A STUDY ON
“MANJAL KAMALAI”

Dissertation Submitted To
THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY
Chennai – 32

For the Partial fulfillment in Awarding the Degree of
DOCTOR OF MEDICINE (SIDDHA)
(Branch – IV, Kuzhanthai Maruthuvam)

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MARCH - 2009
INTRODUCTION

Siddha dates back to 2000 B.C originated in southern India. The word siddha is derived from “siddhi” meaning an object to be attained “perfection” (or) heavenly bliss”.

The principles of siddha system it is a holistic approach. This system is treated should be oriented not merely to disease but should also take into account the pt his Environment, sex, age, Habits, mental frame habit, diet and physical condition.

Medicine has drawn richly from traditional cultures. The traditional tamil system evolved with the development of mankind that are truly Indian in origin and the development are Siddha and Ayurvedha.

Siddha system is practiced in the tamil speaking areas of south India.

Siddha systems approaches and analyses the ailments on the basis of the “Tiridhosa theory of disease” The dhosas or humours and vatham, pitham, kabam.

The three humours are derived from “pancha bootha” by the combination of five bhoothas.

Siddha system is based upon “Food is medicine” medicine is food” and later on pancha boothic and Tridosha theory.

The Dosha’s or humours are vatha (wind) pitha (gall) and Kapha (mucus).

When these in perfect balance and harmony a person is said to be healthy.

Siddhar’s humoural pathology explains that all diseases are caused by the mixture of the three cardinal humours, vil, vadha, pitha, kabam.

The above three humours are present in the rat win 1: 1/2 : 1/4. It will disturb the normal condition and will result in disease.

Thiruvalluvar also mentioned the above fact in his thirukkural.

“இன்றைய வகையான வரலாறு வெர்பு வேதியாக வாஸ் வரணாக அணிமை வரண்ட நாய்க”.

The three humours are nourished by their respective elements present in food.

“ஏரங்கள் பராமரிக்க பெரிய வேதியம் காந்து வெள்ளை இரண்டு நன்னை”. 
They are the fundamental principles of creation preservation and destructive in the universe. The creative force in the physical body is known as vatham, the protective force is pitham and the destructive one is kabam. The tridosha theory is important point to be considered in health and disease.

This tridhosha is causation of signs and symptoms is a great boom for Indian physicians for diagnosis and prognosis of disease.

Siddha’s narrated in detail the classification Aetiology, Pathology, Pathogenesis, complications treatment and diet to be followed.

Their aim is to have disease free physical and psychological make up to prevent illness and to bestow Immortality.

This illustrated in the verse.
One that cures physical ailment is medicine
One that cures psychological ailment is medicine
One that prevents ailment is medicine
One that bestow immortality is medicine
-Thirumantiram.

The clinical methods of siddha system through which the correct diagnosis is made out are called as “ENVAGAI THERVUGAL” they are

“பயில் பாத்ரை நீரியம் கெடளி விளை
மையம் குண்டுல்போனங் பாத்ததுக்கங்கும்.”
“Children’s health” Tomorrow’s wealth”

So the central and state government is giving importance to children for this growth and development and gives interest to preventive measures against disease.

To reduce the child mortality rate the concept of Siddha medicine will be very much developed to the concept of modern medicine for this aspect the author select the disease. MANJAL KAMALAI from Kuzhandhai Maruthuvam.
AIM AND OBJECTIVES

“Several tones of theory cannot equal a one of practice”

- Swami Sivanandha.

I have chosen the disease MANJAL KAMALAI for study because there is a unshakable. Truth among the people that this disease is curable only in our traditional medicine of Siddha system of medicine from 2000 BC to 2009.

India is a tropical country having larger population in the world. Where people of different socio-economic status and are found. The down trodden poor children who live in density populated areas with poor sanitary facilities lack of personal and environmental hygiene are the common victims of this disease.

If proper attention has not been given it may even leads to fatal condition. So immediate care should be taken as soon as the act break of this disease is noticed in children.

I have selected the disease MANJAL KAMALAI it is compared with modern medicine and its prognosis with our trial medicine

The medicine is easily prepared from our homes. Cheep and best and also safe.

OBJECTIVES

1. To study the MANJAL KAMALAI its etiology symptomatology, pathogenesis, prognosis and prevention.

2. To know how the disease alters the normal conditions of mukkutram, poripulangal.

3. To study the relationship of age, sex, race environment, socio economic status and familial incidence of the disease.

4. To analyse the incidence of this disease among the children of age group 1-12 yrs.

5. To study my trial medicine its action biochemical and pharmacological laboratories.

6. To make awareness about the prevention of the disease.

7. To have clinical trial medicine is children.

As a Siddha pediatrician had an extra personal interest in the study of a new drug for this common pediatric disease.
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கரித்து கற்பிள்ளை

According to Bala vagadam: Dr. Pon Gurusironmani
1. Oothu kamalai
2. Varal kamalai
3. Manjal kamalai

According to Yugi vaidha chinthamani

According to Agasthiyar 2000
Manjal Noi is classified into 8 kinds they are,

1. Vatha Kamalai
2. Pitha Kamalai
3. Kapha Kamalai
4. Vatha kapha Kamalai
5. Pitha kapha Kamalai
6. Mukkutra Kamalai
7. Manjal Kamalai
8. Azhagu Kamalai
9. Sengamala Kamalai
10. Kumba Kamalai
11. Gunma Kamalai
12. Oothu Kamalai
13. Varal Kamalai.

The symptoms of each type, which are similar to the symptoms that are explained by Yugi vaidhya chinthamani.
According to vaidhya sara sangiragam

Kamalai is classified into 5 types.
1. Varal kamalai
2. Vatha kamalai
3. Pitha kamalai
4. Aiyya kamalai
5. Manjal kamalai

According to Dhanvanthri vaidhyam – by Dr. S. Venkatarajan.

Kamalai classified into 5 types they are,
1. Vatha kamalai.
2. Pitha kamalai
3. Silathma kamalai
4. Mukkuttru kamalai
5. Kumba kamalai

Manjal kamalai is not described in Dhanvanthri

According to Roga nirmaya saram (Roga nithanam) by Dr. Mahadeva pandithar.

Kamalai classified into 13 types, as described in Yugi Vaidhya Chinthamani.

Signs and symptoms of Manjal kamalai

According to Bala vagadam

1. Yellowish discolouration of the sclera, conjunctiva, tongue and urine.
2. Pyrexia
3. Vomiting
4. Lethargy
5. Anasarca
6. Rhinorrhoea
7. Abdominal pain
8. Dropsy
9. Anaemia
10. Constipation
The other types mentioned in Bala vadagam

**Oothu Kamalai**

“அறு கண்டவப் பலர்கைப் பொழுது கண்டுக்கறி பலர் கண்டவப் பாதம் பாட்டும் பிள்ளையும் வழியாக பாட்டும், அல்லது பலர் கண்டவப் பாதம் பாட்டும் பிள்ளையும் வழியாக பாட்டும்.

Varal Kamalai

“கூறுக்குறி புரிபத்தியும் குடி புரிபத்தியும் - புனிதமான புரிபத் புனிதமான புரிபத்தியும் கூறுக்குறி புரிபத் புனிதமான புரிபத்தியும்”.

Oedema of the face and lower extremities
1. Excessive appetite, flatulence
2. Dysentery
3. Altered sensorium
4. Swelling all over the body (Anasarca)
5. Sore tongue
6. Pryexia
7. Malaise

Varal Kamalai

1. Dark yellow coloration of the eye
2. Lassitude
3. Excessive thirst
4. Dryness of the body.

According to Yogi vaidhya chinthamani

Prodromal signs and symptoms

“புற்றுக்கூடா குண்ட்குறிக் கால்வாசல்
பாதம் புரிபத்தியும் - புனிதமான புரிபத் புனிதமான புரிபத் புனிதமான புரிபத் புனிதமான புரிபத்
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கூறுக்குறி புனிதமான புரிபத்

1. Pallor of the palm, sole, face and body
2. Lassitude
3. Shivering
4. Dyspnoea
5. Constipation  
6. Yellow coloration of the face  
7. Oedema  
8. Fatiguability  
9. Generalised tiredness  
10. Heaviness of the head.

**Signs and symptoms**

According to Agasthiyar – 2000

1. Yellow coloured urine  
2. Oedema of face and lower extremities  
3. Yellowish discoloration of the conjunctiva  
4. Loss of appetite  
5. Sweating over the face
6. Pallor of the eyes
7. Dyspnoea on exertion
8. Dryness of the tongue and body
9. Pallor of the body.

According to Athma rakshamirtha vaidhya sara sangiragam:

- by Kanthaswami mudhalayar.

“...”

That is oedema all over the body, dryness of the tongue, diarrhoea, thirst, yellow coloured urine etc.

According to Siddha maruthuvam – by Dr. Kuppuswamy mudaliyar and Noi naadal part II – By Dr. Shanmugavelu:

Both were explained kamalai as in Yugi chinthamani.

1. Yellowish discolouration of urine.
2. Oedema of the body.
3. Yellowish discolouration of the face, tongue, conjunctiva, and extremities
4. Shining of the face
5. Anorexia
6. Loss of libido
7. Mental apathy
8. Constipation
9. Turmeric Odour in the breathing,

In Anubava vaidhya deva ragasiyam part IV – by Seetha ram prasath

He described Kamalai as in Jeeva Rakshamirtham

1. Yellowish discolouration of the face, tongue and extremities
2. Shining of the face
3. Oedema.
4. Aversion to food
5. Lethargy
6. Loss of libido
7. Constipation

Pathogenesis(பாதோஜெனிஸ்):

...
Taking of Pitha porugal in excessively which are having the taste of sour, salt and pungent, occupation wandering in the hot sun, sleeplessness during night etc. these activities vitiating the Pitham, then kabam is also deranged then these two thathus affects viyanan. It also affects blood. Liver, bile and produce jaundice.

