NATIONAL INSTITUTE OF SIDDHA
Tambaran sanatorium, Chennai - 47
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CHENNAI - 600 032

A STUDY ON
KALLADAIPPU
(DISSertation SUBJECT)

For the partial fulfillment of the requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)
BRANCH I– MARUTHUVAM
SEPTEMBER - 2007
ACKNOWLEDGEMENT

I express my sincere thanks to chief siddhar Sivan, for the performs what is appointed for me.

I express my thanks to The Tamil Nadu Dr. M. G. R. Medical University
Chennai 32

I express my thanks to our director Prof. Dr. V. Arunachalam M.D. (s)
National Institute of Siddha, Tambaram sanatorium – 47.

I would like to express my immense gratitude to our respectable Head of the department Prof. Dr. K. Manikavasagam, M.D. (s) whose excellent guidance and valuable suggestion have enabled me to complete this dissertation in good shape.

I whole heartedly thanks to Dr. M. Logamanian M.D. (s) Asso. Prof. of NIS for her constant motivation and help in doing this work.

I also express my sincere thanks to Dr. G. Ujeevanam M.D. (s) for his encouragement in doing this study.
I acknowledge my thanks to Prof. Dr. C. Venkataraman, the director of C.L.Baid medha pharmacy college, Thoraibakkam, Chennai – 96, for his support for pharmacological and phytochemical study.

I my sincere thanks to Mr. Thirunavakarusu and Mr. Kannathasan and research student of C.L.Baid medha pharmacy college, Thoraibakkam, Chennai – 96, for his support for pharmacological and phytochemical study.

My sincere thanks also go to Mr. P. Jayapal MSc Asst. Prof. of Biostatics in NIS, Tambaram for his guidance in this study.

I fully heartily thanks to my parents, friends and relatives who are behind these success to work.
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INTRODUCTION

Siddha system of medicine is one of the ancient systems contemporaneous with those of the submerged lands, Egyptian, Mesopotamian, Chinese and Grecian medicines. The unique nature of the system is its continuous service to humanity for more than five thousand years in combating diseases, and in maintaining its physical, mental and mortal health.

The Siddha medicine is well founded on the basic principles of nature and its elements after a careful and thorough study of the human system.

The ancient Tamil in their quest for knowledge for longevity had developed two ways, by which man can achieve mastery over nature; the one is the yogic way and other through medicines. Yogies is known as Siddhars. Hence the system of medicine profounded by them came to be known as siddha system of medicine.

Siddha is a Tamil word that is derived from its root Siddhi – which means perfection in life or heavenly bliss. Siddhars attained eight kinds of supernatural powers. The persons who had attained eight kinds of miraculous power in the life are known as Siddhars.

The earliest medical treatise in Tamil was propounded by Sivanar, who was the first to preside over the ancient first Tamil academy. It was followed by a number of works of immortal Siddhars.

Siddha system dealing not only science of life, nature of universe, astronomical data, cosmic dance, atomic theory, space travel, alchemy and kayakalpa and medicines et.
Agastya siddhar who is the chief of the Siddhars’ school is said to have been a celebrated philosopher and physician who laboured amongst the Tamil in southern India.

The man is said to be the microcosm and the world the macrocosm; because what exists in the world exist in man; or in other words there is nothing in the macrosom of nature that is not contained in man. So man must be looked upon as an integral part of universal nature and not as anything separate or different from the latter. In the organism of man, these forces may act in an abnormal manner and cause diseases there by. Similarly, in the great organism of the cosmos, they may act abnormally likewise and bring about disease on earth. Seven planets exercise special power over some part of the body to cause diseases or diseases accordingly to their influences on the three humours in the system.

The human body is composed ninety six thatwas or constituent principles in nature including elements, bodily and mental organs, faculties matter etc.

The three humours are vadham, pittam and kabham. They are the three fundamental principles and essential factors in the composition and constitution of the human body. Three humours are representing respectively the air, the fire and the water of the five elements. Which form the connecting link between microcosm or man and macrocosm or world? The external air, heat and water correspond to the internal vatham, pitham and kabam.

Man is thus linked with external world and any change in the elementary condition of the external world has its corresponding changes in the human organ (humours). The normal order of vadham, pitham and kabam are in the proportion of 1: ½:¼ (4:2:1) respectively. Any changes in this proportion are some to bring about disease or death. But the maintenance of their normal proportion gives vitality of the organism and assures the prevention health and longevity of life.
The siddhars materia medica is based on human pathology. It asserts that all substances of the animal, the herbs and the mineral kingdoms contain one or more of these three humours in their composition. They should play an important role in the maintenance of these humours in the men and women. Siddhars knowledge is on minerals, metals and plants suitable combination of these things on processing and the preparation of medicine. According to the classification of the five elements of nature was of a very high order as can be learned from the lines of the ancient siddha medicine.

Super intellectual siddhars devised and utilized special technique for diagnosis a disease known as envagai thervugal. These eight factors play a vital role in finding out disease and deranged life elements.

Urinary calculus is known to mankind since antiquity. The earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 B.C.

*Kalladaippu* is most common disease of the present society Due to sedentary life style and abnormal diet habits.

Clinical features of *kalladaippu* are lower abdomen pain, dysuria, oliguria, burning micturition, nusea, vomiting, haematuria and fullness of abdomen.

In the contemporary world, till now no internal medicine is found for the renal calculus. In spite of new approach in diagnosis and management like Extra Corporeal Shock-wave Lithotripsy (E. C. S. L) and endoscopy surgery, but it still continues to cause significant morbidity with high tendency of reoccurrence and this kind of treatment procedure not reach to low socio economic status families, who are mostly affected renal calculus.

The contribution of Yoogi in the field of regional and humoral pathology regarding the disease *kalladaippu* is highly commendable; Siddha
medicine is ancient duration system of medicine for centuries has provided proven treatment of disease like *kalladaippu* without morbidity.

We must praise, safe guard and follow the Siddha system of medicine and all so furnish it to take up with the up to date advancement.

So the author of this dissertation work has selected *kalladaippu* under the neer arugal noi disease, expected by siddhar Yoogi in Yoogi vaithiya sindhamani perunool -800 and clinical entity comparable to urolithiosis in modern medicine. The drug *kalladaippu thool* was selected in Anupoga vaithiya navaneetham.
AIM AND OBJECTIVES

The aim of this work is to select the case of kalladaippu patients and administer them with the trial drug, as per the line of treatment and analysis clinically and experimentally to prove efficacy of the drugs. Siddha literature revealed so many drugs for kalladaippu; among them the author has selected kalladaippu thool.

The main objective of the patient study is to create awareness about Siddha medicine and highlight the efficacy of Siddha medicine for the disease of kalladaippu among the public with basic intention in mind. The following specific objectives have been drawn.

To collect and compare varies siddha and modern medicine.

To have an idea about incidence of kalladaippu with reference of age, sex, residence (Nilam), occurrence and duration of diseases.

To have a detailed study of investigation, diagnosis and prognosis of disease.

To study through investigation by Siddha fundamental and modern investigation during and after treatment in all patients.

To study the urine analysis, hematological study and ultrasonography for all patients.

To study the biochemical analysis of trial drug.

To study pharmacological diuretic study and acute toxicity study of trial drug.

To conduct the clinical trial of kalladaippu thool on kalladaippu.
The disease *kalladaippu* is placed under siruneer noi or moothira noi. Which is classified in to,

1. Neerinai arukkal noi (Oligurial diseases)
2. Neerinai perukkal noi (Polyurial diseases)

This has been mentioned by Therayar in his ‘Theran karisal’ as follows

"தீிக திகம்பர காமரகத முன்னிலிங்கன வீரேநாம்
தீிக மண்டவினால் தீிக பச்சிகைப்பான்
தீிகப் யோயிறு மார்ச்சும் தீிக க்ளிகைப்பான்"

-தீிக கிரீீீ்

*Kalladaippu* is one of the Neerinai arukkal noigal

Verupeyar (synonym): Achmari

**சுவூப்பு (DEFINITION)**

According to the text Siddha maruthuvam by Dr. Kuppusamy. There is gradual or suddenly obstruction to the flow of urine, pain with burning sensation in the urethral tract, low back pain, renal angle pain, and sands like crystal’s deposit in urine. These are characteristic features of *Kalladaippu*.
Agasthiyar says the definition of *kalladaippu* as sand likes crystals deposited in urine, followed by small size of stones are excreted in urine. Stones are stagnated in kidney, ureter, urinary bladder and urethra. Pain with burning sensation start in urethral orifice followed to agonizing pain occurs during the stone moving in urethral tract from the bladder, when the stone removed pain also relieved.

Large concentration of minerals in the bladder or kidney produces calculus or gravel. It is altered with difficulty in passing in urine.

- T. S. Sambasivam Pillai.

*Kalladaippu* is defined as pain in and around the umbilicus, fever, dysuria and urine smelling like that of goat's urine.

- Jeevanachamirtham.
NOI VARUM VAZHIKAL (AETIOLOGY)

"முதல் சிறுருவ மின்பருதியும்
நூற்றோர் வைணவ யாத்தி விளக்காந்துதான் புராணம்
நூற்றோர் மாதத்தில் வாழ்வுகின்று புரேல்
அதன் நிறைந்திரும் கலன்னுப்பருதியை”

-புத்தி காலரிசிசி தீர்த்தனை

Stone formed by derangement of humours of vatham and pittam.

"குப்பிலிக்காரங்க கலன்புப்பல் பாதிய விளக்கம்
குப்பிலிக்காரம் பினாத்திருப்பார் காலத்து
குப்பிலிக்காரங்க வண்ணமுள்ள புத்தி விளக்கம்
நூற்றோர் பாதிய மக்கள் விளக்கம்
நூற்றோர் வண்ணமுள்ள புத்தி விளக்கம்
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நூற்றோர் வண்ணமுள்ள புத்தி விளக்கம்
-புத்தி காலரிசிசி தீர்த்தனை

It is worth while to mention the poem of Yoogimamunivar who is authority of Siddhars regional and humoral pathology. He has revealed about this disease since 14th century.

Yoogimamunivar says that as blood clotted in urinary bladder due to urinary tract diseases followed by swelling of urinary bladder, urinary stones are formed in urinary tract induced by humour of vatham and pittam.

"நூற்றோர் குப்பிலிக்காரங்க கலன்புப்பல்
குப்பிலிக்காரங்க கலன்புப்பல் முக்கியம்
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குப்பிலிக்காரங்க கலன்புப்பல் குப்பிலிக்காரங்க
-புத்தி காலரிசிசி தீர்த்தனை
In this explains that mind plays a major role in causing many diseases and connection between body and mind and soul is established i.e., the mukkutram deranged by internal factors such as sexual perversion, anger and robbery.

“The causes mentioned here, 
- Contaminated water
- Stone crystals, bone, hair and sand mixed food.
- Decayed food
- Carbohydrate food
- Humour of vali induced food

Siddha maruthuva surukkam explained that urination is one of the 14 visceral reflexes. When one suppress this visceral reflex it lapses into manifestation of morbidity, which comprises inflammation of bladder,
anuria, arthralgia, pain in genital region and deranged of keezh nokku kaal. This leads to the formation of calculus.

The author also explain that ejaculation of semen is one of the 14 visceral reflexes when one suppress this reflex it causes fever, retention of urine which favours urinary calculi, chest pain, arthralgia, and white discharge.

"கொழிக்கை வடமால்கி கனேற்றுக்காக
்பாசமாய் கைகள் சுருக்கி
மார்த்தாமும் மாநிலமுடி
நின்ற பழுவதற்காக
பிக்கும் பயில்பெருந்து
துாரை வடமால்கினால்
நீரின் முறை குறைவு”
-நேற்றுக்கான சீவா

“கொழிக்கை வடமால்கின் குழல்பான்
சுருக்கி வடமால்கின்
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சால்ப் பிக்கும் பயில்பெருந்து
பல்பழுவை சுருக்கி பயில்பெருந்து
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நீரின் முறை
-நேற்றுக்கான சீவா

According to noi vilakkam

- Derangement if humour in blood,
- Excessive indulgence in sexual activity or sexual perversion,
- Trauma on testes,
- Suppression of urine and semen
• Inflammation of bladder
• Syphilis (Mega noi)
• Stagnation of urine in urinary tract.
• Dryness of semen causes the formation of stones.
• Increased intake of food that cause flatulence.

**POTHU KURI KUNANGAL**

• Gradual or sudden obstruction to flow of urine,
• Unbearable pain (agonizing pain) in the penis
• Excruciating pain and swelling is experienced at tip of penis if the calculus attempts to expel.
• Colicy pain radiating from loin to groin, lower abdomen, urethra and genitalia if the calculus is irregular with sharp projection.
• Burning and scanty micturation and haematuria.

**SYNDROMES ASSOCIATED WITH KALLADAIPPU**

Excessive growth of muscles in chest region, back of trunk, umbilicus and anal and urethral orifice followed by stricture of urethral orifice like a sand like crystals blocked in urethra. Dysuria, body pain, impairment of conscious, tiredness, and giddiness occurs.
The most experienced of Siddhars, Yoogimamunivar who has studied the disease according to regional and humoral pathology classifies *kalladaippu* into 4 types there are

1. **Vaikodam Kalladaippu**
2. **Paipudam Kalladaippu**
3. **Veyyai Kalladaippu**
4. **Paipudam Kalladaippu**

1. **Vaikodam Kalladaippu** *(VALI KALLADAIPPU)*

   "காலாடைப்பு வாடைகணங்கள் காலாடைப்புக் காலாடைப்பு
   பொழுதுகாலாடைப்பு காலாடைப்பு
   பொழுதுகாலாடைப்பு காலாடைப்பு
   பொழுதுகாலாடைப்பு

   தென்பெற்று பொழுது காலாடைப்பு
   தென்பெற்று பொழுது
   தென்பெற்று
   தென்பெற்று"

   - Retention of urine.
   - Lower abdomen pain.
   - Pain with swelling of penis.
   - Agonizing pain.
   - Breathlessness.
   - Abdomen distention.
   - Excretion of urine with mucus.

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   தென்பெற்று
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   பொழுதுகாலாடைப்பு
   பொழுதுகாலாடைப்பு

   தென்பெற்று
   தென்பெற்று
   தென்பெற்று
   தென்பெற்று"

   - Retention of urine.
   - Lower abdomen pain.
   - Pain with swelling of penis.
   - Agonizing pain.
   - Breathlessness.
   - Abdomen distention.
   - Excretion of urine with mucus.
2. பிற்கு கால்கள்பே (AZHAL KALLADAIPPU)

"அக்காலகுரு குற்றக்கலா வெள்ளாண்மை
ஆப்பாகுரு வெள்ளாண்மை வெள்ளாயலை
பல்பாகுரு குற்றக்கலா பெருமாக
்பன்பாகுரு வெள்ளாண்மை வெள்ளாயலை
தலாகுரு வெள்ளாயலை வெள்ளாண்மை
நூறாகுரு குற்றக்கலா வெள்ளாயலை
காலாக்குரு குற்றக்கலா குற்றக்கலா வெள்ளாயலை"

- Stone blocked in urinary tract followed by oliguria.
- Pain in urethra like an insertion of burning iron bar, followed by sweating all over body.
- Anuria, Agonizing pain.
- Blood stained calculus stagnated in urethra followed by piercing and boring pain with sensitiveness.

3. இற்கும் கால்கள்பே (IYYA KALLADAIPPU)

"காலாக்குரு குற்றக்கலா வெள்ளாயலை
ffects குற்றக்கலா குற்றக்கலா வெள்ளாயலை
காலாக்குரு வெள்ளாயலை குற்றக்கலா வெள்ளாயலை
பல்பாகுரு வெள்ளாயலை வெள்ளாயலை
பல்பாகுரு வெள்ளாயலை குற்றக்கலா வெள்ளாயலை
பல்பாகுரு வெள்ளாயலை குற்றக்கலா வெள்ளாயலை
பல்பாகுரு வெள்ளாயலை குற்றக்கலா வெள்ளாயலை"

- Abdomen distention like a bow with pain.
- Pain in all joints of upper and lower limb and hip joints.
- Pain in penis, sweating all over body.
- White coloured stone excreted in urine.
4. Mukkuttra Kalladaippu (MUKKUTTRA KALLADAIPPU)

"Mukkuttra Kalladaippu is a disease characterized by severe pain in the urethra, dysuria, oliguria, crystals excreted in urine like small sands, handful crystals excreted in urine daily. It is a fatal disease.

In Thanvanthri vaithiyam, kalladaippu is classified to four types they are

Kallerippu (KALLERIPPAAN)

"Kallerippu is a disease characterized by severe pain in the urethra, dysuria, oliguria, crystals excreted in urine like small sands, handful crystals excreted in urine daily. It is a fatal disease."
• Increased iyyam and azhal kutram dries the urine and semen forming calculi
• Sudden or gradual obstruction in urinary tract
• Dysuria
• Pain in umbilicus
• Fever
• Anorexia.

**SILETHUMA ACHMARI**

"தீர்மானத்திற்குச்செல்லும் மலர் கோயில்களில் கிட்டுக்கொள்ளும் சிறைக்குற்றங்களைப் பதிவு செய்து வெளிப்படுத்திக்கொள்ளப்படும். செய்தியர் பதிவு செய்து பின்னர் பிள்ளைகள் கலந்து கொள்ளும் செய்தியர் தீர்மானத்திற்குச் செல்லும் செய்தியர் தீர்மானத்திற்குச் செல்லும்.

