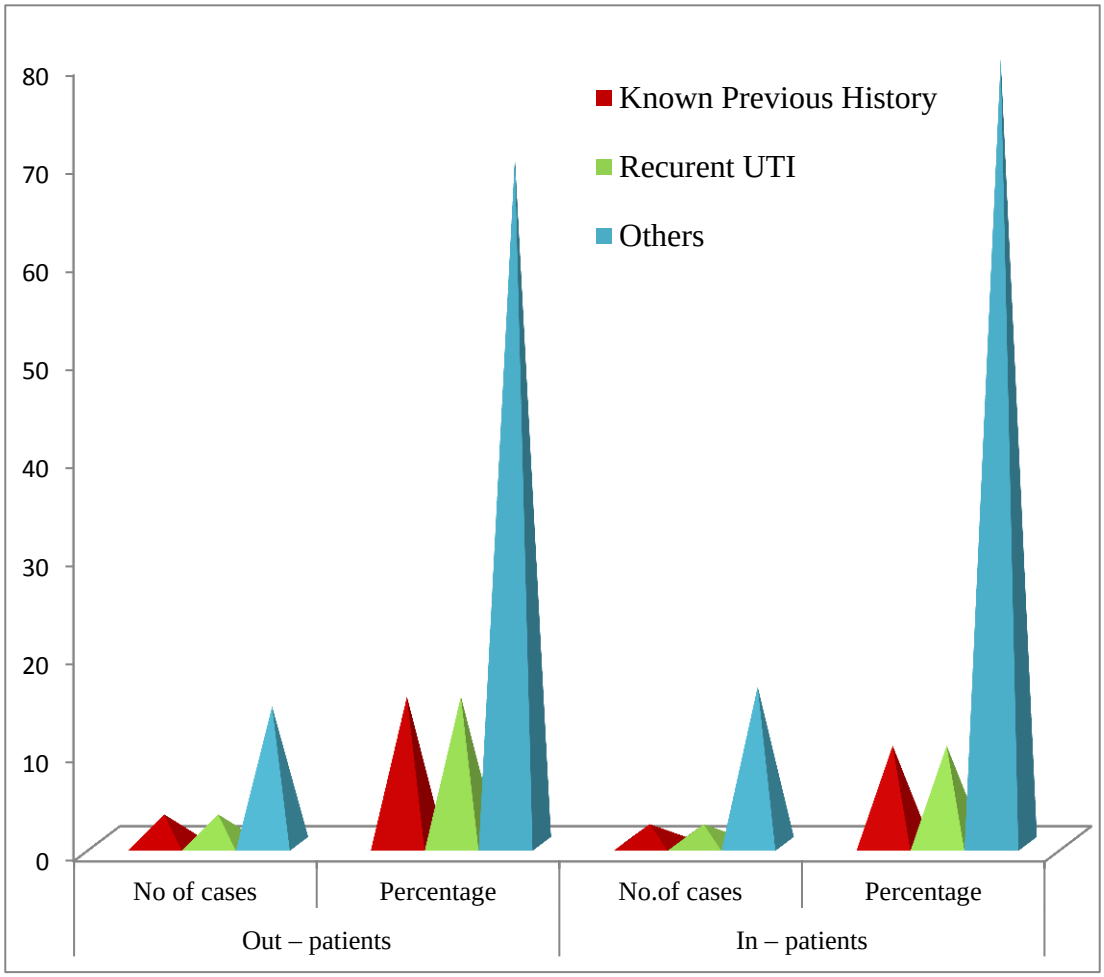


From the above data it is learnt that majority did not have any bad habits and rest were taking alcohol.

TABLE – 8

AETIOLOGICAL FACTORS

S.No	Aetiological Factors	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Known Previous History	03	15	02	10
02	Recurent UTI	03	15	02	10
03	Others	14	70	16	80

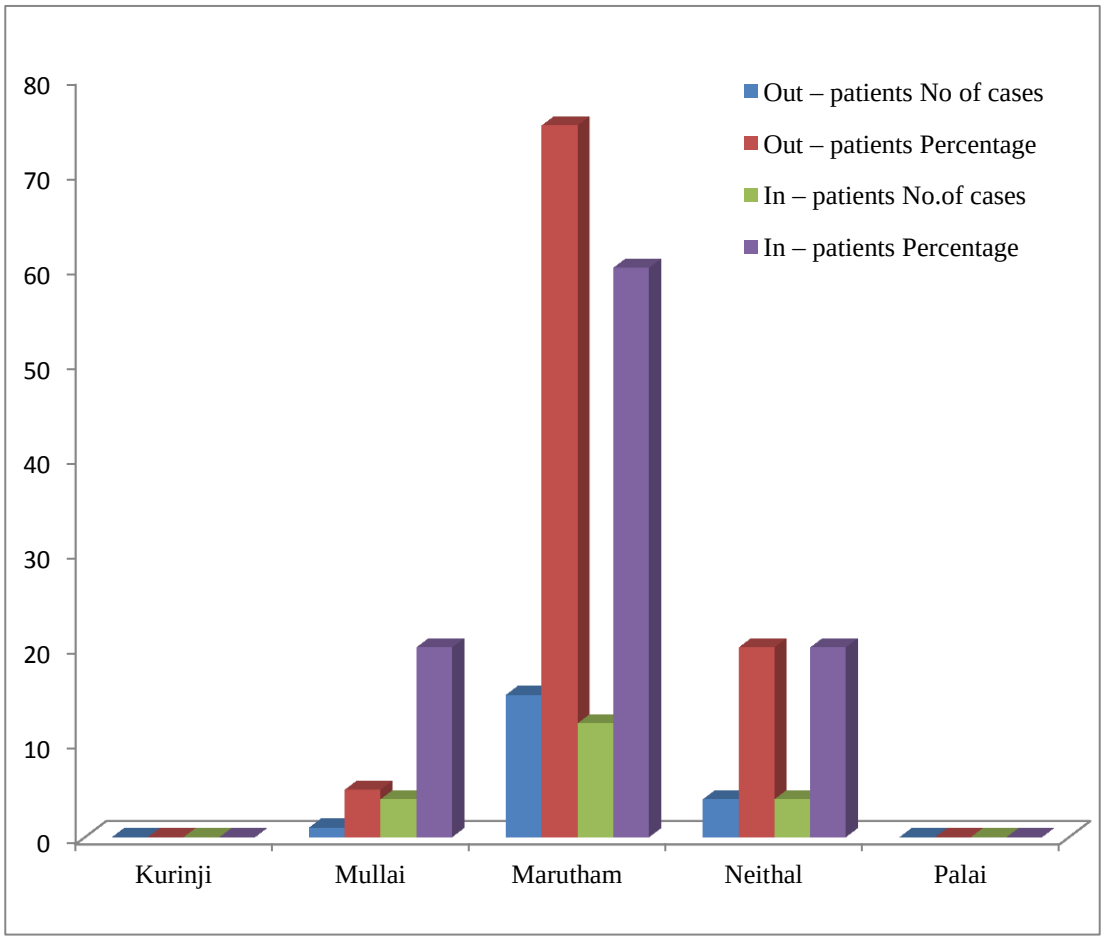


The above table shows that majority of the Out and Inpatients had other such causes.

TABLE – 9

THINAI

S.No	Thinai	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Kurinji	00	00	00	00
02	Mullai	01	05	04	20
03	Marutham	15	75	12	60
04	Neithal	04	20	04	20
05	Palai	00	00	00	00

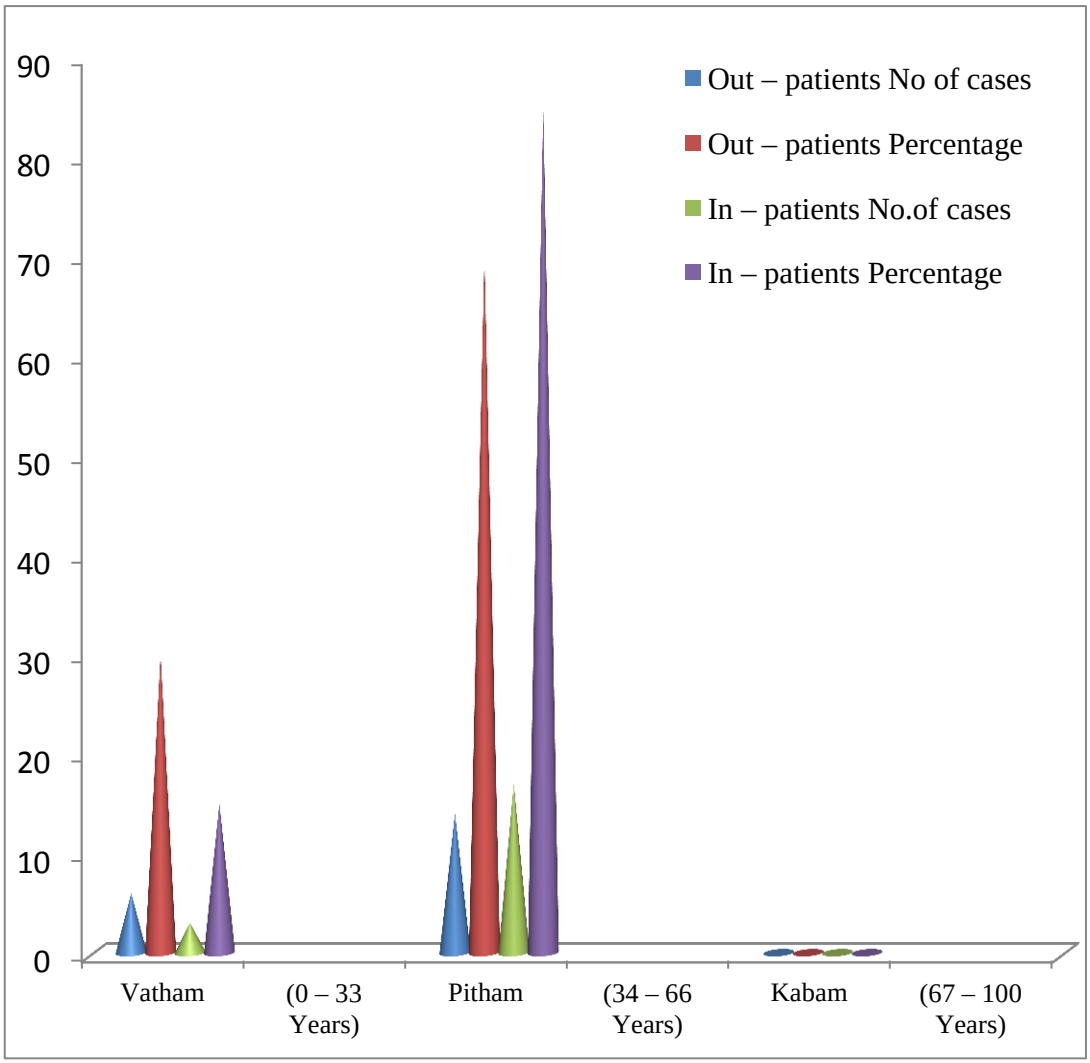


It is observed from the data that majority of the patients suffering from kalladaippu noi were marutha nilam.

TABLE – 10

MUKKUTRA KALAM

S.No	Mukkutrakalam	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Vatham (0 – 33 Years)	06	30	03	15
02	Pitham (34 – 66 Years)	14	70	17	85
03	Kabam (67 – 100 Years)	00	00	00	00

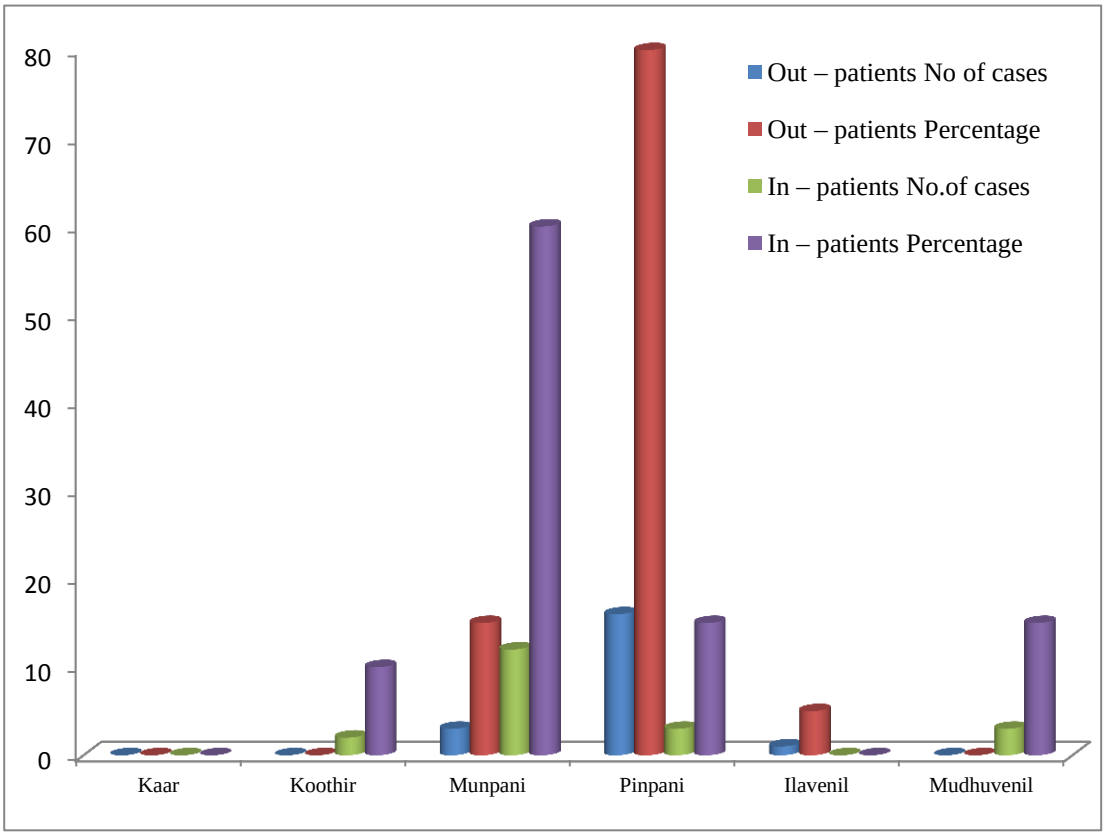


This table reveals that majority of both outpatients and inpatients, suffering from kalladaippu noi were in pitha kaalam (i.e.,) in between 34 – 66 years.

TABLE – 11

PARUVA KALAM

S.No	Paruva kalam	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Kaar	00	00	00	00
02	Koothir	00	00	02	10
03	Munpani	03	15	12	60
04	Pinpani	16	80	03	15
05	Ilavenil	01	05	00	00
06	Mudhuvenil	00	00	03	15

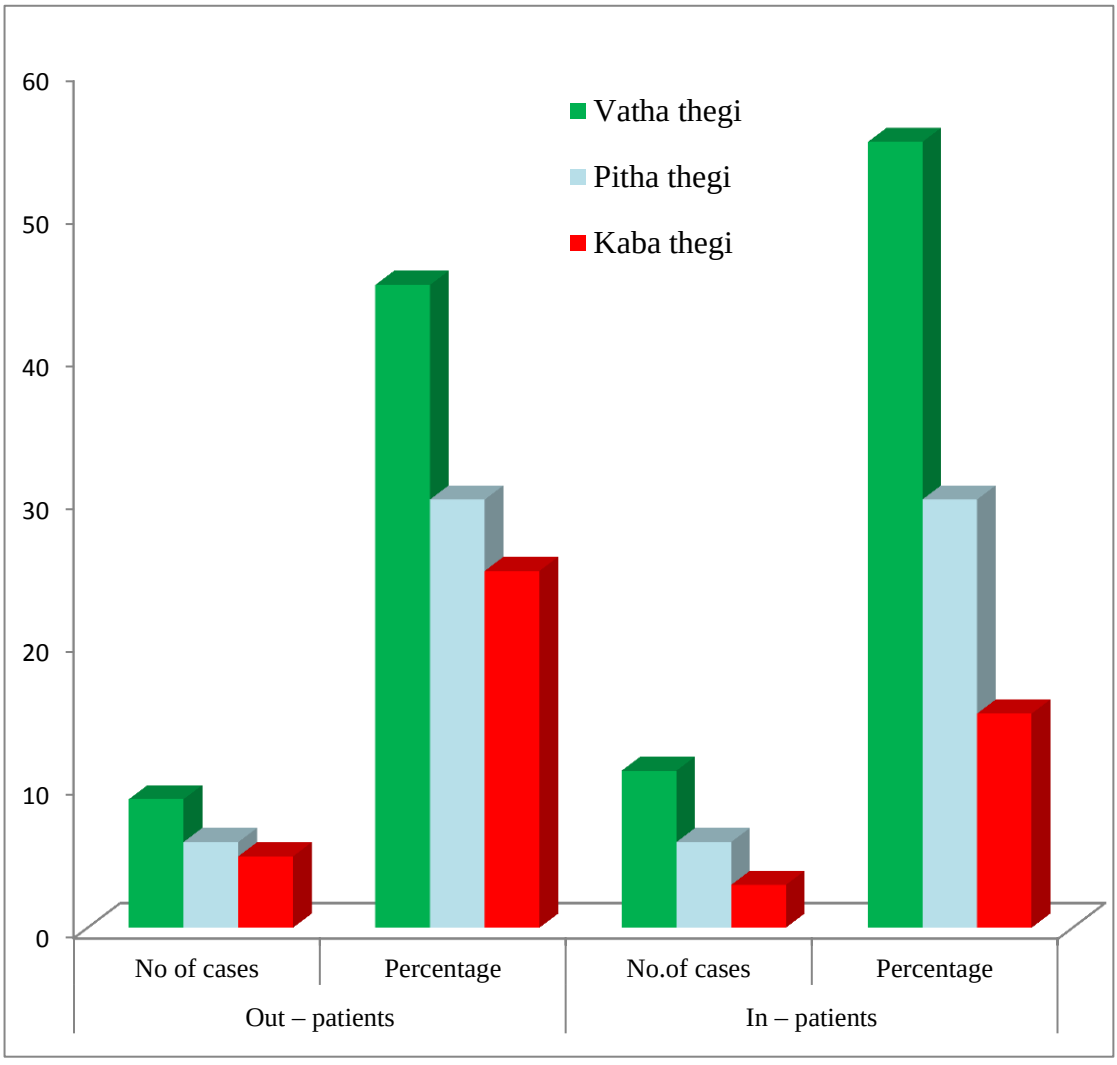


The data reveals that kalladaippu noi most occured during pinpani and munpani kaalam

TABLE – 12

THEGI

S.No	Thegi	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Vatha thegi	09	45	11	55
02	Pitha thegi	06	30	06	30
03	Kabha thegi	05	25	03	15



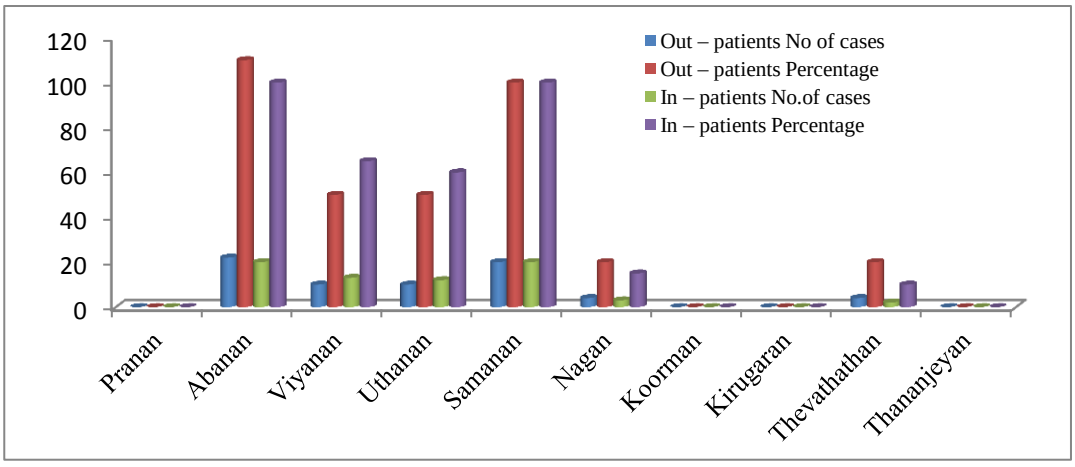
From the above table it is understood that people with vatha thegi was most affected by kalladaippu noi and kaba thegi were least affected