Mukkuttra verubadugal (முக்குட்டிரண் வெற்றப்பகுதி)
Diagnosis (பியாறிப்பி) (சாரணத்தான்)

Piniyarimurai means method to find out the disease
this is based upon three main principles and Envagai thervugal

The three main principle are

1. Poriyal arithal
2. Pulanal arithal
3. Vinathal.

Pori-five organs of perception. They are skin, tongue, eyes, nose and ears
Pulan – functions of five senses, they are touch, taste, sight, smell, and sound.
Examination of the pori and pulan of the patient by pori and pulan of the physician.
Vinathal – asking question concerned with the disease to the patient or asking to his parents or relatives.

Envagai thervugal

“தன் நுழைவை மற்றவற்றுடன் இணைய தற்போது
முச் சுற்றுடன் உள்ளிட்டுமன்மை”
“வேண்டும் இருவரையை இணைய தன் விளையாட்டு வத்குறுத்து”
-சீரமைந்த பகுதியை 1

1. Naadi
2. Sparism
3. Naa
4. Mozhi
5. Vizhi
6. Malam
7. Moothiram

1. Naadi

Naadi is defined as the Uyirthathukkal which are responsible for the existence of life in the physical body. It is mentioned in Noi Nadal Part-I as follows,

“இட்டையை உறிப்பு வசதியுமாறு அண்டவண்ண குறிப்பிட்டதை அதிகமான ஈட்போற்றம் முடுக்க”

So Naadi is the vital force of life. By means of feeling the beating of radial artery with the tip of the three fingers that is index, middle and ring finger at the lower end of the radius.

This Naadi denotes the pulse reading of normal vali, Azhal, Aiyyam and various abnormal condition.
Siddhars laid great emphasis on the study of Naddi for diagnosis, Prognosis and assessing the effect of treatment.

Naddi is classified into five kinds as follows:
1. Vatha Naadi
2. Pitha Naadi
3. Kapha Naadi
4. Pootha Naadi
5. Guru Naadi

In normal practice Vatha Naadi, Pitha Naadi and Kaba Naadi can be observed. The proportion of Vatha, Pitha, Kaba Naadi are 1: \(\frac{1}{2}\): \(\frac{1}{4}\). Pootha Naadi and Guru Naadi are not generally observed in practice, since it cannot be learn without the help of Yogi, Asan or Guru.

The characters of Naadi are elaborately explained in our Siddha literature like “Pithinen Siddar Naadi”.

Each Naadi has its own origin, region, functions, movements and Mathirai (Proportion)

Index finger should be used to feel Vatha Naadi.
Middle finger should be used to feel Pitha Naadi.
Ring finger should be used to feel kaba Naadi.

In Manjal Kamalai the Pitha and kapha Naadi is mostly affected. Hence the Naadi in this disease is Pitha Kabam.

Sparisam is a method of palpation and percussion to know the condition of the body like warmth, fever, chillness, sweating, numbness, paresthesia, dryness of the body, erosion patches, ulcers, oedema, emaciation, swelling, obesity, enlargement of spleen and liver etc.
3. Naa (tongue)

This is the method of Inspection of the tongue mainly gums, teeth, lips, palate etc. To find out the colour changes according to Vatha, pitha, Kapha, and Mukkuttra disease, glossy tongue, coated tongue, flushing, Yellowish discoloration, pallor, fissures, Neoplastic changes and deviation of the tongue etc.

In Jaundice Yellow-coated tongue denotes the anorexia, bittertaste, excessive salivation with vomiting.

4. Niram (colour)

Diagnosis made with help of colour of the body such as pallor, yellow, cyanosis, Vatha, Pitha, Kapha, Thontha and Mukkutra colour etc. In Jaundice skin and sweating is yellow in colour.

5. Mozhi

This is very useful to diagnose a disease with the help of speech and pitch of the tone, In Jaundice speech is not affected.

6. Vizhi

This method is useful to diagnose a disease, by examine the eye such as the colour of the eye, lacrimation, drooping of eye lids and haemorrhage etc. In Jaundice the sclera, conjunctiva become yellow in colour.

7. Malam

In Vatha type, malam is hard, dry, scanty and black in pitha type the stools is loose (semisolid) moderate in quantity yellowish red in colour with fermenting odour, in kapha type the stools are white or clay coloured considerably large in quantity containing lot of mucous and frothy bubbles.

In Thontha type the malam may passes some of the combined features of two dhoshas. In Thridosha of sannibatha Kamalai, the malam may posses some of the features of all three doshas described above.

8. Moothiram (urine)
This investigation is more useful to diagnose the disease with the help of colour, smell, abnormal constituents, froth, excessive or scanty urination, specific gravity, mixing of blood, pus, deposits, sugar, albumin etc.

**Tamil Text**

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“துறுக்க வைத்தியங்கலன்களுக்கு
தனத்து வந்துள்ளது போன்.
மூத்தம் பெற்றும் போன் வைத்திய
பல்குறு சுருக்கம் முற்படது
அனுரத்து மற்றும் மருத்துவத்தில்
பல்வகைகளின் பொருள்
பல்வகையான பொருளைத்
பாதுகாப்பாகவே வைத்தியாக்கும்.
”

**Tamil Text**

“பாதுகாப்பாகவே வைத்தியாக்கும்
பாதுகாப்பாகவே வைத்தியாக்கும்
பாதுகாப்பாகவே வைத்தியாக்கும்
பாதுகாப்பாகவே வைத்தியாக்கும்.

**Tamil Text**

“அது வைத்தியத்தில் முற்படது
அது வைத்தியத்தில் முற்படது
அது வைத்தியத்தில் முற்படது
அது வைத்தியத்தில் முற்படது.

**Tamil Text**

“பாதுகாப்பாகவே வைத்தியா
பாதுகாப்பாகவே வைத்தியா
பாதுகாப்பாகவே வைத்தியா
பாதுகாப்பாகவே வைத்தியா.

**Tamil Text**

“அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட.

**Tamil Text**

“அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட
அது வைத்தியத்தில் முற்பட.
வியூரிலைநார் மென்போர் முறைப்படுத்தாது மேலும் வருந்தாது. உள்ளே உள்ளே மேலும் வருந்தாது. ஆதாரங்கள் என்றால் என்ன என்ன என்றால் என்ன

முடி, பிரிக்கும் புக்கும் நீங்கள்

''சரணாலை பிரித்தகம் பக்தர்
மன்னரா வரச்சம தந்த போல் செய்வதையே பார்க்க முடியுமா
பாத்தெருக்கள் பிள்ளை பெண்கள்
கிளையே பிள்ளை
கிளையே பெண்கள் பலர் சாதனம்
பலரே குரு குருவும் சாதனம்

சீர்தானியாக அன்றால் என்

''அப்படி என்ன விளக்கமாகவும் குறுக்காகவும் விளக்கமாகவும்''

இன்னாலும் வண்ண சிற்று குண்டுநீர்த்தைவை விளக்கப்படுத்துவதற்குச் செல்வூர்கள் சாதனம் அச்சங்கால்

முடி காட்சி என்று தந்த

''அதேட்ச வண்ணத்தை விளக்கப்படுத்து விளக்கமாகவும்

சீர்தானியாக உள்ள்துவமற்றும்

''சாதனம் விளக்கமாக வந்தது என்று
புரபர்காகச் செய்வதையே என்று
சீர்தானியாக விளக்கப்பட்டது
சீர்தானியாக விளக்கப்பட்டது
சீர்தானியாக விளக்கப்பட்டது
சீர்தானியாக விளக்கப்பட்டது

சீர்தானியாக விளக்கமாக விளக்கமாக விளக்கமாக விளக்கமாக விளக்கமாக

நகரங்கள்:

சீர்தானியாக விளக்கமாக விளக்கமாக விளக்கமாக விளக்கமாக விளக்கமாக

புக்கும் என்ன என்ன

''சீர்தானியாக விளக்கமாக 

“அம்பைகள் பரிசை கையேடு பிட்டு”

மிகவும் பிட்டு தர்க்கப்பட்டுள்ள மிட்டை பரிசையில் அடுத்து பிட்டிக்கப்பட்டு அருட்ச.

பின்னர் இன்று வட்டாரம்:

“அம்பைகள் பிட்டு”

தர்க்கப்பட்டுள்ள மிட்டை பரிசையில் அடுத்து பிட்டிக்கப்பட்டு அருட்ச. தர்க்கப்பட்டுள்ள மிட்டையில் அடுத்து பிட்டிக்கப்பட்டு அருட்ச.

“நேர்த்திய மாற்றங்களில் பிட்டு பிட்டிப்பு தர்க்கப்பட்டு உள்ளது”.

“வட்டாரம் பிட்டிக்க பிட்டு தர்க்கப்பட்டு குறுக்க உள்ளது உத்தராக”.

“செய்யப்படும் கொண்டிருந்த பிட்டு பிட்டிப்பு உள்ளது உத்தராக உள்ளது”

In Manjal Kamalai, Pitham is mostly affected and then kabam is affected. Here it is essential to have a thorough knowledge of pitham since these are affected in this disease.