• Calculus in the Ureter or urethra causes hydronephrosis
• Oliguria,
• Reddish white in colour and falls out like semen
• Stones are expelled as 4 or 8 fragments.

**PITHA ACHMARI**

"சிற்றுருவில் பின்னர்களில் பிள்ளைப்போக்கும் செய்தியர் தீர்மானத்திற்குச் செல்லும். செய்தியர் தீர்மானத்திற்குச் செல்லும் செய்தியர் தீர்மானத்திற்குச் செல்லும். செய்தியர் தீர்மானத்திற்குச் செல்லும் செய்தியர் தீர்மானத்திற்குச் செல்லும்.

• Burning sensation in urethra due to azhal kutram
• Burning micturition
• Formation of stone that appear like semicarpus anacardium seeds.

**SUKKILA ACHMARI**

"சங்கிலியும் பொழுதுதொண்டுள்ளில் கொன்றுக்காட்டும் சிற்றுருவினர் சிற்றுருவில் பின்னர்களில் செய்தியர் தீர்மானத்திற்குச் செல்லும். சிற்றுருவில் பின்னர்களில் செய்தியர் தீர்மானத்திற்குச் செல்லும். சிற்றுருவில் பின்னர்களில் செய்தியர் தீர்மானத்திற்குச் செல்லும்.

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• Suppressions of semen during ejaculation, develops on to stones and obstruction in the flow of urine
• Sand like gravels are expelled
• Pallor of the body.
• This is incurable.

CLASSIFICATION ACCORDING TO NOI VILAKAM

“மாறி மாறும் மலரித்து விளக்கும் நெற்று தன்னு விளக்கும் கனவால் மாறும் மலர்நித்தியினும்”

There are four types of kalladaippu according to noi vilakkam

1. Vali kalladaippu,
2. Analakalladaippu,
3. Iyya kalladaippu,
4. Karuneer kalladaippu

அளிக்கமல்லபு (VALI KALLADAIPPU)

“மாறி மாறும் மலரித்து விளக்கும் நெற்று தன்னு விளக்கும் கனவால் மாறும் மலர்நித்தியினும் கற்பிக்கும் கண்டையாக கனவும் பிள்ளைம
மாறிகள் கண்டால் குறிப்பிட்டு விளக்கும்
அளிக்கும் கண்டையாக கனவும் பிள்ளைம
நெற்று தன்னு விளக்கும் மலர்நித்தியினும் கனவால் மாறும் மலர்நித்தியினும்”

• Tongue biting, palpitation and shivering,
• Crushing of the lower abdomen and genital organs
• Dribbling of urine.
• The stones are blackish red colour.
அணலகால்பு (ANALA KALLADAIPPU)

“அணலகால்பு பெறும் பெயர்பிக்கும்
சுருக்கும் அல்லது கால்பண்டத்துலிகம்
சிலையாக குறிப்பிட்டு வருகிறில்
செல்பது முலைத் தம்முள்ள

• Burning micturition,
• Dysuria,
• The stones are reddish black or yellow in colour and passing of smalls stones

இய்யாகால்பு (IYYA KALLADAIPPU)

“இய்யாகால்பு பெறும் சில்லியக்
சுருந்து லைக் கால்பண்டத்துறை
சிலையாக பிளிப்பிட்டு வருகிறில்
பிள்பம் அல்லது தம்முள்ள

• Pricking pain, forcful pain with severe intensity when passing urine,
• Fever with rigors,
• White or honey coloured shining or luminant large size stone expelled.

கருனீர் கால்பு (KARUNEER KALLADAIPPU)

“கருனீர் கால்பு பெறும் சில்லியக்
சிலையாக பிளிப்பிட்டு வருகிறில்
கருனீர் கால்பு லைக் கால்பண்டத்துறை
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
அல்லது பிளிப்பிட்டு வருகிறில்
அல்லது பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
சிலையாக பிளிப்பிட்டு வருகிறில்
உடுப்பு அல்லது பிளிப்பிட்டு வருகிறில்
Sudden or gradual obstruction to flow of urine,
• Swelling of testis,
• Excessive vali kutram breaks the stones into small and large size crystals and expels along with urine.
• Sudden stoppage of urine stream,
• Retention of urine,
• Abdominal pain,
• Loss of taste excessive thirst,
• Pricking pain with swelling of abdomen and testis,
• Retention of urine or anuria may leads to renal failure and fatal

Classification in Jeevaratchamirtham and Anubhava vaithiya devaragasium:

Fives types

1. Vatha achmari,
2. Pitha achmari,
3. Kabha achmari,
4. Shukila achmari,
5. Swargara achmari.
Classification of disease in Indian medicine

-By Dr. Juliusjully

Four types;
1. Vatha achmari,
2. Pitha achmari,
3. Kabha achmari,
4. Shukila achmari.

Classification in siddhar aruvai maruthuvam- four types:
1. Vali kalladaippu,
2. Azhal kalladaippu,
3. Iyya kalladaippu,
4. Venneer kalladaippu.

MUKKUTRA VERUPADUKAL (PATHOLOGY)

The imbalance in one’s diet and fluid intake increases the Azhalkutram. This raised kutram dries up the body fluid and urine resulting in concentration of salts; this further affects the keezh nokku kaal. One of the functions of the keezh nokku kaal is to excrete urine. So when this keezh nokkukaal is affected urine will be obstructed within urinary tract. This favors the deposition of urinary salts to develop in to calculi any where in the kidney or urinary tract.

DIAGNOSIS AND PROGNOSIS

In piniyarium muraikal the following principles are followed in Siddha system. There are
1. கைப்பற்றியறிகைது கை,
2. எக்கல்லையறிகைது கை,
3. மூடையறிகைது கை.

The maruthuvar (physician) should observe the patient, palpate and interrogate the patient thoroughly. This is stressed also understood by this maxim. “Eyes first and most, Hands next and little, mouth last and never”
I, II, PORIYAL ARITHAL AND PULANAL ARITHAL

Poriyal arithal or understanding by the five organs of perception.
Pulanal arithal or understanding by the sense objects. There are

1. Mei- Ooru (Somatic sense)
2. Vaai – Suvai (Taste)
3. kan – Oli (Vision)
4. Mookku – Natram (Smell)
5. Sevi – Oosai (Sound)

1. இறுதிக்கும் (INTEROGATION)

An effective history taking helps one to diagnosis properly. By vinathal the physician should ask the patients native place, mode of living, food habits, personal habits, complaints and duration of illness etc. If the patient is deaf or dumb or if the patient is a child, the particulars should be obtained from his relatives or parents.
Poriyal arithal, pulanal arithal and vinathal are applied through eight special tools of investigation that is envagai thervugal.

ENVAGAI THERVUGAL

“மறுபரிதை சத்தியம் விசாரிழியிற்று
மல்லை கொரியாடண வங்குண்டுவரியும்”
-அமர்கிரி உரு

NAADI

The word pulses means the beating of artery felt with the tip of the fingers, its rate and character go to indicate a person’s condition of health. It is also understood as the beating, throbbing or rhythmical dilatation of arteries as the blood is propel led along them by the contraction of the heart in the living body. The term pulse in medical practice is usually applied to beat or throb felt in radial artery at the wrist; through it may felt over the temporal, carotid, ulnar, brachial, femoral and other artery.
Normally the pulse is recorded in the radial artery in the right hand for the male and left hand for the female by keeping the index finger, the middle finger and the ring finger on it after gently scrubbing the area. It is one unit in vali as felt by index finger and a half unit in azhal as belt by the middle finger and one fourth units in iyya as felt by the ring finger. The different diseases could easily be diagnosed with aid of the pulse.

"அரைய சர்கேசன் மாலும் கூம்பு வரிடா சுந்தரம் குடியர்கள் குடியர்கள் குடியர்கள் குடியர்கள்" -அககிரியின் வரம்

கந்தாகம் பாடல்

Aggravation of valinadi produces symptoms of *kalladaippu*. This is emphasized in Agathiar nadi, Sathaga nadi and Rathina churukkanadi.

"அரையேசேசன் வாகில்பிள்ளையானது அவர்கள் பெரும் பக்தவரை நீலங்கள் நீலங்கள் நீலங்கள்
சேலன் பூவுகள் சேல்பிள்ளையானது
சேலன் பூவுகள் கூம்பு வரிடா
சேலன் பூவுகள் குடியர்கள் குடியர்கள் குடியர்கள் குடியர்கள்
சேலன் பூவுகள் குடியர்கள் குடியர்கள்

"அரையேசேசன் வாகில்பிள்ளையானது அவர்கள் பெரும் பக்தவரை நீலங்கள் நீலங்கள் நீலங்கள்
சேலன் பூவுகள் சேல்பிள்ளையானது
சேலன் பூவுகள் கூம்பு வரிடா
சேலன் பூவுகள் குடியர்கள் குடியர்கள் குடியர்கள்
சேலன் பூவுகள் குடியர்கள் குடியர்கள் குடியர்கள் குடியர்கள்

"அரையேசேசன் வாகில்பிள்ளையானது அவர்கள் பெரும் பக்தவரை நீலங்கள் நீலங்கள் நீலங்கள்
சேலன் பூவுகள் சேல்பிள்ளையானது
சேலன் பூவுகள் கூம்பு வரிடா
சேலன் பூவுகள் குடியர்கள் குடியர்கள் குடியர்கள்

"அரையேசேசன் வாகில்பிள்ளையானது அவர்கள் பெரும் பக்தவரை நீலங்கள் நீலங்கள் நீலங்கள்
சேலன் பூவுகள் சேல்பிள்ளையானது
சேலன் பூவுகள் கூம்பு வரிடா

--கந்தாகம் பாடல்
Aggravation of azhal nadi produces symptoms of kalladaippu,

“இனியை மாற்றுறுப்பு தனிநாள் விலங்கிடையும் காற்று மற்றும் பந்து எத்துக்காடு பார்வுகின்றன இனியை காற்றுக் குறைவான குறைவு க் காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பது காண்பது குறைவான குறைவு காண்பат

Derangement of vali-azhal naadi produces symptoms of kalladaippu.

“புருஷரின் வலிக்குள் விளக்கம்
புருஷரின் வலிக்குள் விளக்கம்
மற்றும் வலிக்குள் விளக்கம்
காண்பது வலிக்குள் விளக்கம்
அல்லது வலிக்குள் விளக்கம்
-இன்று பாதுகாக்க

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

In Kalladaippu patients tenderness over the lower abdomen, renal angle and lumbar region, swelling can be felt in valikalladaippu (may be due to hydronephrosis)
Also patient’s temperature is increased in lower abdomen and in ayyakalladaippu, sweating all over the body at the time of colic.

துண்டு (Tongue)

By the examination of the tongue its colour, size, shape, coating, moisture, movement, ulcer, fissures, crust and condition of teeth and gums can be examined. In kalladaippu if there is constipation (valikalladaippu), the tongue would seem to be coated. Loss of taste in karuneer kalladaippu.

“கற வல்லத்துண்டில் மிகவும் மின்னின்னியுள்ளது
சொல்லல்ல டெட்டு விளையடுக்கின்றது”

தென் (Colour)

Colour of the skin all over body, a local region of affection in conjuntiva, tongue, nail bed and hair etc.

Vali udal- Block colour,
Azhal udal- Yellow or red colour,
Iyya udal – White or yellow

In kalladaippu niram of udal depends up on the body constitution, pallor of the body is observed in sukkila ashmari.

“சுக்கில மின் கோயில்ஸ்காஸ் மலர் மூச்சுக்கியம்
சுக்கிலுடை கோயில்பிட்டு மின் ஸ்கொலீ மூச்சும்”

மேற்புறம் (Speech)

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

மேற்புறம் (Eye)

Examine the colors of eye - like reddish or yellowish discoloration and characters like dryness and lacrimation. Tiredness and redness due to pain is observed in patients with renal colic. In addition one has to be
examine the patients acquits, there may be pallor of eyes due to gross haematuria.

**Stools**

By examining malam, its nature, colour, quantity and presence of blood or pus can be noted. In valikalladaippu constipation and oliguria will be present.

"நிறிக்குத்தார் பிக்கானது கற்குட்டு கொரிக்காரார் விளக்கான விமர்சனம்"

**Urine examination**

Urinary examination is good diagnosis method compare to naadi and other Envagai thervugal. Theraiyar mention below as

Siruneer should be collected in early morning; patient should be eating six tastes of food with regular time and well sleeping over night, urine should be examine with in 3¾hrs. This is quoted as

"அதிசுற்றுச் சிறுமி அதிசுற்றுச் சிறுமி
அளக்க அளக்க அளக்க அளக்க அளக்க
துற்கவை துற்கவை துற்கவை
அதிசுற்றுச் சிறுமி அதிசுற்றுச் சிறுமி
உரிமையுடைய உரிமையுடைய உரிமையுடைய
உரிமையுடைய உரிமையுடைய உரிமையுடைய உரிமையுடைய
உரிமையுடைய உரிமையுடைய உரிமையுடைய உரிமையுடைய
c
-தினு மற்றும் வாழ்கை காக்கம்
Siruneerin pothugunam:

“நீக்க தீர்க்கிறது வெள்ளம் நீண்டு காட்டுமலை
நான் பார்வையில் வைத்துரையே”
- திரு மாத்திராவார் கூறும்


Above the five parameters by which each urine sample should be examined.

**NIRAM (COLOUR)**

**NIRA THOGAI**

“பிரம் விளக்கத்தை காணி விளக்கத்தைப் போன்றால்
நீர்கல்லு நீர்கல்லு பெர்க்குத்து வைத்து”
-திரு மாத்திராவார் கூறும்

1. Yellow,
2. Red,
3. Green,
4. Block,
5. White.

Urine may be any colour mention above.

**கல்லப் பிளிள் துள்ளம் (COLOUR INDICATING URINARY STONES)**

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

“கல்லாளான் கல்லாள் லித்திலில் கல்லாளான்
நான் பார்வையில் கல்லாளான் கல்லாளான கல்லாளான
கல்லாளான கல்லாள் லித்திலில் லித்திலில்
பிளிள் பிளிள் பிளிள்
-திரு மாத்திராவார் கூறும்
EADAI (SPECIFIC GRAVITY)
Urine not thick is considered healthy.

“சில குழுப்பு சில விளக்கம் கிளிக்கினை
கக்கொடுத் தண்டு பொய்வர் கோப்பு தனிக்கை”
-மீதிய பதிக்கவேண்டும் கத்தைம

NURAI (FROTH)

“பாதகரம் பலசிலகாரசுறு பகாதான புக்காதான அதில் புக்கை குறிகளினை
ளஞ்சும்பித் குறிக்கைப்போன பாதாதை”

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

NATRAM (SMELL)

மூச்சுட்டிலிருந்தால்

“மாசாகாதிரா மாயிறா மாசாகாதிரா மால்
உருளடை கம்பள மாகாரகமிக்
கரைகளின் செய் கார்களின் பொல்காரின்
குர்ல்புரள் பிளக்கமன் கார்ட்தா மாகாரது
பொலுக்களின் புர்கைகள் குழு பொல்காரின்
இறக்கமுத் கீர்த்தண்ணம் பொலுக்கள் குல்லை”

-மீதிய பதிக்கவேண்டும் கத்தைம

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

ENJAL (DEPOSITS)

If urine excretion look like curd water white color and sand like deposits in urine indicate stones in kidney. This mention as
NEI KURI

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

"அரோக்கக் கிளாசிக்கெட் நகம்”
“சுரி பிரம் பாசியல் அகில்பிக்கும்”
“எகிபத் கிளாசிக்கெட் நோகமான்கும்”

In kalladaippu patients, oil spreading like ring indicates Azhal neer or snake indicates Vali neer.

КАРЫКИЛАВА, АЗАРЫКИЛАВА (PROGNOSIS)

“சில்பார்க் கிளாசிக்கெட் விரோட்ட நோகமா
கோப்பாய வார்கா கண்டல்லாபு
புல்பா பில்குெட் கண்டல்லாபு
புக்குரலெடு கண்டல்லாபு
மென்பு கூல்லுஞ்சா ஆகியா

புக்குரலெடு கூல்லுஞ்சா விரோட்ட நோகமா
கோப்பாய வார்கா கண்டல்லாபு
தீர்க்கையும் கூல்லா கண்டல்லாபு

-கிளாசிக்கெட் நோகமா குர்கூர்
According to Yoogimamunivar, Vali, Azhal, and Ayya kalladaippu are curable where as Mukkutra kalladaippu is in curable.

**LINE OF TREATMENT**

“கல்லடைப்பு வைப்பு கல்லடைப்பு”

- விற்கும் 800

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

“சுருக்கிசுறு முது வரும்
சுருக்கிசுறு பாது வரும்
அடுத்தத்தான் கம் கா வரும்”

- மோழுடன் -II

As per the above mention, author gives purgation to all patients as their body condition. The author of dissertation has selected trial drug *Kalladaippu thool*, dose of 5gms two times per day with raddish juice for 2 months period.

In siddha system treatment is not only for removable of disease but the prevention and improving the body condition after removal of disease. This is said as kappu, neekam and thiraippu.