TABLE – 13

MUKKUTRAM

A) Derangement of Vatham

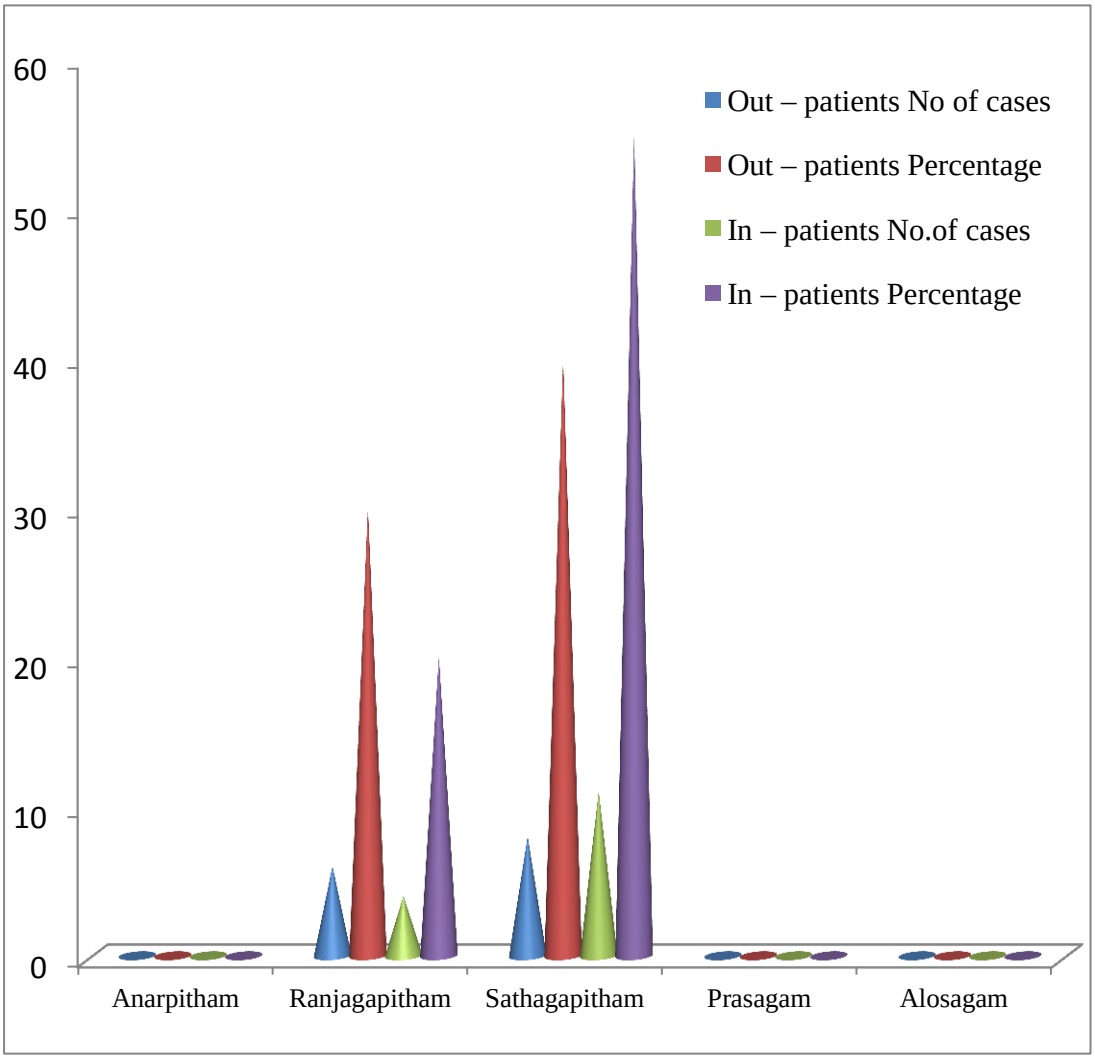
S.No	Vatham	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Pranan	00	00	00	00
02	Abanan	22	110	20	100
03	Viyanan	10	50	13	65
04	Uthanan	10	50	12	60
05	Samanan	20	100	20	100
06	Nagan	04	20	03	15
07	Koorman	00	00	00	00
08	Kirugaran	00	00	00	00
09	Thevathathan	04	20	02	10
10	Thananjeyan	00	00	00	00



The above date reveals that all the patients had derangement in Abanan and Samanan. Viyanana and Uthanan seems to be disturbed in the majority of the patients.

B) Derangement of Pitham

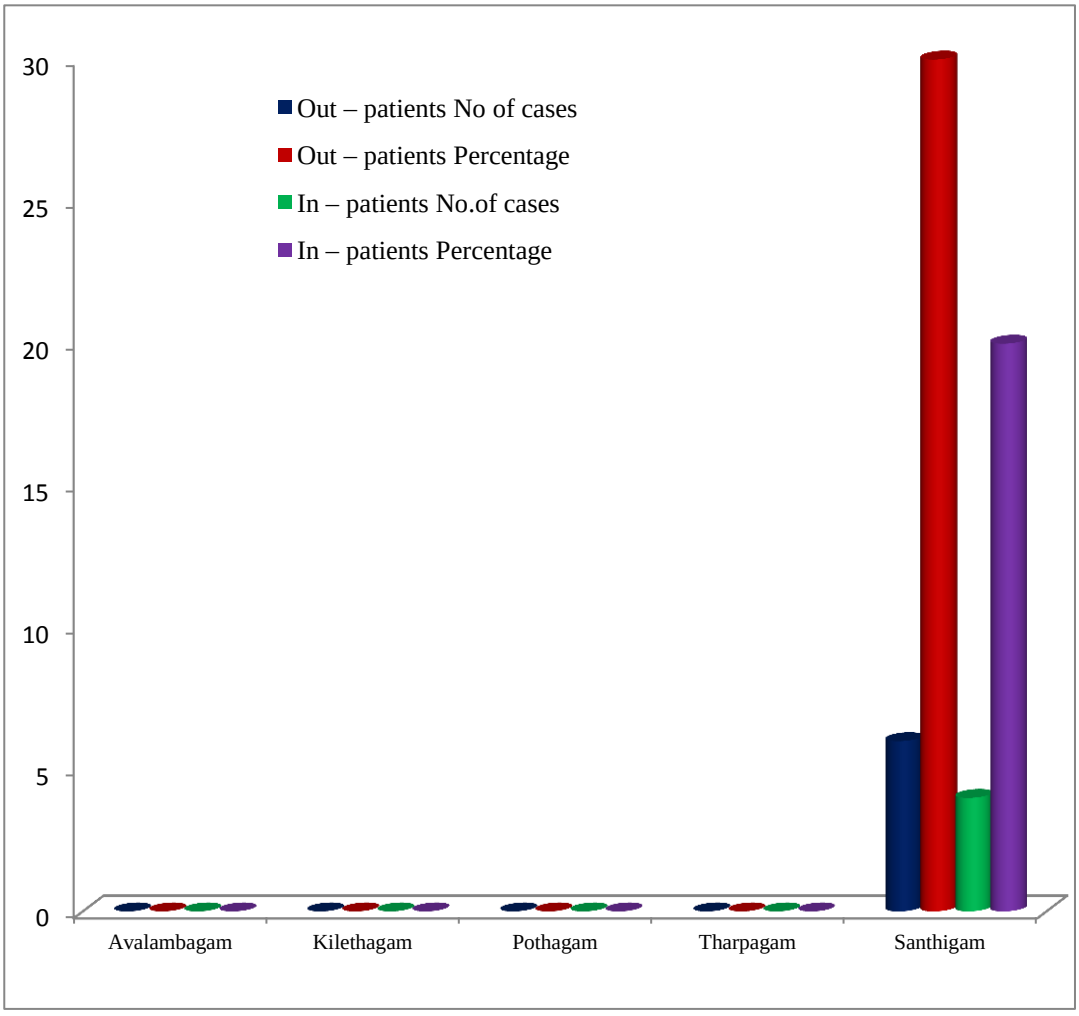
S.No	Pitham	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Analapitham	00	00	00	00
02	Ranjagapitham	06	30	04	20
03	Sathagapitham	08	40	11	55
04	Prasagam	00	00	00	00
05	Alosagam	00	00	00	00



It is revealed from the above table the sathaga pitham was affected in majority of the patients.

C) Derangement of Kapham

S.No	Kabam	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Avalambagam	00	00	00	00
02	Kilethagam	00	00	00	00
03	Pothagam	00	00	00	00
04	Tharpagam	00	00	00	00
05	Santhigam	06	30	04	20

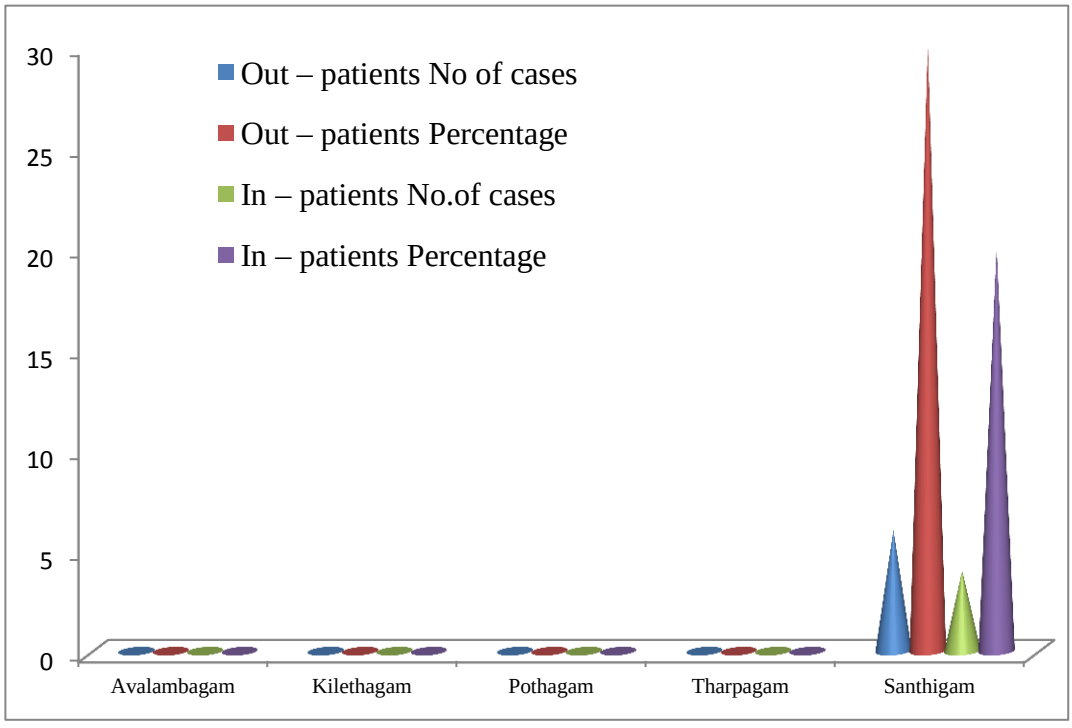


From the above data it is observed that only santhigam was affected in a few number of patients.

TABLE – 14

UDAL THATHUKKAL

S.No	Udal thathukkal	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Saaram	20	100	20	100
02	Seneer	06	30	05	25
03	Oon	00	00	00	00
04	Kozhuppu	00	00	00	00
05	Enbu	05	25	08	40
06	Moolai	00	00	00	00
07	Sukkilam / Suronitham	00	00	00	00

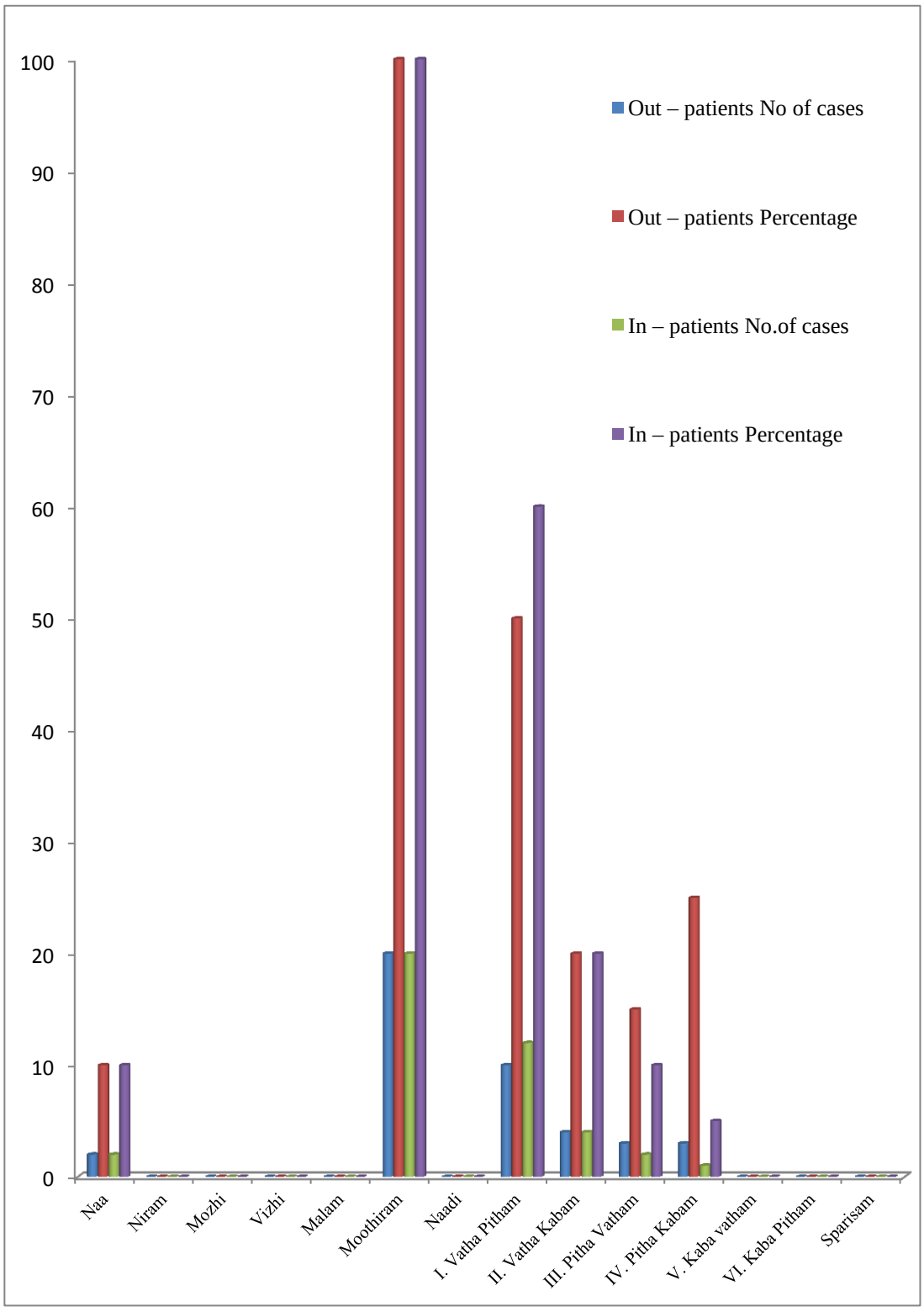


The above table clearly shows that saaram was affected in all patients. Seneer and Enbu Thathu were affected in few patients.

TABLE – 15

ENVAGAI THERVUGAL

S.No	Envagai Therugal	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Naa	02	10	02	10
02	Niram	00	00	00	00
03	Mozhi	00	00	00	00
04	Vizhi	00	00	00	00
05	Malam	00	00	00	00
06	Moothiram	20	100	20	100
07	Naadi	00	00	00	00
	I. Vatha Pitham	10	50	12	60
	II. Vatha Kabam	04	20	04	20
	III. Pitha Vatham	03	15	02	10
	IV. Pitha Kabham	03	25	01	05
	V. Kabha vatham	00	00	00	00
	VI. Kabha Pitham	00	00	00	00
08	Sparisam	00	00	00	00

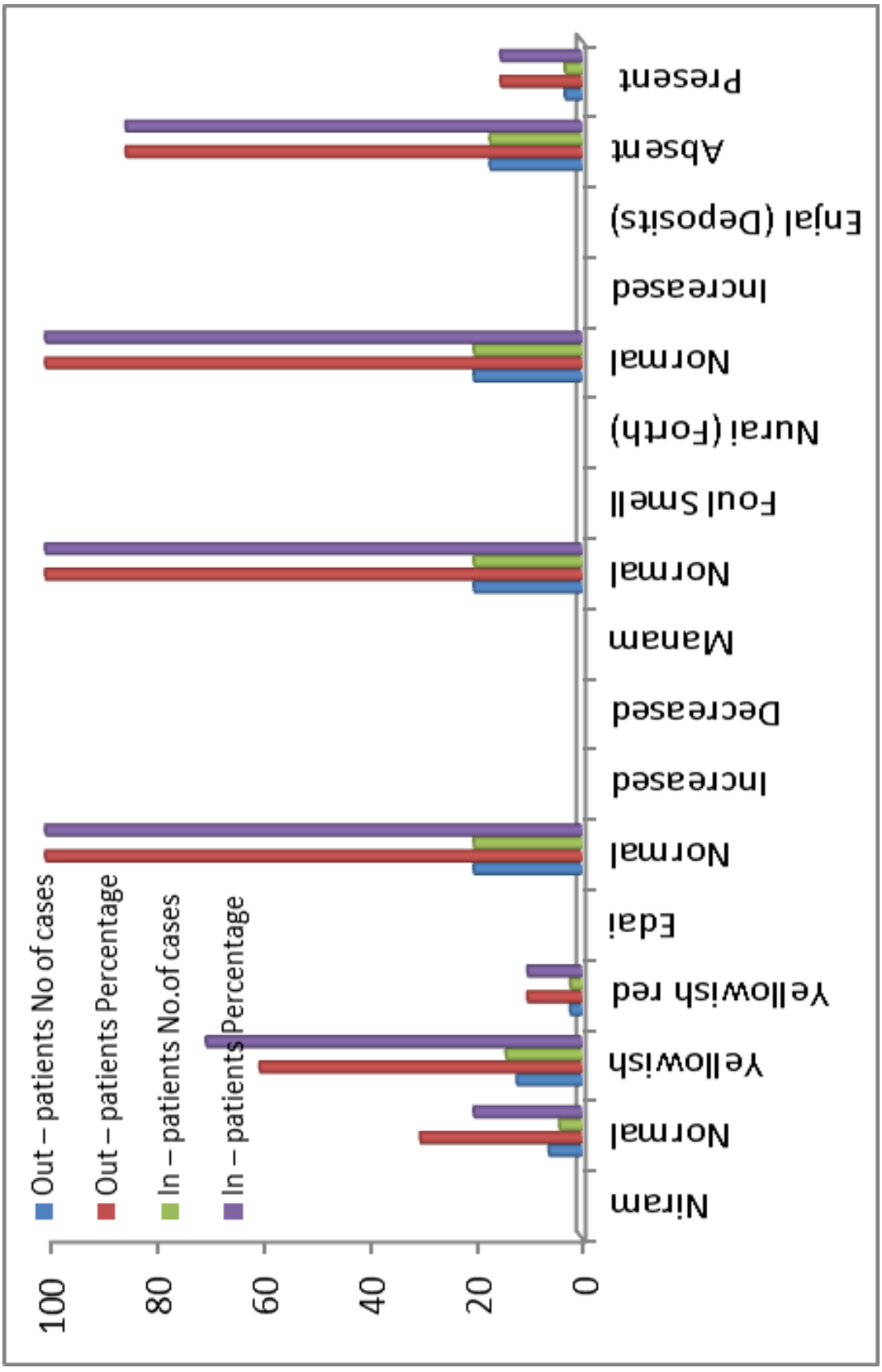


From the above table it can be understood that moothiram was affected remarkably in all the patients considering naadi vatha pitha naadi was observed in majority of the patients.