மத்தியே வேறுபடுத்தம்:

10 நேரானம்:

1. பிட்டையாளர்:
   - புரவநிலை, சுருக்கமானது, புரிந்து உண்மையானது வேறு படையும்
2. அணுபாளர்:
   - சுருக்கமானது மூடாது செய்யப்படும்
3. விலாசாளர்:
   - கல்லாள் அணுபாளர் வேகமானது, கல்லாள் வேகமானது திண்குகின்றது
   - சுருக்கமானது பதில் உயரத்து செய்யப்படும்.
4. சிறந்தாளர்:
   - சுருக்கமானது அணுபாளரின் மூடும்படி அவர் கொள்ளப்படுமதின்
   - சுருக்கமானது மூடும்படியாக உயரத் செய்யப்படும்.
5. கல்லாளர்:
   - பதில்பக்காட்சிகளானது, மூடாது அணுபாளரின் உயரமை செய்யப்படும்.
6. கல்லாளர்:
   - கல்லாளர் சுருக்கமானது செய்யப்படும். கல்லாளர் சுருக்கமானது
   - செய்யப்படும்.
7. கல்லாளர்:
8. மேலும்:
- தமிழ்ச் சொல்லில், தமிழ் பல்லுயிரின்
9. குறைந்திருக்கும்:
- கருவிகள் நூற்றன் நோய் செய்ய, கருவியால் அனுமதித்து அகழ்த்துவது
10. குறைந்திருக்கும்:
- மட்டும் முழு வெளியும் திறன் பல்கலை.
- முன்னிகழ்ற குடும்பருங்கள் வேலும்போன்று பிற்காலங்கள் இரண்டின் வலக்கு செவ்விகளில் தங்கு விளங்குவது.

பிற்பகுதி:

பிற்பகுதியின் பிரிவுகள்: 5 கோடை

1. ஆண்டுப் பிரிவு:
- இரு கலங்களின் இருப்பதாக.
2. ஆண்டுப் பிரிவு:
- அனுமதிக்கப்பட்டு பிரிவு தொகுதிகளை தானியங்கியது
3. வரைபட்டு பிரிவு:
- கதைகளின் செயலங்களைப் போற்று புகழ்த்து
4. வரைபட்டு பிரிவு:
- கலங்களின் பல்கலை செய்திகள்
5. பிரிவு பிரிவு:
- தொன்மை அளவிலானது

பிற்பகுதியின் கோடை:

1. கலங்கள், வேலு, குளிர், வெப்ப இருக்கும் பல்கலையான விளைந்தை
2. வேலு, குளிர் தொன்மை பிரிவுபட்டு
3. வேலு வெப்பமான விளைந்தை எளிதாக
4. தொன்மை தொன்மை

பிற்பகுதியின் கோடை:

1. பொழித்துக்கடி
2. குளிர்
3. வெப்பமான
4. தொன்மை

கலங்கள் (சுவாமி):

1. சுவாமி தலைகுப்பு கலங்கள்:
- குருக்கள், குருப்பு, சுவாமி, தூயமாயுடைய நூற்றன், குருக்கள்
2. சுவாமி அளவிலாந்:
- தொன்மை
3. குறிப்பிட்டு சொல்லையுடைய பண்பு

போட்டியில், தீர்வுகள், சமாதி, கல்விக்க, மொழிபெயர் சான்றிமை மூலமைத்து வல்லிக்கும்.

5. குறிப்பிட்டு குறிப்பிட்டு பண்பு:

சொல்லில் கொண்டு புரியாது வேளை தோற்றியிருக்கு குறிப்பிட்டு பொருளின் பொருளின் தொடர்ந்து சுருக்க ஓரியாக்கல் தன்மை, கைவரிசை, மூலம், நூற்பொருள் பனித்தொடர்புகள்.

5. குறிப்பிட்டு பிரிவுகள்:

5 பிரிவுகள்
1. அவாக்கம்
2. எடுத்துகள்
3. பிரிவுகள்
4. குறிப்பிட்டு
5. உரைக்கை

1. அவாக்கம்:

- குறிப்பிட்டு சொல்லையுடைய பண்புகள் குறிப்பிட்டு குறிப்பிட்டு

2. எடுத்துகள்:

- குறிப்பிட்டு சொல்லையுடைய பண்பு குறிப்பிட்டு குறிப்பிட்டு

3. பிரிவுகள்:

- தனிமை கலவையை தொண்டு

4. குறிப்பிட்டு

- குறிப்பிட்டு சொல்லையுடைய பண்பு தொண்டு

5. உரைக்கை

- குறிப்பிட்டு சொல்லையுடைய பண்பு

ஆனால் (பார்வை):
காலை பகுதிகளைப் பிரிக்கப்பட்டு பிறக்கச் செய்யும் வாய்ப்பு. இதுவே நாட்டில் நூற்றாண்டுகள் வருமாறு.

காலைச்சூழல் அடுத்தும் பகுதிகளைப் பிரிக்கப்பட்டு

1. காலைக் காலையின் (மாத்ருக்) - வெண்மை
2. காலைகள் (தாக்குகள்) - வெண்மை, காலைகள்
3. பின் பின்வரும் - காலைகள், காலை
4. விளையாட்டுகள் - மாற்று, மாற்று
5. இலங்கைகளின் காலை - விளையாட்டு, காலை
6. பிள்ளைகளின் காலை - விளையாட்டு, விளை

காலை அல்லது அருமைகள் காட்சிகளைப் பிரிக்கப்பட்டு

3 அல்லது அல்லது

1. காலைகளின் வருமாறு
2. விளையாட்டுகளின் வருமாறு
3. பிள்ளைகளின் வருமாறு

காலை அல்லது அருமைகள் காட்சிகளைப் பிரிக்கப்பட்டு

நூற்றாண்டு காலைகளின் (அல்லது) நூற்றாண்டு காலைகளின்:

1. போற்று:
   போற்று போற்று போற்று
2. போற்று:
   அருமை, அருமை, அருமை, விளையாட்டு விளையாட்டு விளையாட்டு
3. போற்று:
   போற்று போற்று போற்று
4. போற்று:
   போற்று போற்று
5. போற்று:
   போற்று போற்று
6. போற்று:
   போற்று போற்று
7. போற்று:
   போற்று போற்று

சூழ்லிகள் வருமாறு விளையாட்டு விளையாட்டு

சூழ்லிகள் வருமாறு விளையாட்டு விளையாட்டு
“தமிழ் கட்டணம் பெண்களின் குழந்தைகள்
தமிழுக்கு முன்னிலையில் நூற்றாண்டிற்கு
பெண்கள் பிற்குரத்தை நிறைத்து
பெரும் மருந்தாகத்தை திறன் முறையில்
செய்துள்ளது ஒட்டுகத்தை கத்து
தினசரியிலுள்ள தமிழ் கட்டணம்
கட்டணமே 13 வயதுக்கு முன்னர், குழந்தைகள் வாழ்வாறு.

சிறப்பு கட்டணங்கள்:

1. பிறந்த கட்டணம்
2. அபத்து கட்டணம்
3. விழும் கட்டணம்
4. விளையாட்டு கட்டணம்
5. குருது கட்டணம்
6. அந்த விழும் கட்டணம்
7. அபத்து விழும் கட்டணம்

சிறப்பு விளக்கங்கள்:

1. குடா விளக்கம்
2. குதிகள் விளக்கம்
3. குழந்தை
4. தேவ விளக்கம்
5. குருது
6. விழும்
7. விளையாட்டு
8. அபத்து
9. குருது விளையாட்டு
10. விழும் அபத்து
11. விழும் குருது

முடிக்கும்:

அபத்து அபத்து பார்வையும் இந்தக் குழந்தைகளுக்கு
“செய்யாதலே பாதுகாப்பு” என்று குறிப்பிட்டு தமிழ் வாழ்வாறு  செய்யும்.
திறனிலை பிற்கு அரும் அமைப்பு குழந்தைகளின் விளக்கத்திற்கு பிற்கு நூற்றாண்டிற்கு
திறனிலை பிற்கு நூற்றாண்டிற்கு வாழ்வாறு  குழந்தைகளுக்கு, எதிர்க்கு வாழ்வாறு
பாதுகாப்பில் வாழ்வாறு நூற்றாண்டிற்கு.
1. முதிர்வாண பிள்ளைகள் கிளைப்பிறுத்தக் கூடி கூட்டுவக்கால் வசாத்து விளையாட்டில் அனைத்து, குழந்தைகள் மறுவுலகம் தோறு முதிர்வாணக் வசாத்து விளையாட்டில். தமிழ்ப் பள்ளிகள் பிள்ளைகளுக்கு அதிக தொழில்பாதுகை நடத்தி, குழந்தைகள் மறுவுலகம் விளையாட்டில்.

2. முதிர்வாண்கள் பாறைகள் முதிர்வாண்கள் தமிழ் பனாட்டு சதுரங்கம் விளையாட்டில் அனைத்துக் கலைஞர்கள் பாலாட்டில்.

3. பிள்ளைநழையும் பாலைப்பான பிள்ளைகள், அம்மாளருக்கான மறுவுலகம் விளையாட்டில்.

4. முதிர்வாண்கள் பிள்ளைகள் கூட்டுவக்கால் வசாத்து விளையாட்டில், பிள்ளைநழையும் கூட்டுவக்கால் வசாத்து விளையாட்டில்.

5. பிள்ளைநழையும் பாலைப்பான அனைத்துக் பாலைப்பானைகள் கலைஞர்கள் மறுவுலக விளையாட்டில்.

6. பிள்ளைநழையும் முதிர்வாண்கள் மறுவுலகம் குழந்தைகள் மறுவுலகம் விளையாட்டில் (அனைத்து கலைஞர்கள் கலைஞர்கள்).

சந்தைத்தானம்:

• காலமுக விளையாட்டு குழல், குழல், பூமியால், சேதியால், குழல்கள் விளையாட்டு தொழில்பாதுகை.

• காலமுக நூற்றாண்டு விளையாட்டு விளையாட்டு தொழில்பாதுகை குழல் தொழில்பாதுகை விளையாட்டில் அனைத்து கலைஞர்கள் கலைஞர்கள்.