**DIET**

All patients are strictly advised to follow this diet restriction as highly calcium and oxalate diet likes cabbage, cauliflower, tomato, pees, cashonuts, almonds, grapes, strawberry, pine apple, tea and coffee. Calcium and protein diet likes milk, milk products and non vegetarian foods.
URINARY CALCULUS

PHYSIOLOGICAL ANATOMY OF THE KIDNEY

The kidneys are two bean shaped organs situated retroperitoneal, one on the either side on the posterior abdominal wall. Each kidney weighs about 120 to 150gms and is enclosed in thin but tough fibrous capsules, and concave medial side of the kidney is the hilum where the renal artery enters and renal vein and ureter leave the kidney. The components of kidney are arranged in three layers.

1. Outer cortex

This is dark and granular appearance. It contain renal capsules and convoluted tubules, at intervals cortical tissues penetrate medulla in this form of columns is called column of Bertini.

2. Inner medulla

This gives radially striated appearance as it contains tubular and vascular structures. Medulla mass is divided in to 8 to 18 medullary or malpighian pyramids, the base of pyramids and are connect with cortex and apex projects in to minor calyx.

3. Renal sinus

It consist upper expanded part of ureter called renal pelvis, it subdivided into about 8 minor calyces, and branches of nerves, arteries, and tributary vein and loose connective tissues and fat.

Nephron

Structural and functional unit of the kidney is nephron. Each kidney has a million nephrons. Each nephron begain in cortex as

- A funal like dilatation called the Bowman’s capsule, which enclose a tuft of capillaries the glomerulus. The Bowman’s capsule together with the glomerulus is called the Malpighian corpuscle or Renal corpuscle.
- The renal tubule leaves the Bowman’s capsules and becomes convoluted to form of the proximal convoluted tubule (PCT).
• It then becomes straight and passes down it the medulla as the descending limb of the loop of the Henle, after varying distances before reaching the end of the papilla, it turns round in the form a U-shaped bend, forming loop of Henle, and passes upward towards the cortex, parallel with its former course as the ascending limb of the loop of Henle. Each limb has an outer thick and inner thin portion.

• Thick ascending limb approaches its own, glomerulus, and contact with the afferent and efferent arterioles to form the juxtaglomerular apparatus. It then becomes convoluted to form the distal convoluted tubule.

• The DCT then straightens out and joins by short connecting ducts to form the collecting tubules or collecting ducts (CD). They unit to form larger collecting ducts and descent parallel to loop of Henle and open in the papilla or the duct of bellini in the renal pelvis. The length of the nephron varies from 4 to 6.5cms.
RENAL CIRCULATION

Renal artery arises from the aorta, enters the kidney at the hilum and divided in to an anterior and a posterior branch, which give rise the about five segmental arteries. The segmental artery divided into interlobar arteries, which pass outward in the medulla between the pyramids to reach boundary zone between the medulla and cortex. Here they turn to take a horizontal course uniting with adjacent arteries to form arterial arches called arcuate arteries. Several straight arteries arise from these arches and run radially outward through the cortex. These are called interlobular arteries. From each interlobular artery, numerous afferent arteries arise, and enter the Bowman’s capsule forming glomerular capillary tuft. The afferent arterioles divide in to 4 to 5 large capillaries. Each large capillary divided in to small capillaries, which form the loop, and capillary loop unit to form the efferent arteriole, which leaves the Bowman’s capsule.
The efferent arterioles give rise to renal portal system. The efferent arterioles form a second capillary network surrounding the tubular portion of the nephron; the capillaries of second set are called peritubular capillaries.

The tubular portion of juxtamedullary nephron is supplied by some specialized peritubular capillaries called Vasarecta. Vasarecta arise directly from the efferent arteriole of the juxtamedullary nephrons. The peritubular capillaries drain into the venous system, which include the peritubular venules, interlobular veins, segmental vein and finally the renal vein.

**URINE FORMATION**

Kidney excreted the unwanted substances including metabolic end products and those substances, which are present in excessive quantities in body, through urine. Normally about 1 to 1.5 liters of urine is formed every day. The mechanism of urine formation includes various processes. First, when blood passes through glomerular capillaries, the plasma filtered in Bowman capsule. When this filtrate passes through the tubular portion of nephron, it undergoes varies changes both in quality and in quantity. Many wanted substances like glucose, aminoacid, water and electrolytes are reabsorbed from the tubules, this process is called tubular reabsorption. And some unwanted substances are secreted in to the tubule from peritubular blood vessels. This process is called tubular secretion. The urine formation includes three processes.

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

**1. GLOMERULAR FILTRATION**

When blood passes through the glomerular capillaries the plasma filtered in to the Bowmans capsule. All the substances of plasma are filtered except plasma proteins. The filtered fluid is called Glomerular filtrate. During filtration the substances passes through a three layer of filtrating membrane such as
1. The endothelium of capillary membrane,
2. Basement membrane and
3. Endothelium of visceral layer of Bowman capsule.

The glomerular filtration is called ultrafiltration because minute particles are filtered, but the plasma proteins are not filtered due to larger molecular size than size of the slit pores. The composition glomerular filtrate is similar to that of plasma except in the absence of plasma proteins.

**Glomerular filtrate rate GFR**

The total quantity of filtrate formed in all the nephron of both the kidneys in the given unit of time is called glomerular filtrate rate. The normal value of glomerular filtrate rate is 125 ml / minute or about 180 litres / day.

**Filtration Fraction**

The fraction of the renal plasma which becomes the filtrate is called filtration fraction. It is the ratio between renal plasma flow and glomerular filtrate. It is expressed in percentage.

They filtration fraction = \( \frac{\text{GFR}}{\text{Renal plasma flow}} \times 100 \)

\[ \frac{125 \text{ ml / minute}}{650 \text{ ml / minute}} \times 100 \]

= 19.2%

The normal filtration fraction values from 15 to 20%

**Pressure determining filtration:**

Glomerular capillary pressure, colloidal osmotic pressure in the glomeruli and the hydrostatic pressure in the Bowman’s capsule, which are determine the GFR. Among these pressures, the glomerular capillary pressure favours filtration the glomerular capillary pressure is about 60mm Hg and is highest capillary pressure in the body. The colloidal osmotic pressure and hydrostatic pressure oppose the filtration. The colloidal osmotic pressure exerted by plasma protein in the glomeruli. The plasma proteins are not filtered through the glomerular capillaries, so increased
concentration of proteins in glomerulus during filtration causes the development of colloidal osmotic pressure. It is about 25mmHg. Hydrostatic pressure in Bowman’s capsule is exerted by the filtrate in Bowman’s capsule during filtration. It is about 15mmHg.

**Net filtration pressure:**

The balance between pressure favoring filtration and pressures opposing filtration is called net filtration pressure. This is very essential for the maintenance of GFR so this is otherwise known as effective filtration pressure.

The net filtration pressure = Glomerular capillary pressure – colloidal osmotic pressure + Hydrostatic pressure in Bowman’s capsule

\[ = 60 – (25+15) = 20\text{mmHg} \]

The normal net filtration pressure is about 20mmhg and it varies between 15 and 20mmHg

**Factors regulating GFR:**

Following are the various factors, which regulate or affect the GFR

1. **Tubular glomerular feedback mechanism**

   This is the process in which the GFR is constantly regulated by means of feedback from renal tubule. The macula densa of juxtaglomerular apparatus is responsible for this. When the glomerular filtrate passes through the end portion of thick ascending segment of renal tubule, the macula densa detects the concentration of sodium chloride and accordingly alters the GFR. If the concentration of sodium chloride is more, macula densa causes constriction of afferent arteriole and filtration rate decreases, the constriction of afferent arteriole may be due to the secretion of thromboxane A2 from macula densa.

2. **Glomerular capillary pressure**

   The GFR is directly proportional to glomerular capillary pressure. When glomerular capillary pressure is increased the GFR is also increased, in turn depends upon the renal blood flow
3. Colloidal osmotic pressure
   The GFR is inversely proportional to colloidal osmotic pressure exerted by protein. During dehydration or increased plasma protein level, colloidal osmotic pressure is more and GFR is reduced.

4. Hydrostatic pressure in Bowman’s capsule
   GFR is inversely proportional to this. The hydrostatic pressure in Bowman’s capsule is increased in conditions like obstruction of urethra and oedema of kidney beneath renal capsule.

5. Renal blood flow
   This is the most important factor necessary for glomerular filtration. And, the GFR is directly proportional to this.

6. Constriction of afferent arteriole
   This reduces the blood flow to the glomerular capillaries and this in turn reduces GFR.

7. Constriction of efferent arteriole
   If efferent arteriole is constricted; initially there is an increase in GFR.

8. Systemic arterial pressure
   However, increase in mean arteriole pressure up to 180mmHg or reduction up to 60mmHg does not alter renal blood flow or GFR. This is due to auto regulatory mechanism. Variation in pressure above 180mmHg or below 60mmHg affects the renal blood flow and GFR because auto regulating mechanism fails beyond this range.

9. Sympathetic stimulation
   The mild or moderate stimulation of sympathetic nerves does not causes any significant change either in renal blood flow or in GFR. This is due to auto regulation. Strong sympathetic stimulation causes severe constriction of the blood vessels needs to increase filtration initially, but latter decreases.

10. Surface area of capillary membrane
    GFR is directly proportional to the surface area of the capillary membrane.
Tubular reabsorption

When the glomerular filtrate passes through the tubular portion of nephron, both quantitative and qualitative changes occur. Large quantity of water, 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 more 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. So the tubular reabsorption is called the selective absorption. Depending upon the degree of reabsorption, the various substances are classified into three categories.

1) High threshold substances

The food substances like glucose, amino acid, acetoacetate ions and vitamins are completely reabsorbed do not appear in urine under normal condition. These substances appear in urine, only if their concentration in plasma is abnormally high or in renal diseases. So these substances are called high threshold substances.

2) Low threshold substances

The substances such of urea, uric acid and phosphate are reabsorbed to little extent. These substances appear in urine even under normal conditions. Such substances are known as low threshold substances.

3) Non threshold substances

The metabolic end products like creatinine are not at all reabsorbed and are excreted in urine irrespective of their plasma level. These substances are called non threshold substances.

Mechanism of reabsorption
The mechanisms involved in tubular reabsorption are of two types
1. Active reabsorption and
2. Passive reabsorption.

1. Active reabsorption
   The movement of molecules is against the electrochemical gradient. This needs liberation of energy. And the energy is derived from ATP. The substances reabsorbed actively from the renal tubule are sodium, calcium, potassium, phosphates, sulphates, bicarbonates, glucose, amino acids, ascorbic acid, uric acid and ketone bodies.

2. Passive reabsorption
   In this process, the movement of molecules is more along the electrochemical gradients. This process does not need energy, the substances reabsorbed by passive transport are chloride, urea, and water.

Site of reabsorption
   Substances reabsorbed from proximal convoluted tubule. Glucose, amino acids, sodium, potassium, calcium, bicarbonates, chlorides, phosphates, uric acid and water are reabsorbed from proximal convoluted tubule. The substances reabsorbed from loop of Henle are sodium chloride. The substances reabsorbed from distal convoluted tubule are sodium, bicarbonate and water.

Tubular secretion
   Some substances secrete in to the lumen from the peritubular capillaries through the tubular epithelial cells. These known as tubular secretion or tubular excretion.

1. Potassium is secreted actively by sodium – potassium pump in distal convoluted tubule and collecting duct.

2. Ammonia is secreted in the proximal convolute tubule.
3. Hydrogen ions are secreted in the proximal and distal convoluted tubules. Maximum hydrogen ion secreted in proximal tubule.

Thus by the process of glomerular filtration, selective reabsorption and tubular secretion urine is formed in the nephron. It is also concentrated by counter current mechanism and ADH. Finally it passes through ureter into the urinary bladder and is stored there until it is voided out.
URINARY CALCULUS

Urinary calculus is stone–like body composed of urinary salts bound together by a colloid matrix of organic material, it consist of nucleus around which concentric layers urinary salt are deposited. Urinary stones have afflicted humankind since antiquity, with earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 BC.

Until the 1980s, urinary stones were a major problem, with a significant proportion of patients requiring extensive surgical procedures and a sizable losing their kidney. The advent of extra corporeal techniques for stone destruction and refinements in endoscopic surgery, however have greatly decreased the morbidity associated with stone surgery.

EPIDEMOLOGY

INTRINSIC FACTORS

Hereditary

Several disorders that cause renal stones are hereditary. Familial renal tubular acidosis are associated with nephrolithosis in almost 70% of patients. Cystinuria, Xanthinuria and Dehydroxyadenuria are disorders cause renal stone.

Age and sex

The peak incidence of urinary calculus occurs in the twenties to forties. About three males are affected for every female because increased serum testosterone causes increased endogenous oxalate production in male, increased urinary citrate in women.

EXTRINSIC FACTORS

Geography

The prevalence of urinary calculi is higher in those who live in mountainous, desert, and tropical areas.
Climate and seasonal factors

Price and associates found that the incidence of urinary calculi was higher during the summer months. High temperatures increase perspiration, which may result in concentrated urine. This promotes increased urinary crystallization. Parry and Lister suggest that increased exposure of sun light cause increased production of 1, 25-dihyrdooxvitamin D3 and increased urinary calcium excretion. This may cause higher incidence of urolithiosis in summer months.

Water Intake

Urine dilution by increased water intake may increased ion activity coefficients and hence urinary crystallization, water diuresis reduces the average time of residence of free crystal particles in urine dilutes components of urine that may crystals. Minerals contents of water may contribute to cause stone disease. (E.g. sodium chlorides) Zinc in an inhibitor of calcium crystallization.

Diet

Ingestion of excessive amounts of purines, oxalates, calcium, phosphates, and other elements often results in excessive excretion of these components in urine causes increased incidence of calculi.

Occupation

Lonsdale (1968b.c) indicated that urinary calculi are much more likely to be found in individuals who have sedentary occupations. The highest were found in cooks and engineering room personnel. The risk of calcium oxalate and uric acid stones formation in Astronauts because of hypercalciuria, hypocitraturia, decreased pH, and lower urinary volumes.

PHYSICAL CHEMISTRY

Urinary stones do not occur unless crystals of the offending substance form in urine. For crystals to occur, the urine should be supersaturated with the salt in consideration. Increases in urinary excretion of chemicals that constitute the crystals result in an increased in potential for crystallization. Urine does not need to be continuously supersaturated
for crystals to form or grow: Intermittent super saturations, as is seen during of dehydration or after meals, is sufficient. Crystallizing potential for calcium oxalate is related not so much to the concentration of calcium or oxalate in urine but to chemical activity of ions in solution. The compounds such as citrate and phosphate form complexes with calcium, and elements such as magnesium and sodium form complexes with oxalate, effectively reducing the free ionic concentration of each.

Urinary crystals can be seen in most urine specimens, particularly after storage, yet most individuals do not form stones. Stone formers as a group excrete larger crystals and crystals aggregates than healthy individuals. Normal subjects have inhibitors of crystal formation, growth, and aggregation in urine such as low-molecular-weight compounds such as citrate and pyrophosphate and larger molecules, such as glycosaminoglycans, nephrocalcin, and Tamm – Horsfall protein. Urine from patients with recurrent calcium and oxalate stones tends to have higher calcium and oxalate saturation and lower inhibitors than urine from patients without stones and a mathematically derived saturation - inhibition index has been reported to discriminate between the groups with better than 90% accuracy.

Free crystals formed within the kidney do not have the ability to grow to a size large enough to occlude a collecting duct and, form a stone in a free – flowing urinary system. Crystal aggregation and retention within the kidney are prerequisites for urinary crystals to be converted to urinary calculi. Crystal aggregation is enhanced in individual who lack inhibitors of aggregation. The urinary glycoproteins nephrocalcin and Tamm-Horsfall protein are potent inhibitors of crystal aggregation in simple solutions, whereas citrate and magnesium are inhibitors of crystal growth.

Anatomical abnormalities, such as medullary sponge kidney or ureteropelvic junction obstruction, or increased “stickiness” of the tubular epithelium can predispose to increased crystal retention. Urate and calcium oxalate crystals anchor to surrgaces of cultured renal epithelial cells and may adhere in vivo to tubular cells or urothelium. Although not
Proven, bacterial infection may promote calcium oxalate stone formation by increasing urinary matrix, which, in turn, promotes crystal adherence. Finally, altered transport of calcium and oxalate by renal epithelial cells may result in intracellular or interstitial crystallization. These are retained in the kidney and can become the nidus for stone formation.

**TYPES OF CALCULUS**

**CALCIUM OXALATE STONES**

This is the most common type of stones. 39% of patients are calcium oxalate type stone. 14% of patients are combined with calcium phosphate. They are irregular in shape and covered with sharp projection, which tend to cause bleeding. The surface of the stone is discoloured by pigments of altered blood. It is very hard and absorbs X-rays well.

**PATHOPHYSIOLOGY OF STONE FORMATION**

**I. HYPERCALCIURIA**

Between 30% and 60% of all patients with calcium oxalate kidney stones have increased urinary calcium excretion in the absence of raised serum calcium levels. Hypercalciuria has been defined as the excretion of greater than 4mg calcium per kg body weight per day or greater than 7 mmol in men and 6 mmol in women (Parks and Coe, 1986). Final definition is the excretion of urinary calcium of greater than 0.11 mg/100 ml of glomerular filtrate. Hypercalciuric nephrolithiasis suffer from multiple disturbances in renal tubular function a disturbance in phosphate transport, and accelerated 1, 25 – dihydroxyvitamin D3 synthesis, resulting in increased intestinal calcium absorption.