TABLE - 16

Neerkuri

S.No	Type of Test Result	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Niram				
	Normal	06	30	04	20
	Yellowish	12	60	14	70
	Yellowish red	02	10	02	10
02	Edai				
	Normal	20	100	20	100
	Increased				
	Decreased				
03	Manam				
	Normal	20	100	20	100
	Foul Smell				
04	Nurai (Forth)				
	Normal	20	100	20	100
	Increased				
05	Enjal (Deposits)				
	Absent	17	85	17	85
	Present	03	15	03	15

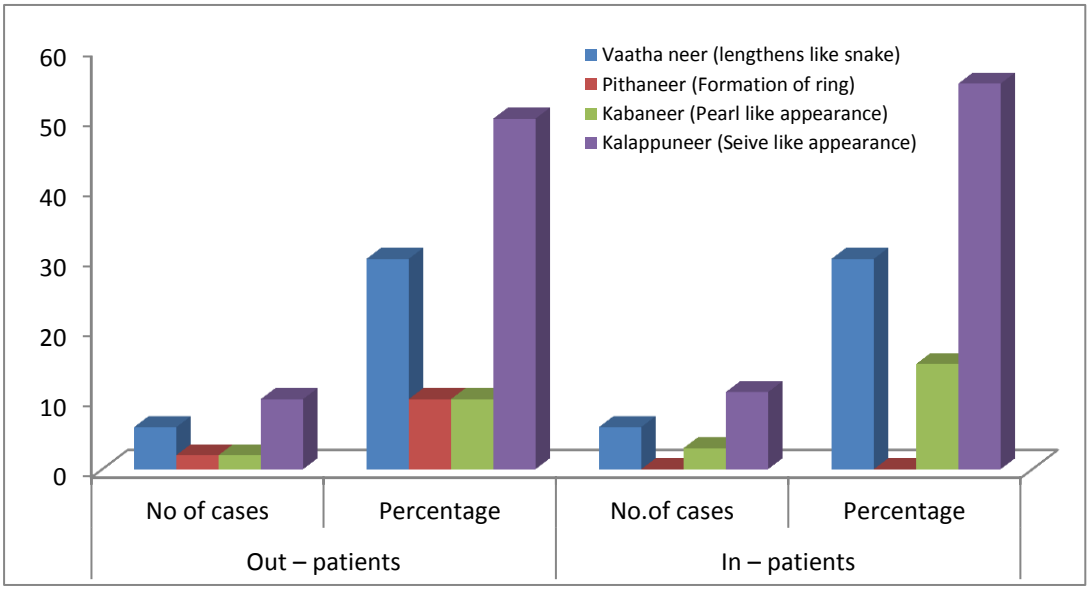


Yellowish coloured urine were noticed in majority of patients. Smell, forth were normal in all of them. RBCS were found to pass in urine of 2 out-patients and 1 in patient.

TABLE - 17

Neikuri

S.No	Neikuri	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Vaatha neer (lengthens like snake)	06	30	06	30
02	Pithaneer (Formation of ring)	02	10	00	00
03	Kabaneer (Pearl like appearance)	02	10	03	15
04	Kalappuneer (Seive like appearance)	10	50	11	55

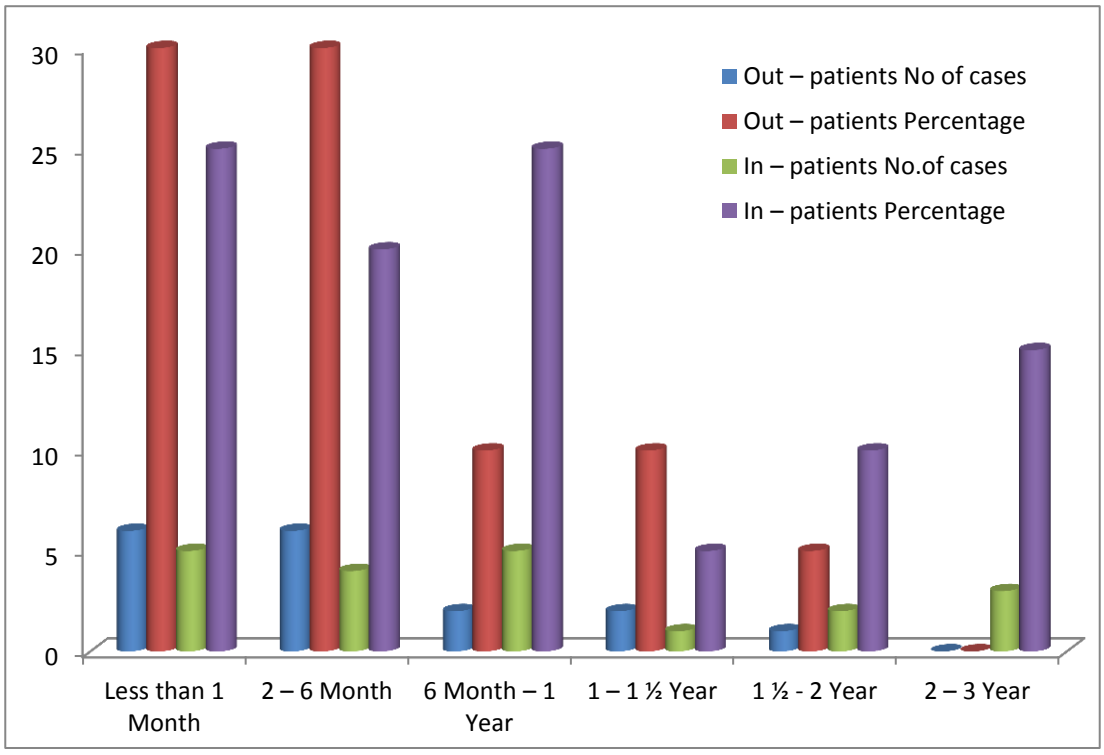


The above data reveals that seive like appearance was found in most of the cares and snake like appearance which indicates vathaneer. Kabaneer pearl like appearance were observed only in 3 outpatients and pitha neer were observed only 2 in out patient.

TABLE - 18

Duration of illness

S.No	Type of Characters	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Less than 1 Month	06	30	05	25
02	2 – 6 Month	06	30	04	20
03	6 Month – 1 Year	02	10	05	25
04	1 – 1 ½ Year	02	10	01	05
05	1 ½ - 2 Year	01	05	02	10
06	2 – 3 Year	03	15	03	15



From the above table it is understood that majority of out-patients expeienced the symptoms of this diseases. During time period of 0-6 months whereas the in patients between 6-12 months. Only a few in patients suffered from the symptoms for more than a year.

TABLE – 19

Types of pain

S.No	Type of Pain	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	Severe Pain	07	35	09	45	-	-	-	-
02	Moderate Pain	07	35	08	40	03	15	01	05
03	Mild Pain	06	30	03	15	-	-	-	-
04	No Pain	-	-	-	-	-	-	-	-

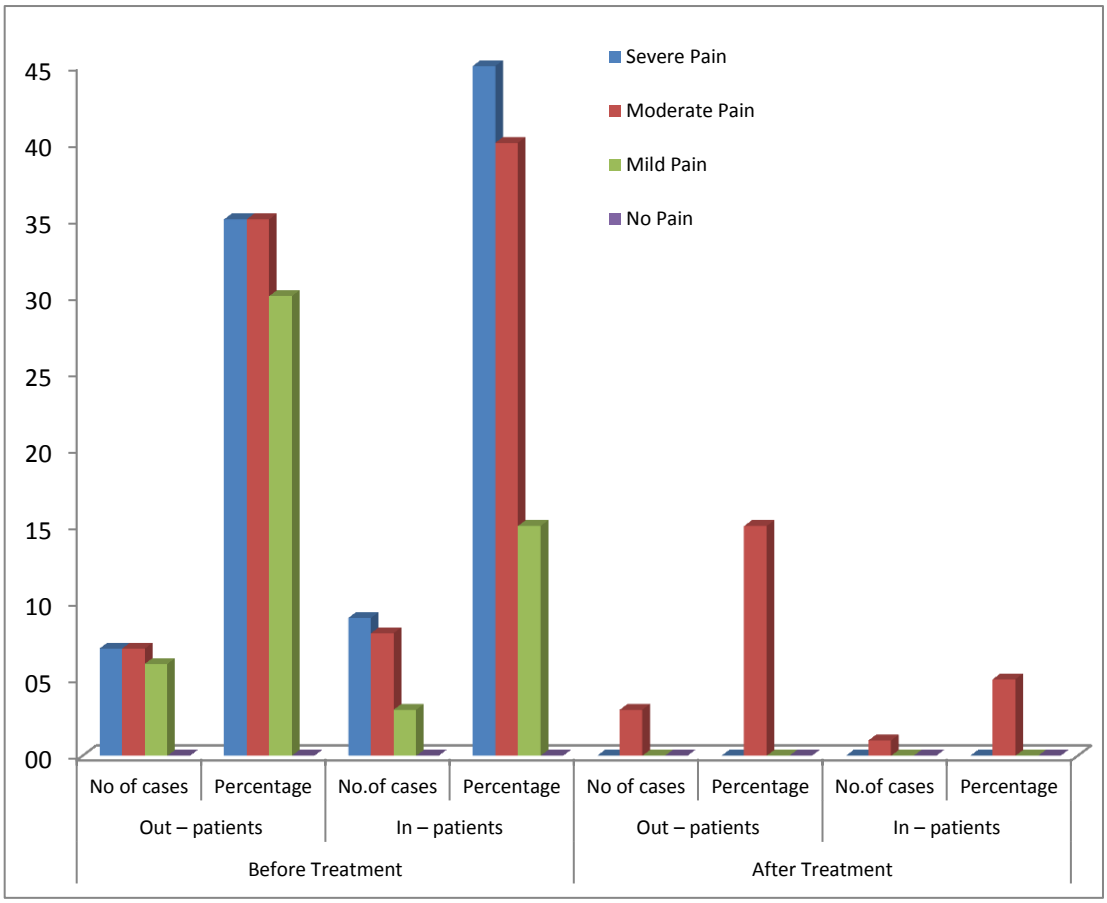
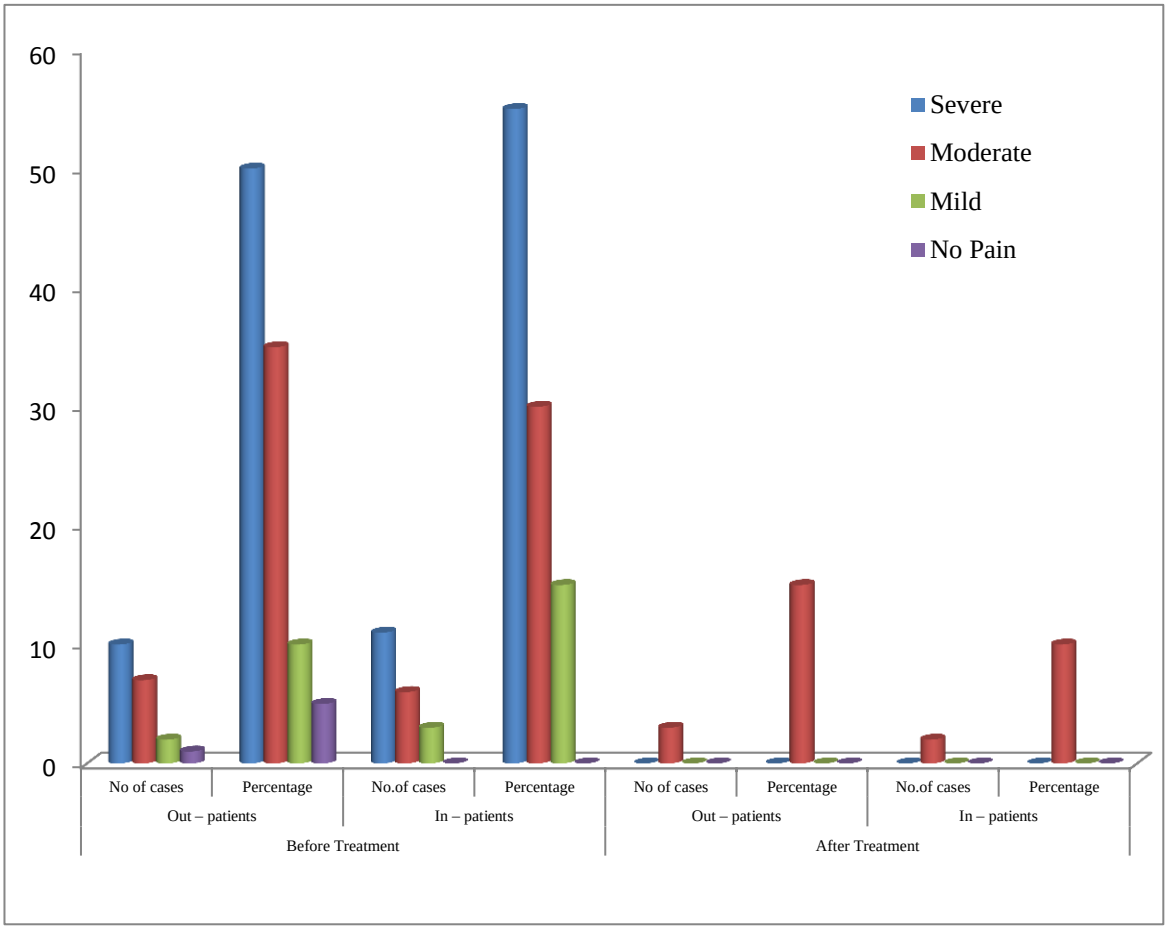


TABLE - 20

Types of Dysuria

S.No	Severity Dysuria	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	Severe	10	50	11	55	00	00	00	00
02	Moderate	07	35	06	30	03	15	02	10
03	Mild	02	10	03	15	00	00	00	00
04	No Pain	01	05	00	00	00	00	00	00

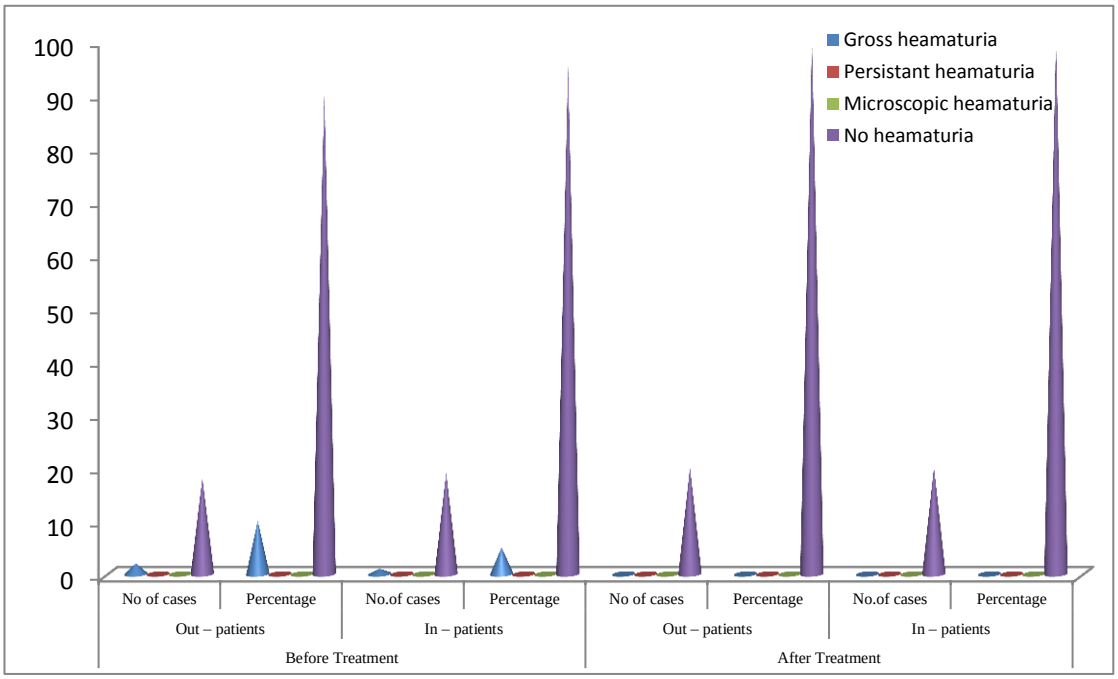


In the present study majority of the cases presented with severe dysuria on their first visit. On their subsquient visists the symptoms decreased and ultimately at the end of if study period there were only 15% of cases with mild dysuria.

TABLE – 21

Types of Heamaturia

S.No	Type of Heamaturia	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	Gross heamaturia	02	10	01	05	00	00	00	00
02	Persistant heamaturia	00	00	00	00	00	00	00	00
03	Microscopic heamaturia	00	00	00	00	00	00	00	00
04	No heamaturia	18	90	19	95	20	100	20	100

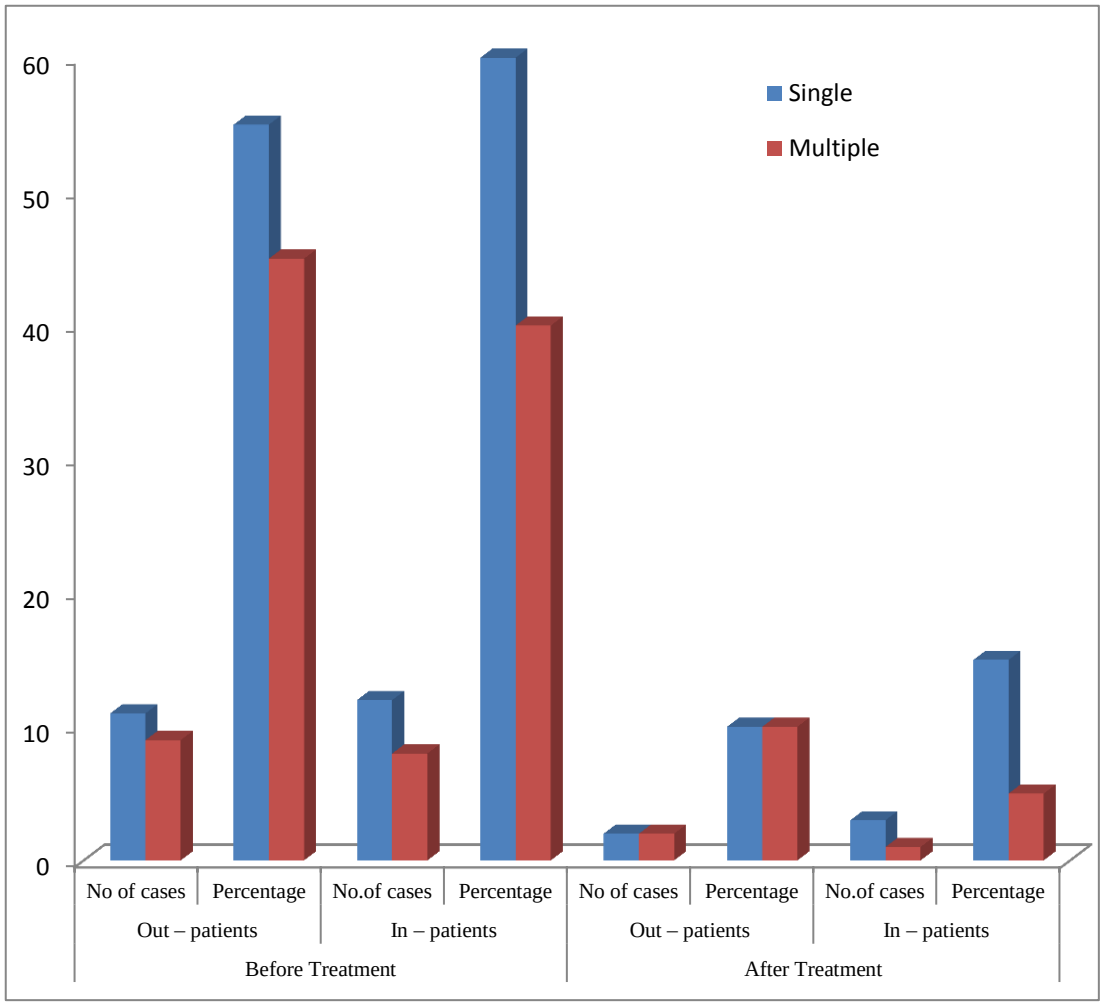


It was noted during the study that 10% of the out patients and 5% of the in-patients had gross haematuria. After treatment the urine of all the patients were normal.