• தமிழ்ப்பள்ளிகள் தொழில்பாதுகை விளையாட்டு குழல் மறுவுலகம் விளையாட்டில் அனைத்து கலைஞர்கள் கலைஞர்கள் விளையாட்டில்.

• தோற்றப்பட்டை நூற்றாண்டு விளையாட்டு குழல் மறுவுலகம் விளையாட்டில்.

1. சிறைத்து காலமுக
2. குழல்பார்வை, வேல்க்குழல் குழல்
3. கூட்டுவக்கால் குழல்
4. சிறைத்து காலமுக (புதினைத்துடன்)
5. குழல்பார்வை, வேல்க்குழல் புதினை, சென்றுபார்வை, கம்பாமுதி, கிளைப்பிறுத்தது
6. குழல் தொழில்பாதுகை காலமுக
7. சேதியால் சேதம் குழல்
8. கானுந்து குழல் தொழில்பாதுகை, புதினை, மும்பாயின் (ந) காலமுக
9. கானுந்து பேராம், பேராம் புதினை காலமுக
LIVER AND BILIARY SYSTEM
DEVELOPMENT AND FUNCTION

MORPHOGENESIS:

The liver and biliary system originate from a cluster of cells that cap a ventral diverticulum in the primitive fore gut. The hepatic anlage (pars hepatic) appears during the 4th wk of gestation as a duodenal diverticulum. Within the ventral mesentery proliferation of cells forms anastomosing hepatic cords, with the network of primitive liver cells, sinusoids, and septal mesenchyme establishing the basic architectural pattern of liver lobule.

The solid cranial portion of the hepatic diverticulum eventually forms hepatic glandular tissue and the intra hepatic bile ducts. The caudal portion (pars cystica) becomes the gall bladder, cystic duct and common bile duct.

The hepatic lobules are identifiable at the 6th gestational wk. The liver reaches a peak relative size at the 9th wk at about 10% of the fetal weight.

The bile canalicular structures that include microvilli and junctional complexes are specialized loci of the liver cell membrane these appear very early in gestation and by 6-7 wk. large canaliculari bounded by several hepatocytes are seen.

The intra hepatic bile ducts are derived through branching of the hepatic duct, formation is complete by the 3rd month. The cystic duct and the gall bladder are fully reconalized by the 7th – 8th wk.

In the hepatic excretory (biliary) system intercellular bile canalliculi empty in to the smallest bile ductules, which unite to form inter lobular bile ducts that follow the terminal branches of the portal vein. At the hilum of the liver, the intra hepatic
ducts leave the branches of the portal vein and merge to form the extra hepatic biliary system.

The ducts of the right and left lobes form the common hepatic duct. The common bile duct is formed from the merger of the common hepatic duct and cystic duct. It runs along the right edge of the lesser omentum, terminating as the intra mural papilla of vater.

Union of the biliary tract with the pancreatic ducts forms the ampulla of vater which with the sphincter of oddi, regulates the flow of bile into the intestine, prevents entry of bile into the pancreatic duct, and inhibits reflex of intestinal contents into the ducts.

The transport and metabolic activities of the liver and facilitated by the structural arrangement of liver cells. Cords, which are formed by rows of hepatocytes, separated by sinusoids that converse toward the tributaries of the hepatic vein (the central vein) located in the center of the lobule.

This establishes the pathways and patterns of flow for substances to and from the liver. Plasma proteins and other plasma components are secreted by the liver.

Absorbed and circulating nutrients arrive through the portal vein or the hepatic artery and pass through the sinusoids and past the hepatocytes to the systemic circulation at the central vein.

Biliary components are transported via the series of enlarging channels from the bile canaliculi through the bile ductula to the common bile duct.

Bile secretion has been noted at the 12th gestational wk. the major components of bile vary with stage of development. Near term, cholesterol and phospholipids content is relatively low and low concentrations of bile acids, the absence of bacterially derived (secondary) bileacids and the presence of unusual bile acids reflect low rates of bile flow and immature bile acid synthesis.

Fetal hepatic blood flow is derived from the hepatic artery and from the portal and umbilical veins, which from the portal sinus, The portal venous inflow is directed mainly to the right lobe of the liver. Umbilical flow is primarily to the left.

The ductus venosus shunts blood from the portal and umbilical veins to the hepatic vein bypassing the sinusoidal network.

The ductus venosus becomes obliterated when oral feedings are initiated the oxygen saturation is lower in portal than in umbilical venous blood .accordingly the right hepatic lobe has lower oxygenation and greater hematopoietic activity than the left hepatic lobe. Sinusoidal endothelium is the site of large macrophases which become the kupfer (Retiullo – endothelial) cell network.
The liver constitutes 5% of body weight at birth but only 2% in the adult. Early in gestation (7th wk) hematopoietic cells outnumber functioning hepatocytes in the hepatic anlage.

The hepatocytes are smaller (~20 µm) than at maturity (30-35µm) and contain less glycogen. Near term, the hepatocytes dominate the organ, and cell size and glycogen content increase. Hemotopoiesis is virtually absent by the 2nd postnatal month in full-term infants. As the density of hepatocytes increases with gestational age, the relative volume of the sinusoidal network decreases.

**ANATOMY**

**Liver.**

**SYNONYM:**

Hepar (the term hepatic)

**DEFINITION:**

Liver is the largest gland in the body.
Situated in the right upper quadrant of the abdominal cavity.
Most of it is covered by the ribs and costal cartilages. Except in the upper part of the epigastrium.

**LOCATION:**

It occupies in the right. Hypochondrium greater part of the epigastrium. And extends into the left hypochondrium. Up to the left lateral line.

**SHAPE:**

It is wedge shaped, It resembles a four sided pyramid laid on the one side. With its apex directed towards the left.

**COLOUR:**

Reddish brown
Soft and very friable

**WEIGH:**
1400 – 1800 grams in males
1200 – 1400 grams in females
A range of 1000 -2500 gram.
In foetus – 1/25 of body weight
In adult – 1/40 of body weight

EXTERNAL FEATURES:

Mainly it has two surfaces.

1. Diaphragmatic (In contact with the diaphragm)
2. Visceral (In contact with several abdominal viscera)

DIAPHRAMATIC SURFACE:

It is boldly convex, molded to the under surface of the diaphragm.
It is subdivide into.

1. Anterior
2. Posterior
3. Superior
4. Inferior
5. Right.

The inferior surface is well defined because it is demarcated by the sharp inferior border.

VISCERAL SURFACE:

It is flat and slopes downwards forwards and to the right from the posterior surface.

There is no clear dividing line.

ONE PROMINENT BORDER:

The inferior border is prominent border
It is sharp anteriorly
It separates the anterior from the inferior surface
It is laterally rounded
It separated the right from the inferior surface.

The sharp anterior part is marked by 2 notches
i) Inter lobar notch, ii) Cystic notch

i) **INTER LOBAR NOTCH:**

The notch of ligamentum teres.

ii) **A CYSTIC NOTCH:**

i) The fundus of the gall bladder.
   In the epigastrium the inferior border extends from the left 8\textsuperscript{th} costal cartilage.
   To the right 9\textsuperscript{th} costal cartilage.

ii) Other borders are rounded and ill defined.

**LOBES:**

Liver is divided into the 2 lobes

i) Right lobe

ii) Left lobe

   - By the falciform ligament

   Inferiorly – By the fissure for ligament terus.
   Posteriorly – By the fissure for ligament venous.

   Anterior

   And superior

   - It is larger
   - 5/6 part of liver
   - It has 2 additional lobes
     i) caudate lobe
     ii) quadrate lobe.

   Caudate lobe
   - It is situated on the posterior surface
   - Bounded by the groove for inferior venacava

   Left – By the fissure for ligamentum venosum.

   Inferiorly – porta hepatics.
   It is continuous with
   Above – superior surface
   Below – Right
   - Behind the porta hepatics

   It connect to the right lobe of liver by the caudate process.

   Below and left it presents small rounded elevation called the papillary process.

**QUADRATE LOBE:**
situated inferior surface
- Rectangular shape
- It is bounded
Anterior- by the inferior border
Posterior – by the porta hepatic

PORTA HEPATIS:

It is a deep transverse fissure
- situated in inferior surface of the Rt lobe of the liver
- It between the
Above - caudate lobe
Below – quadrate lobe

LEFT LOBE:
- It is smaller
- Only 1/6 part of liver
- It is flattened
- Its inferior surface near the fissure for ligamentum venosum.
- A rounded elevation called the omental tuberosity (tuber omen tale)

I PERITONEAL RELATIONS:

The liver is mostly covered by peritoneum.
It include “Bare area”,

i) Bare area:
- The posterior surface of the right lobe of liver.
- Limited by coronary and right triangular ligament.

a) Groove for inferior vena cava
   It between caudate and main bare area

b) Gall bladder
   Inferior surface of rt lobe of liver.

c) Porta hepatic
   It lines of reflects of peritoneum.

ii) PERITONEAL LIGAMENTS:

a) Falciform ligament:
   - antero superior surface of the liver
   - to anterior abdominal wall and diaphragm.

b) Left triangular ligament:
- Superior surface of Lt lobe of liver.
- To diaphragm.

c) **Right triangular ligament:**

- Lateral part of the posterior surface of Rt lobe to diaphragm.

d) **Coronary ligament:**

- Superior and Inferior surface
- Bare area of liver

e) **Lesser omentum**

**II VISCCERAL RELATIONS:**

i) **Anterior surface**:

- It is triangular and slightly convex
- Related to xyphoid process and anterior abdominal wall, diaphragm on eachside
- Drawn from xiphisternal joint to the 10th rib in mid axillary line
- Join to 8th rib
- Falci form ligament is attached little

ii) **Posterior surface**:

- it is triangular and concave
- vertical impression in middle
- It is related to diaphragm & Rt supra renal gland near groove for inferior vena cava.
- Groove for Inferior venacava – upperpart of liver caudate lobe- related to crura of diaphragm.
  Fissure for ligamentum venosum – very deep
- In front of caudate lobe
  Posterior surface of the left lobe is marked by oesophageal impression.

iii) **Superior surface:**
- quadrilateral and concave
- In middle cardias impression is present.