Hypercalciuria has three types, such as

1. Absorptive hypercalciuria,
2. Renal hypercalciuria
3. Resorptive hypercalciuria,
1. Absorptive hypercalciuria

In absorptive hypercalciuria, the primary abnormality is increased calcium absorption. In absorptive hypercalciuria type 1, intestinal hyperabsorption of calcium exists, whether or not the patient is on a calcium-restricted diet. Intestinal magnesium absorption is normal in patients with absorptive hypercalciuria, but oxalate absorption is increased. Vitamin D increases calcium and magnesium, but not oxalate, absorption in both the jejunum and ileum. Absorptive hypercalciuria type 2 is a variant of this disorder wherein patient's exhibit increased urinary calcium excretion while on their normal diet but normal calcium excretion on a low-calcium, low-sodium diet. In the final subcategory, absorptive hypercalciuria type 3, the serum phosphate is low, suggesting that the increased intestinal calcium absorption is the result of stimulation in vitamin D production as the result of the lowered serum phosphate.

2. Renal hypercalciuria

In this condition, the underlying abnormality is a primary renal wasting of calcium. The consequent reduction in circulating serum calcium stimulates PTH production. Two facts must be stressed; first, intestinal absorption of calcium is increased in both absorptive and renal hypercalciuria; second, parathyroid function is suppressed in absorptive hypercalciuria but stimulated in renal hypercalciuria. These two criteria elevated fasting urinary calcium levels and stimulated parathyroid function serve to distinguish renal from absorptive hypercalciuria.

3. Resorptive hypercalciuria

This syndrome is synonymous with subtle hyperparathyroidism. Hypercalciuria results from excessive PTH dependent bone resorption as well as enhanced intestinal absorption of calcium.

4. Idiopathic hypercalciuria

The syndrome of renal phosphate leak, elevated 1, 25 dihydroxyvitamin D3, and absorptive hypercalciuria has been demonstrated in members of a Bedouin tribe in which intermarriage is common. Idiopathic
hyper calciuria may be inherited as an autosomal trait, although the pattern can reflect polygenic control of calcium excretion as well.

II. HYPERCALCAEMIA NEPHROLITHIOSIS

CAUSES

1. Primary hyperparathyroidism
   These patients are between 39% and 78% with early presented of renal calculi. These patients with higher level of 1, 25-dihydroxyvitamin D3 and greater calcium absorption tend to have renal stones.

2. Malignancy associated hypercalcemia
   Malignancy –associated hyper calciuria is an exceedingly rare cause of renal stones. The most common cause of malignancy- associated hypercalcaemia, even in patients with skeletal metastasis, is production by the tumour of a bone resorbing substance called PTH- related polypeptide.

3. Sarcoidosis and other granulomatous diseases
   The sarcoid granuloma produces 1, 25-dihydroxyvitamin D3, it causing increased calcium absorption, hypercalcaemia, and hypercalciuria.

4. Hyperthyroidism
   About 5% to 10% of patients with hyperthyroidism develop hypercalcimia. Hypercalcaemia and hypercalciuria result from a stimulation of bone resorption mediated by thyroxine and tri-iodothyronine.

5. Glucocorticoid – induced hypercalcemia
   Glucocorticoid excess leads to increased bone resorption, decreased bone formation, and osteopenia. Glucocorticoid also has direct stimulatory effect on parathyroid gland.

6. Pheochromocytoma
   Hypercalcaemia when seen in patients with pheochromocytoma, occur most often in patient with multiple endocrine neoplasma type 2, in which primary hyperparathyroidism, medulary carcinoma of thyroid, adrenal gland tumour coexit.
7. Immobilization

Prolonged bed rest can lead to hypercalcaemia, as the result of increased bone turnover. Hypercalcaemia is seen most often when another condition, such as Paget’s disease- with accelerated bone turnover-primary hyperthyroidism, or malignancy, coexists in an immobilized patient.

III. HYPEROXALURIA

CAUSES:

Primary hyperoxaluria is a rare genetic disorder resulting from increased hepatic production of oxalate. Enteric hyperoxaluria occurs in patients with short bowel syndrome or malabsorption. Finally, a group of patients with recurrent idiopathic calcium oxalate lithiasis exhibits mild hyperoxaluria or increased transport of oxalated by red blood cells.

1. Primary hyperoxaluria

Two types of primary hyperoxaluria exit. Primary hyperoxaluria type1 is an autosomal reessive inborn error of metabolism characterized by nephrocalcinosis and oxalosis. The diseases are characterized by increased urinary excretion of oxalic, glycolic, and glyoxylic acids. Primary hyperoxaluria type I is due to a defect of the enzyme alanine-glyoxylate aminotransferase (AGT) in the liver.

In normal human liver, AGT catalyzes the transamination or detoxification of glyoxylate to glycine, a function that it can perform only if it is located in the peroxisome. Its deficiency in primary hyperoxaluria results in glyoxylate’s being oxidized to oxalate.

Primary hyperoxaluria type II or L-glyceric aciduria is a much rarer variant of the disease. Deficiencies of the hepatic enzymes D-glycerate dehydrogenase and glyoxylate reductase lead to increases in urinary oxalate and glycerate excretion.

2. Enteric hyperoxaluria

Ingested oxalate is absorbed through the stomach and the colon. Malabsorption from any cause, including small bowel resection, intrinsic
disease, or jejunoileal bypass, increases the colonic permeability of oxalate as the result of exposure of the colonic epithelium to bile salts. Furthermore, loss of calcium in the feces results in the presence of less calcium in the intestinal lumen, allowing oxalate to exist in a soluble form. The hyperoxaluria from small bowel malabsorption often exceeds 1 mmol/day and causes recurrent nephrolithiasis, nephrocalcinasis, and renal oxalate deposition.

3. Mild metabolic hyperoxaluria

Mild hyperoxaluria is as least as important a factor in the pathogenesis of idiopathic calcium oxalate stones as hypercalciuria. Baggio and colleagues are found an increase in oxalate self exchange across red blood cell membrane in 79% of patients with idiopathic calcium oxalate stones. The oxalate absorption was increased with increased calcium absorption. Dietary restriction of oxalate results in decreased oxalate excretion.

IV. HYPERURICOSURIA

Uric acid promotes calcium oxalate crystallization by facilitating the formation nuclei. Sodium acid urate may produce calcium oxalate stone disease by nullifying the effectiveness of naturally occurring inhibitors of calcium oxalate crystal growth. Excessive dietary intake of purine is main cause of hyperuricosuria. Between 80% and 90% of patients with hyperuricosuria nephrolithiosis are men. Patients with mixed with uric acid and oxalate stones have lower urinary pH than patients with pure calcium oxalate stones.

V. HYPOCITRATURIA

Hypocitraturia has been reported in 15% to 63% of patients with stones. Urinary citrate is normally greater in women than in men. Hypocitraturia define as citrate excretion of less than 0.60 mmol (115mg) in men and 1.03 mmol (200mg) in women. Acidosis is probably the most important etiologic factors in hypocitraturia. In patients with inflammatory bowel disease and chronic diarrhea, intestinal alkali loss results in metabolic acidosis. Thiazide- induced hypokalaemia and intracellular
acidosis are other cause of decreased urinary citrate excretion. A diet rich in animal protein may produce an acid load. Strenuous physical exercise and sodium intake can likewise produce hypocitraturia. Urinary tract infection with bacteria degrading citrate lowers urinary citrate excretion.

The primary mechanism of action of citrate is as a complexing agent for calcium. Calcium citrate complexes are considerably more soluble than calcium oxalate. Citrate inhibits the spontaneous nucleation of calcium oxalate, crystal growth and aggregation of calcium oxalate and phosphate.

VI. HYPOMAGNESURIA

Many experimental studies have suggested that administration of magnesium salts prevents stones disease. The most common cause of hypomagnesuria is inflammatory bowel disease associated with malabsorption. Most patients with hypomagnesuria also have hypocitraturia.

VII. SEX HORMONES AND RENAL STONES

Calcium oxalate renal stones occur much more frequently in men than in women because increased serum testosterone levels resulted in increased endogenous oxalates production by liver and increased intestinal absorption of calcium in men, increased urinary citrate concentration in urine in women.

CALCIUM PHOSPHATE STONES

Calcium phosphate stones composed predominantly around 10% of stones of renal origin. Although some amount of calcium phosphate is often found in calcium oxalate calculi, pure calcium phosphate stone are rare. It is usually smooth and dirty white, and easy to see on radiopaqhic film.

Causes:

Renal tubular acidosis is common cause for this type of stone. Up to 70% of adults with renal tubular acidosis have kidney stones. This stone
may be seen with oxalate and stuvite stones. Stone formation typically occurs in papillary tips and in medulla. Stone formation is result of hyper calciuria, hypocitraturia, and increased urinary pH. Hypercalciuria is result of systemic acidosis on bone demineralisation and secondary hyperthyroidism. Hypocitraturia result from a primary defect in renal tubular citrate transport, again, the result of metabolic acidosis. Hypocitraturia is probably the most important metabolic factor for stone formation in patient with renal tubular acidosis. The defect in proximal tubular bicarbonate resorption is associated with this type of stone.

Diagnosis: the patient has hypokalaemia, hyper chloremia, metabolic acidosis and urinary pH of 5.5.

**URIC ACID STONE**

These are hard, smooth and often multiple. Their colour varies from yellow to reddish brown. The pure uric acid stone are radiolusent most of stone with calcium so they radiolusant shadow.

**Causes:**

The principal cause of uric acid crystallization is supersaturation of urine with respect to undissociated uric acid. The normal 24-hour urinary uric acid excretion is between 500 and 600mg per litre of urine. Patient with uric acid stone often have prolonged periods of acidity in urine. The pH of urine in patient with uric acid stones was 5.5 ±0.4 as compared with 6.0±0.4 in patients who form calcium oxalate stones. Three factors are involved in uric acid urolithiosis. First, patients tend to excrete excessively acid urine at relatively fixed, low urinary pH. Second, they may absorb, produce, or excrete more uric acid than patients without gout or uric acid stones. Third, urinary volume is diminished in these patients. The combination of these factors is ideal for the crystallization of uric acid in the urine. The frequency of uric acid stones in gout is about 20%. Myeloproliferative disorders such as acute leukemia are an important cause of severe hyperuricosuria, particularly in childhood.
STRUVITE STONES (INFECTION STONES)

The stone is composed of magnesium, ammonium, and phosphate, mixed with carbonate.

Pathogenesis

Two conditions must coexist for the crystallization of struvite- a urine pH of 7.2 or above and the presence of ammonia in the urine. A second mechanism by which bacterial infection may induce stone formation is by increasing crystal adherence. Urease producing bacteria hydrolyze urea to carbon dioxide and ammonium molecules. Two molecules of ammonia are produced from one molecule of urea; neutralization of the base is incomplete. As a result of this, the urinary pH rises.

Clinical presentation

Struvite calculus accounts are the majority of staghorn stones observed in most countries. They can grow quite large and may fill the collecting system. Struvite stones can form on a nidus of calcium oxalate stones and can grow quite rapidly. Most infection stones are radiopaque, but poorly mineralized matrix stones are faintly radiopaque or radiolucent. Women, perhaps because of their increased susceptibility to urinary tract infections, are more commonly affected than men. The presence of a foreign body in the urinary tract and neurogenic bladder are main causes for struvite calculi.

Patients with struvite stones may present acutely with fever, loin pain, dysuria, frequency, and hematuria. Metabolic abnormalities are present in patients with mixed calciumoxalate and struvite stones but not in patients with pure struvite stones.

CYSTINE STONES

They are uncommon Cystine stones account for about 1% of all urinary calculi in the United States and occur only in patients who have cystinuria. Cystinuria is an autosomal recessive disorder of transmembrane cystine transport manifested in the intestine and in the kidney. Cystine stones are radiopaque, although less so than calcium
oxalated stones. The radiopacity is the result of the disulfide bond in cystine. The stones are hexagonal, translusant white colour appear in acid urine and it is yellowish or pinkish color when first removed but greenish hue color when exposed to air and have a waxy appearance and very hard. They are often multiple, are large, and may form staghorns. Cystinuria can cause renal stones in childhood, but the peak of clinical expression is in the second and third decades. The cystine stones form because cystine is poorly soluble within the range of normal urinary pH.

**XANTHINE STONES**

These are extremely rare. They are smooth and round, brick—red in color and a radiolucent. Xanthinuria is an inborn error of metabolism, inherited as an autosomal recessive trait and characterized by a deficiency of xanthine oxidase. Serum uric acid levels are low, averaging less than 1.5 mg/dl. Serum and urine levels of xanthine and hypoxanthine are significantly increased. Xanthine stones develop because xanthine is less soluble than hypoxanthine.

**MATRIX CALCULI**

Matrix calculi are found predominantly in individuals with infections caused by urease-producing organisms. Proteus species are especially likely to be associated with matrix calculi. These stone composed of coagulated mucoids with little crystalline component. They are radiolucent and may be confused with uric acid calculi. Their association with alkaline urinary tract infection, however, usually assists in making a presumptive diagnosis because uric acid calculi are usually formed in acidic, sterile urine. Stones composed of β-2-microglobulin, a protein that is filtered and appears in the urine, may form in the kidneys of uremic patients.

**CLINICAL PRESENTATION**

**Acute stone episode**

A urinary calculus usually presents with an acute episode of renal or ureteral colic as the result of a stone obstructing the urinary tract. There
are five locations where stones can be impacted in the urinary tract. First, stones may become impacted in calyx of the upper urinary tract. Symptoms are abdominal distended and painful and create hematuria. The second area in which a calculus may become impacted is the ureteropelvic junction. A third area of impaction is at or near the pelvic brim, where the ureter begins to arch over the iliac vessels posteriorly into the true pelvis. The fourth area, especially in females, is the posterior pelvis, where the ureter is crossed anteriorly by the pelvic blood vessels and by the broad ligament; finally, the most constricted area through which the urinary calculus must pass is the ureterovesical junction, which is the most common site of impaction. Other impaction is urinary bladder and urethra.

**Renal colic**

The partially obstructing, continuously moving calculus appears to create the greatest amount of colic. As the stone moves to the midureter, pain generally tends to radiate to the lateral flank and abdominal area. When ureteral stones are near the bladder, patients often develop the symptoms of urinary frequency and urgency. Because of the autonomic nervous system transmits visceral pain. Nausea and vomiting are commonly associated with renal colic.

**Physical signs**

Pulse rate and blood pressure may be elevated because of pain and agitation. Examination of the abdominal reveals moderate deep tenderness on palpation over the location of the calculus and the area of the loin.

**INVESTIGATION**

**Blood**
- Calcium, Phosphate, Uric acid, Urea and Electrolytes
- Parathyroid hormone.

**Urine**
- Protein, Glucose, Blood, and Amino acid.
24-hr Urine examination
    Urea, Creatinine clearance, Sodium, Calcium, Oxalate, and Uric acid.

Urine culture

Stone analysis: Chemical composition

Plain abdominal radiograph

Intravenous Urogram

Computed Tomography

Ultrasonography

CT scan

Modern methods of stone removal for Kidney stones:

    Percutaneous nephrolithotomy, spiral CT, extracorporeal shock-wave lithotripsy. Open surgery for renal calculi are Pyelolithotomy, extended pyelolithotomy, nephrolithotomy.

Treatment for Ureter calculi:

    Endoscopic stone removal, ureteroscopic stone removal and lithotripsy in situ. Open surgery is ureterolithotomy.
MATERIALS AND METHODS

1. AIMS
   a) Primary Aim

   To estimate the efficacy of *Kalladaippu thool* in the treatment of *Kalladaippu*.

   b) Secondary Aim

   To find out the side–effects of the trial drug, if any.

2. POPULATION AND SAMPLE

   The population consists of all patients with *Kalladaippu* diagnosed by clinical symptoms and Ultrasonography satisfying the inclusion and exclusion criteria mentioned below. The sample consists of *Kalladaippu* patients attending the IPD/OPD of Ayothisoss Pandithar Hospital of the National Institute of Siddha, Chennai – 47.

3. SAMPLE SIZE

   The trial size will be 30 patients.

4. INCLUSION CRITERIA

   1. *Vatha, Pittha* and *Khaba kalladaippu*.

   2. Aged between 20 and 60 years.

   3. Willing to be admitted in the hospital for 8 weeks or willing to attend the O.P.D once a week for 8 weeks.

5. EXCLUSION CRITERIA

   1. Severe infection

   2. Kidney diseases

   3. Diabetes insipitus

   4. Pregnancy
6. WITHDRAWAL CRITERIA

1. Obstructive anuria
2. Severe haematuria
3. Severe pain

7. TRIAL DRUG AND DURATION

*Kalladaippu thool* (powder) 5 g with 50 ml of raddish juice, twice a day, for 8 weeks.

8. CLINICAL ASSESSMENT AND INVESTIGATION

a) CLINICAL ASSESSMENT

IN SIDDHA ASPECT

1. *Vatha Kalladaippu*: Retention of urine, lower abdominal pain with distention, pain with swelling of penis, agonizing pain, breathlessness and excretion of urine with mucus.

2. *Pittha Kalladaippu*: Stone blocked in urinary tract followed by oliguria, pain in urethra like an insertion of burning iron bar, sweating all over the body, anuria, agonizing pain, blood stained calculus excreted in urine with piercing and boring pain and hyperalgia.