TABLE – 22

Number of Stones

S.No	Type of Stones	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	Single	11	55	12	60	02	10	03	15
02	Multiple	09	45	08	40	02	10	01	05

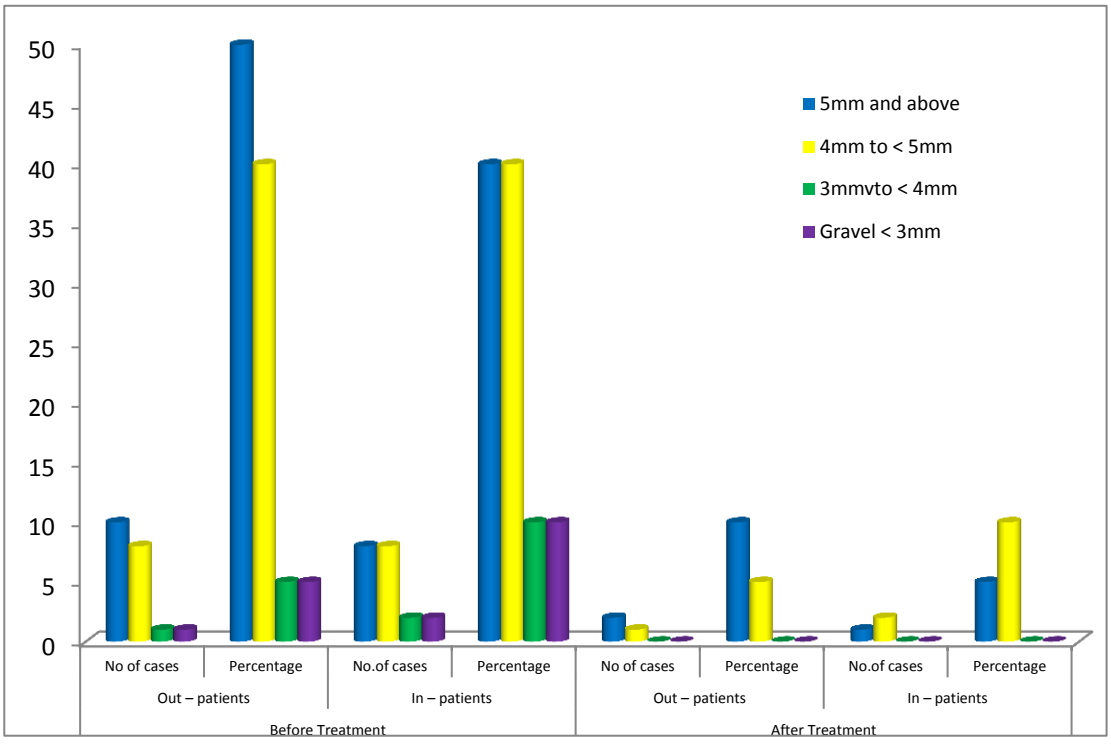


Most of the USG reports of the cases revealed single stones and multiple stone. But after the study there were only few cases with stones with reduction in size of stones.

TABLE – 23

Size of Stones

S.No	Size of Stones	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	5mm and above	10	50	08	40	02	10	01	05
02	4mm to < 5mm	08	40	08	40	01	05	02	10
03	3mm to < 4mm	01	05	02	10	00	00	00	00
04	Gravel < 3mm	01	05	02	10	00	00	00	00

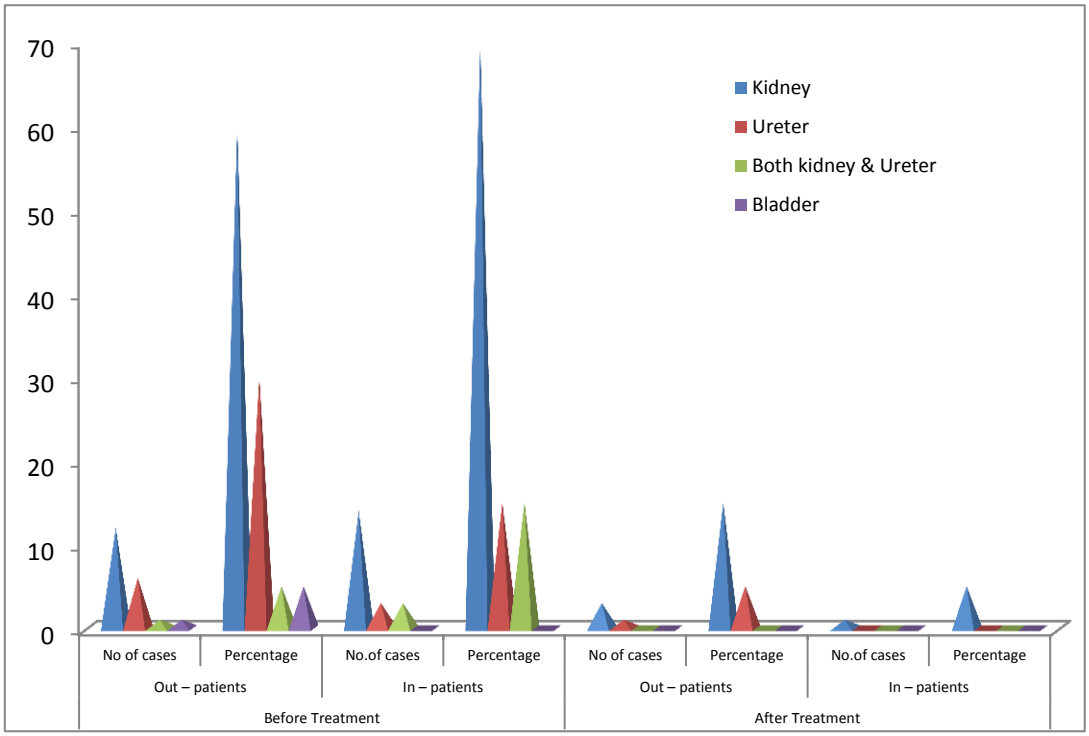


The USG reports of the cases before treatment revealed that in majority of the cases of size of stone 5mm and above. 4mm to <5mm was second major in the cases. At the end of study 10% of out patient and 5% of in patients presented with stone at the size of 5 and above. But the size were reduced comparing to the before treatment report.

TABLE – 24

Position of stones

S.No	Size of Score	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	Kidney	12	60	14	70	03	15	01	05
02	Ureter	06	30	03	15	01	05	00	00
03	Both kidney & Ureter	01	05	03	15	00	00	00	00
04	Bladder	01	05	00	00	00	00	00	00

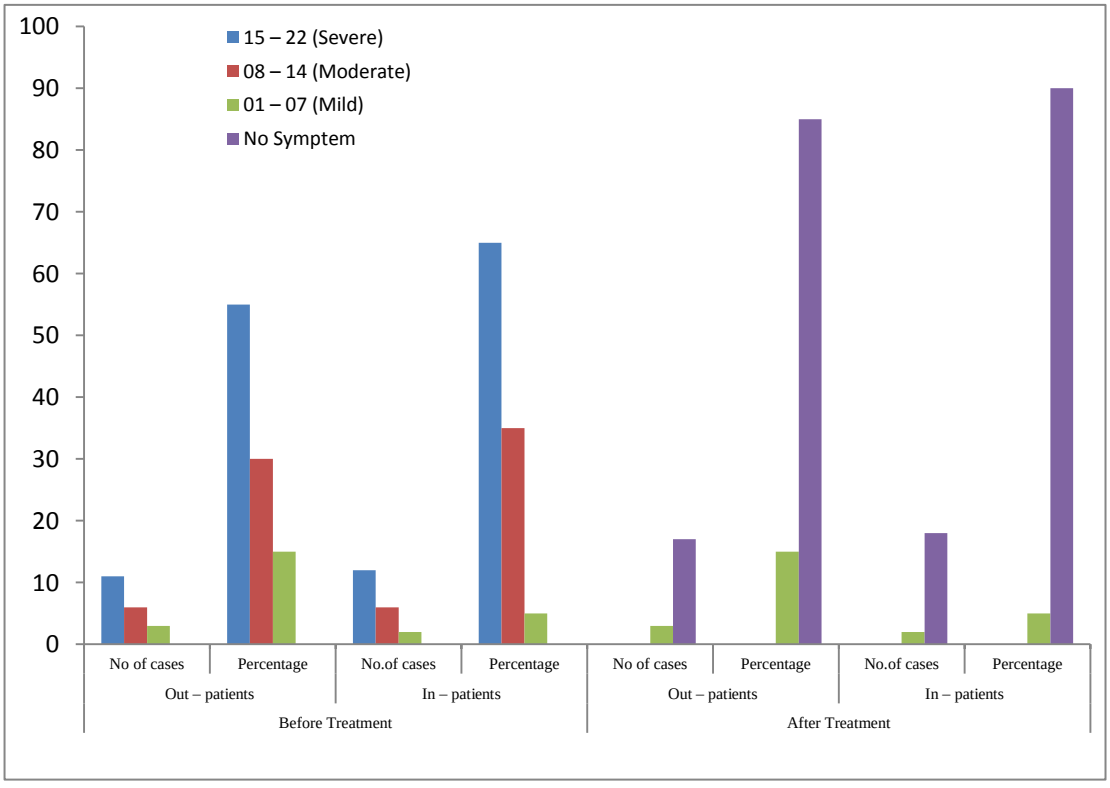


Before treatment majority of the cases were presenting Renal calculi especially in the calyees of the kidney. Ureteric calculi were found in 30% of out patients and 5% of in patient.

TABLE - 25

Urolithiasis symptoms score

S.No	Types of Score	Before Treatment				After Treatment			
		Out – patients		In – patients		Out – patients		In – patients	
		No of cases	%	No.of cases	%	No of cases	%	No.of cases	%
01	15 – 22 (Severe)	11	55	12	65	00	00	00	00
02	08 – 14 (Moderate)	06	30	06	35	00	00	00	00
03	01 – 07 (Mild)	03	15	02	05	03	15	02	05
04	No Symptom	00	00	00	00	17	85	18	90

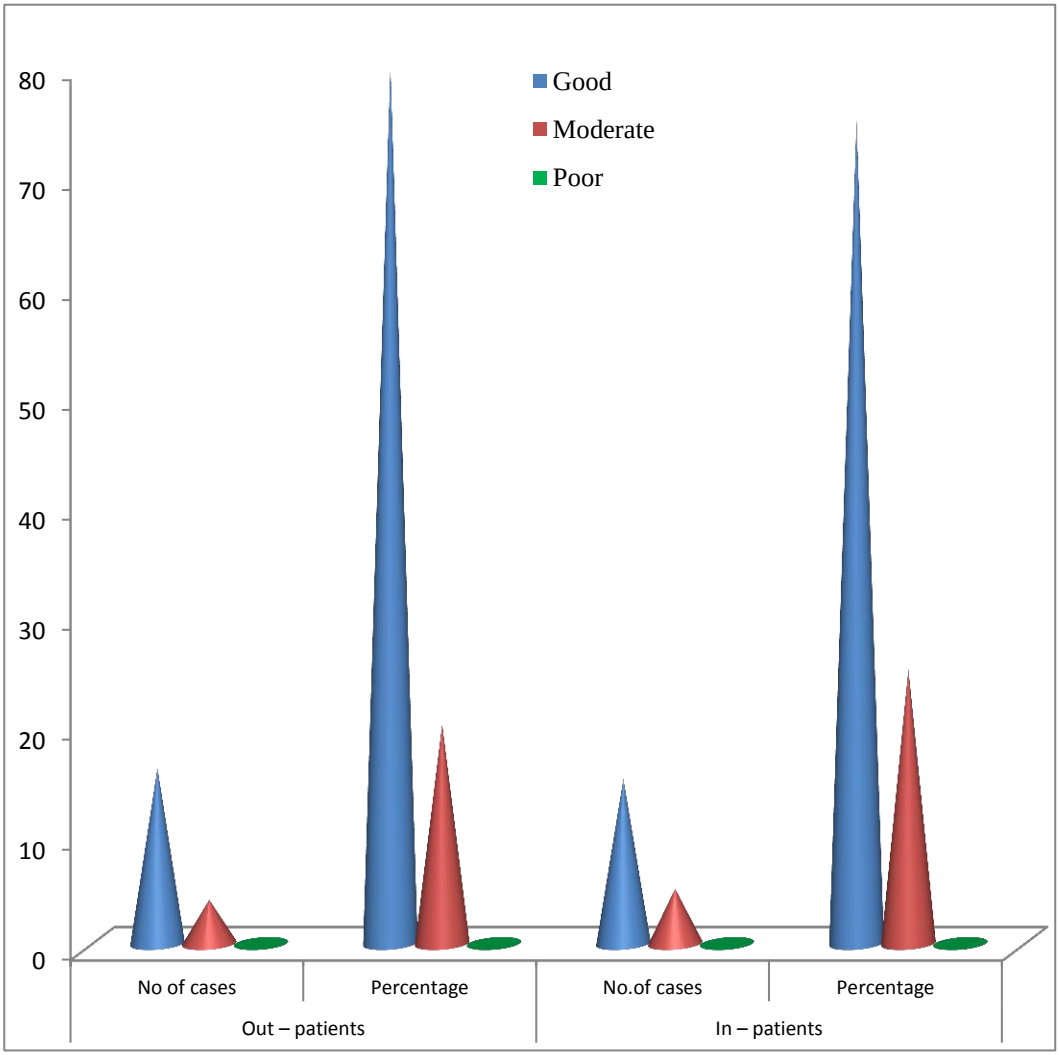


The above tubular column reveals that the score severe high before treatment in majority of cause. It was noticed that the score gradually decreased during the course of the study and ultimately 85% of out patients and 90% of in patients had score (ie) no symptoms

TABLE - 26

Grading outcomes of the study

S.No	Type of Characters	Out – patients		In – patients	
		No of cases	Percentage	No.of cases	Percentage
01	Good	16	80	15	75
02	Moderate	04	20	05	25
03	Poor	00	00	00	00



From the above observations it is determined that good response was noted in 75% of out-patients and 85% of in patients. Moderate response was observed in 25% of out patients and 15% of in patients. Hence, it can be concluded that the trial medicine, Nilakumilaver kudineer is very effictive in curing kalladaippu noi.

CASE SHEET OF 20 OUT PATIENTS TREATED FOR KALLAIDAIPPU NOI

SNo	OP.No	Name	Age	Sex	Starting of Treatment	End of Treatment	No of Days Treated	Result
01	6749	Gabreal Ganesan	48	M	21.01.2016	19.02.2016	30 Days	Good
02	9209	Aavudaiappan	30	M	28.01.2016	26.02.2016	30 Days	Fair
03	11609	Sangaralingam	36	M	04.02.2016	04.03.2016	30 Days	Good
04	12967	Ismail	40	M	08.02.2016	08.03.2016	30 Days	Good
05	10907	Joys Gracy	34	F	02.02.2016	02.03.2016	30 Days	Good
06	14148	Latha	43	F	11.02.2016	11.03.2016	30 Days	Good
07	13799	Kumar	47	M	10.02.2016	10.03.2016	30 Days	Good
08	21310	Raja	28	M	04.03.2016	02.04.2016	30 Days	Good
09	20374	Sudalai	58	M	01.03.2016	29.03.2016	30 Days	Good
10	9053	Samikkannu	40	M	28.01.2016	26.02.2016	30 Days	Good
11	11638	Malika	37	F	04.02.2016	04. 03.2016	30 Days	Good
12	16904	Shahul Hameed	35	M	19.02.2016	19.03.2016	30 Days	Good
13	18009	Ramachandran	60	M	23.02.2016	23.03.2016	30 Days	Good
14	16456	Sundari	30	F	18.02.2016	18.03.2016	30 Days	Good
15	23579	Nehrumani	52	M	12.03.2016	10.04.2016	30 Days	Good
16	23076	Vincent John	60	M	10.03.2016	08.04.2016	30 Days	Good
17	9660	Banupriya	30	F	29.01.2016	27.02.2016	30 Days	Fair
18	3611	Durairaj	48	M	11.01.2016	11.02.2016	32 Days	Fair
19	22817	Anandhi	48	F	09.03.2016	07.04.2016	30 Days	Good
20	14835	Sellaiyah	51	M	15.02.2016	15.03.2016	30 Days	Good

BLOOD INVESTIGATIONS OF OUT – PATIENTS

S. No	Op. No	Before Treatment							After Treatment						
		WBC Total Cells/ Cub.mm	WBC – DC%			ESR		Hb %	WBC Total Cells / Cub.mm	WBC – DC%			ESR		Hb%
			P	L	E	1/2 hr	1hr			P	L	E	1/2 hr	1hr	
1	6749	7900	65	32	3	13	17	12.5	8100	62	34	4	5	7	13
2	9209	8400	62	34	4	12	20	10.5	8000	59	38	3	10	22	12.0
3	11609	8100	58	41	1	12	20	9	7700	64	33	3	30	3	12.4
4	12967	7500	57	41	1	14	22	12	8700	55	43	2	28	32	12.3
5	10907	9500	66	28	6	18	20	9.6	9500	63	30	7	20	25	13
6	14148	9700	66	26	8	20	23	13	8800	55	39	6	26	32	13.8
7	13799	8500	67	29	4	35	41	14.5	8000	60	35	5	12	20	12.8
8	21310	7900	64	31	5	31	35	10.5	8500	60	38	2	18	25	13.5
9	20374	8700	59	38	3	18	20	9	8000	61	37	2	23	28	12.5
10	9053	7500	69	28	3	20	23	9.7	8500	62	35	3	26	30	11
11	11638	8500	64	34	2	26	30	12.3	7500	60	38	2	24	31	11.8
12	16904	8300	66	30	4	18	26	13.5	7300	56	38	6	27	32	13.5
13	18009	7000	65	30	5	28	33	14	8800	66	28	6	20	29	14
14	16546	8300	59	38	3	12	18	10	9300	62	35	3	16	22	11.5
15	23579	7100	57	40	3	22	30	11	9000	67	38	5	19	25	14.5
16	23076	7500	58	40	2	20	23	9.1	8300	65	30	5	22	28	10
17	9660	7200	60	35	5	32	40	10.5	9500	67	28	5	23	31	9.6
18	3611	9000	65	32	3	22	30	11.5	7800	54	43	3	19	28	12
19	22817	9100	52	41	7	30	35	12.5	8100	52	41	7	26	33	10.5
20	14838	9800	65	30	5	20	30	10	7200	59	38	3	23	30	11