INFERIOR SURFACE:

- Quadrilateral
- Left – downwards and backwards
- Impression – neighting viscera

Concave gastric impression for stomach
Fissure for ligamentum teres for portal vein quadrate lobe is related to 1st part of duodenum
Pylorus
Omentum

Porta hepatic, gall bladder fossa lodges the gall bladder.
Rt lobe inferior surface – colic impression
Rt. kidney - Renal impression

Second part of duodenum – duodenal impression Right surface.
- Quadrilateral & convex
- Diaphragm Opposite the 7th to 11th rib

BLOOD SUPPLY:

- 20% of blood supply from hepatic A
- 80% Portal vein

BILE PIGMENT METABOLISM:

i) The breakdown of haemoglobin liberates the porphyrin moiety in the form of a tetra pyrrol ring. Which is split and reduce to form the bile pigment.

SECRETION OF BILE:

i) Hepatocytes
   (The parenchyma consists of rows of Liver cells)
ii) The two rows of cords of the cells surrounded by the sinusoids
iii) A cleft like lumen exists between the apposed cell membranes of the two rows of hepatic cells.
iv) This cleft called the bile canaliculus.
v) The liver cells discharge their secretion called BILE.
BILE

Fresh human bile is a clear golden yellow liquid formed.
It is secreted by the liver
It is slightly viscous and bitter taste
Its pH varies from 7.5 to 8.5
About 5000 to 700ml are secreted daily by the liver
500 ml is stored in the gall bladder.
It is concentrated and periodically discharged into the small intestines

The bile in gall bladder is more viscous and has a greenish tinge. Because of
the presence of bile pigments.

DURING DIGESTIN:

The gall bladder contracts to supply bile to the intestines. Via the common
bile duct. The bile mixes with the pancreatic juice and exerts important influence on
the digestion of fats of food.

BILE CONTAINS:

1 to 4 % of solids
25% are inorganic constituents main in organic constituents are
Bicarbonates and chlorides of sodium

i)Potassium
   The remaining part contains
i) organic materials
   Bile – Bile salts
Bilirubin  
Cholesterol  
Small amount of phospholipids  
Fats  
Fatty acids  
Mucin  
Urea  
Enzyme – alkaline phosphates

The inorganic constituents \{ Are products of excretion And Bile pigments

And are removed from blood by the liver cells by biliary excreting.

The bile sates And bile cholesterol \} Are formed in the polygonal cells of liver.

The liver alkaline phosphate also originates from these cells. Mucin is formed from the epithelium of bile duct and mucosa of gall bladder. The cholesterol of bile is synthesized by the liver. Composition of hepatic bile and gall bladder bile.

<table>
<thead>
<tr>
<th>Constituents</th>
<th>Percentage of Total Bile Hepatic Bile (P^H 7.1 to 8.5)</th>
<th>Percentage of Total Bile Gall Bladder Bile (PH 5.5 to 7.7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity</td>
<td>1.009 to 1.013</td>
<td>1.026 to 1.032</td>
</tr>
<tr>
<td>Water</td>
<td>97.00</td>
<td>85.92</td>
</tr>
<tr>
<td>Solids</td>
<td>2.52</td>
<td>14.08</td>
</tr>
<tr>
<td>Bile acids</td>
<td>1.93</td>
<td>9.14</td>
</tr>
<tr>
<td>Mucin and bile pigments</td>
<td>0.53</td>
<td>2.98</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.06</td>
<td>0.26</td>
</tr>
<tr>
<td>Fats and fatty acids</td>
<td>0.14</td>
<td>0.32</td>
</tr>
<tr>
<td>Inorganic salts</td>
<td>0.84</td>
<td>0.65</td>
</tr>
<tr>
<td>pH</td>
<td>7.1 to 8.5</td>
<td>5.5 to 7.7</td>
</tr>
</tbody>
</table>

Gall Bladder and the storage of Bile.

The liver secretes between 500 – 1000 ml of Bile per day. Which travels down the hepatic ducts and passes along the cystic duct to be stored in the gall bladder.

**Gall bladder:**

It is a pear shaped dilated sac
Lined by mucous membrane a thin layer of smooth muscle fibers and fibro elastic tissue. The mucosal epithelium consists of columnar cells showing microvillus infolded basal membrane and numerous mitochondria features. The characteristic of cells involved in active transport. The capacity of gall bladder is 50ml It communicates the cystic duct. The mucosa of the duct shows spiral folds. Act as a spiral value (Valve of Heister) The cystic duct joints the hepatic duct to form the common bile duct. It joints the main pancreatic duct do open into the duodenum. Bile is secreted continuously by the liver. It is required only intermittently by the intestine. Therefore when not required it is stored in the gall bladder.

**Regulation of Biliary secretion:**

The bile is permitted to enter the duodenum is determined by two factors.
1. the rate of formation of bile by the liver or choleresis
2. The evaluation of stored bile from the gall bladder on chore cyst kinesis.

**THE RATE OF SECRETION OF HEPATIC BILE:**

(CHOLERESIS)

-In man is about 13 to 65 ml per hour is influenced by several factors.
- Which increase the output of bile without necessarily changing its concentration is called choleretics.
- Affecting the solid contents is called hydro choleretics.
- Bile salts act as powerful choleretics.
- This biliary ductal component of bile secretion is under neuro hormonal control. Hydrochloionic acid acid chime in the duodenum have also been shown to increase water and electrolyte secretions of bile.
- Hepatocrinin having a potent choleretic effect on the liver.

**EVACUATION OF GALL BLADDER BILE (Cholecys to Kinesis)**

-The duodenum is brought about by neural and hormonal control.
- Which bring about evacuation of stored bile in to the duodenum by contracting the gall bladder are called cholagogues or a cholecys to go gues.
  - Intrinsic extrinsic nerves are involved in cholecystokinesis.
  - The reflexes through either intrinsic plexuses or by extrinsic afferents impringing on vassal nuclei (probably raso –vagal) to inhibit the gall bladder contractions.
  - (Choledochod wodenal sphincter)

**FUNCTIONS OF BILE:**
Bile salts are most important for their role in digestion and absorption, particularly of fats. To provide the alkaline pH for neutralization of acid chime.

**EMULSIFICATION OF FATS:**

The detergent or hydrotropic property of bile salts helps to lower the interfacial tension between the oil and water phases in a mixture.

**FORMATION OF MICELLAR SOLUTIONS:**

They activate pancreatic lipase; they are known to be absorbed on to the surface of the lipid globules in the emulsion and the biolytic activity of pancreatic lipase.

**SECRETION OF PANCREATIC JUICE:**

Bile salts in the duodenum stimulate secretion of pancreatic juice. Secretion of pancreatic lipase.

**ENTEROKINASE:**

Bile salts increase the release of Enterokinase from the microvillous membrane of the duodenal mucosal cells.

**EXCRETION:**

Bile acts as an important vehicle for the excretion of numerous drugs, toxins, and salts of heavy metals such as copper, zinc, and mercury.

**LAXATIVE:**

Mild laxative effect on the gut by stimulating peristalsis in the small bowel.

**ANTISEPTIC ACTION:** Inhibits the growth of certain bacteria.

**BLOOD SUPPLY TO THE LIVER:**

Blood enters the liver through the portal vein and the hepatic artery.

**PORTAL VEIN:**

It is formed by the union of the superior mesenteric vein. Which drains blood from the major part of the gastrointestinal tract and the splenic vein. Which is the venous drainage of the spleen, pancreas, gall bladder, and stomach. Superior mesenteric vein drains mainly into the right lobe of the liver. Splenic vein mainly goes to left lobe. The portal vein devices into branches. Which will lie within the portal
tracts and form sinusoids between the plates of hepatic cells. The various sublobular veins unite to form the hepatic vein.

HEPATIC ARTERY:
The arterial blood to the liver and supplies the protect tracts and the bile ducts. If anastomoses within the liver parenchyma is a complex manner draining both into the sinusoids as well as into the branches of the portal vein.

It is the common venous outflow of the liver.

NERVUS INNERVATION OF LIVER:
The nerve supply to the liver is arranged in two plexuses at the hilum of the organ, the anterior plexus from left celiac ganglion.
The lefty vougues provide the parasympathetic supply to this plexus.
Both the anterior and posterior plexuses join together to provide intra hepatic innervation.
The right phrenic nerve also supplies sensory fibres to the liver.
FUNCTIONS OF LIVER

1. STORAGE FUNCTION:

   Many substances are stored in liver
   -Glycogen
   -Amino acids
   -Iron
   -Folic acid
   -Vitamins A, B12 and D.

2. SYNTHETIC FUNCTION:

   -Plasma proteins
   -Blood group substances
   -Clotting factors
   -Somatomedin
   -Heparin
   Are synthesized in liver.

3. SECRETION OF BILE:

   Liver secretes bile, It contains bile salts, Bile pigments, cholesterol, fatty acids and lecithin.
   -The bile salts are required for digestion and absorption of fats in the intestine.

4. METABOLIC FUNCTION:
Carbohydrate
Proteins
Lipids
Vitamins
Many of hormones in liver.

5. EXCRETORY FUNCTION:

Liver excretes cholesterol,
Bile pigments
Heavy metals (lead, arsenic, bismuth)
Toxins
Bacteria – typhoid
Virus

6. HEMOPOIETIC FUNCTION:

Heat is produced in liver due to metabolic actions.