3. *Khaba Kalladaippu*: Abdominal distention with pain like a bow, pain in all joints of upper and lower limbs, lumbar region, hip region and penis, sweating all over body and white colored stone excreted in urine.

b) INVESTIGATION

1. BLOOD
   TC (cells/cumm), DC (%), Hb (g %), ESR (mm/hr), Blood Sugar (R) (mg %), Blood Urea (mg %), Serum Creatinine (mg %) and Serum Cholesterol (mg%).

2. URINE

   Reaction (pH), Albumin, Sugar and Deposits.
3. MOTION

Ova, Cyst, and Occult blood.

4. ULTRA SONOGRAPHY

5. IN SIDDHA SYSTEM

Envagai thervukal: Naa, Niram, Mozhi, Vizhi, Malam, Moothiram (neerkuri and neikuri), Sparism and Naadi.

9. CONDUCT

Kalladaippu patients satisfying the inclusion and exclusion criteria will be admitted to the trial. Informed consent will be obtained from the patients. Lab investigation will be carried out before treatment and at the end of the treatment.

For the I.P. patients trial drug will be administered by the doctor and clinical assessment will be done weekly. The trial drug will be issued to the O.P. patients for 7 days at a time. Patients will be asked to come for clinical assessment once in 7 days. Also, they will be asked to bring back unconsumed drug at each visit and return them.

11. FORMS

Form I: Selection proforma - Used before admission.

Form II: Assessment proforma – Used once in a week during treatment.

12. ANALYSIS

Paired $X^2$-test for changes in signs and symptoms.

Paired t-test for changes in objectives parameters.
OBSERVATION AND RESULTS

30 cases of kalladaippu were treated with kalladaippu thool 5gms with 50ml raddish juice, two times for 8 weeks in NIS. The observation are tabulated, the patient who had positive ultrasonography, positive urine examination finding of crystals, R B C, albumin and puscells were included in this group. Tables were formed with reference of age, sex, diet history and personal habits seasonal incidence, distribution of land, clinical laboratory investigation and diagnosis.

DISTRIBUTION OF CASES BY GENDER

TABLE: 1

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</table>

Distribution of cases by gender
### DISTRIBUTION OF CASES BY AGE

**TABLE: 2**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td>21 to 30</td>
<td>5</td>
</tr>
<tr>
<td>31 to 40</td>
<td>11</td>
</tr>
<tr>
<td>41 to 50</td>
<td>6</td>
</tr>
<tr>
<td>51 to 60</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
</tr>
</tbody>
</table>

**Distribution of cases by age**

![Bar chart showing the distribution of cases by age]
### DISTRIBUTION OF CASES BY AGE AND SEX

**TABLE: 3**

<table>
<thead>
<tr>
<th>Age(yr)</th>
<th>Cases</th>
<th>Male No</th>
<th>Female No</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>20 to 30</td>
<td>3</td>
<td>10.0</td>
<td>2</td>
<td>5</td>
<td>16.6</td>
</tr>
<tr>
<td>31 to 40</td>
<td>9</td>
<td>30.0</td>
<td>2</td>
<td>11</td>
<td>36.6</td>
</tr>
<tr>
<td>41 to 50</td>
<td>6</td>
<td>20.0</td>
<td>0</td>
<td>6</td>
<td>20.0</td>
</tr>
<tr>
<td>51 to 60</td>
<td>3</td>
<td>10.0</td>
<td>5</td>
<td>8</td>
<td>26.6</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>70.0</td>
<td>9</td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

**Age and sex distribution**

![Age and sex distribution chart](chart.png)
### DISTRIBUTION OF CASES BY FOOD HABITS

**TABLE: 4**

<table>
<thead>
<tr>
<th>Food habits</th>
<th>Cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetarian</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>Non vegetarian</td>
<td>26</td>
<td>86.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

---

**Distribution among Food habit**

![Bar chart showing distribution among food habits](image)
## DISTRIBUTION OF CASES BY SEASONAL

### TABLE: 5

<table>
<thead>
<tr>
<th>Paruvakalam</th>
<th>Cases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Elavenil kalam</td>
<td>10</td>
<td>33.3</td>
</tr>
<tr>
<td>Muthuvenil kalam</td>
<td>10</td>
<td>33.3</td>
</tr>
<tr>
<td>Kaar kalam</td>
<td>8</td>
<td>26.6</td>
</tr>
<tr>
<td>Koothir kalam</td>
<td>2</td>
<td>6.6</td>
</tr>
<tr>
<td>Munpani kalam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pinpani kalam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

**Distribution among paruva kalam**

![Bar chart showing distribution among paruva kalam]

- Elavenil kalam: 33.3%
- Muthuvenil kalam: 33.3%
- Kaar kalam: 26.6%
- Koothir kalam: 6.6%
- Munpani kalam: 0%
- Pinpani kalam: 0%
DISTRIBUTION OF CASES BY DURATION OF DISEASE

TABLE: 6

<table>
<thead>
<tr>
<th>Duration in months</th>
<th>Cases</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 3</td>
<td>19</td>
<td></td>
<td>63.3</td>
</tr>
<tr>
<td>3 to 6</td>
<td>7</td>
<td></td>
<td>23.3</td>
</tr>
<tr>
<td>6 to 9</td>
<td>2</td>
<td></td>
<td>6.6</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td></td>
<td>100.0</td>
</tr>
</tbody>
</table>

Distribution among duration of illness

![Bar chart showing the distribution of cases by duration of disease]
**DISTRIBUTION OF CASES BY THINAI**

**TABLE: 7**

<table>
<thead>
<tr>
<th>Thinai (Land)</th>
<th>Cases</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kurinchi (Hill)</td>
<td>4</td>
<td>13.3</td>
<td></td>
</tr>
<tr>
<td>Mullai (Forest)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Marutham (Fertile)</td>
<td>1</td>
<td>3.3</td>
<td></td>
</tr>
<tr>
<td>Neithal (Costal)</td>
<td>25</td>
<td>83.3</td>
<td></td>
</tr>
<tr>
<td>Paalai (Desert)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
<td></td>
</tr>
</tbody>
</table>

**Distribution among Thinai**

![Graph showing distribution among Thinai](image-url)
**DISTRIBUTION OF CASES BY YAKKAI**

**TABLE: 8**

<table>
<thead>
<tr>
<th>Yakkai</th>
<th>Cases</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valiazhal</td>
<td></td>
<td>24</td>
<td>80.0</td>
</tr>
<tr>
<td>Azhalvali</td>
<td></td>
<td>6</td>
<td>20.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

**Distribution of cases by naadi**

![Graph showing distribution of cases by naadi]

- Valiazhal
- Azhalvali
### DISTRIBUTION OF CASES BY CLINICAL FEATURES

**TABLE: 9**

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Clinical features</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Abdominal pain</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>Abdominal distention</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Dysuria</td>
<td>18</td>
</tr>
<tr>
<td>4</td>
<td>Oliguria</td>
<td>16</td>
</tr>
<tr>
<td>5</td>
<td>Yellowish urination</td>
<td>18</td>
</tr>
<tr>
<td>6</td>
<td>Haematuria</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>Pyuria</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>Nausea</td>
<td>9</td>
</tr>
<tr>
<td>9</td>
<td>Vomiting</td>
<td>2</td>
</tr>
</tbody>
</table>

![Bar chart showing the distribution of cases by clinical features]
### DISTRIBUTION OF CASES BY NAADI

**TABLE: 10**

<table>
<thead>
<tr>
<th>Naadi</th>
<th>Cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vali azhal</td>
<td>24</td>
<td>80.0</td>
</tr>
<tr>
<td>Vali iyyam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Azhal vali</td>
<td>6</td>
<td>20.0</td>
</tr>
<tr>
<td>Azhal iyyam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Iyya vali</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Iyua Azhal</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

![Bar chart showing distribution of cases by Naadi]
### DISTRIBUTION OF CASES BY NEERKURI

**TABLE: 11**

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Neerkuri</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>1</td>
<td>Niram Yellowish</td>
<td>22</td>
</tr>
<tr>
<td>2</td>
<td>Edai Oliguria</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>Manam Tamarind</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>Nurai</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>Enjal</td>
<td>11</td>
</tr>
</tbody>
</table>

**Distribution of cases among neerkuri**

![Bar chart showing distribution of cases among neerkuri]
## DISTRIBUTION OF CASES BY NEERKURI

### TABLE: 12

<table>
<thead>
<tr>
<th>Neikuri</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Vali</td>
<td>0</td>
</tr>
<tr>
<td>Azhal</td>
<td>0</td>
</tr>
<tr>
<td>Iyyam</td>
<td>3</td>
</tr>
<tr>
<td>Thontham</td>
<td>0</td>
</tr>
<tr>
<td>Other Slowly spread</td>
<td>27</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
</tr>
</tbody>
</table>

### Distribution of cases among neikuri

![Distribution of cases among neikuri](image-url)
DISTRIBUTION OF CALCULI IN URINARY SYSTEM

TABLE: 13

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Site</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Both kidneys</td>
<td>11</td>
<td>36.6</td>
</tr>
<tr>
<td>2</td>
<td>Right kidney</td>
<td>9</td>
<td>30.0</td>
</tr>
<tr>
<td>3</td>
<td>Left kidney</td>
<td>9</td>
<td>30.0</td>
</tr>
<tr>
<td>4</td>
<td>Right ureter</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>5</td>
<td>Left ureter</td>
<td>2</td>
<td>6.6</td>
</tr>
</tbody>
</table>

Distribution of cases among site of calculus

![Bar chart showing distribution of calcui among different sites]
RESULT OF CLINICAL FEATURES AFTER TREATMENT

TABLE: 14

<table>
<thead>
<tr>
<th>RESULT</th>
<th>NO. CASES</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOOD IMPROVEMENT</td>
<td>28</td>
<td>93.3</td>
</tr>
<tr>
<td>MODERATE IMPROVEMENT</td>
<td>2</td>
<td>6.6</td>
</tr>
</tbody>
</table>

RESULT AMONG CASES

![Diagram showing result among cases with bars for good and moderate improvement]
### REPORTS OF URINARY INVESTIGATION

**Table: 15**

<table>
<thead>
<tr>
<th>S. NO</th>
<th>OP.NO</th>
<th>URINE INVESTIGATION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td><strong>BEFORE TREATMENT</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>ALBUMIN</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>PUS CELLS</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>ALBUMIN</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>PUS CELLS</strong></td>
</tr>
<tr>
<td>1</td>
<td>R. 8855</td>
<td>Nil</td>
</tr>
<tr>
<td>2</td>
<td>S. 359</td>
<td>Nil</td>
</tr>
<tr>
<td>3</td>
<td>S. 511</td>
<td>4 to 5</td>
</tr>
<tr>
<td>4</td>
<td>S. 2319</td>
<td>Nil</td>
</tr>
<tr>
<td>5</td>
<td>S. 3568</td>
<td>Nil</td>
</tr>
<tr>
<td>6</td>
<td>S. 4030</td>
<td>Trace</td>
</tr>
<tr>
<td>7</td>
<td>S. 4356</td>
<td>4 to 6</td>
</tr>
<tr>
<td>8</td>
<td>S. 5492</td>
<td>Nil</td>
</tr>
<tr>
<td>9</td>
<td>S. 8457</td>
<td>2 to 4</td>
</tr>
<tr>
<td>10</td>
<td>S. 9707</td>
<td>1 to 2</td>
</tr>
<tr>
<td>11</td>
<td>T. 101</td>
<td>Trace</td>
</tr>
<tr>
<td>12</td>
<td>T. 1201</td>
<td>2 to 4</td>
</tr>
<tr>
<td>13</td>
<td>T. 1362</td>
<td>2 to 4</td>
</tr>
<tr>
<td>14</td>
<td>T. 3053</td>
<td>2 to 3</td>
</tr>
<tr>
<td>15</td>
<td>T. 3220</td>
<td>1 to 2</td>
</tr>
<tr>
<td>16</td>
<td>T. 5894</td>
<td>2 to 4</td>
</tr>
<tr>
<td>17</td>
<td>T. 6924</td>
<td>3 to 4</td>
</tr>
<tr>
<td>18</td>
<td>T. 8061</td>
<td>2 to 4</td>
</tr>
<tr>
<td>19</td>
<td>T. 8379</td>
<td>Trace</td>
</tr>
<tr>
<td>20</td>
<td>T. 8456</td>
<td>Nil</td>
</tr>
<tr>
<td>21</td>
<td>T. 9592</td>
<td>2 to 4</td>
</tr>
<tr>
<td>22</td>
<td>U. 1119</td>
<td>Trace</td>
</tr>
<tr>
<td>23</td>
<td>U. 1656</td>
<td>3 to 3</td>
</tr>
<tr>
<td>24</td>
<td>U. 4075</td>
<td>Trace</td>
</tr>
<tr>
<td>25</td>
<td>U. 5278</td>
<td>3 to 4</td>
</tr>
<tr>
<td>26</td>
<td>U. 7537</td>
<td>2 to 4</td>
</tr>
<tr>
<td>27</td>
<td>U. 9107</td>
<td>3 to 4</td>
</tr>
<tr>
<td>28</td>
<td>V. 2027</td>
<td>2 to 3</td>
</tr>
<tr>
<td>29</td>
<td>V. 5386</td>
<td>2 to 4</td>
</tr>
<tr>
<td>30</td>
<td>V. 6251</td>
<td>Nil</td>
</tr>
</tbody>
</table>

Out of 30 patients of urine examination 6 patients had albumin, 11 patients had crystals and 2 patients had pus cells in urine before treatment.

After treatment 100% patients were normal report.
# ULTRASONOGRAPHY REPORTS IN BEFORE AND AFTER TREATMENT

<table>
<thead>
<tr>
<th>S. No</th>
<th>O P No</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Before treatment</th>
<th>After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>R. 8855</td>
<td>Mr. Kumar</td>
<td>41</td>
<td>M</td>
<td>3×3 mm and 4×4 mm Sized calculi seen in mid calyx of right kidney and left kidney.</td>
<td>Normal report.</td>
</tr>
<tr>
<td>2</td>
<td>S.511</td>
<td>Mr. Anandan</td>
<td>30</td>
<td>M</td>
<td>5mm and 7mm sized calculi seen in upper and lower pole of Rt. kidney. 7mm size calculus seen in upper pole of Lf. Kidney, 7mm size in Rt. V. U.J</td>
<td>3mm size calculus seen in mid pole of Rt. kidney. 2mm size calculus seen in upper pole of Lt. kidney. Stone removed in urine.</td>
</tr>
<tr>
<td>3</td>
<td>S. 2319</td>
<td>Mr. Rajan</td>
<td>29</td>
<td>M</td>
<td>Bilateral renal small calculi</td>
<td>Normal report.</td>
</tr>
<tr>
<td>4</td>
<td>S.3568</td>
<td>Mr. Chandrasekar</td>
<td>45</td>
<td>M</td>
<td>4.4 mm and 4.7mm sized calculi seen in lower calyx and mid calyx of Lt. kidney with Calyceal system dilated.</td>
<td>Normal study.</td>
</tr>
<tr>
<td>5</td>
<td>S. 4030</td>
<td>Mrs. Atchammal</td>
<td>54</td>
<td>F</td>
<td>A calculus 8mm size seen in Rt. V.U junction with pelvic Calyceal system and Ureter dilated.</td>
<td>Normal study.</td>
</tr>
<tr>
<td>6</td>
<td>S. 5492</td>
<td>Mr. Dhanasekar</td>
<td>35</td>
<td>M</td>
<td>9mm size calculus seen in collecting system of Lt. kidney.</td>
<td>4mm size calculus seen in Lt. kidney.</td>
</tr>
<tr>
<td>7</td>
<td>S.8457</td>
<td>Mr. Sathish Kumar</td>
<td>24</td>
<td>M</td>
<td>5mm and 5mm sized calculi seen in upper calyx of Rt. kidney and Lt. kidney.</td>
<td>Normal study.</td>
</tr>
<tr>
<td>8</td>
<td>S. 9707</td>
<td>Mr. Richard</td>
<td>50</td>
<td>M</td>
<td>5mm and 4mm sized calculi seen in mid calyx of Rt. kidney. 5mm size calculus seen in lower calyx of Lf. Kidney.</td>
<td>4mm size calculus seen in mid calyx of Rt. kidney. 5mm size calculus seen in lower calyx of Lt. kidney.</td>
</tr>
<tr>
<td>9</td>
<td>T. 101</td>
<td>Mrs. Pattammal</td>
<td>60</td>
<td>F</td>
<td>Multiple calculi seen in mid pole of Rt. kidney, largest size 8mm. 1.3 mm size calculus seen in Rt. proximal Ureter junction with hydronehrosis.</td>
<td>Ureter stone moved to bladder.</td>
</tr>
<tr>
<td>10</td>
<td>T.1201</td>
<td>Mr. Brimanaidu</td>
<td>41</td>
<td>M</td>
<td>5mm size calculus seen in lower pole of Lt. kidney.</td>
<td>3.2 sizes calculus seen in lower pole of Lt. kidney</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>T.1362</td>
<td>Mr. Velraja</td>
<td>60/M</td>
<td>5mm size calculus seen in Rt. kidney</td>
<td>4mm size calculus seen in Rt. kidney</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>T.3053</td>
<td>Mr. Rashid</td>
<td>39/M</td>
<td>8mm size calculus seen in lower calyx of Rt. Kidney</td>
<td>Normal study</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>T.5894</td>
<td>Mrs. Epsi</td>
<td>58/F</td>
<td>A clump of calculi in size of 2.3 cm seen in pelvis of Rt. kidney</td>
<td>2.5 cm size calculi seen in Rt. kidney</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>T.8061</td>
<td>Mr. Unnikrishnan</td>
<td>35/M</td>
<td>Two calculi size of 5mm and 8mm are seen in Rt. mid calyx. 4mm size calculus seen in Rt. upper ureter.10 mm size calculus seen in Rt. V. U junction with Hydronephrosis 7mm calculus seen in Rt. mid calyx.</td>
<td>Two calculi size of 5mm and 6mm seen in Rt. mid calyx. 4mm size calculus seen in Rt. upper calyx. 6mm size calculus seen in Lt mid calyx. No Hydronephrosis, Ureter appears normal.</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>T.8379</td>
<td>Mr. Lourthu mary</td>
<td>60/F</td>
<td>Two calculi size of 8mm and 9mm seen in left lower calyx.</td>
<td>Normal study</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>T.8456</td>
<td>Mrs.Patchaiammal</td>
<td>56/F</td>
<td>8mm size calculus seen in Rt. kidney. 7 to 8mm size calculus seen in Lt kidney.</td>
<td>4mm and 5mm size calculi seen in Rt. and Lt kidney.</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>U. 7537</td>
<td>Mr. Sathesh kumar</td>
<td>25/M</td>
<td>5mm and 6mm size calculi seen in Rt. and Lt kidney</td>
<td>Normal study</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>U. 9107</td>
<td>Mrs. Mushina sheri</td>
<td>25/F</td>
<td>A calculus size of 5mm seen in Rt. mid calyx.</td>
<td>Normal study</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>V. 6251</td>
<td>Mr. Durai raj</td>
<td>23/M</td>
<td>1.5 cm size calculus seen in Lt lower calyx</td>
<td>9mm size calculus seen in Lt lower calyx</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>ID</td>
<td>Name</td>
<td>Age</td>
<td>Gender</td>
<td>Calculi Details</td>
<td>Urololgical Status</td>
</tr>
<tr>
<td>----</td>
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<td>--------------------------------------------------------------------------------</td>
<td>----------------------------</td>
</tr>
<tr>
<td>1</td>
<td>S. 359</td>
<td>Mr. Ranganathan</td>
<td>35/M</td>
<td></td>
<td>5mm size calculus seen in right mid calyx. Left Ureter and Calyceal system dilated</td>
<td>Stone removed in urine on 8/12/06</td>
</tr>
<tr>
<td>2</td>
<td>U. 5278</td>
<td>Mr. Raja</td>
<td>42/M</td>
<td></td>
<td>8.5 and 6mm size calculi seen in upper calyx of Rt. kidney. 5mm and 6mm size calculi seen in mid and lower calyx of Lt. kidney. 10mm size calculus seen in Lt mid Ureter.</td>
<td>Stone removed in urine.</td>
</tr>
<tr>
<td>3</td>
<td>S.511</td>
<td>Mr. Anandan</td>
<td>30/M</td>
<td></td>
<td>5mm and 7mm sized calculi seen in upper and lower pole of Rt. kidney. 7mm size calculus seen in upper pole of Lt. Kidney. 7mm size in Rt. V. U. J</td>
<td>Stone removed in urine.</td>
</tr>
<tr>
<td>4</td>
<td>T. 101</td>
<td>Mrs. Pattamal</td>
<td>60/F</td>
<td></td>
<td>Multiple calculi seen in mid pole of Rt. kidney, largest size 8mm. 1.3 mm size calculus seen in Rt. proximal Ureter junction with hydroureter.</td>
<td>Stone removed in urine.</td>
</tr>
</tbody>
</table>