BIO CHEMICAL ANALYSIS

S.No	OP NO	Blood (Sugar) mgs%		Blood (Urea (mgs%)		Total Cholesterol (mg%)		Serum Creatinine (mg s%)	
		BT	AT	BT	AT	BT	AT	AT	BT
1	6749	86	106	19	21	162	175	0.8	0.7
2	9209	85	100	21	20	160	165	0.7	0.8
3	11609	90	106	30	28	178	180	0.5	0.6
4	12967	125	100	14	20	150	162	1.0	1.3
5	10907	80	122	20	26	170	175	1.2	1.0
6	14148	100	100	28	26	162	160	1.0	1.3
7	13799	118	123	20	22	151	156	0.8	0.6
8	21310	101	117	23	20	202	198	1.2	0.5
9	20374	146	120	18	20	165	170	1.1	0.7
10	9053	132	142	25	23	183	176	0.7	0.7
11	11638	96	100	25	26	163	160	0.6	1.0
12	16904	114	110	30	28	180	185	0.4	0.8
13	18009	86	95	22	20	202	200	0.8	1.0
14	16546	90	126	30	33	160	160	0.6	0.7
15	23579	97	112	14	20	202	200	0.8	1.0
16	23076	130	140	29	32	184	185	0.7	0.8
17	9660	100	115	27	30	171	165	0.6	0.5
18	3611	110	120	29	31	165	168	0.5	0.7
19	22817	80	77	20	24	189	185	0.8	1.0
20	14838	86	95	20	26	202	200	1.2	1.0

URINE ANALYSIS

S.No	OP No	Urine Analysis											
		Before Treatment						After Treatment					
		Alb	Sug	P.C	E.C	RBC	Cast/ Crystal	Alb	Sug	P.C	E.C	RBC	Cast/ Crystal
1	6749	NIL	NIL	NAD	2-3	NAD	NAD	NIL	NIL	NAD	1-2	NAD	NAD
2	9209	NIL	NIL	1-4	4-5	2-3	NAD	NIL	NIL	NAD	NAD	NAD	NAD
3	11609	Traces	NIL	1-2	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
4	12967	Traces	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
5	10907	NIL	NIL	2-5	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
6	14148	NIL	++	NAD	1-2	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
7	13799	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
8	21310	++	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
9	20374	NIL	NIL	1-3	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
10	9053	NIL	++	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
11	11638	NIL	NIL	NAD	2-5	5.0	NAD	NIL	NIL	NAD	NAD	NAD	NAD
12	16904	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
13	18009	NIL	NIL	1-3	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
14	16546	Traces	NIL	1-5	NAD	NAD	NAD	NIL	Traces	NAD	NAD	NAD	NAD
15	23579	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
16	23076	NIL	++	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
17	9660	NIL	NIL	NAD	2-3	4-5	NAD	NIL	NIL	NAD	NAD	NAD	NAD
18	3611	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
19	22817	NIL	NIL	1-4	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
20	14838	++	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD

CASE SHEET OF 20 IN PATIENTS TREATED FOR KALLAIDAIPPU NOI

S.No	IP.No	Name	Age	Sex	Starting of Treatment	End of Treatment	No of Days Treated	Results
01	323	Pirapammal	60	F	08.02.2016	05.03.2016	26 Days	Good
02	328	Pappathi	38	F	03.02.2016	22.02.2016	20 Days	Good
03	299	Subbhuraj	55	M	04.02.2016	28.02.2016	25 Days	Good
04	276	Datchanamoorthi	53	M	02.02.2016	03.03.2016	31 Days	Good
05	275	Paulraj	60	M	02.02.2016	02.03.2016	30 Days	Good
06	973	Subbhammal	60	F	19.02.2016	01.03.2016	29 Days	Good
07	468	Lakshmanan	58	M	22.02.2016	26.03.2016	34 Days	Fair
08	499	Kamalam	50	F	25.02.2016	16.03.2016	20 Days	Good
09	550	Latha	30	F	29.02.2016	09.04.2016	39 Days	Fair
10	656	Subbulakshmi	42	F	10.03.2016	03.04.2016	20 Days	Good
11	590	Indhiran	48	M	04.02.2016	30.03.2016	25 Days	Good
12	671	Isaki	56	M	03.02.2016	25.03.2016	22 Days	Good
13	678	Durai	49	M	12.03.2016	14.04.2016	32 Days	Good
14	734	Nayinar	52	M	18.03.2016	16.04.2016	30 Days	Good
15	974	Perumal	56	M	19.03.2016	09.04.2016	20 Days	Good
16	746	Sujatha	30	F	19.03.2016	14.04.2016	25 Days	Good
17	796	Lakshmi	58	F	25.03.2016	20.04.2016	25 Days	Good
18	740	Saraswathi	60	F	25.03.2016	23.04.2016	28 Days	Good
19	793	Balammal	29	F	28.03.2016	26.04.2016	30 Days	Fair
20	950	Ponraj	42	M	03.03.2016	01.04.2016	30 Days	Good

BIO CHEMICAL ANALYSIS

S. No	Ip. No	Before Treatment							After Treatment						
		WBC Total Cells/ Cub.mm	WBC - DC%			ESR		Hb %	WBC Total Cells / Cub.mm	WBC – DC%			ESR		Hb %
			P	L	E	1/2 hr	1hr			P	L	E	1/2 hr	1hr	
1	323	8600	62	31	7	38	42	10.2	9000	55	43	2	32	40	11
2	328	8800	50	40	10	25	31	9.2	8200	62	35	3	16	19	9.5
3	299	7800	67	30	3	6	17	12.8	8500	68	29	3	8	13	11.8
4	276	7000	62	30	8	36	42	9.8	8600	58	38	14	27	32	11.2
5	275	8000	55	30	15	30	38	13.8	8500	68	26	16	12	19	10.2
6	973	8100	60	35	5	12	16	11.8	7100	60	33	7	22	25	10.6
7	468	8300	61	27	12	30	32	10.4	7700	68	28	13	12	20	11
8	499	8800	72	15	13	32	37	11.5	6800	58	40	2	20	23	9.8
9	550	7000	58	40	2	25	31	12.4	7500	67	31	2	12	19	12.6
10	656	7100	64	32	4	12	25	10	7800	65	33	2	6	17	10
11	590	8800	66	28	6	12	20	11	8100	65	30	5	8	15	11.5
12	671	8300	54	28	18	26	30	11	9100	62	35	3	32	38	12
13	678	8500	60	31	9	11	18	9.5	8000	55	30	15	30	38	12.5
14	734	7200	64	35	1	10	15	12	7900	58	40	2	15	28	11
15	974	7000	54	31	15	28	32	11	7200	52	33	15	16	20	10.5
16	746	6500	52	37	1	20	29	10.8	7800	70	22	8	16	19	12
17	796	6800	70	27	3	8	19	9.5	8400	60	36	4	20	23	11.5
18	740	7600	64	35	1	11	15	13.5	7700	56	42	2	8	13	10.8
19	793	7100	59	36	15	18	25	10	7500	68	30	2	16	21	12
20	950	8400	64	34	2	15	18	11.2	8400	62	37	1	21	24	12

BIO CHEMICAL ANALYSIS

S.N0	IP No	Blood (Sugar mgs%)		Blood (Urea (mgs%)		Total Cholesterol (mg%)		Serum Creatinine (mgs %)	
		BT	AT	BT	AT	BT	AT	AT	BT
1	323	104	110	19	18	115	95	0.5	0.7
2	328	120	116	33	35	170	182	0.8	0.6
3	299	94	120	18	22	144	140	1.2	1.0
4	276	107	110	35	33	110	128	0.8	0.5
5	275	125	100	15	12	215	218	0.6	0.8
6	973	123	120	33	28	235	230	1.0	0.8
7	468	99	102	23	28	183	170	0.6	0.5
8	499	125	110	24	22	174	168	0.7	0.5
9	550	150	140	35	33	200	204	1.2	0.9
10	656	101	125	26	28	207	210	0.6	0.5
11	590	101	120	30	28	220	200	0.8	0.6
12	671	98	114	28	32	180	200	0.4	0.6
13	678	130	142	26	28	167	170	0.8	0.7
14	734	110	108	26	22	143	140	1.2	1.0
15	974	137	130	30	34	160	146	0.7	0.5
16	746	113	133	15	21	125	117	0.5	0.6
17	796	100	94	25	21	164	140	0.5	0.8
18	740	126	130	18	20	176	161	0.3	0.6
19	793	104	110	27	32	245	220	0.7	0.8
20	950	100	98	33	38	220	200	1.2	0.8

URINE ANALYSIS

S.No	IP No	Urine Analysis											
		Before Treatment						After Treatment					
		Alb	Sug	P.C	E.C	RBC	Cast/ Crystal	Alb	Sug	P.C	E.C	RBC	Cast/ Crystal
1	323	NIL	NIL	1-2	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
2	328	NIL	NIL	1-4	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
3	299	NIL	NIL	1-2	1-2	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
4	276	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
5	275	Traces	NIL	2-5	NAD	NAD	NAD	Traces	NIL	NAD	NAD	NAD	NAD
6	973	++	NIL	NAD	NAD	5-6	NAD	NIL	NIL	NAD	NAD	NAD	NAD
7	468	NIL	NIL	NAD	2-3	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
8	499	NIL	++	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
9	550	NIL	NIL	5-7	NAD	5.0	NAD	NIL	NIL	1-2	NAD	NAD	NAD
10	656	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
11	590	++	NIL	2-4	2-5	NAD	NAD	NIL	NIL	NAD	0-1	NAD	NAD
12	671	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
13	678	NIL	NIL	2-4	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
14	734	NIL	NIL	NAD	NAD	5.2	NAD	NIL	NIL	NAD	NAD	NAD	NAD
15	974	Traces	NIL	2-3	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
16	746	NIL	NIL	NAD	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
17	796	NIL	NIL	2-5	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
18	740	NIL	++	NAD	1-2	NAD	NAD	NIL	NIL	NAD	0-1	NAD	NAD
19	793	NIL	NIL	2-3	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD
20	950	NIL	NIL	1-2	NAD	NAD	NAD	NIL	NIL	NAD	NAD	NAD	NAD

DISCUSSION

The present study is "To evaluate the efficacy of Clinical Randamized control trial drug **NILAKUMILAVER KUDINEER** (Internal)" in the treatment of kalladaippu,(Urolithiasis). Out of 20 out patients and 20 in patients were selected based on clinical features and modern investigations parameters and siddha diagnostic methods (envagai thaervugal) were carried out after the disease.It is confirmed by ultrasonagram. The trial drug **NILAKUMILAVER KUDINEER** was prepared and given to the patients. The reports of urine, blood and general details were collected from the patients before and after treatment. The urolithiasis symptom score of each patient before and after treatment were compared to assess the therapeutic value of the trail drug **"NILAKUMILAVER KUDINEER"** .

AGE

In this study, among the 20 out-patients, Kalladaippu noi was found to be the most common in the age group, 41-50 years (35%) and in case of inpatients, it was predominant (65%) in the age group, of 51-60 years.

SEX

It was observed from the study that among the 20 outpatients majority of them (70%) were male and (30%) were female. Whereas among inpatients majority of them were females (50%) and (50%) were male cases.

RELIGION

In the current study the majority of both outpatients (80%) and inpatients (90%) were hindus. Only a minority of them were Christians (10%) and muslims (10%) among outpatients and inpatients.

OCCUPATION

From the data collected during the enrolment of patients it was learnt that 25% of the outpatients were engaged in private jobs and 35% of the inpatients were home makers. The rest of them were of coolies (25% of OP and 30% of IP), farmers (20% of OP and 15% of IP), and retired employees (5% of OP and 5% of IP).

SOCIO-ECONOMIC STATUS

The present study revealed that of (50% OPD and 45% IPD) and were hailing from the poor class. The second majority were from middle sector (30% of outpatients and 40% of inpatients). Patients from rich sector were only 20% and the visited OP to attain treatment.

FOOD HABITS

Out of the 40 cases who were recruited for the study majority of them (60% of outpatients and 75% of inpatients) were taking mixed diet comprising both vegetarian and non-vegetarian items.

PERSONAL HABITS

From the history taken from the patients it was noted that 45% of outpatients and 50% inpatients were not involved in any bad habits. 15% of outpatients and 20% inpatients had alcoholies.

AETIOLOGICAL FACTORS

It was learnt that 70% of outpatients and 80% of inpatients had others causes of Kalladaippu noi. 15% of outpatients and 10% of inpatients had known previous history of Kalladaippu noi. In the Recurrent UTI (15% of OP and 10% of IP), Kalladaippu noi might be attributed to other causes such as altered food habits, inadequate water intake and drinking contaminated water / hard water.

THINAI

Among the patients who were selected for the trial 75% of the outpatients and 60% of the inpatients were from marutha nilam. 20% of inpatients were from mullai nilam and 20% of inpatients were from neithal nilam.

MUKKUTRA KAALAM

It is vivid from the history of the patients that majority of them (70% of outpatients and 85% of inpatients) were in Pitha Kaalam (34 – 66 years). The rest of them (30% of outpatients and 15% of inpatients) fell under Vatha kaalam (0 – 33 years).

PARUVAKAALAM

In general, Kalladaippu noi occurs in all the seasons. But in the present study, it was found to be the commonest during pinpani kalam (80% outpatients 15% of inpatients) and munpani kaalam (15% of outpatients 60% of inpatients). Only 5% of outpatients suffered from this disease during Ilavenil and 10% koothir kaalam.

THEGI (CONSTITUTION OF BODY)

In this study,Vatha thegi patients were found to be the most affected by Kalladippu noi (50% of outpatients and 60% of inpatients), The second major were Pitha thegi (25% of outpatients and 20% inpatients). Only a few of them (25% of outpatients and 20% of inpatients) were Kabha thegi.

MUKKUTRAM

A. DERANGEMENT OF VATHAM

In the contemporary study, it was noted that all the patients under treatment had disturbances in Abaanan and Samaanan, which was the primary cause for oliguria, dysuria and formation of calculi. Viyaanan was affected in 50% of outpatients and 65% of inpatients which was the cause for renal colic experienced by the patients. Derangement in Dhevathathan (20% of outpatients and 10% of inpatients) caused tiredness in the patients and disturbances in Uthaanan resulted in nausea / vomiting in 50% of outpatients and 60% inpatients.

B. DERANGEMENT OF PITHAM

It was noted that Sadhagam was affected in 40% of outpatients and 55% of inpatients resulting in dysuria, renal colic and difficulty in their regular work in day to day life. Ranjagam was found to be disturbed in 30% of outpatients and 20% of inpatients.

C. DERANGEMENT OF KAPAM

Santhigam was found to be affected in 30% of outpatients and 20% of inpatients, causing joint pain in elderly patients.

UDAL THATHUKKAL

Among the patients selected for the study Saaram was affected in all patients causing sluggishness. Enbu thaathu was found to be affected in 30% of outpatients and 25% of inpatients and they suffered from arthralgia. Senneer was found to be affected in 25% of outpatients and 40% of inpatients.

ENVAGAI THAERVUGAL

In this study, Moothiram was affected in all the patients.Naa was affected in 10% of outpatients and 10% of inpatients featuring fissures in their tongue. Vatha pitha naadi was felt in most of the patients (50% of outpatients and 60% of inpatients).The second major type of naadi was Pitha vatha naadi (15% of outpatients and 10% of inpatients).It was noticed that only 20% of the outpatients 20% of the inpatients had vatha kabha and 5% of inpatients affected by Kaba pitha and Pitha kabha naadi.

NEERKURI

It was noted that the urine colour was yellowish in 60% of outpatients and 70% inpatients and it was normal in 30% of outpatients and 20% of inpatients. Edai, Manam and Nurai were normal in all the patients. There were no deposits found in the samples of all the patients.

NEIKURI

When Neikuri was tested in the urine samples of the patients it was observed that the urine samples of majority of them (50% of outpatients and 55% of inpatients) revealed that kalapu neer. (30% of outpatients and 30% of inpatients) Vatha neer.

DURATION OF ILLNESS

Majority of the patients experienced symptoms of Kalladaippu noi only in the past 2-6 months period (30% of outpatients and 20% of inpatients) and 6 months – 1 year (10% of outpatients and 25% of inpatients). 30% of outpatients and 25% of inpatients suffered from the disease less than one month.

TYPE OF PAIN

Through investigations in patients it was observed that 35% of the inpatients and 45% of outpatients suffered from severe pain and 35% of outpatients and 40% of inpatients experienced moderate pain on their first visit. 30% of outpatients 15% inpatients experienced mild pain. But at the the end of treatment only 15% of outpatients 5% of inpatients of the total 40 patients were found with moderate pain. Majority of them (80%) were relieved from renal colic.

SEVERITY OF DYSURIA

On their first visit majority of the cases (50% of outpatients and 55% of inpatients) experienced severe dysuria and moderate dysuria (35% of outpatients and 30% of inpatients). On their subsequent visits the symptom decreased and finally at the end of the trial only 15% of the outpatients experienced mild dysuria and 10% of inpatients experienced moderate pain.

NUMBER OF STONES

Most of the ultrasonogram reports of the patients (55% of outpatients and 60% of inpatients) revealed single stones and 45% of outpatients and 40% of inpatients revealed multiple stones on the day of enrolment. At the end of the trial only a few cases (10% of outpatients and 15% of inpatients with single stone, 10% of outpatients and 5% of inpatients with multiple stones) presented with stones in their USG reports.

SIZE OF STONES

The USG reports of the cases before treatment showed that majority of the cases (50% of the outpatients and 40% of the inpatients) presented with stone of size 4 to 5mm . But after treatment only 10% of outpatients and 5% of inpatients had stones of size 4to 5mm .

POSITION OF STONES

The USG reports of the patients before treatment revealed stones which were mostly (60% of outpatients and 70% of inpatients) present in the calyces of kidney.Ureteric calculi were comparatively lesser (30% in outpatients and 15% in inpatients) and only 5% of outpatients and 15% of inpatients had stones both in kidneys and ureter. After treatment 15% of outpatients and 5% of inpatients had renal calculi and 5% of outpatients had ureteric calculus.

STONE ANALYSIS

Three stones were received from 3 different patients at the end of the treatment. Three stones were analysed in a well reputed laboratory to determine the composition of stones. The analysis of the stones indicated the presence of calcium oxalate. The stone analysis reports of all the three patients are enclosed as Annexure VI.

PRECIPITATING FACTORS

In the present study, the personal history of patients and laboratory investigations revealed that the occurrence of Kalladaippu noi might be attributed to altered food habits , drinking contaminated water / hard water, inadequate water intake, suppressing seminal discharge, and poor health awareness.

TREATMENT

The trail medicine selected for the clinical study was **NILAKUMILAVER KUDINEER -50ml** BD morning & evening . The present study proved the therapeutic values of the trial medicine which is evident from the absence of calculi and symptoms associated with kalladaippu noi in majority of the patients. Good response was noticed in 50% of outpatients and 65% of inpatients and moderate response in 25% of outpatients and 15% of inpatients. The good and moderate response of trial drug in treating Kalladaippu noi is attributed to the lithotriptic , diuretic and analgesic effects of the trial medicine.

SUMMARY

A study on Kalladaippu noi with the following Clinical trial medicine was undertaken for my dissertation work at the Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai.

The trial drug is **NILAKUMILAVER KUDINEER -** 50ml BD morning & evening (internal).

The study was conducted

- To find therapeutic out the efficacy of the trial medicine in dissolving or disintegrating the calculus.
- To know the efficiency of the medicine in spontaneous expulsion of the calculi.
- To know about the recurrence (or) any adverse effects in the patients during the course of treatment with the trial medicine

The patients were showed good prognosis within a short period. Renal colic and burning micturation reduced within 5 days of treatment. It was observed that all other signs and symptoms relieved at the end of course of the treatment with the trial medicine and strict diet restriction (pathiyam). From the clinical examination and enquiring the patients, it was noted that stones were broken into fragments and expelled out in the form of sand grains and gravels with urine. It was evident from the laboratory and ultra-sonographic investigations that the trial medicines helped in the spontaneous expulsion of calculus in some of the patients.

Good response was noticed is in 80% of OPD and 75% of IPD and moderate response in 20% of outpatients and 25% of inpatients. The patients were advised to take the trial medicine, **NILAKUMILAVER KUDINEER** after food. As the ingredients included in the trial drug have lithotriptic , diuretic and analgesic effects, they should have compensated the deranged pitham in Kalladaippu and initiated diuretic action to expel the stone. The patients were advised to restrict diet to avoid recurrence.