7. HEMOPOIETIC FUNCTION:

In fetus (hepatic stage) the blood cells are produced in liver.

8. HEMOLYTIC FUNCTION:

RBC life span is 120 days
It is destroyed by the kupffer’s cells are the reticulo endothelial cell in liver.

9. DEFENSIVE AND DETOXIFICATION FUNCTIONS:

- Detoxification of foreign bodies
- Bacteria are swallowed digested phagocytosis
- Antibody production
- Detoxification → Removal of toxic property.

i) Metabolic degradation
ii) Toxic substances converted into non-toxic materials → conjugation with glucuronic acid or sulphates.
JAUNDICE

Jaundice refers to the yellow appearance of the skin, sclera and mucous membranes resulting from an increase bilirubin concentration in the body fluids. It is detectable when the plasma bilirubin exceeds 50µ mol /l (3mg ld1).

Internal tissues and body fluids are colored yellow but not the brain as bilirubin does not cross the blood brain barrier other then in the immediate neonatal period.

MECHANISMS PRODUCING JAUNDICE

- Haemolysis
- Impaired hepatic bilirubin transport
- Hepato cellular damage
- Cholestasis (impaired bile flow)

HAEMOLYTIC JAUNDICE

This results from increased destruction of red blood cells or their precursors in the narrow causing increased bilirubin production.

Jaundice due to haemolysis is usually mild because a healthy liver can excrete a bilirubin load 6 times greater than normal before unconjugated bilirubin accumulates in the plasma.

Exceptions to this occur in the newborn when the hepatic bilirubin transport mechanism is immature and in patients with liver disease.

CLINICAL FEATURES:
- Increased excretion of bilirubin
- Stercobilinogen leads to normal colored stools
  -(or) Dark stools
  Increased urobilinogen excretion causes the urine to turn dark on standing as urobilin is formed.
  - Pallor due to anemia
  - Splenomegaly – due to excessive reticulo endothelial activity.

**INVESTIGATION:**

The plasma bilirubin is less than 100µ moll (6mg ld1)
Liver Function test – Normal
No bilirubinuria
Unconjugated Hyper bilirubinemia present
Haemolytic anaemia present.

**MANAGEMENT:**

i) Splenectomy results in
  - preistant anemia
  - Severe hemolytic or aplastic crisis
  - Family members are died from this disease.
  - Evidence of chole cystitis chole cystitis, choledolithiasis

**HEPATO CELLULAR JAUNDICE:**

Hepato cellular jaundice results from inability of the liver to transport bilirubin in to the bile as a result of liver cell damage. Bilirubin transport across the hepatocytes may be impaired. Between uptake of bilirubin (on conjugated) and transport of conjugated bilirubin in to the canaliculi.

Swelling of cells and oedema resutlions from the disease may cause obstruction of the biliary canaliculi concentration in the blood of unconjugated and conjugated bilirubin is increased. Bilirubin transport is disturbed.

Acute parenchymal liver diseases due to viral hepatitis,

  Prolonged alcohol abuse
  Chronic hepatitis
  Cirrhosis

**CHOLESTATIC JAUNDICE:**

It is a failure of bile flow between the hepatocyte and the duodenum. Jaundice becomes progressively severe in unrelieved cholestasis because conjugated bilirubin is unable to enter the bile canaliculi and passes back into the blood. Failure of clearance of unconjugated bilirubin arriving at the liver cells.
AETIOLOGY:

- Failure of the hepatocytes to generate bile flow.
- Obstruction to bile flow in the bile ducts in the portal tracts.
- Obstruction to bile flow in the extra hepatic bile ducts between the porta hepatic and the papillae of water

CLINICAL FEATURES:

Early Features
Jaundice
Dark urine
Pale stools
Pruritus

Late features:

Xanthelasma and xanthomas
Mal absorption
Weight loss
Steatorrhoea
Bleeding tendency
Fever
Rigors
Pain
Biliary infection
Hepatic abscess

INVESTIGATION:

Ultrasoundography – donot show dilated bile duct
Antimitochondrial antibody
ERCP is the best investigation is the biliary tract is dilated.
Dilatation extends to the lower common bile duct.
TTC is used only of ERCP is unavailable

MANAGEMENT:

This depends on the underlying cause of the cholestasis

UNUSUAL FORMS OF CHOLESTASIS:

CHOLESTASIS OF PREGNANCY:
It is caused by an inherited susceptibility of the patient’s liver cells to oestrogens. This condition is sometimes precipitated by oral contraceptives. Pruritus is the dominant symptom.

Jaundice.

Inching almost always starts in the third trimester of pregnancy and remits within about 2 weeks of delivery. Pruritus can be relieved with cholestyramine no harm comes to the fetus but the condition tends to recur in subsequent pregnancies.

---

**VIRAL HEPATITIS**

**Hepatitis A (Infective Hepatitis)**

Viral hepatitis A (HAV) accounts for about 150,000 of the 500,000-600,000 new cases of viral hepatitis that occur each year in the United States. The hepatitis caused by HAV is an acute illness (acute viral hepatitis) that never becomes chronic. At one time, hepatitis A was referred to as "infectious hepatitis" because it could be spread from person to person like other viral infections. Infection with hepatitis A virus can be spread through the ingestion of food or water, especially where unsanitary conditions allow water or food to become contaminated by human waste containing hepatitis A (the fecal-oral mode of transmission). Hepatitis A typically is spread among household members and close contacts through the passage of oral secretions (intimate kissing) or stool (poor hand washing). It also is common to have infection spread to customers in restaurants and among children and workers in day care centers if hand washing and sanitary precautions are not observed.

**Hepatitis A** - Caused by the hepatitis A virus, Hepatitis A is often spreads because of poor personal hygiene habits, such as not washing hands after a bowel movement. You can also get hepatitis A by eating foods or drinking beverages contaminated with the virus. Hepatitis A is a common form of viral hepatitis in the United States This disease is responsible for serious health problems.

**Signs and Symptoms:**

- Loss of appetite
- Nausea
Vomiting
Fever
Weakness
Tiredness
Aching in the abdomen

**Less common symptoms include:**

Dark urine
Light-colored stools
Fever
Jaundice (a yellow appearance to the skin and white portion of the eyes)

Diagnosis of viral hepatitis is based on symptoms, physical findings as well as blood tests for liver enzymes, viral antibodies, and viral genetic materials.

**HEPATITIS A VIRUS**

- Picorna virus
- Entero viruses
- HAV is Highly infectious
- 27 nm
- Spread by the faecal – oral route

Nucleic Acid: It is a RNA virus.

Incubation Period: 2-4 wks
- Children are commonly affected.

**HEPATITIS “A” VIRUS**

![HEPATITIS “A” VIRUS](image)

**PREVENTION:**

- Active immunization with an Inactivated virus vaccine (Harrix)

**HEPATITIS “B” VIRUS**

- Only hepadna virus causing infection in humans
- DNA virus
Size - 42 nm

Incubation period - 4-20 Weeks.

mode of spread - Blood transfusion.
- Blood products
- Parenteral drug abusers
- Tattooing
- Acupuncture
- In adequately sterilised needles.
- sexual intercourse
- male homosexuals

Trans placental spread - mother to child transmission

CHRONIC INFECTION - Chronic liver disease.

PREVENTION:
- Hepatitis B vaccine containing HBs Ag (Engerix) (Active immunisation)
- Intramuscular injection of Hyperimmune serum globulin prepared from blood containing anti – HBs Should be given within 24 hours or at most a week.
- Hyper immune globulin – active – passive immunisation.

HEPATITIS “C “VIRUS
Group - Flavivirus
Nucleic acid - RNA virus
Size - 30 – 38nm
Incubation period - 2 – 26 weeks
mode of transmission - Blood products
- Parenteral drug abusers
- Sporadic infection
- Sexual and vertical transmission

CHRONIC INFECTION - Chronic liver disease
PREVENTION - No vaccination

HEPATITIS D VIRUS
Group - Defective virus
- Incomplete virus
Nucleic acid - RNA
Size - 35nm
Incubation - 6-9 wks
mode of transmission - Blood transfusion
- Sexual and vertical
CHRONIC INFECTION - Chronic carriers of the hepatitis B virus
- Acute hepatitis
- Chronic hepatitis
- Cirrhosis

PREVENTION - Prevented by prevention of hepatitis B virus infection

HEPATiTIS E VIRUS
- RNA Virus
Size - 27nm
Incubation period - 3 – 8 wks
mode of transmission - By Faecal – oral route
- Water borne hepatitis
Prevention - No vaccine.

OTHER VIRUSES:
- Cytomegalovirus
- Epstein – Barr Virus – cause abnormal liver function
- Herpes simplex
- Yellow fever virus.

CLINICAL FEATURES:
- Jaundice by a few days to 2 wks
- Chills, headache, malaise
- Anorexia
- Nausea
- Vomiting
- Diarrhoea
- Abdominal pain
- Enlarged liver
- Enlarged cervical lymph nodes
- Splenomegaly in children
- Arthralgia
- Serum Sickness syndrome
- Urticaria
- Poly arthritis
- Dark yellow urine
- Yellow tint to the sclerae

INVESTIGATIONS:
- A plasma Aminotransferase 400 U / L
- Plasma bilirubin
- Alkaline phosphatase 250 U / L
- Prothrombin time for liver damage

Serological tests:

It can Identity Hepatitis Antigens.

COMPLICATIONS:
- Fulminant hepatic failure
- Cholestatic hepatitis
- Hyper bilirubinemia (Gilbert’s Syndrome)
- A plastic anaemia
- Connective tissue disease.
- Renal failure
- Chronic hepatitis
- Cirrhosis (Hepatitis B,C and D viruses)
- Hepato cellular carcinoma.