19 Patients are taken ultrasonography after treatment. Among 30 patients, in 9 patients calculi are totally reduced, in 9 patients calculi are reduced in size and number and one patient calculus not reduced. Among 30 patients, ureter stone removed in urine in 4 patients. 9 patients are not taken U S G after treatment.
DISCUSSION

Urinary calculus is known to mankind since antiquity. The earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 B.C. *Kalladaippu* is most common disease of the present society. In spite of new approach in diagnosis and management like Percutaneous nephrolithotomy, spiral CT, extracorporeal shock-wave lithotripsy but it still continues to cause significant morbidity with high tendency of recurrence. *Kalladaippu* commonly affected in middle and low class family, they do not do these advance procedure.

The contribution of yoogi in the field of regional and humoral pathology regarding the disease *kalladaippu* is highly commendable. Siddha medicine is ancient duration system of medicine for centuries have provided proven treatment of disease like *kalladaipu*. Therefore clinical trial was conducted on kalladaippu patients with *kalladaippu thool*, 30 patients of *kalladaippu* patients were selected.

1. DISTRIBUTION OF CASES BY GENDER

The gender distribution was 2.3: 1(M: F) in total of 30 patients, i.e. 21 males and 9 females because increased serum testosterone causes increased endogenous oxalate production in male, increased urinary citrate in women.

2. DISTRIBUTION OF CASES BY AGE

A total of 30 patients of varying age group were included in this study, the maximum distribution of *kalladaippu* occurs evenly in the 31 to 40, 41 to 50 and 51 to 60 years of age groups, 25 patients were in the age groups(e.g) 83.3% of the total number comes under the study. Several authors have reported 30 to 50 years of age group as the period of maximum incidence of *kalladaippu*, these periods are mainly in pittha period (33.4 yr to 66.4 yr) of human life as per the “Rathina surukkanadi”.

“அர்க்கிருந்திருந்திருந்த மட்டுமே புகழ்வட்டிடமாய் காணவும்”
- கான்நின்ற சார்மாந்தம் காண்கத்தா
3. DISTRIBUTION OF CASES AMONG FOOD HABITS

According to table 4, 26 patients were on non-vegetarian foods, these are induced vatham and pittham causes for *kalladaippu*. These food are having mainly protein and fat substances, it can induce humour of Vatham and pitham.

Animal protein increase urinary calcium, oxalates and uric acid and decrease urinary citrates excretion in urine due to protein contains sulphur contain amino acid, it increases endogenous acidosis.

Excessive in take of beverage likes coffee, tea, milk and alcohol are induces *kalladaippu* because of tea contain large amount of oxalates, coffee increase urinary calcium excretion, milk contains large amount of calcium and it has lactose potent stimulator of calcium absorption.

4. DISTRIBUTION OF CASES AMONG SEASON

While distribution of *kalladaippu* was highest in Elavenil, Muthuvenil and Mazhi kalaam, out of 30 cases 28 cases belong to these periods because of in Elavenil kalaam iyyam comes in normal, in Muthuvenil kalaam vatham increased and iyyam decreased, in Mazhi kalaam pitham increased above in normal level. Our literature mention as Vatham and pittham is one main cause for *kalladaippu*.

“எந்தெந்த மலைகளில் இருந்து அடுத்த சிற்றினங்கள் வெய்வு பார்க்க”

Price and associates found that the incidence of urinary calculi was higher during the summer months. High temperatures increase perspiration, which may result in concentrated urine. This promotes increased urinary crystallization.

5. DISTRIBUTION OF CASES AMONG NILAM

Out of 30 cases 25 belonged to neithal nilam because in our literature reveals this land is a place of aggravation of Vatham and also the
food substances obtained here containing salt. Minerals contents of water may contribute to cause stone disease.

These environmental factors, diet factors and personal habits leads to derangement of Vatham and pitham, these humours are main predisposing factors for \textit{kalladaippu}.

6. DISTRIBUTION OF CASES AMONG NEERKURI AND NEIKURI

On examination by envagai thervugal, the moothiram had been affected in all patients due to derangement of pitha kutram and abana vayu causing abdominal pain, yellowish urination, dysuria, Oliguria, nausea and vomiting.

7. DISTRIBUTION OF CASES AMONG CLINICAL FEATURES

As per the above data 100% of patients were affected abdominal pain and above 50% of patients were affected by yellowish urination, dysuria and Oliguria, after treatment abdomen pain relieved in 93.3% of patients, where other symptoms were relieved in 100% of patients.

The presence of albumin, crystals and pus cells come down to normal under treatment with trial drug in 100% of cases.

8. ULTRASONOGRAPHY REPORT

Out of 30 cases 19 patients were taken ultrasonography, after treatment with trial drug with in 8 weeks of periods, stone completely dissolved in 9 patients, ureter stone removed in urine in 4 patients and stone reduced in size and numbers in 9 patients. Other 9 patient’s clinical symptoms were totally reduced.

9. BIOCHEMICAL STUDY

\textbf{Potassium:}

The trial drug having potassium salts, these are good diuretics and reduce urinary excretion of calcium and kidney stone recurrence.

Potassium citrate prevents upper urinary tract calculi this proved by Lee YH, HuangWc and Tsai JY in J. urology.1999 may: 161.
Citrates:
The drugs having Citrates is most important complexor of calcium in urine and reduce ionic calcium concentration, sodium citrate salts mixed with alkaline salt of trial drug causes metabolic alkalosis decrease mitochondrial uptake of citrate and increase urinary citrate excretion that prevention of recurrent calcium oxalate stones.

Magnesium:
Magnesium salts produce more favorable Magnesium to calcium ratio in urine in condition that offers relative protection against stone formation. Magnesium decrease renal tubular citrate resorption through the chelatoin of citrate and this increase urinary citrate excretion in urine. It salts lowered the relative saturation urine with calcium oxalate or brushite.

Phosphate:
Phosphates were used in the hope of binding dietary calcium in the intestinal thus reducing its absorption. Urinary phosphorus excretion is markedly increased during therapy with alkaline phosphate which results in an increasing in the inhibitor activity of calculus in urine perhaps because of increased renal excretion of pyrophosphate and citrate.

Zinc:

10. PHARMACOLOGICAL STUDY:

Test drug *kalladaippu thool* (1000mg/kg/p.o) was given in water the diuretic activity was not significant at the end of 5 and 10 hrs when compared to urea (1000mg/kg/p.o).

However an enhanced diuresis was observed when test drug *kalladaippu thool* was along with radish juice with a Lipschits value more then 1 at the end of 5 and 10 hrs of examination.
11. ACUTE ORAL TOXICITY STUDY:

_Kalladaippu thool_ at the dose of 2000 mg/kg/po did not exhibit any mortality in rats. As per the OECD 423 guideline the dose is said to be Unclassified under the toxicity scale.

12. TREATMENT:

All ingredients of trial drug are having diuretic and lithotriptic effects, they were prepared as per the formula seen in the Siddha literature.

13. A Patients were advised to take plenty of water and asked to follow strict diet restriction during and after treatment to prevent the recurrence of stones.

14. The clinical study was hope encouraging. This is only preliminary approach and study may be undertaken with the same drug in large number of patients, which may prove to be more effective in treatment.
SUMMARY

- The clinical trial was conducted in 30 patients of renal calculus with trial drug of *kalladaippu thool* in dose of 5gms twice a day with adjuvant of 50ml raddish juice.

- Clinical diagnosis of *kalladaippu* was done in the basis of clinical features described in our text book.

- Urine and haematological examination and ultrasonography were done before and after treatment of trial drug.

- The various Siddha aspect examinations were carried out and recoded.

- *Kalladaippu* in Siddha aspect was correlated with urinary calculus in modern aspect.

- In biochemical study the trial drug had magnesium, potassium, citrates and tannin. Which are confirmed the clinical prognosis in renal calculus.

- In pharmacological study showed that the trial drug had well diuretic action without adverse effect in dose of one gram per kg.

- The medicine chooses for clinical treatment and management of *kalladaippu* by *kalladaippu thool*.

- Observation of the trial drug in the clinical study was clinically effective.
CONCLUSION

- The clinical trial of *kalladaippu thool* was conducted in 30 patients for 8 weeks of period.

- In the study 93.3 % of patients were clinically good improvement and 6.6% were moderate in improvement with trial drug. There were no clinically significant adverse reactions noted with trial drug.

- In Siddha and modern aspect of urine investigation in all patients after treatment came down to normal.

- Out of 30 patients, 19 patients were taken ultrasonography after treatment, among 19 patients, stone dissolved completely in 47.3 % (9) and reduced size and number in 47.3 % (9) of patients. Among 30 patients ureter stone removed in 13.3 % (4) with short period of treatment.

- In biochemical study the drug has potassium, magnesium, citrates and tannin were confirmed. This helps in clinical prognosis of renal calculus.

- The diuretic action of the trial drug had very good efficacy with anubanam in animal study and without toxic effect.

- Expenditure of the trial drug is low in cost and easily preparable but highly efficacy in *kalladaippu*.

- The clinical study was encouraging to do the deep study in large sample of kalladaippu patients with trial drug with proper diagnosis.
PREPARATION AND PROPERTIES OF TRIAL DRUG

KALLADAIPPUTHOOL (கல்லாட்டைப்புத்தூல்)

INGREDIENTS

1. அச்சரண்டு (Ash of Achyranthus aspera) -2 palam (70gms)
2. பூமல் (Ash of Plantain barks) -2 palam (70gms)
3. மூலன் (Ash of Palm flowering stalks) -2 palam (70gms)
4. கொரிலியில் (Ash of tried Brinjal’s plants) -2 palam (70gms)
5. பலைத்துப்பு (Powder of Pale catechu) -2 palam (70gms)
6. பூர்இச்சோல் (Purified Borax powder) -2 palam (70gms)
7. ஆலுமனு (Purified Alumen) -2 palam (70gms)
8. பொத்துப்பு (Purified Rock salt) -2 palam(70gms)
9. பொட்டுசோல் (Purified Sodium carbonates) -2 palam(70gms)

METHOD OF PREPARATION

Above all ingredients were taken in to equally and made powder form by rock mortar.

DOSAGE: 5gms (1 to 1.5 varagan eadai), twice a day for 8 weeks.

ADJUVANT: 50ml of Raddish juice

MEDICINAL USES: Urinary calculus, anuria, burning micturition, urethral stricture and tumor in urinary tract.

- அதிபாசல் குறுக்கு பாய்ந்து இடுவது, குருடாக்கும் பதால், பாகம் 76.
- கம்பி பத்து குருடாக்க அருகிய நிலை
1.தாழ்ந்தயை (ACHYRANTHUS ASPERA)

Family: Amaranthaceae
Part of uses: Whole plants
Taste: Bitter, pungent and astringent,
Potency: Heat
Pirivu: Pungent
Action: Astringent, diuretic and alterative

மருத்துவக் குறிப்பிட்டு (Therapeutic effects)

“அழியர் தரும்பல் ஆனையர் கிளி பிரிவுகள் அகில மக்கள் கோரிக் குடும்பங்கள் பிரிவுகளில் பிரிவுகளின் மக்கள் தகவல் மேல்பின் பல்லும் வர்த்தகி வகையை அளிக்கும் மீத்தவர் தவறான நீள்வாய்ந்த மருத்துவம் விளைவு”

- (தரும்பல்)

Medicinal uses:

Decoction of whole plants is good diuretics in renal calculus.
It contains a large percentage of alkaline ash and it mainly contains potash salt. Decoction of whole plants is a good diuretic found efficacious in renal dropsies and general anasarca.

Ashes of plants with water, jaggery cures dropsies such as anansarca, ascites.

-Indian materia medica, by Dr. K.M. Nadkani.

It is useful in renal and vesical calculi, flatulence, colic, painful inflammation, dropsy, and vomiting.

- Indian medicinal plants, Volume-1. Vaidyaratnam, P.S. Varier’s.
2. AMYLUM (MUSA PARADISIACA)

Family: Mimosaceae
Part of uses: Fruit, leaves and stems
Taste: Astringent
Potency: Cold
Pirivu: Pungent
Action: Styptic, anti Vatha, diuretic, refrigerant and astringent.

Therapeutic effects

It cures ascites, burning micturition, haematuria and abdominal ulcer. Its salt is a diuretic and lithotriptic action, it prepared from plantain bark ashes. Plantain ash contains phosphoric anhydride, lime, alkalies, iron, and chlorine etc.

Ash of husk of ripe fruit contains carbonates of potash and soda, chloride of potassium, alkaline phosphates with alittle sulphate, lime, silica, earthy phosphates etc. Ashes produced by burning the plant contain potash salt and are therefore useful in acidity, heartburn and colic. It ashes is a potash salt uses for nephritis, uraemia and ascities.

-Indian materia medica By Dr.K.M.Nadkarni.

The ashes obtained by burning plant are anti scorbutic, anthelmintic, and stomachic, and are useful in hyperacidity, heartburning, colic, and verminosis. The inflorescence axis (stem) is very specific for renal and vesical calculi.

-Indian medicinal plants, Vaidyaratnam, P.S.Varier.
3. **BORASSES FLABELLIFER**

**Family:** Palmaeae  
**Part of uses:** Root, flowering stalk, juice, bark and fruits.  
**Taste:** Astringent  
**Potency:** Cold  
**Pirivu:** Sweet  
**Action:** Astringent, diuretic, demulcent, nutritive and refrigerant.

**Therapeutic effects**

"பாரை மார் பாதுகாப்பு, கருஞ்சம குழன்  
கிளிக்கும்கள் பிரிகள் பிரித்து பலவனால்  
பரிசம் குடிக்கவும் புகைகற்கமுகும் பாரை மின்னில்  
பலையுடன் காண்நாள் பலராகின்" - (அத.)