The pharmacological study revealed that the trial medicine had lithotriptic, diuretic and analgesic effects. The acute toxicity study revealed that the trial drug did not produce any toxic effects. Accordingly, the patients under treatment also did not experience any adverse effects during the course of treatment with the trial medicine.

In this study, it has been proved that the trial medicine, **NILAKUMILAVER KUDINEER (Internal)** is highly effective and economically viable in curing KALLADAIPPU NOI.

CONCLUSION

The following conclusions have been drawn from the Open Labelled Phase II, RCT study on "The evaluation of efficacy of the trial drug, **(internal)** in **NILAKUMILAVER KUDINEER** treating **KALLADAIPPU NOI**", was carried out at the PG Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai as my dissertation work.

1. The ingredients incorporated in the trial medicine helped to cure Kalladaippu noi by compensating the increased pitham which is attributed to the diuretic, lithotriptic action, analgesic effect, as mentioned in the various siddha literatures - **Annexure I**. (Page No – 114)
2. The biochemical analysis of trial medicine revealed that the presence of various minerals like Calcium, Chloride, and Ferrous Iron - **Annexure II**. (Page No – 117)
3. The acute toxicity study on the trial medicine revealed that it had no toxic effects – **Annexure III.**
4. The pharmacological study on trial medicine revealed the lithotriptic, diuretic and analgesic effect of the trial medicine – **Annexure IV**.
5. The Ultra sonogram reports of patients before treatment and after treatment were compared to evaluate the prognosis in patients – **Annexure V**
6. The analysis of the stones of some of the patients revealed that the presence of calcium oxalate - **Annexure VI**.
7. Clinically, the trial medicine is free from side effects as no patient experienced side effects during the course of treatment.
8. The result showed good response in 80% of outpatients and 75% of inpatients and moderate response in 20% of outpatients and 25% of inpatients.

The trial medicine was found to have lithotriptic, diuretic analgesic effect, and have properties to compensate the increased pitham, which is one of the important causes for Kalladaippu. From this study, it has been proved that the trial medicine, **is highly effective a NILAKUMILAVER KUDINEER and economically viable in curing Kalladaippu noi.**

ANNEXURE - I

PREPARATION OF TRIAL MEDICINE

The purified drug is coarsely pounding or milling. For the preparation of decotion 25gms of the powder is boiled and make kashayam.

NILAKUMILAVER KUDINEER(Internal)

The following raw drug required for the preparation of trial medicine N**ilakumilaver** were purchased.

Name of the Drug	Nilakumilaver
Botanical Name	Gmelina asiatica
Family Name	Verbenaceae
Parts used	Roots , Leaves
Quantity	350gms
Pharmacological action	Anti inflammatory, Diuretic, Alterative, Demulcent, Aromatic

Characters ;

சுவை : சிறுகைப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

Dosage:

50ml twice a day

Duration:

30 days.

நிலக்குமிழ்

‘‘பேதியோடு கண்சொருகல் பேராத சீதளமும்
வாதவலி கொட்டாவி மாந்தமும் போம் - ஓதச்
சலக்குமிழைக் யொத்த தனக்கருங்கள் மாதே
நிலக்குமிழைக் கையிலெடு நீ’’

இதனை நீரினில் சேர்த்து அலச நீரானது குழகுழப்புத் தன்மையடையும். இதனை கொடுத்துவர வெட்டை நீர்க்கடுப்பு நீர்ப்பைதாபிதம் நீங்கும்

வேறு பெயர்கள்

அமுதச்சோகிதம்
செடிக்குமிழ்
சாரதி
குமிள்
நிலக்குமிழ்

(சாம்பசிவம் பிள்ளைத்தமிழ்)

நிலக்குழிழவேர் குடிநீர்

சேரும் சரக்கு

நிலக்குமிழ் வேர்

ANNEXURE - II

BIO-CHEMICAL ANALYSIS OF NILAKUMILAVER KUDINEER

Preparation of the extract:

5gms of the drug was weighed accurately and placed in a 250ml clean beaker. Then 50ml of distilled water was added to it and dissolved well. Then it was boiled well for about 10 minutes. It was cooled and filtered in a 100ml volumetric flask and then it is make up to 100ml with distilled water this fluid was taken for analysis.

QUALITATIVE ANALYSIS

S.NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	**TEST FOR CALCIUM** 2ml of the above prepared extract is taken in a clean test tube. To this add 2ml of 4% Ammonium oxalate solution	A white precipitate is formed	Indicates the presence of calcium
2.	**TEST FOR SULPHATE** 2ml of the extract is added to 5% Barium chloride solution.	No white precepitate is formed	Absence of sulphate
3.	**TEST FOR CHLORIDE** The extract is treated with silver nitrate solution	No white precipitate is formed	Absence of chloride
4.	**TEST FOR CARBONATE** The substance is treated with concentrated Hcl.	No Brisk effervessence is formed	Absence of carbonate
5.	**TEST FOR STARCH** The extract is added with weak iodine solution	Blue colour is formed	Indicates the presence of starch
6.	**TEST FOR FERRIC IRON** The extract is acidified with Glacial acetic acid and potassium ferro cyanide.	No blue colour is formed	Absence of ferric iron
7.	**TEST OF FERROUS IRON** The extract is treated with concentrated Nitric acid and Ammonium thiocyanate solution	Blood red colour is formed	Indicates the presence of ferrous iron

8.	**TEST FOR PHOSPHATE** The extract is treated with Ammonium Molybdate and concentrated nitric acid	No yellow precipitate is formed	Absence of phosphate
9.	**TEST FOR ALBUMIN** The extract is treated with Esbach's reagent	No Yellow precipitate is formed	Absence of Albumin
10.	**TEST FOR TANNIC ACID** The extract is treated with ferric chloride.	Blue black precipitate is formed	Indicates the presence of tannic acid
11.	**TEST FOR UNSATURATION** Potassium permanganate solution is added to the extract	It gets decolourised.	Indicates the presence of unsaturated compound
12.	**TEST FOR THE REDUCING SUGAR** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and add 8-10 drops of the extract and again boil it for 2 minutes.	Colour change occurs.	Indicates the presence of Reducing sugar
13.	**TEST FOR AMINO ACID** One or two drops of the extract is placed on a filter paper and dried well. After drying, 1% Ninhydrin is sprayed over the same and dried it well.	No Violet colour is formed	Absence of Amino acid
14.	**TEST FOR ZINC** The extract is treated with Potassium Ferrocyanide.	No white precipitate is formed	Absence of Zinc.

Inference:

The extract prepared from the given sample **NILAKUMILAVER KUDINEER** contains Calcium, starch, ferrous iron, tannic acid, reducing sugar and unsaturated compounds. Biochemical Analyis report was done by **Department of Bio Chemistry, Government Siddha Medical College, Palayamkottai.**

ANNEXURE – III

PHARMACOLOGICAL ANALYSIS

EVALUATION OF ANTILITHIATIC EFFECT OF NILAKUMILAVER KUDINEER ON 1%ETHYLENE GLYCOL INDUCED LITHIASIS IN ALBINO RATS

INTRODUCTION

Urinary stone disease has afflicted humankind since antiquity and can persist, with serious medical consequences, throughout a patient's lifetime. In addition, the incidence of kidney stones has been increased in western societies in the last five decades,in association with economic development. Most calculi in the urinary system arise from a common component of urine, e.g.calcium oxalate (CaOx), representing up to 80% of analyzed stones (1).Currently, open renal surgery for nephrolithiasis is unusual and used only rarely since the introduction of extracorporeal shockwave lithotripsy (ESWL), which has revolutionized urological practice and almost become the standard procedure for eliminating kidney stones. However, in addition to the traumatic effects of shock waves, persistent residual stone fragments and the possibility of infection, suggest that ESWL may cause acute renal injury, a decrease in renal function and an increase in stone recurrence (2,3).

A number of vegetable drugs have been used in India and elsewhere which claim efficient cure of urinary stones (4). In the indigenous system of medicine, the Nilakumilaver Kudineer is reported to be useful in the treatment of urinary stones.

However, so far no systematic study has been reported regarding the antiurolithiatic property of Nilakumilaver Kudineer. In the present study, an effort has been made to establish the scientific validity for the antiurolithiatic property of Nilakumilaver Kudineer using ethylene glycol induced hyperoxaluria model in rats.

Materials and methods

- *Preparation of choornam*
- *Pharmacological screening for antiurolithiatic activity*
- *Animal selection*

For acute toxicity studies, Wistar albino mice of either sex weighing between 25 and 30 g were selected and healthy adult male Wistar albino rats weighing between 150 and 200 g were selected for the antiurolithiatic activity. The animals were acclimatized to standard laboratory conditions (temperature:25±2 ◦C) and maintained on 12-h light:12-h dark cycle. They were provided with regular rat chow(Lipton India Ltd., Mumbai,India) and drinking water ad libitum. The animal care and experimental protocols were in accordance with Institutional Animal

Ethical Committee (IAEC).

Acute toxicity studies

The acute oral toxicity study (5)was carried out as per the guidelines set by Organization for Economic Cooperation and Development(OECD)received from Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA).One-tenth of the median lethal dose (LD50)was taken as an effective dose(6)

Ethylene glycol induced urolithiasis model

Ethylene glycol induced hyperoxaluria model (7) was used to assess the antilithiatic activity in albino rats. Animals were divided into eight groups containing six animals in each.

ANTI LITHOLYTIC ACTIVITY

Antilithiotic activity

Urolithiasis is the third most common disease of the urinary tract and it is affecting the human beings since the earliest days. The recognition of different types of urinary calculi also resulted in more varieties of medical treatment. Still recurrence rates continue to be high with one of every two patients having another stone within 5 years. So, there is a need to develop new drugs to prevent the recurrence of kidney stones. In most cases of the urolithiasis, the common component is either calcium oxalate (75-90%) or magnesium ammonium phosphate to an extent of 5-10%. Many remedies have been employed during ages to treat urinary stones. Most of the remedies were taken from plants and proved to be useful.

Chemicals and apparatus

Ethylene glycol was obtained from Merck Ltd., Mumbai, India. All other chemicals and reagents used were analytical grade and procured from approved chemical suppliers. Apparatus such as the metabolic cages (Tecniplast, Italy), semiautoanalyzer (Metrolab, 1600-DR), cold centrifuge (Remi Instruments, C-30BL), UVspectrometer (Shimadzu Scientific Instruments, UV-3600) were used in the study

Animals

Male Wistar albino rats (120-150gm) were used for this experiment. They were maintained at 25 ± 2° C and relative humidity of 45 to 55% and under standard environmental conditions (12 hr. light 12 hr. dark cycle). The animals had free access to food (Sri Venkateswaraa Enterprises, Bangalore, India) and water *ad libitum*.

Treatment protocol

Method : Ethylene glycol and ammonium chloride induced hyperoxaluria model was used to induce calcium oxalate urolithiasis.

Groups – 5 groups of 6 animals each

Group – I **:** Treated as normal control received 10ml/kg of normal saline through orally

Group – II : Urolithatic control received calculi inducing treatment for 28 days, comprised of 0.75% v/v ethylene glycol with 1% w/v ammonium chloride in drinking water *ad libitum* for three days to accelerate lithiasis followed by only 0.75% v/v ethylene glycol for 25 days

Group – III : treated as Test - I received 100mg/kg of *Nilakumilaver Kudineer* with 2 ml of sterile water through orally for 28 days + received calculi inducing treatment for 28 days, comprised of 0.75% v/v ethylene glycol with 1% w/v ammonium chloride in drinking water *ad libitum* for three days to accelerate lithiasis followed by only 0.75% v/v ethylene glycol for next 25 days

Group- IV **:** treated as Test - 2 received 200mg/kg of *Nilakumilaver Kudineer* with 2 ml of sterile water through orally for 28 days + received calculi inducing treatment for 28 days, comprised of 0.75% v/v ethylene glycol with 1% w/v ammonium chloride in drinking water *ad libitum* for three days to accelerate lithiasis followed by only 0.75% v/v ethylene glycol for next 25 days

Collection and analysis of urine

On the 28th day of calculi induction treatment, all animals were kept in individual metabolic cages and urine samples of 24 h were collected. The collected urine samples were measured for following parameters.

Urine volume

Animals were placed in separate metabolic cages for 24 h and total urinary volume was measured using the measuring cylinder and reported in ml.

Urine pH

Uric acid crystals were found to deposit most frequently in the concentrated acid urine. Thus, the acidity of the urine was tested using the pH meter.

Urinary oxalate

The 1 ml of urine was acidified beforehand by concentrated HNO3 to solubilize crystals and then adjusted to pH 7 by NaOH in the presence of color indicator, the bromothymol blue. About 2ml of saturated CaSO4 and 14 ml of pure ethanol were added to precipitate oxalate overnight. The samples were centrifuged at 450× *g* for 10 min and then filtered on filter paper. The precipitate obtained was solubilized in 10 ml of water acidified by 2ml of concentrated sulfuric acid. The samples were titrated by a solution of KMnO4.

Urine calcium

It was estimated by using commercially available standard kit of Biolab diagnostics Pvt. Ltd. Tarapur (India) as per o-cresolphthalein complexone method. Determination of urine calcium was done by using CHARIOT prince autoanalyser.

Urine magnesium

It was estimated by using commercially available standard kit supplied by Biolab diagnostics Pvt. Ltd. Tarapur (India) as per Calmagite method. Determination of urine magnesium was done by using CHARIOT prince autoanalyser.

Collection and Serum analysis

After urine collection period, blood was obtained from the retro-orbital under anaesthetic condition and animals were sacrificed by cervical decapitation. Serum were separated by centrifugation and analyzed.

Serum uric acid

It was estimated by using commercially available standard kit of Biolab diagnostics Pvt. Ltd. Tarapur (India) as per Colorimetric enzymatic method and analysed by CHARIOT prince autoanalyser.

Serum creatinine

It was estimated by using commercially available standard kit as per Urease/salisylate method. Determination of serum creatinine was done by using CHARIOT prince biochemistry autoanalyser.

Blood urea

It was estimated by using commercially available standard diagnostic kit of Biolab Diagnostics-India using diacetymonoxime colorimetric end-point method. Determination of Blood urea was done by using CHARIOT prince biochemistry autoanalyzer.

Kidney homogenate analysis

The abdomen was cut open to remove both kidneys from each animal. Isolated kidneys were cleaned off extraneous tissue and rinsed in ice-cold physiological saline. The left kidney was finely minced and 10 % homogenate was prepared in Tris-Hcl buffer (0.02 mol/l, pH 7.4). The homogenate was used for measurement of various biochemical parameters.

Estimation of biochemical markers

The homogenate was used to assay the marker enzymes in serum, urine and tissue constituents like ACP, Alkaline phosphate (ALP), Aspartate aminotransferase (AST),

Alanine aminotransferase (ALT) and Lactate dehydrogenase (LDH) were estimated respectively using different types of enzyme marker kits.

STATISTICAL ANALYSIS

Data expressed as Mean ± S.E.M. The data were analyzed using one-way analysis of variance (ANOVA) followed by Dunnett's test and $p<0.05$ considered as statistical significant.

Effect of Decoction of *Nilakumilaver Kudineer* on urinary excretion

Group	**Treatment**	**Dose**	**Urine**	
			Calcium (mg/dl)	**Oxalate (mg/dl)**
I	Control (Distilled water)	2 ml/kg, p.o.	1.842 ± 0.010	0.841 ± 0.011
II	Urolithiatic Control (Gentamycin + CPD)	80mg/kg, i.p.	10.669 ± 0.177	3.314 ± 0.080
III	Standard (Cystone)	750 mg/kg, p.o.	9.110 ± 0.047	0.174 ± 0.007
IV	Gentamycin + ***Nilakumilaver Kudineer***	80mg/kg, i.p + 100 mg/kg, p.o.	4.462 ± 0.103	1.587 ± 0.027
V	Gentamycin + ***Nilakumilaver Kudineer***	80mg/kg, i.p + 200 mg/kg, p.o.	7.520 ± 0.071	2.619 ± 0.127

n = 6. Values are expressed as ± S.E.M.

***P < 0.0001 vs. Control (followed by two way ANOVA).

Effect of Decoction of *Nilakumilaver Kudineer* on Serological Parameters

Group	Treatment	Dose	Serum	
			Urea (mg/dl)	**Creatinine (mg/dl)**
I	Control (Distilled water)	2 ml/kg, p.o.	43.017 ± 0.169	0.663 ± 0.018
II	Urolithiatic Control (Gentamycin)	80mg/kg, i.p.	173.912 ± 0.330	3.173 ± 0.081
III	Standard (Cystone)	750 mg/kg, p.o.	48.163 ± 0.092	0.542 ± 0.007
IV	Gentamycin + ***Nilakumilaver Kudineer***	80mg/kg, i.p + 100 mg/kg, p.o.	32.270 ± 0.106	0.357 ± 0.008
V	Gentamycin + ***Nilakumilaver Kudineer***	80mg/kg, i.p + 200 mg/kg, p.o.	34.318 ± 0.160	0.448 ± 0.010

n = 6. Values are expressed as ± S.E.M.

***P < 0.0001 Vs. Control (followed by two way ANOVA).

ACUTE TOXICITY STUDY

Acute oral toxicity refers to those adverse effects occurring following oral administration of a single dose of a single dose of a substance or multiple doses given within 24 hours. Acute toxic class method (OECD guidelines 423, (2000) was followed to arrive at the maximum safety dose of the drug extracts. Three Wistar strain female albino rats (8-12 weeks old, 180-200g body weight) were used in each group. Single dose (2g/kg) of the *Nilakumilaver Kudineer* was orally administered to overnight fasted (food but not water withheld) animals while control animals received the vehicle (0.3%w/v CMC). Animals were observed individually after dosing at least once during the first 4 hrs and daily thereafter, for a total of 14days. Body weights of the animals were recorded. The other observations include changes for skin, fur, eyes and mucous membranes, respiratory, circulatory and autonomic and central nervous system and somatomotor activity and behavior pattern. At the end of 14 days, all animals were subjected to gross necropsy.

Statistics

Data are expressed as mean ± SEM; data analysed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with test compounds.

* Values were considered significant at $P < 0.5$.

Results

Acute toxicity study

All of the rats fed with the food sample showed normal general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin and fur.

TABLE - 1

Hematological values of *Nilakumilaver Kudineer* in the acute toxicity study

S. No	Parameter	Control	Sample 2g/kg
1	White blood cells ($x10^3$/µl)	9.36±0.54	8.85±0.36
2	Hemoglobin (g/dl)	11.50±0.26	10.75±0.84
3	Mean corpuscular volume	60.45±2.3	62.58±1.9
4	Mean corpuscular hemoglobin conc. (g/dl)	34.56±0.86	31.36±0.25
5	Platelet ($x10^5$/µl)	5.60±0.52	5.70±0.60
6	Red blood cell ($x10^6$/µl)	3.87±0.24	3.75±0.30

Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight

No significant different from normal control

Table - 2

Blood chemical values of food sample in the acute toxicity study

S. No	Parameter	Control	Sample 2g/kg
1	Glucose (mg/dl)	148.75±3.96	145.60±3.58
2	BUN(mg/dl)	34.26±1.23	36.58±1.42
3	Creatinine(mg/dl)	0.46±0.06	0.50±0.12
4	Total protein (g/dl)	5.48±0.23	5.62±0.17
5	Albumin (g/dl)	3.49±0.62	3.68±0.24
6	Total bilirubin (mg/dl)	0.26±0.02	0.32±0.04
7	AST (u/l)	141.5±3.76	144.25±2.43
8	ALT (u/l)	86.36±1.75	89.47±1.26
9	ALP (u/l)	75.57±2.16	78.64±1.88

Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight

No significant different from normal control

Discussion and conclusion

In acute toxicity study for 14 days, at a dose of 2g/kg of *Nilakumilaver Kudineer* sample were chosen for the experiment. In the aspect of general behaviours, the rats treated with food sample at a single dose had no signs of behavior changes and toxic signs. The treated groups revealed no significant differences in body weight gain. The increase in body weight may have resulted from physiological changes in rats such as metabolism, food and water intake. However, the result from animal health monitoring in the entire period of 14days showed no sign of morbidity and diseases.