MANAGEMENT:

BED REST:

Should be continued until symptoms and signs have disappeared.

DIET:

Glucose
Fruit drinks
Light diet
Good protein intake.
Sugar cane Juice.
NEONATAL JAUNDICE

Jaundice occurs when the liver cannot Excrete bilirubin

2 Types

i) Physiology
ii) Pathology

PHYSIOLOGY: -

Elevation of un conjugated bilirubin concentration during the first week.
1) Increase bilirubin load on liver cells.
2) Defective hepatic uptake of bilirubin from plasma.
3) Defective bilirubin conjugation
4) Defective bilirubin Excretion.

PATHOLOGICAL:

i) Conjugated hyper bilirubinemia
ii) Un conjugated hyper bilirubinemia.

Un conjugated hyper bilirubinemia.

More than 5mg / dL / 24 hours
CAUSES: -

i) Increased production.
   - Feto maternal blood group in compatibility
   - Rh, ABO
   - Hereditary Spherocytosis
   - Non-Spherocytic anaemia
   - Sepsis
   - Increased entero hepatic circulation

ii) decreased clearance.
   - Inborn errors of metabolism
   - Drugs and hormones.

COMPLICATIONS:

1. Increase in environment and body temperature
2. Retinal damage
3. Bronze baby syndrome.
4. Electric shock.

CONJUGATED HYPER BILI RUBINEMIA:

Total bilirubin Direct > 2mg / dl.

It is associated with.

ii) Biliary atresia.
   - Congenital atresia of bile duets

Clinical Features:

- Clay coloured stools
- Anaemia
- Fat soluble vitamin defiency.

Liver biopsy:

Bile plugs in dilated ducts.

KERNICTERUS:

The Jaundice is presumably a consequence of metabolic and physiological adjustments after birth. In Extreme cases a brain – damaging condition known as kernicterus.
This condition has been rising in recent years due to inadequate detection and treatment of neonal hyperbilirubinemia.

Neonatal Jaundice is a risk factor for hearing loss.

LIVER HISTOLOGY
MATERIALS AND METHODS

This study was carried out at the department of Kuzhanthai maruthuvam. To standardize the efficacy of Jeeraga Chooranam and Mookirattai ver kudineer. The following protocols are maintained as materials for the dissertation.
Identification of the trial drug for study and its confirmation.
Trial drug preparation.
Biochemical studies
Pharmacological studies
Clinical trials.

COLLECTION OF TRIAL DRUG AND IDENTIFICATION

The raw drugs and fresh plant leaves for the trial drug and collected and the botanical identity was confirmed.

PREPARATION OF TRIAL DRUG

The raw drugs and fresh plant leaves are purified and dried well and then powdered and filtered in a cloth to arrive a fine powder. The roots are dried and then Halfly powder form.

CLINICAL TRIALS

Twenty cases of both sexes in the age group below twelve years were selected from the out patient session and admitted in the post – graduate Kuzhanthai Maruthuvam ward.

EVALUATION OF CLINICAL PARAMETES

1. Cases with yellowish urine are taken for study.
2. The diagnosis was confirmed by clinical and laboratory criteria.

SIDDHA CLINICAL DIAGNOSIS

i. Mukkutra Niligal
ii. Envagai theruvagal
iii. Vdal Kattu Nilaigal
iv. Nilam
v. Kaalam
vi. Mummalam [ malam, moothiram and viyarvai]
vi. Vayathu [Age]
vi. Thega nilai (udal vanmai)

INVESTIGATION

Urine
Bile salt
Bile pigment
Hemoglobin percentage
Serum bilirubin
    Total  :
    Direct :
    Indirect :

For the purpose of differential diagnosis, the following tests were carried out
i)    SGOT
ii)   SGPT
iii)  Serum Alkaline Phosphates
iv)   Australian Antigen (HBs Ag)

CASE PROFORMA

Specific signs and symptoms of the three varieties of kamalai are given. Among the three varieties, Manjal kamalai have been taken for research study with Jeeraga Chooranam, Mookirattai ver Kudineer.

PREPARED TRAIL MEDICINES
**OBSERVATION AND RESULTS**

Results were observed with respect of the following criteria

1. Sex distribution
2. Age distribution
3. Religion distribution
4. Kaalam
5. Paruva kaalam
6. Thinai
7. Socio economic status
8. Clinical features
9. Derangement of Uyirthathukkal
10. Udal kattugal
11. Envagai thervugal
12. Neerkur, Neikuri reference

1. Sex distribution

<table>
<thead>
<tr>
<th>S.No</th>
<th>Sex</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male child</td>
<td>11</td>
<td>55%</td>
</tr>
<tr>
<td>2</td>
<td>Female child</td>
<td>9</td>
<td>45%</td>
</tr>
</tbody>
</table>

For this study 20 patients were selected 11 were male and 9 were female

Graph showing sex distribution:

2. Age distribution:

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age</th>
<th>No.of case</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0 to 1 month kappu and sengeerai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>1 month – 1 year sengeerai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>1 year – 3 years thalattu, mutham, varugai, sappani</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>3 years – 6 years Ambuli, chitri, sirupillai, Sirudher, Oonjal, pillai</td>
<td>Male 4 Female 2</td>
<td>Male 20% Female 10%</td>
</tr>
<tr>
<td>5</td>
<td>6 years – 12 years pethai, pethumbai, mangal, siruparuvam</td>
<td>Male 7 Female 7</td>
<td>Male 35% Female 35%</td>
</tr>
</tbody>
</table>

Out of 20 cases, 14 cases (70%) belongs to 6 - 12 years and 6 cases (30%) belongs to 3 – 6 years age group.

3. Religion distribution:

<table>
<thead>
<tr>
<th>S.No</th>
<th>Religion</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Hindu</td>
<td>17</td>
<td>85%</td>
</tr>
<tr>
<td>2</td>
<td>Christian</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>3</td>
<td>Muslim</td>
<td>2</td>
<td>10%</td>
</tr>
</tbody>
</table>
Out of 20 cases, 17 cases (85%) belong to Hindu, 1 case (5%) belonged to Christian and 2 case (10%) belong to Muslim region.

4. Kaalam

<table>
<thead>
<tr>
<th>S.No</th>
<th>Kaalam</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Vatham</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Pitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Kabam</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

All the cases were in vatha kaalam.

5. Paruva kaalam

Month of admission in the in-patient ward is taken for the following data

<table>
<thead>
<tr>
<th>S.No</th>
<th>Paruvakaalam</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Kaar(Avani,Puratiasi)</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>2.</td>
<td>Koothir kaalam(Iypasi,karthigai)</td>
<td>9</td>
<td>45%</td>
</tr>
<tr>
<td>3.</td>
<td>Munpani(Margali,Thai)</td>
<td>9</td>
<td>45%</td>
</tr>
<tr>
<td>4.</td>
<td>Pinpani(masi,pankuni)</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>5.</td>
<td>Elavenil(chithirai,vaikasi)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Muthuvenil(Aani,Adi)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

The maximum incidence of manjal kamalai was during koothir kaalam and munpani kaalam.

6. Thinai (place)

<table>
<thead>
<tr>
<th>S.No</th>
<th>Thinai</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Kurinji(Hilarea)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Mullai(Forest area)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Marutham(Fertile area)</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Neithal(coastal)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Palai(Deset area)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Among the 20 cases, all belonged to maruthanilam, even though the literature says no disease coil occur in “Maruthanilam”, If did not correlate well with this study.

7. Socio-economic status.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Socio-economic status</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Poor</td>
<td>18</td>
<td>90%</td>
</tr>
<tr>
<td>2.</td>
<td>Middle class</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>3.</td>
<td>Rich</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>
According to this study 90% of cases were poor and 10% of cases from middle class socio economic status.

8. Table showing clinical features

<table>
<thead>
<tr>
<th>S.No</th>
<th>Signs and Symptoms</th>
<th>During admission No.of cases.</th>
<th>During discharge No.of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Nausea</td>
<td>20</td>
<td>Nil</td>
</tr>
<tr>
<td>2.</td>
<td>Vomitting</td>
<td>15</td>
<td>Nil</td>
</tr>
<tr>
<td>3.</td>
<td>Fever</td>
<td>16</td>
<td>Nil</td>
</tr>
<tr>
<td>4.</td>
<td>Yellow coloured conjunctiva sclera and tongue</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>5.</td>
<td>Yellow coloured urine</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>6.</td>
<td>Anorexia</td>
<td>20</td>
<td>Nil</td>
</tr>
<tr>
<td>7.</td>
<td>Constipation</td>
<td>4</td>
<td>Nil</td>
</tr>
<tr>
<td>8.</td>
<td>Puritus</td>
<td>4</td>
<td>Nil</td>
</tr>
<tr>
<td>9.</td>
<td>Abdominal pain</td>
<td>16</td>
<td>Nil</td>
</tr>
<tr>
<td>10.</td>
<td>Irritability</td>
<td>20</td>
<td>Nil</td>
</tr>
<tr>
<td>11.</td>
<td>Liver tenderness</td>
<td>6</td>
<td>1</td>
</tr>
</tbody>
</table>

9. Distribution of Uyirthathukkal
a). Table showing derangement of vatham

<table>
<thead>
<tr>
<th>S.No</th>
<th>Vatham</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Pranan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Abanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Viyanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Uthanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>5.</td>
<td>Samanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>6.</td>
<td>Naagan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Koorman</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8.</td>
<td>Kirukaran</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>9.</td>
<td>Devathathan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10.</td>
<td>Thananjeyan</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

In all the 20 cases Abanan, viyanan, Uthanan, samanan and kirukaran were affected.

b) Table showing the derangement of pithamn.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Pitham</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Anal pitham</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Ranjagam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Saathagam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Prasagam</td>
<td>20</td>
<td>100%</td>
</tr>
</tbody>
</table>
5. Alosagam

Among the 5 types of pitham all were affected except Alogagam.

c) Table showing the derangement of Kabam

<table>
<thead>
<tr>
<th>S.No</th>
<th>Kabam</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Avalambagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Kilethagam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Pothagam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Tharpagam</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>5.</td>
<td>Santhigam</td>
<td>3</td>
<td>15%</td>
</tr>
</tbody>
</table>

Kilethagam and pothagam were affected in all the cases, tharpagam was affected in 2 patients santhigam was affected in 3 patients.