**Medicinal uses:**

It cures vayu gunnam, burning micturition, dental disease and chronic fever. Ashes of flowering stalk are diuretics. Salt prepared from ashes of flowering stalk diluted with water then filter it pure ash less diluted water exposure to sun light. It contains albuminoids gum and fat. Juice is diuretics, cooling, stimulant, and antiphlogestic.

It uses for dropsy and inflammatory condition. Ashes of flavoring stalk are useful in enlarged spleen.

- Indian materia medica  
  By. Dr. K.M. Nadkarni.

The ash obtained by burning the inflorescence is good antacid, antiperiodic, and useful in hyperacidity, heartburn, biliousfever, splenomegaly, hepatomegaly, and skin diseases.

- Indian medicinal plants, volume-1  
  Vaidyaratnam, P.S. Varier’s.
4. கத்திக்கு (SOLANUM MELONGENA)

Family: Solanaceae

Part of uses: All parts of plant

Taste: Bitter and astringent

Potency: Heat

Pirivu: Pungent

Action: Stimulant, hypnotic and expectorant.

இயற்கைக் குறிப்பிட்டு (Therapeutic effects)

“கத்திக்கு பிக்கும் கருவக்கு திக்குரின்
நிலைக் பிக்கும் கருவக்கு கருவக்கு பிக்கும்
பிக்கும் கருவக்கு பிக்கும் கருவக்கு
பிக்கும் கருவக்கு” - (அட)

Chemical constituents:


Ripe ones are carbonesanous and bilias. Burnt fruits are like in digestion, purgative, slightly ciliious and beneficial in phlegm, wind and obesity.

-Indian materia medica

Medicinal uses: It leaves used in diuretic drugs.
5. *कंकनकलकुंप (UNCARIA CAMBIR)*

Family: Rubiaceae

Part of uses: Stem

Taste: Bitter astringent

Potency: Heat

Pirivu: Pungent

Action: Astringent.

**Therapeutic effects**

"..."

- Indian materia medica by Dr. K.M. Nadkarni

**Medicinal uses:**

It cures diarrhea, anuria, oral ulcer, burned ulcer, blood diseases and increase concentration of semen.

It contains the active principle- Catechu- tannic acid 22 to 50% p.c, catechine 7 to 33 p.c., quercetin, a yellow – colouring principle, catechu-red, gambier- fluorescein, wax and oil etc.

- Indian materia medica by Dr. K.M. Nadkarni
5.  சொத்திகை (SODIUM BIBORATE)

It is composed of boric acid and soda.
Taste:   Sweet with astringent.
Potency: Heat
Pirivu:  Pungent
Action: Astringent, diuretic, antacid, local sedative, antiseptic and emmenagoge.

யாராஜ் சிலையாய் (Therapeutic effects)

“சொத்திகை சொய்ல் பெருமையாய் சொத்திகையாய்
பெருமையாய் சொன்னாய் பெருமையாய் சொத்திகையாய்
நீங்களால் பெருமையாய் சொய்ல் சொய்ல்
பெருமையார் கோப்பிலும் சொத்திகை
- Siddha materia medica

Purification:
It fried by heat, still water contents removal.

Medicinal uses:
It cures scabies, eight types of gunmam, giddiness, dysentery, urinary calculus, burning micturition, dental diseases, worm’s infection, snake poison and epilepsy.

Vengara parpam cures pitha diseases like burning micturition and kalladaippu.

Borax is given orally in dose varying from 10 to 30 grains in acidity of stomach, dyspepsia and intestinal organism. It commonly mixed in decoction for kalladaippu.
6. புருஷரா (ALUMEN)

Characters:

It is colorless, transparent crystals, with acid. Alum is a general name for a class of double sulphates containing aluminium and such metals as potassium, ammonium, iron, etc.

Taste: Sweetish astringent

Potency: Heat

Pirivu: Pungent

Action: Astringent, diuretic, haemostatic and anti spasmodic.

Therapeutic effects

"சேரம்புறும் காயம் சுற்றி பாதாரியன சுருக்கம்காணாம் காட்டிக்காரம் காலைவிளங்கு பாதாரியன் குளையாரிக்கு மட்டும் ஒருவராக காணக்கொண்ட சதுக்கம் சேர்மயத்தான பரங்கிறார்க் கூறு"

-Siddha materia medica

Purification:

It makes in to power form and mixed with water then boiled well up to solid consistence and tried with sun light.

Medicinal uses:

It cures gingivitis, fileriasis, eye diseases, heart diseases, Vatham, fever, peptic ulcer and tumor. Padikara parpam has been used for urinary calculus, urethral stricture, anuria and burning micturition.

-Siddha materia medica.

It is useful in leucorrhoea, haematuria, haemoptysis, menorrhagia, gastric and intestinal catarrh and other haemorrhages.

-Indian materia medica, by Dr. K.M. Nadkarni.
7. சோடியம் (SODIUM CHLORIDE IMPURA)

Character:

It is found in small white crystalline grains or transparent cubes. It is brownish white externally and white internally. It has a pure saline taste and burns with a yellow flame. Taste: Salt in taste.

Potency: Heat

Pirivu: Pungent,

Action: Diuretic, laxative, carminative and stomachic.

வைரோக்கம் (Therapeutic effects)

"அளவு கற்கலை மற்றும் கிரேடுகைகளுடன் குழிப்ப்புக்களுக்கு வருபட்டும் விலையும் விளையுடன் காரின்கள் கண்டுபிடிப்பை சுட்டையும் மூடாமல் கிரேடுகை குழிப்ப்ப்பு விளையை பாதுகாப்பாம்

-Siddha materia medica

Purification:

It made in to powder form, mixed with anna kaadineer, kept in to the sun light for three Dayes, then washed with fresh water and tried with sun light.

Medicinal uses:

It cures eight types of gunmam, dyspepsia, kaba pitham, tumour, three thosam, constipation, poison, sukila noi and eczema.

In small dose it is highly carminative, stomachic and digestive. It possesses stronger purgative property. -Indian materia medica

By. Dr. K.M. Nadkarni.
8. **Sodium Carbonate** (SODIUM CABONATE)

Medicinal uses: Sowkaram was prepared by fuller earth, pooneeru, potassium nitrates and lime.

**Therapeutic effects**

Fuller earth is one of the ingredients of sowkaram so it has same action.

Constituents: Fullar earth contains 25 p.c. of sodium carbonate with certain impurities such as organic matter, sulphate of soda, potash.

Action: It is antacid, alterative, and diuretic.

**Medicinal uses:**

It is useful in dyspepsia with vomiting, diarrhea, and flatulanse. It is efficient remedy in urinary diseases as uric acid, gravel and suppression of urine.

- Indian material medica by Dr. K.M. Nadkarni.
ADJUVANT:
RADDISH JUICE (RAPHANUS SATIVAS, Linn):

Family: Cruciferae
Part of uses: All parts of plant
Taste: Pungent
Potency: Cold
Pirivu: Pungent
Action: Diuretic, Laxative. Stimulant, Stomach and Aphrodisiac

 Tamil Text: (Therapeutic effects)

“ஏரத்து கவுப்பு மறையுமியிலும் குடர்ந்த கூடையால் ஏரத்து தம்பியை மண்டகள் இருந்து உண்டு 
இறவடையிலியில் பரித்தெதி காலனித்து புகழ்பண்டு 
நான்மின்னம் சிக்குத்து கார்”

-Siddha materia medica

Constituents:
Fresh vegetable contain 91.00 p.c of moisture.
Completely tried material contain etherextract-4.00p.c,
Albuminoids-18.00p.c, Solublecarbohydrates-52.66p.c,
Woody fibre- 9.34 p.c, and Ash-16.00 p.c.
Seeds and wood contain fixed oil and sulphureted volatile oil

Medicinal uses:
Root juice given for urinary complaints like dysurea and strangury.

-Indian materia medica
By.Dr. K.M.Nadkarni.

Root juice 34ml to 100ml is increased urination.
Root is contains rich in vitamin C and E it increase immunological and antibacterial activity of white blood cells by increasing their motility.
## QUALITATIVE ANALYSIS OF ACIDIC/BASIC RADICALS AND PHYTOCHEMICAL CONSTITUENTS IN TEST DRUGS

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Observation</th>
<th>Inference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Test for Calcium:</strong></td>
<td>2 ml of extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxide solution.</td>
<td>White precipitate is formed</td>
</tr>
<tr>
<td><strong>Test for Sulphate:</strong></td>
<td>2 ml of the extract is added to 5% barium chloride solution.</td>
<td>White precipitate is formed</td>
</tr>
<tr>
<td><strong>Test for Chloride:</strong></td>
<td>The extract is treated with Silver nitrate solution</td>
<td>White precipitate is formed</td>
</tr>
<tr>
<td><strong>Test for carbonate:</strong></td>
<td>The substance is treated with Conc. HCl.</td>
<td>Effervescence is formed</td>
</tr>
<tr>
<td><strong>Test for Starch:</strong></td>
<td>The extract is added with weak iodine solution</td>
<td>Blue colour is not formed</td>
</tr>
<tr>
<td><strong>Test for Iron (Ferric):</strong></td>
<td>The extract is treated with glacial acetic acid and potassium ferrocyanide</td>
<td>Blue colour is formed</td>
</tr>
<tr>
<td><strong>Test for Iron (Ferrous):</strong></td>
<td>The extract is treated with Conc. HNO₃ and ammonium thiocynate</td>
<td>No Blood red colour is formed</td>
</tr>
<tr>
<td><strong>Test for phosphate:</strong></td>
<td>The extract is treated with</td>
<td>Yellow precipitate is formed</td>
</tr>
<tr>
<td>Test for Tannic acid</td>
<td>The extract is treated with Ferric chloride</td>
<td>Blue black precipitate is formed</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Test for Unsaturation</td>
<td>1 ml of Potassium permanganate solution is added to the extract.</td>
<td>Does not get decolourised</td>
</tr>
<tr>
<td>Test for saponins:</td>
<td>Dilute extract + 1 ml of distilled water and shake well.</td>
<td>Froth formation</td>
</tr>
<tr>
<td>Test for sugars:</td>
<td><strong>Benedict method</strong>; 5 ml of Benedict solution heated gently then add 8 drops of diluted extract then heated in a boiling water bath.</td>
<td>No colour change</td>
</tr>
<tr>
<td>Molisch test;</td>
<td>Dilute extract + 2 drops of Molisch + 3 ml conc. H₂SO₄.</td>
<td>No Reddish violet zones appeared</td>
</tr>
<tr>
<td>Test for steroids:</td>
<td>Liberman Burchard test; Dilute extract + 2 ml acetic anhydride + conc. H₂SO₄.</td>
<td>No Formation of red colour</td>
</tr>
<tr>
<td>Test for amino acids:</td>
<td>Dilute extract + 2 ml of Ninhydrin’s soln.</td>
<td>Formation of violet colour</td>
</tr>
<tr>
<td>Test for proteins:</td>
<td>Biuret method; 1 ml of dilute</td>
<td>Formation of Violet</td>
</tr>
<tr>
<td>Test for Flavanoids</td>
<td>Dilute extract + 2 drops of conc. HCl and gently heated.</td>
<td>No formation of pink colour</td>
</tr>
<tr>
<td>---------------------</td>
<td>----------------------------------------------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Test for phenol;</td>
<td>Dilute extract + 2 drops of FeCl₃ soln.</td>
<td>Deep green colour is formed</td>
</tr>
<tr>
<td>Test for Tannins;</td>
<td>Dilute extract + 2 ml of 10% lead acetate adds.</td>
<td>White precipitate formed</td>
</tr>
<tr>
<td>Test for alkaloids;</td>
<td>Mayer’s method; 1 ml of dilute extract + 1 ml reagent.</td>
<td>No appearance of cream colour precipitate</td>
</tr>
<tr>
<td></td>
<td>Dragendorff’s method; 1 ml of dilute extract + 1 ml of reagent.</td>
<td>No appearance of orange colour precipitate</td>
</tr>
<tr>
<td>Citrate:</td>
<td>2 ml dilute extract add 0.5 ml of sulphuric acid, and 3 ml of KMNO₄ warm until the colour of KMNO₄ is discharged and add 10% of sodium nitro preside in 1 M of sulphamic acid make in to alkaline with strong ammonia.</td>
<td>Violet colour turns in to violet in blue.</td>
</tr>
<tr>
<td>Solution</td>
<td>Effect</td>
<td>Test Result</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-----------------------------------------------</td>
<td>----------------------------</td>
</tr>
<tr>
<td><strong>Magnesium:</strong></td>
<td>2ml dilute extract of sample + 1ml of diluted ammonia</td>
<td>White precipitate formed</td>
</tr>
<tr>
<td><strong>Ammonia:</strong></td>
<td>2ml dilute extract of sample add with 2M NAOH and 0.3ml of potassium tetraiodomercurate solution and allowed stand for 5 minutes.</td>
<td>Yellow colour developed</td>
</tr>
<tr>
<td><strong>Zinc:</strong></td>
<td>2ml dilute extract of sample add with 1.5 ml of 2 M Hcl, 5ml of distilled water and diluted sodium hydroxide.</td>
<td>White precipitate formed</td>
</tr>
</tbody>
</table>
PRECLINICAL PHARMACOLOGICAL AND
TOXICOLOGICAL STUDY OF KALLADAIPPU THOOL
FOR DIURETIC ACTIVITY IN RATS

Index

1.0 Materials and Methods
   1.1 Test drugs
   1.2 Preparation of drugs for dosing
   1.3 Drugs and Chemicals
   1.4 Experimental animals
   1.5 Acute oral toxicity study
   1.6 Diuretic activity

2.0 Results
   2.1 Preliminary phytochemical screening
   2.2 Acute oral toxicity study
   2.3 Diuretic activity

3.0 Discussion

4.0 Reference
1.0 MATERIALS AND METHODS

1.1 Test Drugs

The test drug used in the study was processed by the methods prescribed in standard text books of siddha medicines.

1.1 Kalladaippu thool

*Kalladaippu thool* was prepared by the method described in Anuboga vaidhya navaneetham 9th part, page no.72.

1.2 Preparation of drug for dosing

Drug used for the study was suspended each time with 1% (w/v) solution of sodium carboxy methyl cellulose before administration.

1.3 Drugs and chemicals

Fine chemicals used in these experiments were obtained from Sigma Chemicals Company, U.S.A. Other analytical grade chemicals were obtained from S.d. Fine Chemicals Ltd., Mumbai.

1.4 Experimental animals

Colony inbred wistar rats of either sex weighing 200 - 250 g were used for the pharmacological and toxicological studies. The animals were kept under standard conditions 12:12 (day/night cycles) at 22°C room temperature, in polypropylene cages. The animals were fed on standard pelleted diet (Hindustan Lever Pvt Ltd., Bangalore) and tap water *ad libitum*. The animals were housed for one week in polypropylene cages prior to the experiments to acclimatize to laboratory conditions. The experimental protocol was approved by the Institutional Animal Ethical Committee (IAEC).
1.5 Acute oral toxicity study

Acute oral toxicity was conducted as per the OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 animals of a single sex per step. Depending on the mortality and/or moribund status of the animals, on the average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. This procedure results in the use of a minimal number of animals while allowing for acceptable data based scientific conclusion.

The method uses defined doses (5, 50, 300, 2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which cause acute toxicity.

Wistar albino rats of either sex weighing 200-250 g were fasted overnight, but allowed water *ad libitum*. Since the formulation is relatively non toxic in clinical practice the highest dose of 2000 mg/kg/p.o (as per OECD guidelines “Unclassified”) was used in the acute toxicity study.

The animals were observed closely for behavioural toxicity, if any by using FOB (Functional observation battery).

1.6 Diuretic activity in rats (Lipschitz test)

A method for testing diuretic activity in rats has been described by Lipschitz *et al.*, 1943. The test is based on urine excretion in test animals and compared to rats treated with a high dose of urea. The “Lipschitz value” is the quotient between urine excretion by test animals and urine excretion by the urea control.

Male wistar rats weighing 150-200 g were used. Three animals per group are placed in metabolic cages provided with SS wire mesh bottom and funnel to collect the urine. SS sieves are placed on the funnel to retain
feces and allow the urine to pass. The animals were fed with standard pelleted diet and water *ad libitum*. 15 hrs prior to the experiment food and water were withdrawn. The animals were divided into 4 groups with 3 animals in each group and the following regimen of treatment was instituted.

- **Group I** Control (Radish juice) 5 ml/per animal
- **Group II** Urea (Standard) 1g/kg/p.o
- **Group III** Test drug in water 1000 mg/kg/p.o
- **Group IV** Test drug (1000 mg/kg/p.o) in radish juice (5ml/per animal)

For screening procedures 2 groups of three animals were used for the study. The test drug *Kalladaippu thool* and standard drug urea were administered orally at the doses of 1g/kg, respectively. Additionally 5 ml of 0.9% sodium chloride per 100 g body weight was given by gavage. Urine excretion was recorded after 5 and 10 hrs.

Urine volume excreted per 100 g body weight was calculated for each group. Results are expressed as “Lipschitz value” ie, the ratio of test/urea. Indices of 1.0 and more are regarded as a positive effect.
2.0 Results

2.1 Preliminary basic, acidic radicals and phytochemical studies

The qualitative chemical analysis and acidic, basic radical’s assay of the drugs showed the presence of phytoconstituents and minerals as depicted in (Table 1).