The albino Wistar rats were healthy as shown by the normal appearance of general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin fur.

With regards to hematological values, most of values in treated groups were normal in comparison with the control group. Significantly, some values were different from those of the control group such as RBC, MCV, MCHC, and platelet. However, such values are within the normal ranges. These variations may have resulted from variation among animal groups (Feldman et al., 2000) (Inala et al., 2002). Therefore, these results suggest that the test drug did not cause hematological or immunological defects in rats.

Furthermore, blood chemical examination was performed in order to evaluate any toxic effects on liver. In this study, the levels of these blood chemical values were minor changes and remained within the normal range (Casley and King, 1980) (Levine, 1995) (Angkhasirisap et al., 2002).

In conclusion, *Nilakumilaver Kudineer* sample given orally to Wistar rats did not produce toxicities.

DIURETIC ACTIVITY OF NILAKUMILAVER KUDINEER

Introduction

Medicinal plants can be important sources of unknown chemical substances with potential therapeutic effects. Besides, the World Health Organization has estimated that over 75% of the world's population still relies on plant-derived medicines, usually obtained from traditional healers, for basic health-care needs

(1). The study of plant species with diuretic effects is still a fruitful research in search of new diuretics. Diuretics are the drugs that increase the rate of urine flow; clinically useful diuretics also increase the rate of excretion of Na+ (natriuresis) and an accompanying anion, usually Cl-. Most clinical applications of diuretics aim to reduce extracellular fluid volume (edema) by decreasing total body Nacl content. Although continued administration of diuretic causes a sustained net deficit in total Na+, the time course of natriuresis is finite because renal compensatory mechanisms brings Na+ excretion in line with the Na+ intake, a phenomenon known as diuretic braking. Diuretics alter the excretion of other cations (e.g. K+, H+, Ca2+, Mg2+), anions (e.g. Cl, HCo3- and H2Po4) and uric acid. In addition diuretics may alter renal hemodynamics indirectly mediated by local prostaglandins synthesis(2).

Despite the popular use of this Nilakumilaver Kudineer preparation, there are no data about the pharmacological effect of Nilakumilaver Kudineer on diuretic activity.
The aim of the present study was to evaluate the potential diuretic and natriuretic activities Nilakumilaver Kudineer on different experimental animal.

Material and Methods

Experimental animals

Healthy male albino rats weighing 180-200 g were used for the study. The animals were maintained in polypropylene cages of standard dimensions at a temperature of 37 ± 1°C and standard 12h : 12h day/night rhythm. The animals were fed with standard rodent pellet diet (Hindustan Lever Ltd.) and water *ad libitum*. Prior to the experiment, the animals were acclimatized to the laboratory conditions. The experimental protocol was approved by Institutional Animal Ethical Committee (IAEC) constituted under CPCSEA.

Drug Treatment

The Nilakumilaver Kudineer at the dose levels of 200, 400 mg/Kg body wt., p.o. was administered once daily for three consecutive days. Furosemide (20 mg/Kg; p.o.) was used as standard for diuretic activity. Control group of animals (n=6) received normal saline (10 ml/Kg)

Experimental design

The animals were divided into 4 groups of 6 rats each as follows; Group I: received only 10ml/kg normal saline Group II: received Furosemide 20 mg/kg,
Group III: received PS 200 mg/kg body weight p.o., Group IV: received PS 400 mg/kg body weight p.o

Diuretic activity

Rats were fasted overnight and treated with vehicle, Furosemide and PS stated above along with normal saline (50 ml/kg). The rats were placed in metabolic cages and the urine samples were collected for 24h, measured using a standard measuring cylinder. The amount of urine (in ml) collected for 24 h was compared and tabulated (3).

Natriuretic activity

Estimation of Sodium and Potassium content of the urine samples of all groups of animals were done by using a laboratory model flame photometer. The ratio of Na+/K+ is calculated for Natriuretic activity. A value greater than 2.0 indicates a favorable Natriuretic effect. Ratio greater than 10.0 indicates a potassium sparing effect (4).

Statistical analysis

The results were expressed as mean ± S.E.M. Statistical comparisons were made by means of newmann keuls multiple range tests.*p* values smaller than 0.05 was considered as significant.

Table no:1 Diuretic activity of NILAKUMILAVER KUDINEER (urine Volume) in 24 Hours

Group	**Treatment**	**Urine volume**
I	Normal saline 10ml/kg	7.80±0.60
II	Frusemide 20mg/kg	12.60±0.85**
III	PC 200mg/kg	12.05±0.72**
IV	PC 400mg/kg	11.80±0.65**

Values are Mean ± SEM, n=6, **p<0.01.

Table 2: Natriuretic activity of Nilakumilaver Kudineer

Treatment	Na+	K+	Na+/K+
Normal Saline 10ml/kg	1.70±0.05	0.68±0.02	2.50
Frusemide 20mg/kg	3.20±0.08**	0.85±0.04**	3.76
PC 200mg/kg	1.95±0.06*	0.70±0.01ns	2.78
PC 400mg/kg	2.90±0.12**	0.68±0.02ns	2.22

Values are Mean ± SEM, n=6, *p<0.05, **p<0.01, NS - not significant

RESULTS AND DISCUSSION

Table -1 shows the urine volume collected in 24 hours for all the groups. It is evident that the Nilakumilaver Kudineer treated groups excreted more urine than the control groups. The PC at 200 and 400 mg/kg exhibited comparable effect with that of the reference drug Furosemide 20 mg / kg and the results were statistically significant. Table -2 shows the sodium and potassium content of the urine for all groups. The amount of Sodium excreted was increased for Furosemide treated group; statistically significant rise in Na+ excretion was also noticed for PC treated groups. The potassium content excreted in the urine was statistically insignificant for all the groups. The Natriuretic effect was calculated by employing the formula Na+ / K+. It was found that the Nilakumilaver Kudineer treated groups possess favorable Natriuretic effect. The present study showed that the PC significantly increases the urine output and excretion of urinary sodium and had no effect on the urinary potassium excretion. Diuretics have two separate connotations; increase urinary per se and net loss of solute (i.e. electrolyte) and water (i.e. saluretic). These two processes are involved in the suppression of renal tubular reabsorption of electrolytes, water and low molecular weight organic compounds into the blood stream and a consequence; promote the formation of urine (5). An attempt to extrapolate the diuretic action of plant extract from rats to man using the activity of Furosemide in the organism as a guideline has been reported. The results clearly shows that the PC at doses of 200 and 400 mg / kg produced significant dose dependent increase in urinary excretion and urinary

sodium loss but no effect on urinary potassium loss with respect to control and standard drug treated groups. The data demonstrates that the PC has diuretic effect, Natriuretic effect but no potassium sparing effect and is as potent as Furosemide. This indicates the use of PC as a diuretic agent based on a sound mechanistic background. Also the excretion of potassium ions was similar to the untreated group, which rule out the possibility of hypokalemia and associated ototoxicity (6).

Conclusion

From the above results, it is concluded that Nilakumilaver Kudineer used by folklore traditionally showed significant diuretic activity. The experimental evidence obtained in the laboratory model could provide a rationale for the traditional use of this plant as diuretic.

ANALGESIC ACTIVITY OF *NILAKUMILAVER KUDINEER* AGAINST ACETIC ACID INDUCED WRITHING REFLUX IN MICE

Analgesic activity of *Nilakumilaver Kudineer* at a dose of 100 mg/kg and 200 mg/kg was evaluated by acetic acid induced writhing reflux in mice. Painful reactions in animals may be produced by the chemicals such as phenylquionolone, bradykinin etc. like that, acetic acid pain reaction which is characterized as a writhing response. Construction of abdomen, turning of trunk (twist) and extension of hind legs are taken as reaction to chemically induced pain. Analgesics (both narcotic and non-narcotic) inhibit writhing response.

Requirements

Animal: Swiss albino mice (20 – 25g) either sex

Drugs and chemicals: Diclofenac sodium (standard),

Acetic acid (1%), *Nilakumilaver Kudineer*

Method

Treatment protocol

Group- 1: Treated as normal control received 10ml/kg of normal saline through orally

Group- 2: treated as standard control received 10mg/kg of diclofenac sodium through orally

Group- 3: treated as Test-I received 100mg/kg of *Nilakumilaver Kudineer* with 2 ml of sterile water through orally

Group- 4: treated as Test- II received 200mg/kg of *Nilakumilaver Kudineer* with 2 ml of sterile water through orally

Both dose of *Nilakumilaver Kudineer* were administered one hour prior to the acetic acid administration. Note the onset on writhing. Record the numbers of abdominal contractions, trunk twist and extension of hind limbs as well as the number of animals showing such response during a period of 10 minutes were noted.

Statistics

Data are expressed as mean ± SEM; data analyzed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with extracts.

* Values were considered significant at $P < 0.01$.

Table - 1

Analgesic activity of *Nilakumilaver Kudineer* against acetic acid induced writhing reflux in mice

Treatment	**Dose (mg/kg)**	**No. of writhing**	**% reduction in reaction time**
Group I Normal saline	Inject 1%v/v acetic acid 1ml/100g of body weight	35.2±2.6	-
Group II Standard	10mg/kg Diclofenac sodium through orally	7.5±0.7	78.69***
Group III *Nilakumilaver Kudineer*	100mg/kg administered through orally	13.6±1.2	61.36***
Group IV *Nilakumilaver Kudineer*	200mg/kg administered through orally	10.4±1.5	70.45***

Values are expressed as mean ± SEM

Values are analysed by one way ANOVA followed by Dunnet's multiple range tests

*** Values were considered significant at $P < 0.001$.

Results

The table values show that analgesic activity of *Nilakumilaver Kudineer* at a dose of 100mg/kg and 200mg/kg by acetic acid induced writhing reflex. The result reveals that both doses of Nilakumilaver Kudineer possess significant analgesic activity at $P < 0.001$.

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Sundays : 6.30 am to 1.00 pm

7, Manakavalam Pillai Hospital Road,
Palayamkottai.

Pt.Name: Mr.Sankaralingam Age : 36/ M

Ref.by. Dr.K.Annapoorani M.D.(S)., Date:22.03.16.

URINE STONE ANALYSIS REPORT:

Calcium Oxlate (Stone)	Present.
Non Oxlate Calciam (Stone)	Not Present.
Carbonate (Stone)	Not Present.
Uric acid (Stone)	Present .
Bilirubin (Stone)	Not Present.
Cholesterol (Stone)	Not Present.

Lab Technician

Please Repeat Samples if results are not correlating with clinical findings

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Pt.Name: Mrs.Sundari Age : 30/ F

Ref.by. Dr.K.Annapoorani M.D.(S)., Date:30.03.16.

URINE STONE ANALYSIS REPORT:

Calcium Oxlate (Stone)	Present.
Non Oxlate Calciam (Stone)	Not Present.
Carbonate (Stone)	Not Present.
Uric acid (Stone)	Not Present .
Bilirubin (Stone)	Not Present.
Cholesterol (Stone)	Not Present.

Lab Technician

Please Repeat Samples if results are not correlating with clinical findings

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Pt.Name: Mr.Kumar Age : 47/ M

Ref.by. Dr.K.Annapoorani M.D.(S)., Date:25.03.16.

URINE STONE ANALYSIS REPORT:

Calcium Oxlate (Stone)	Present.
Non Oxlate Calciam (Stone)	Not Present.
Carbonate (Stone)	Not Present.
Uric acid (Stone)	Present .
Bilirubin (Stone)	Not Present.
Cholesterol (Stone)	Not Present.

Lab Technician

Please Repeat Samples if results are not correlating with clinical findings

AARTHI SCANS

AN ISO 9001 ORGANISATION

Before Treatment

Name: MRS.BANUPRIYA | Date: 02-Nov-2015

Age : 30/F | ID : AS/VPI/CT/095307

Ref.By: Dr. DHAMOTHARAN.T. BSC. MBBS.

Thank you for your referral.

CT SCAN KUB- PLAIN

Volume scan was taken from xipisternum to pubis without IV contrast

Right kidney is normal in size and measures 9.6 X 3.9cms.
4 tiny calculi (2mm) noted in upper, middle and lower calyces.
No evidence of parenchymal thinning or scarring is seen.
No evidence of hydroureteronephrosis is seen.
Right ureter is not dilated. Right vesico-ureteric junction appears normal.

Left kidney is normal in size and measures 9.7 X 3.6cms.
Mild hydroureteronephrosis.
Two tiny (2mm) calculi noted in middle calyx.
Two calculi noted in lower ureter at S3 level, distal one measuring 9 x 5 x 4mm (density 1600HU), proximal one measuring 3 x 3mm.
No evidence of parenchymal thinning or scarring is seen.
Left vesico-ureteric junction appears normal.

The bladder is normally distended. No evidence of calculus or diverticulum. No abnormal wall thickening.

Uterus is normal for the age. No obvious exophytic lesion seen.

Both ovaries are increased in size with multiple small follicles in periphery.
Right ovary measures 3.5 x 2.6cm. Left ovary measures 3.8 x 2.8cm

Minimal free fluid in pouch of Douglas.

There is no mass or lymphadenopathy seen in the retroperitoneum.

No free fluid in abdomen.

Liver, GB, Spleen, pancreas and adrenals appear normal.

AARTHI SCANS

AN ISO 9001 ORGANISATION

Name: MRS.BANUPRIYA	Date: 02-Nov-2015
Age: 30/F.	ID : AS/VPI/CT/095307
Ref.By: Dr. DHAMOTHARAN.T. BSC. MBBS.	

IMPRESSION:

- Left lower ureteric calculi causing mild left hydroureteronephrosis.
- Bilateral tiny renal calculi.
- Polycystic appearance of both ovaries.
- Minimal free fluid in pouch of Douglas.

DR. ARUNKUMAR GOVINDARAJAN, MDRD.,
CONSULTANT RADIOLOGIST
Ph: 9940056116

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• THANJAVUR : 22/1, Pudukottai Rd, Ph: 279914, 279917, Mobile : 87544 38504, 99529 69814
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• RAJAPALAYAM : 64, Kamaraj Nagar, 2nd Street, Ph : 04563-225101, Mobile : 99401 10504

Note :This imaging modality is having its own limitations, Hence this report should be correlated with clinical features and other parameters

AFTER TREATMENT

AARTHI SCANS

AN ISO 9001 ORGANISATION

Name:MRS.BANUPRIYA	Date : Apr 02 2016
Age :30Y / Female	ID :AS/VPI/US/0426
Ref By :Dr K.Annapoorani MD(S)	

USG ABDOMEN -

LIVER:
Is normal in size and uniform echo texture. Intrahepatic biliary radicles and CBD appear normal. Portal and hepatic veins appear normal.

GALL BLADDER:
Is adequately distended. No internal echoes are seen. Wall thickness is normal.

PANCREAS:
Appears normal in size and it shows uniform echo texture.

SPLEEN:
Is normal in size and uniform echogenicity.

KIDNEYS:
RT.Kidney measures 11.4 x 5.2cms. **Pelvicalyceal system and ureter are dilated on the right side..**

LT.Kidney measures 9.9 x 5.5cms.

Cortico medullary differentiation is maintained on both sides. Pelvicalyceal system on left side appears normal.

BLADDER:
Is normal contour. No intra luminal echoes are seen. Urinary bladder wall thickness is normal.

PROSTATE:
Normal for the age.

RIF:
Appears normal. No free fluid.

IMPRESSION:

No calculus seen

DR. M.KANDHA KUMAR,MBBS.,DMRD.,
CONSULTANT RADIOLOGIST.
FOOT NOTE: patient's identity is not verified. Report is not valid for medical-legal purpose.

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Before Treatment

AARTHI SCANS

AN ISO 9001 ORGANISATION

Name: MR.VINCENT JOHN	Date: 12-Jan-2016
Age: 60/M	ID : AS/VPI/CR/001961RR
Ref.By: Dr. Self	

USG ABDOMEN

LIVER:
Is normal in size and uniform echo texture.
Intrahepatic biliary radicles and CBD appear normal.
Portal and hepatic veins appear normal.

GALL BLADDER:
Is adequately distended. No internal echoes are seen. Wall thickness is normal.

PANCREAS:
Appears normal in size and it shows uniform echo texture.

SPLEEN:
Is normal in size and uniform echogenicity.

KIDNEYS:
RT. Kidney measures 9.8 x 5.5cms. **Calculi of size 0.5cms seen in lower calyx & 0.6cms seen in upper calyx & 0.4cms seen in middle calyx of right kidney.** LT.Kidney measures 9.1 x 4.7cms.
Cortico medullary differentiation is maintained on both sides.
Pelvicalyceal system on both sides appears normal.

BLADDER:
Is normal contour. No intra luminal echoes are seen. Urinary bladder wall thickness is normal.

PROSTATE:
Measures 4.2 x 3.2 x 2.5cms. Vol: 19.2cc.

RIF:
Appears normal. No free fluid.

IMPRESSION:

- **Right renal calculus.**

DR. K.MANOHARAN, MD, DMRD.,
CONSULTANT RADIOLOGIST.
Foot note: Patients identity is not verified. Report is not valid for medical-legal purpose.

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Note :This imaging modality is having its own limitations, Hence this report should be correlated with clinical features and other parameters

After Treatement

BARANI SCANS

Serving with Humanity

Name	Mr.VINCENT JOHN	*DATE*	12.04.2016
Age/Sex	60Y/M	*ID /BS/CT*	5412
Ref : by	DR K.ANNAPOORANI MD(S)		

USG ABDOMEN

Thanks for reference

Liver

Liver parenchyma shows normal size and morphology. Diffuse parenchymal hyperechogenicities is seen in liver. No evidence of focal lesion is seen. IHBR are not dilated. Portal vein and its major branches appear normal

GB:

Gall bladder appears normal. No abnormal echogenecity or evidence of calculus seen. CBD is not dilated.

Pancreas

Pancreatic parenchyma appears normal. Pancreatic duct is not dilated. No evidence of calcification or abnormal echogenecity is seen.

Spleen:

Parenchyma appears normal in size and echogenecity. No evidence of focal lesion is seen.

KIDNEYS:

Right kidney measures 8.6x4.2 cms. Left kidney measures 8.2x4.4 cms. Parenchymal echoes are normal. CMD is maintained. No evidence of calculus is seen. Pelvicalcyceal system is normal. Ureters are not dilated.

Urinary Bladder:

Bladder appears normal. No evidence of calculus is seen. No significant wall thickening is seen.

Uterus & Ovaries:

Uterus is normal in size and measures 8.0x5.0x3.9 cms. Endometrium (6mms) and myometrium appears normal

Both ovaries are normal in size normal echo pattern. Right ovary is measuring 2.6x1.5 cms and left ovary is measuring 3.2x1.2 cms.

Follicles measuring about 10x6 mms present in right ovary and about 7x5 mms present in left ovary. No dominant follicles in both ovaries.

No free fluid present in POD.

Retroperitoneal structures appear normal.

No significant inflammatory changes or mass in RIF.

No significant free fluid in abdomen and pelvis.