2.2 Acute oral toxicity study

*Kalladaippu thool* at the dose of 2000mg/kg/po did not exhibit any mortality in rats. As per OECD 423 guidelines the dose is said to be “Unclassified” under the toxicity scale. Hence further study with higher doses was not executed.

2.3 Diuretic study

In the diuretic study there was no significant diuretic activity was observed when radish juice alone is given at the end of 5 and 10 hrs observation. Where as urea (1 g/kg/p.o) showed significant diuretic activity at the end of 5 and 10 hrs of observation. When the test drug *Kalladaippu thool* (1000 mg/kg/p.o) was given in water the diuretic activity was not significant at the end of 5 and 10 hrs when compared to standard urea (1g/kg/p.o). However an enhanced diuresis was observed when the test drug *Kalladaippu thool* was given along with radish juice with a Lipschitz value more than 1 at the end of 5 and 10 hrs of observation. This study clearly establishes the enhanced diuretic activity of *Kalladaippu thool* when it was given with radish juice as “anupanam”. The animals received radish juice alone (5 ml/per animal) did not exhibit significant diuretic activity.
Discussion

The *Kalladaippu thool* is said to be clinically useful siddha formulation for diuresis. In the present study the acidic and basic radicals and phytochemical studies revealed the presence of K$^+$, Na$^+$, Ca$^{++}$, Fe$^{+++}$, Mg$^{++}$, Zn$^{++}$, Sulphate, Chloride, Carbonate, Phosphate, Citrate, Ammonium radicals and phytoconstituents like tannins, tannic acid, phenols etc.

In clinical practice the drug is given at the divided doses of 10 g/day in radish juice. The radish juice is used as “anupanam” for enhancing the absorption and bioavailability of the test drug. Administration of radish juice alone did not exhibit any significant diuretic activity but when co-administered with *Kalladaippu thool* exhibited significant diuretic activity. Similarly *Kalladaippu thool* administered in water did not exhibit significant diuretic activity when compared to standard drug urea.

The “Lipschilz value” of present study shows a $\pi/U$ ratio of more than one (1) which is considered to be a significant index to rate a drug as diuretic.

There is good correlation in diuretic activity of *Kalladaippu thool* in both clinical and experimental study when co-administered with radish juice. The presence of Na$^+$ and K$^+$ ions and other basic and acidic radicals present in the formulation may be responsible for the diuretic activity of *Kalladaippu thool*. The exact mechanism of diuretic action of *Kalladaippu thool* should be investigated further.
### Table

Diuretic activity of *Kalladaippu thool* in rats

<table>
<thead>
<tr>
<th>Groups</th>
<th>5 hrs</th>
<th>10 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (Radish)</td>
<td>0.7167±0.2927</td>
<td>2.017±0.4070&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Urea (std)</td>
<td>1.33±0.1751&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.333±0.3882&lt;sup&gt;a,c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Test drug (water)</td>
<td>1.23±0.28&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>1.367±0.1633&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Test drug (radish)</td>
<td>1.733±0.2066&lt;sup&gt;a,d,e&lt;/sup&gt;</td>
<td>3.550±0.2811&lt;sup&gt;a,d,e&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

n = 6, values are expressed as mean ± S.D followed by One way Anova using Turkey’s multiple comparison method.

P<0.001 was considered significant.

ap< 0.001, bp< 0.01, cp<0.001, dp>0.05, ep<0.001

a - all groups vs control
b,c - test (water) vs standard
d - test (radish) vs standard
e - test (water) vs test (radish)
NATIONAL INSTITUTE OF SIDDHA, 
CHENNAI 47

AN OPEN TRIAL OF KALLADAIPPU THOOL FOR THE TREATMENT OF KALLADAIPPU (URINARY CALCULUS)

FORM-I SELECTION PROFORMA


7. Occupation: ____________________

8. Address: __________________________________________

9. COMPLAINTS AND DURATION: __________________________

PAST HISTORY


12. Recurrent renal calculus: ☐ ☐

13. Prolonged immobilization: ☐ ☐

14. Kidney transplantation: ☐ ☐

15. Healed renal tuberculosis: ☐ ☐
### ENDOCRINE DISORDERS

<table>
<thead>
<tr>
<th>Question</th>
<th>1. Yes</th>
<th>2. No</th>
</tr>
</thead>
<tbody>
<tr>
<td>16. Hyper parathyroidism:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Cushing syndrome:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### CONGENITAL DISORDER HISTORY

<table>
<thead>
<tr>
<th>Question</th>
<th>1. Yes</th>
<th>2. No</th>
</tr>
</thead>
<tbody>
<tr>
<td>18. Medullary sponge kidney:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Myelo proliferative disorder:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### TREATMENT HISTORY

<table>
<thead>
<tr>
<th>Question</th>
<th>1. Yes</th>
<th>2. No</th>
</tr>
</thead>
<tbody>
<tr>
<td>20. Oral diuretics:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21. Non-steroid anti-inflammatory drugs:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. Surgery for urinary calculi:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Extracorporeal shock wave lithotripsy:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### FAMILY HISTORY

<table>
<thead>
<tr>
<th>Question</th>
<th>1. Yes</th>
<th>2. No</th>
</tr>
</thead>
<tbody>
<tr>
<td>24. Gout:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25. Cystinuria:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26. Xanthinuria:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### DIET HISTORY

#### Vegetarian foods

<table>
<thead>
<tr>
<th>Question</th>
<th>1. No</th>
<th>2. Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>27. High intake of dairy products:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28. Low calcium diet:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29. Nuts</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Non-vegetarian foods

<table>
<thead>
<tr>
<th>Question</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>30. Meat:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31. Poultry:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32. Fish:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Drinks
33. Alcohol:  [ ]  [ ]  [ ]
34. Tea:  [ ]  [ ]  [ ]
35. Coffee:  [ ]  [ ]  [ ]
35. Milk:  [ ]  [ ]  [ ]

GENERAL EXAMINATION
37. Weight (kg):  [ ]  [ ]  [ ]
38. Temperature (°F):  [ ]  [ ]  [ ]
39. Pulse rate / minute:  [ ]  [ ]  [ ]
40. Heart rate / minute:  [ ]  [ ]  [ ]
41. Respiratory rate / minute:  [ ]  [ ]  [ ]
42. Blood pressure (mmHg):  [ ] / [ ] [ ]
43. Anaemia:  [ ]  [ ]  [ ]
44. Jaundice:  [ ]  [ ]  [ ]
45. Cyanosis:  [ ]  [ ]  [ ]
46. Lymphadenopathy:  [ ]  [ ]  [ ]
47. Pedal oedema:  [ ]  [ ]  [ ]
48. Clubbing:  [ ]  [ ]  [ ]
49. Jugular vein pulsation:  [ ]  [ ]  [ ]

VITAL ORGANS EXAMINATION:
50. Heart  1. Normal  2. Affected
51. Lungs  1. Normal  2. Affected
53. Liver  

54. Kidney  

55. Spleen  

56. Stomach  

57. Urinary bladder  

**CLINICAL EXAMINATION**

**SIGNS AND SYMPTOMS**

58. Abdominal pain  

59. Severity:  
   0. NA  
   1. Mild  
   2. Moderate  
   3. Severe  

60. Sites:  
   0. NA  
   1. Loin  
   2. Renal angle  
   3. Lower abdomen  

61. Radiating areas:  
   0. NA  
   1. Groin  
   2. Thigh  
   3. External Genitalia  

62. Abdominal distention  

63. Dysuria  

64. Oliguria  

65. Yellowish urination  

66. Haematuria  

67. Pyuria  

68. Nausea  

69. Vomiting  

70. Fever  

71. Sweating
## SIDDHA SYSTEM OF EXAMINATION

### IYMPORI

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>72. Mei</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>73. Vaai</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>74. Kan</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>75. Mookku</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>76. Sevi</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
</tbody>
</table>

### KANMENTHIRIUM

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>77. Kai</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>78. Kaal</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>79. Vaai</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>80. Eruvai</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
<tr>
<td>81. Karuvaai</td>
<td>[ ]</td>
<td>[ ] ___________________</td>
</tr>
</tbody>
</table>

### PARUVA KAALA

1. Elavenil kalam [ ]
2. Muthuvenil kalam [ ]
3. Mazhai kalam [ ]
4. Kulir kalam [ ]
5. Munpani kalam [ ]
6. Pinpani kalam [ ]

### THINAI

1. Kurunji [ ]
2. Mullai [ ]
3. Marutham [ ]
4. Neithal [ ]
5. Palai [ ]

### YAKKAI

1. Vali [ ]
2. Azhal [ ]
3. Iyam [ ]
4. Valiazhali [ ]
5. Valaiyam [ ]
6. Azhalval [ ]

10. Mukkutram

85. **GUNAM**

1. Sathuva gunam  2. Rajo gunam

3. Tamo gunam

**UYIR THATHUKKAL VALI**

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>86. Pranan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>87. Abanan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>88. Samanan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>89. Udhanan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>90. Viyanan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>91. Nagan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>92. Koorman</td>
<td></td>
<td></td>
</tr>
<tr>
<td>93. Kirukaran</td>
<td></td>
<td></td>
</tr>
<tr>
<td>94. Devathathan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95. Tananjeyan</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**AZHAL**

<table>
<thead>
<tr>
<th></th>
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<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>96. Anala pittham</td>
<td></td>
<td></td>
</tr>
<tr>
<td>97. Prasaka pittham</td>
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<tr>
<td>98. Ranjaka pittham</td>
<td></td>
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</tr>
<tr>
<td>99. Aalosaka pittham</td>
<td></td>
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</tr>
<tr>
<td>100. Saathaka pittham</td>
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</tbody>
</table>
### IYAM

<table>
<thead>
<tr>
<th>No.</th>
<th>Area</th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>101</td>
<td>Avalambagam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>102</td>
<td>Kilethagam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>103</td>
<td>Pothagam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>104</td>
<td>Tharpagam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>105</td>
<td>Santhigam</td>
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### UDAL THATHUKKAL

<table>
<thead>
<tr>
<th>No.</th>
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<tbody>
<tr>
<td>106</td>
<td>Saaram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>107</td>
<td>Chenneer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>108</td>
<td>Oon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>109</td>
<td>Kozhuppu</td>
<td></td>
<td></td>
</tr>
<tr>
<td>110</td>
<td>Enbu</td>
<td></td>
<td></td>
</tr>
<tr>
<td>111</td>
<td>Moolai</td>
<td></td>
<td></td>
</tr>
<tr>
<td>112</td>
<td>Sukkilam / Suronitham</td>
<td></td>
<td></td>
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</tbody>
</table>

### ENVAGAI THERVUKAL

<table>
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<th>No.</th>
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<tr>
<td>113</td>
<td>Naa</td>
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<tr>
<td>114</td>
<td>Niram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>115</td>
<td>Mozhi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>116</td>
<td>Vizhi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>117</td>
<td>Niram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>118</td>
<td>Thanmai</td>
<td></td>
<td></td>
</tr>
<tr>
<td>119</td>
<td>Niram</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
120. Eadai  
1. No  
2. Yes  

121. Manam  

122. Nurai  

123. Enjal  

124. Neikuri:  
1. Vatham  
2. Pitham  
3. Khabam  
4. Kalappu kuttram  
5. Mukkuttram  

125. Sparisam:  
1. Mithaveppam  
2. Miguveppam  
3. Thatpam  

126. Naadi:  
1. Vatham  
2. Pitham  
3. Khabam  
4. Vathapittham  
5. Vathakhabam  
6. Pitthavatham  
7. Pitthakhabam  
8. Khabavatham  
9. Khabapittham  
10. Mukkuttram  

**INVESTIGATION**  
**BLOOD**  

127. TC (cells /cumm):  

128. P  
129. L  
130. E  
131. B  
132 M.  

133. Hb (gms %):  

134. ESR (mm/hr):  
1. 1/2hr  
2. 1hr  

135. 135.1hr  

136. Blood Sugar (R) (mg %):  

137. Blood Urea (mg %):  

138. Serum Creatinine (mg %):  

139. Serum Cholesterol (mg %):
URINE

140. Reaction:  1. Acid □  2. Alkaline □

141. Albumin:  0. Nil □  1. Trace □  2. + □  3. ++ □
              4. +++ □

142. Sugar:  0. Nil □  1. Trace □  2. + □  3. ++ □
              4. +++ □

143. Pus cells □ □

144. Epithelial cells □ □

145. RBC □ □

146. Crystals □ □

MOTION

147. Ova  1. Yes □  2. No □

148. Cyst □ □

149. Occult blood □ □

ULTRA SONOGRAPHY:

__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________

150. ADMITTED TO TRIAL:  1. Yes □  2. No □

If yes

152. S. No: □ □

153. I.P / O.P  1. I.P □  2. O.P □

154. Drug issued for OP patient (g): □ □

155. Station

156. Date:

157. Signature of Medical Officer
NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47

AN OPEN TRIAL OF KALLADAIPPU THOOL FOR THE TREATMENT OF KALLADAIPPU (URINARY CALCULUS)

FORM II- ASSESSMENT PROFORMA

1. O.P.No / I.P No: ________  2. Bed No: ________  3. S.No: 

4. Name: ____________________

5. Date of admission to the trial:

6. Date of assessment:

7. Week of assessment:

CLINICAL ASSESSMENT

SIGNS AND SYMPTOMS

8. Abdominal pain  1. No☐  2. Yes☐


10. Sites: 0. NA☐  1. Loin☐  2. Renal angle☐  3. Lower abdomen☐

11. Radiating areas: 0. NA☐  1. Groin☐  2. Thigh☐

   3. External Genitalia ☐
1. Yes                2. No

12. Abdominal distention
13. Dysuria
14. Oliguria
15. Yellowish urination
16. Haematuria
17. Pyuria
18. Nausea
19. Vomiting
20. Fever
21. Sweating

INVESTIGATIONS (only at the end of treatment)

BLOOD

22. TC (cells /cumm):


28. Hb (gms %):

ESR (mm/hr): 29. 1/2hr 30. 1hr

31. Blood Sugar (R) (mg %):

32. Blood Urea (mg %):

33. Serum Creatinine (mg %):
34. Serum Cholesterol (mg %):

URINE

35. Reaction:  1. Acid
               2. Alkaline

36. Albumin:  0. Nil
               1. Trace
               2. +
               3. ++
               4. +++

37. Sugar:  0. Nil
           1. Trace
           2. +
           3. ++
           4. +++

Deposit

1. Yes  2. No

38. Pus cells

39. Epithelial cells

40. RBC

41. Crystals

MOTION

1. Yes  2. No

48. Ova

49. Cyst

50. Occult blood

51. ULTRA SONOGRAPHY:

____________________________________________________________________
____________________________________________________________________
____________________________________________________________________
____________________________________________________________________
## INVESTIGATION IN SIDDHA SYSTEM

### NEERKURI

<table>
<thead>
<tr>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>42. Niram</td>
<td>□</td>
</tr>
<tr>
<td>43. Edai</td>
<td>□</td>
</tr>
<tr>
<td>44. Manam</td>
<td>□</td>
</tr>
<tr>
<td>45. Nurai</td>
<td>□</td>
</tr>
<tr>
<td>46. Enjal</td>
<td>□</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1. No</th>
<th>2. Yes</th>
</tr>
</thead>
</table>

| 4. Kalappu kuttram | □ | 5. Mukkuttram | □ |

52. Drugs returned (capsules):

53. Drugs issued (capsules):

54. Station: ____________

56. Signature of the Medical Officer

55. Date: ____________
CONSENT FORM

Certificate by Investigator

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

Date: _____________
Signature: ______________
Station: _____________
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 the 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 *Kalladaippu Thool* for *Kalladaippu*.

Date: _____________
Signature: ______________
Station: _____________
Name: _________________
Signature of witness: ______________
Date: _____________
Name: _________________
Station: _____________
Relationship: _______________
URINARY CALCULUS REMOVED IN URINE

O. P NO: U5278, Name: Mr. Raja

O. P. No: T101, Name: Mrs. Pattammal
O. P. No: S. 359, Name: Mr. Ranganathan

O. P. No: S. 511, Name: Mr. Anandan
ACHYRANTHUS ASPERA

ASH POWER OF ACHYRANTHUS ASPERA
MUSA PARADISIACA

ASH POWDER OF MUSA PARADISIACA
BORASSES FLABELLIFER

ASH POWDER OF BORASSES FLABELLIFER
SOLANUM MELONGENA

ASH POWDER OF SOLANUM MELONGENA
Sodium biborate (Before purification)

Sodium biborate (Purified)
ALUMEN

PURIFIED ALUMEN
PURIFIED SOWKARAM

KALLADAIPPU THOOL
BIBLIOGRAPHY

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2. Thirumoolar karukadai vaithiyam
3. Siddha maruthuvanga surukkam
4. Siddha maruthuva noi vilakkam
5. Thanvanthiri vaithiya kaviam
6. Agathiyar naadi
7. Rathinasurukkanaadi
8. Theriar gunavagadam
9. Anupogavaithiya navaneetham
10. Matchamuni 800
11. Gunapadam Mooligai
    - Dr K.S. Murugesa mudaliar
12. Gunapadam thathu
    - R. Thiyaragan
13. Yugi Vaidhiya chinthamani
14. Noi nadal Noi mudhal nadal thirattu, part -1 and part-2
    - Dr. M. Shanmugavelu
15. Siddha maruthuvam
    - Dr. K. N. Kuppusamy mudaliar
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34. Urology
   Dr. Campbell

35. Dorland’s illustrated medical dictionary.

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