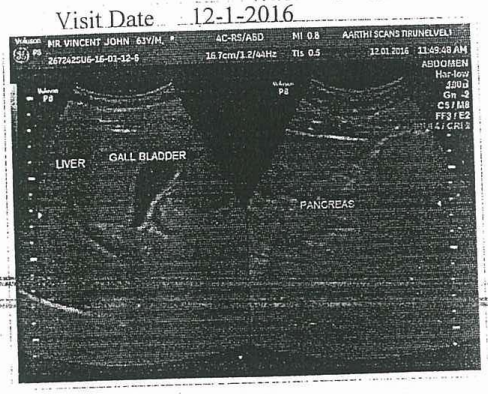
9-B, Thiruchendur Road, (Near Murugankuruchi Signal)
Palayamkottai, TIRUNELVELI - 627 002.
Ph : 0462 - 2583222, 0462 - 4000014 email : baraniscans@yahoo.com

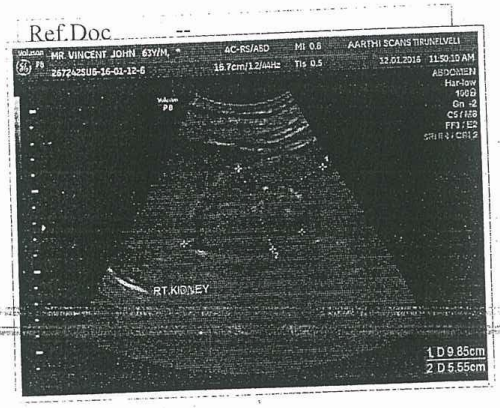
AARTHI SCANS

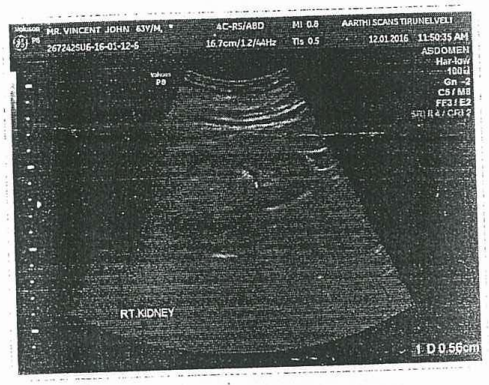
AN ISO 9001 ORGANISATION

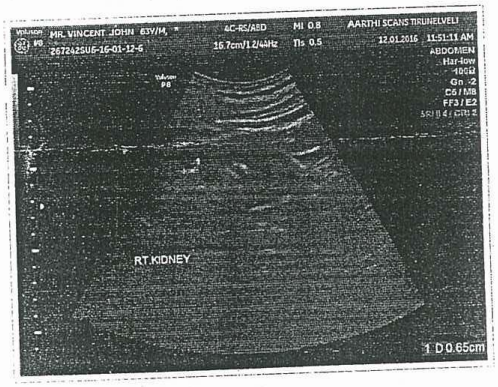
Name \ ID MR. VINCENT JOHN 63Y/M \ 267242SU6-16-01-12-6 Age / Sex 0 / O

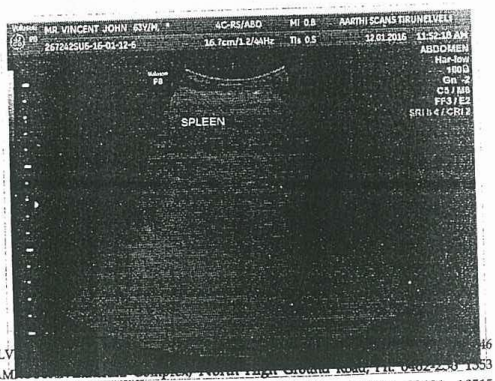
Visit Date 12-1-2016 Ref.Doc --

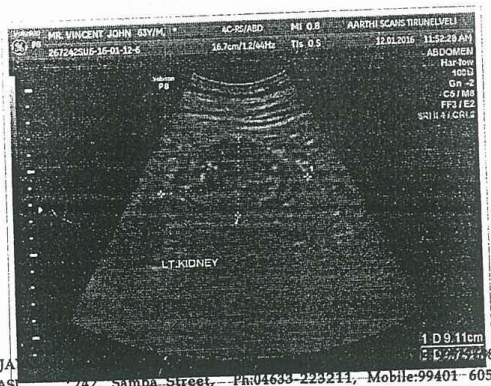












- TIRUNELV ... 46
- THANJA ... 814
- PALAYAM ... 1553
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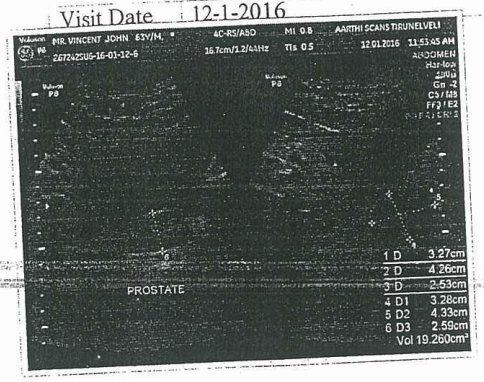
AARTHI SCANS

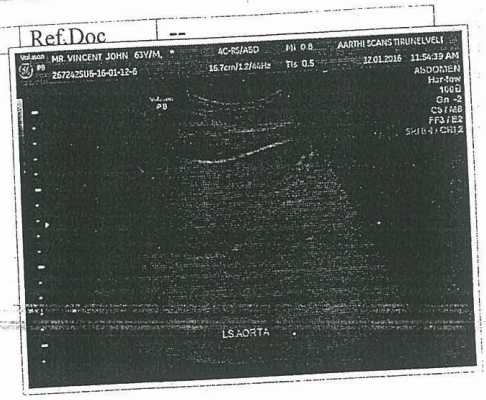
AN ISO 9001 ORGANISATION

Name \ ID : MR. VINCENT JOHN 63Y/M \ 267242SU6-16-01-12-6 Age / Sex

0 / O

Visit Date : 12-1-2016 Ref.Doc : --





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GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
PALAYAMKOTTAI, TIRUNELVELI DISTRICT
DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU (UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM-I

(SCREENING AND SELECTION PROFORMA)

1.Name :_____ 2.Age: ____3.gender: _____ 4.Phone no: _____

5. OP No: ———————— 6. IP No: _____ 7. S.No: _____

INCLUSION CRITERIA:

- Age :30 - 60Yrs
- Sex : Both male and female
- Colicky pain: "loin to groin".
- Nausea, vomiting and sweating
- Hematuria
- Pyuria: pus in the urine.
- Dysuria: burning on urination when passing stones
- Oliguria: reduced urinary volume
- Patients who are willing for admission and stay in IPD for 20 days or willing to attend OPD
- Patients who are willing to undergo radiological investigation and give blood for laboratory investigations.
- Patient willing to sign the informed consent stating that he/she will consciously stick to the treatment during 20 days but can opt out of the trial of his/her own conscious discretion.

EXCLUSION CRITERIA

- In women, gynecologic processes that include ovarian torsion, ovarian cyst and ectopic pregnancy.
- In men, testicular tumor, epididymitis or prostatitis

- Appendicitis
- Cholecystitis
- Diverticulitis
- Colitis
- Pyelonephritis
- Perinephric abscess
- Cystitis and UTI
- Sexually transmitted diseases
- Pelvic inflammatory disease
- Post renal azotemia: the blockage of urine flow through a ureter.

DATE :

STATION : **SIGNATURE OF HOD**

SIGNATURE OF INVESTIGATOR **SIGNATURE OF LECTURER**

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,

PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM l A

HISTORY PROFORMA ON ENROLLMENT

1. Serial No of the case: _____ 2. OP/IP No:_____

3. Name: _____ 4. Gender: Male ☐ Female ☐

5. Age (years): _____ DOB ☐☐ ☐☐ ☐☐☐☐
Date Month Year

6.Address: ----------

7.A.Occupation: ---------- B. Nature of work----------

8. Educational Status: A) Illiterate ☐ B)Literate ☐

9.Height:---------- cms 10.Weight:----------kg

11. Complaints and Duration:

_____

_____

_____

12. Past History

Hypertension _____

Diabetes mellitus _____

Asthma _____

PT _____

HABITS

A) Smoking : 1. Yes ☐ duration _____ years; Number - _____ 2. No ☐

B) Alcoholism:1. Yes ☐ duration _____ years; Quantity- ____ ml 2. No ☐

C) Tobacco chewing: 1. Yes ☐ duration _____ years 2.No ☐

D) Betel chewing : 1. Yes ☐ duration _____ years 2.No ☐

13. Diet style: A.Pure vegetarian ☐ B.Non-vegetarian ☐ C. Mixed diet ☐

14. Drug history: Had the patient been treated before with allopathy drug?

A) Yes ☐ 2) No ☐

15 Marital status **:** 1.Married ☐ 2.Unmarried ☐

16. Family history :

Whether this problem runs in family? 1. Yes ☐ 2.No ☐

If yes, mention the relationship of affected person(s) - _____

18. Bowel habits & micturition: Normal ☐

History of habitual constipation 1.Yes ☐ 2.No ☐

History of frequent diarrhoea 1.Yes ☐ 2.No ☐

History of frequent dysuria 1.Yes ☐ 2.No ☐

19. Psychological state: Normal ☐ Anxiety ☐ Depression ☐

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
PALAYAMKOTTAI, TIRUNELVELI DISTRICT
DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM II & II-A

CLINICAL ASSESSMENT ON ENROLLMENT AND ON VISITS

1. S.NO : _____ 2. OP/IP NO : _____

3. Name : _____ 4. Age : _____

5. Gender : _____ 6. Date of assessment : _____

SIDDHA SYSTEM OF EXAMINATION

1**. ENVAGAI THERVU: [EIGHT-FOLD EXAMINATION]**

I. NAADI: [PULSE PERCEPTION]

	0^{st}Day	07^{th}Day	15^{th} Day	21^{st} Day	28^{th} Day	30^{th} Day
Vali						
Azhal						
Iyyam						
Vali Azhal						
Azhal vali						
Iyya vali						
Vali Iyyam						
Azhal Iyyam						
Iyya Azhal						

II. NAA:[TONGUE]

	0th Day	07th Day	14th Day	21st Day	28th Day	30th Day
Colour	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale
Taste	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None
Coating	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Fissure	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Saliva	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased
Dryness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Glossitis	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Baldness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

III.NIRAM: [COLOUR COMPLEXION]

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted / Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale

IV.MOZHI: [VOICE]

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Medium/ High/ Low / Pitched	Medium/ High/ Low/ Pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched

V.VIZHI: [EYES] (Lower palpabrel conjunctiva)

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale

VI. MALAM; [BOWEL HABITS / STOOLS]

	0^{th} Day	07th Day	14th Day	21stDay	28th Day	30th day
Colour	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale
Consistency	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery
Stool bulk	Normal / Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced
Constipation	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Diarrhoea	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

VII. URINE EXAMINATION:

NEERKURI	**0th Day**	**07th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Niram [Colour]	White/ Yellowish/ Straw coloured / Crystal clear	White/ Yellowish/Straw Coloured / Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish / Straw coloured/ Crystal clear
Manam [Odour]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Nurai [Froth]	Nil/ Reduced / Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased
Edai [Sp.gravity]	Normal/ Increased/ Reduced	Normal/ Increased /Reduced	Normal/ Increased /Reduced	Normal/ Increased / Reduced	Normal/ Increased / Reduced	Normal/ Increased/ Reduced
Enjal [Deposits]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Volume	Normal/ Increased/ Reduced	Normal/ Increased /Reduced	Normal/ Increased /Reduced	Normal/ Increased / Reduced	Normal/ Increased / Reduced	Normal/ Increased/ Reduced

NEIKURI	**0th Day**	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Serpentine fashion						
Annular/Ringed fashion						
Pearl beaded fashion						
Mixed fashion						
Other fashion						

VIII. SPARISAM: [PALPATORY PERCEPTION]

0th Day	**7th day**	**14th Day**	**21st Day**	**28thDay**	**30th day**
Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat

5. THEGI: [TYPE OF BODY CONSTITUTION]

Vatham predominant		Kabam predominant	
Pitham predominant		Thondha udal	

6.NILAM: [LAND WHERE PATIENT LIVED MOST]

Kurinji ☐ Mullai ☐ Marutham ☐ Neithal ☐ Palai ☐

(Hilly terrain) (Forest range) (Plains) (Coastal belt) (Arid regions)

7. KAALAM:

Kaarkalam - ☐ Pinpanikalam - ☐

Koothirkalam - ☐ Ilavenil - ☐

Munpanikalam - ☐ Muthuvenil - ☐

8. GUNAM:

Sathuvam - ☐ Rasatham - ☐ Thamasam - ☐

9.IMPORIGAL (SENSORY ORGANS):

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Mei (Skin)						
Vai (Buccal Cavity)						
Kann (Eye)						
Sevi (Ear)						
Mooku (Nose)						

10. KANMENDRIYAM (MOTOR ORGANS)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Kai (upper limb)						
Kaal (lower limbs)						
Vai (buccal cavity)						
Eruvaai (excretory organs)						
Karuvaai (reproductive organs)						

11.KOSANGAL(Sheath)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Annamaya Kosam						
Pranamaya kosam						
Manomaya kosam						
Vignanamaya kosam						
Ananthamaya kosam						

12. MUKKUTRAM:[AFFECTION OF THREE HUMORS]
A)VATHAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Praanan						
Abaanan						

Viyaanan						
Udhaanan						
Samanan						
Naagan						
Koorman						
Kirukaran						
Devathathan						
Dhananjeyan						

B) PITHAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Analapitham						
Ranjagam						
Saathagam						
Praasagam						
Aalosagam						

C) KABAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Avalambagam						
Kilaethagam						
Pothagam						
Tharpagam						
Santhigam						

1.SEVEN DHATHUS: (7 SOMATIC COMPONENTS)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Saaram [Chyme]						
Senneer [Blood]						
Oon [Muscle]						
Kozhuppu [Fat]						
Enbu [Bones]						
Moolai [Bone marrow]						
Sukkilam / Suronitham / Sperm / Ovum						

14. SYSTEMIC EXAMINATION:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Locomotor system						
Cardiovascular system						
Respiratory system						
Gastro intestinal system						
Central nervous system						
Urogenital system						
Endocrine system						

15. GENERAL EXAMINATION:

	0^{th} Day	**7th Day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Height (cms)						
Weight (kg)						
Temperature (F^0)						
Pulse rate (per min)						
Heart rate (per min)						
Respiratory rate (per min)						
Blood pressure (mm/Hg)						
Pallor						
Jaundice						
Clubbing						
Cyanosis						
Lymphadenopathy						
Pedal edema						
Jugular vein pulsation						

16. CLINICAL SYMPTOMS (USS):

Urolithiasis Symptom Score
(Circle relevant number on each line)

1. Pain/colic	0- No pain	1- Mild pain	2 -Moderate pain	3- Severe pain
2. Haematuria	0 -No haematuria	1- Microscopic	2- Persistent	3 -Gross
3. Dysuria	0 -No dysuria	1- Mild dysuria	2- Moderate dysuria	3- Severe
4. Stone		1- Single stone	2- Multiple stone	
5. Size of stones	0- Gravel < 03mm	1-3 mm to < 4 mm	2-4 mm to < 5 mm	3-5 mm and above
6. Position of stone in kidney	0- no stone in kidney	1-Pelvic ureteric junction	2 Pelvis of kidney	3 Calyces of kidney
7. Position of stone in ureter	0- no stone in ureter	1- Lower part of ureter	2- Middle of ureter	3- Upper part of ureter
8. Position of stone in bladder	0 -no stone in bladder	1 -Base of bladder	2- Intramural ureter	

Total scoring – 22, 1-7 mild, 8-14 moderate,15-22 severe. Symptoms score – (Some of 8 circled numbers)

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL

PALAYAMKOTTAI,TIRUNELVELI DISTRICT

POST-GRADUATE DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM III-LABORATORY INVESTIGATIONS

1	Sl.No		2	OP/IP No	
3	Bed No		4	Name	
5	Age		6	Gender	

I. Haemotological Investigations

		Before Treatment	After Treatment
1	TC (cells/mm)		
2	DC (%)		
	a)Neutrophils		
	b)Lymphocytes		
	cMmonocytes		
	d)Eosinophils		
3	ESR(mm)		
	a)1/2 hour		
	b)1 hour		
4	Haemoglobin		
5	Blood sugar		
6	Blood urea		
7	Serum creatinine		
8	Serum cholesterol		

II.URINE EXAMINATION

		Before Treatment	After Treatment
1	Albumin		
2	Sugar		
3	Epithelial cells		
4	Pus cells		
5	Red blood cells		
6	Casts/Crystals		

III.USG

	IMPRESSION	Before Treatment	After Treatment
1	Size		
2	Grade		
3	Residual Urine Volume		
4	Others		

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL, PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

CONSENT FORM-IV A

Certificate by Investigator

I certify that I have disclosed all details about the study in the terms readily understood by the patient.

Date: …….. Signature: …….

Name: ……..

Consent by Patient

I have been informed to my satisfaction, by the attending physician, the purpose of the clinical trial, and the nature of drug treatment and follow-up including the laboratory investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included

As a subject in the clinical trial of **NILAKUMILAVER KUDINEER for the management of KALLADAIPPU (UROLITHIASIS)**

.

Date: …….. Signature:

……

Name: …….

Date: ……. Signature of Witness: …….

Name…….. ……

Relationship: …….

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM
PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM IV B

WITHDRAWAL FORM

Name: _____ OPD/ IPD number: _____

Age : _____ Sex : _____

Date of trial commencement: _____

Date of withdrawal from trial: _____

Reasons for withdrawal:

- Long absence at reporting : Yes ☐ No ☐
- Irregular treatment : Yes ☐ No ☐
- Shift of locality : Yes ☐ No ☐
- Increase in severity of symptoms : Yes ☐ No ☐
- Development of severe adverse drug reactions : Yes ☐ No ☐

Date :

Station :

SIGNATURE OF INVESTIGATOR **SIGNATURE OF HOD**

SIGNATURE OF LECTURER

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
PALAYAMKOTTAI, TIRUNELVELI DISTRICT
DEPARTMENT OF MARUTHUVAM

PRECLINICAL AND PHASE-II RANDOMIZED OPEN CLINICAL STUDY ON KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM IV C
PATIENT INFORMATION SHEET

- Urolithiasis is refers to the entire clinical picture of the formation and passage of crystal agglomerates called renal calculi or stones in the urinary tract.
- The main factor causing this disease in to the absence of some proteins, glycosaminoglycans, pyrophosphate and citrate present in urine which help to keep otherwise salts in solute.
- Many herbal and mineral siddha medicines are currently practiced by the siddha practioners for Diabetes mellitus.
- The trial drug is prescribed only with evidence of siddha literature.
- The trial drug is prepared at the Gunapadam lab of government siddha medical college & hospital,palayamkottai, under the direct supervision of teaching faculties of Maruthuvam and Gunapadam Dept.

Details of the trial drug:

NILAKUMILAVER KUDINEER

DOSAGE : 50ml twice a day

DURATION : 30 days

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM IV E

ADVERSE DRUG REACTION FORM

Name: _____ OPD/ IPD No : _____

Age: _____

Date of trial commencement: _____

Date of withdrawal from trial: _____

Description of adverse reaction:

_____

_____

Date:

Station:

SIGNATURE OF INVESTIGATOR **SIGNATURE OF HOD**

SIGNATURE OF LECTURER

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
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PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON
KALLADAIPPU(UROLITHIASIS) WITH NILAKUMILAVER KUDINEER

FORM IV –E
(DRUG COMPLIANCE FORM)

Name : _____ Age/ Sex : _____ S. No : _____

OPD/ IPD No : _____ Date : _____ Bed No : _____

Name Of The Drug : **NILAKUMILAVER KUDINEER**

Drugs issued date :

Drugs returned date :

S.NO	DATE	DRUG TAKEN TIME	
		MORNING TIME	EVENING TIME
Day 1			
Day 2			
Day 3			
Day 4			
Day 5			
Day 6			
Day 7			
Day 8			
Day 9			
Day 12			

S.NO	DATE	MORNING/TIME	EVENING/TIME
Day 13			
Day 14			
Day 15			
Day 16			
Day 17			
Day 18			
Day 19			
Day 20			
Day 21			
Day 22			
Day 23			
Day 24			
Day 25			
Day 26			
Day 27			
Day 28			
Day 29			
Day 30			

Date :

Station :

SIGNATURE OF INVESTIGATOR :

SIGNATURE OF HOD :

SIGNATURE OF LECTURER :

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