10. Table showing the conditions of udal kattugal

<table>
<thead>
<tr>
<th>S.No</th>
<th>Kabam</th>
<th>No of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Saaram</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Senner</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Oon</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Kozhuppu</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Enbu</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Moolai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Sukkila / suronitham</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

In all the cases saram and senneer were affected.

11. Envagai thervugal

<table>
<thead>
<tr>
<th>S.No</th>
<th>Envagai thervugal</th>
<th>No of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Naadi: Pitha kabam Kaba pitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Sparisam</td>
<td>20</td>
<td>20%</td>
</tr>
<tr>
<td>3.</td>
<td>Naa</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Niram</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>5.</td>
<td>Mozhi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Vizhi</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>7.</td>
<td>Malam</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>8.</td>
<td>Moothiram</td>
<td>20</td>
<td>100%</td>
</tr>
</tbody>
</table>

Sparism, Naa, Niram, vizhi, Moothiram were affected in all the 20 Patients. Malam was affected in 5 Patients.

12. Neerkuri, Neikuri reference
Urine samples of all the 20 cases were shows dark yellow colour. Ring (Azhal) sign in Neikuri.

The following clinical investigations were carried out at the time of admission and discharge.

1. Serum bilirubin test
2. S. G. OT
3. S.G. P.T
4. Serum Alkaline phosphatase test
5. HBs Ag

Among the 20 cases studied the results were observed as follows

<table>
<thead>
<tr>
<th>S.No</th>
<th>Results</th>
<th>No.of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Good relief</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td>2.</td>
<td>Fair relief</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>3.</td>
<td>Poor relief</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Among the 20 cases, admitted in the inpatient ward. 16 cases (80% showed good result and 4 cases (20%) showed fair result. The results were based on clinical improvement.

**DISCUSSION**

The three varieties of kamalai noi, Manjal kamalai is a clinical entity described by siddhars as pitha type of disease. Which is taken for clinical study in the post graduate department of Kuzhanthai Maruthuvam, Govt.Siddha Medical College, Palayamkottai. By its clinical features like fever, loss of appetite, nausea,
weakness yellowish discoloration of sclera. Conjunctiva, Urine mucosa and under the tongue are clinically identical with Infective hepatitis. A clinical entity described in modern medical literatures.

This study comprises primarily a survey of literatures in both siddha and modern aspects. In siddha texts like Balavagadam, Yugi Vaidhya chinthamani, Agasthiyar 2000 wealth of information of this clinical entity is available.

Modern text book like Nelson paediatrics, Essential paediatrics – O.P.Ghai, Fundamentals of paediatrics partha’s and current pediatric diagnosis and treatment Lange’s etc. were availed for modern aspects.

Siddhars – the ancient scientists, out of their experience recorded the undue exposure to sunlight, excessive intake of spices, sour, salt and pungent taste items are the causative factors. Modern medical science attributes a viral cause for this disease. Among 20 cases, the history given by most of the patients coincided much with the observations of Siddhars.

Generally in India there are many existing predisposing factors which produce jaundice. Economically poor social status and illiteracy are mainly responsible for the poor knowledge about Jaundice. Improper hygienic measures like unpreserved water supply and drainage system in most places and improperly preserved food items, contaminated needles and blood products favour the spread and development of this disease.

Siddhars laid great emphasis on Noi anuga vithi Ozhukkam.

“இனிக்கு குருக்கு வாய்க்கு மார்பாற்கு
 செய்யப் பூ வாழிக்குரு – வாய்க்கு பிடி”

- பரந்தக்குகள் திரும்புவரை

Non scientific treatment of jaundice like salt restriction undue starvation, lead to complications and thus increasing morbidity and mortality. Alcoholism and administration of hepato – toxic drugs cause damage to the liver, lack of facilities for routine screening of HBs Ag in blood donors, and commercial sexual workers play an important role in the spread of Infections jaundice. Accurate labeling of jaundice in every case is essential to study the nature and to take preventive measures.

This has given a clue that extensive experimental studies by researchers of bio-medical fields on the above points may bring new facts on the susceptibility of the individual for the development of this particular clinical entity. Among 20 patients admitted, 14 patients had given a history of contact with infected persons, and 6 patients given a history of persistent exposure to sunlight, and all had taking excessive spicy, sour and pungent taste items.
AGE, SEX AND SOCIO-ECONOMIC STATUS

Among the 20 patients, 6 patients belonged to the age group of 3 – 6 years, 14 patients belonged to the age group of 6-12 years, the entire group of patients belonged to the vatha period.

Among 20 cases admitted 11 were male children and 9 were female children. Among 20 cases admitted for study, most of them belonged to the low income group.

PARUVAKAALAM

Pitham ‘thannilai valarchi adaithal’ (setting its season) period is Kaar kaalam, piranilai valarchi adaithal period is Koothir kaalam. In this study out of 20 patients, 1 patients were treated in Muthuvenil Kaalam, in this study out of 20 patients, 1 patients were treated in Muthuvenil kaalam, 9 patients were treated in koothir kaalam and 9 patients were treated in Munpani kaalam. The modern medical science also states that infective hepatitis commonly occurs in the autumn (September and October) (Third season of the year i.e., August, September and October).

AETIOLOGY

Generally from the history given by the patients, this disease is due to the dietetic, extrinsic and other factors (described in Siddha aspect), which also cause vitiation of Pitham and kabam. Siddhars have also stated the same cause described above for this disease Manjal kamalai that it is due to the derangement of Pitham, that affects the blood and forming this disease. The etiology of Manjal kamalai is based upon the Mukkutram is Siddha system of medicine.

In modern medical science, it is based upon the viral infection. The mode of infection is through stools contaminated water, blood transfusion and sexual act. Anatomical and physiological changes take place in the liver due to this infection.

In both systems, it is stated that any factor which causes damage to the liver, produce this disease. Hence the causes of this disorder given in Siddha system is more or less corresponding to that of modern medical science.

NILAM (FIVE KINDS OF LANDS)

Almost all the cases came from marutha nilam. This may be due to the location of the hospital is at Tirunelveli. So in order to access the geographical distribution in its true perspective a wider study is felt essential, which may be taken by future workers as a separate study. According to Siddhars thought marutha nilam is the safest place to maintain good health. But nowadays air pollution, water contamination and improper drainage have spoilt it.
No history of dyspnoea was given by any patient. So there is no involvement of Piranan.

In some of the cases there was history of constipation and hence involvement of Abanan.

In some cases complaints of body pain and pruritus were present. So involvement of Viyanan is inferred.

Nausea and vomiting were present in all cases. It shows involvement of Uthanan.

Loss of appetite, indigestion was present in all cases. So involvement Samanan.

None of the case was affected by Nagan.

None of the case was affected by Koorman.

In all cases nausea, vomiting, and salivation from the mouth were also present. It shows involvement of Kirukaran.

Devathathan was not affected in Manjal Kamalai.

PITHAM:
Loss of appetite and dyspepsia were recorded in all cases. It shows involvement of anal pitham.

RANJAGAM
In all the cases serum bilirubin was above normal. So involvement of Ranjaga pitham.

SATHAGAM
In all the cases mental depression was present. So involvement of sathagam.

ALOSAGAM
No history of disturbed vision was noted in any case. So no involvement Alosagam.

PRASAGAM
There was yellowish discoloration of the conjunctiva, sclera and body with dry skin noted in all cases. It shows involvement of Prasagam.

KABAM
AVALAMBAGAM
There is no sign of respiratory involvement in all cases. It shows that avalambagam was not affected.

KILETHAGAM
There is a feature of dyspepsia in all cases. So the involvement of kilethagam.

POTHAGAM
In all cases complaints of bitter taste sensation were present. It shows involvement of Pothagam.

THARPAGAM
Among 20 cases 3 patients complains, there was burning sensation their eyes. So involvement of Tharpagam.

SANTHIGAM
Among 20 patients, 2 patients complaints of joint pain. So Santhigam was affected.

UDAL KATTUGAL
As per the signs and symptoms seen in 20 cases, the Saaram and senneer were affected in all cases. But the literature says if any one of the thathu was affected, others were also affected.

ENVAGAI THERUVUGAL
NAADI

“அச்சுவரைம் ஒளித்திரியாளர் விளங்கிய பாதை அறநூற்று கால்வாய்வு வடிவில்லாதோ?”

- விழாவியன் பத்தை 1 (பத்து வருவாய்)

சுருக்கு நுண்மை நிலையானது நான் பெருந்தோற்ற காலாக வணந்தை விளங்கியது.

SPARISM

In Manjal kamalai temperature was elevated at the time of onset. That is due to excessive pitham.

According to modern medical science. Fever was present during the onset of this disease due to viral infection. Palpable liver and tenderness was noted in Manjal kamalai.

NAA

In Manjal Kamalai excessive salivation was present with bitter taste. Tongue is yellow in colour. These are due to involvement of Pitham. These symptoms were noted in all patients.

NIRAM

In all the cases due to involvement of Pitham, the body was mildly yellow in colour.

MOZHI

Not affected in all 20 cases.

VIZHI

Vizhi was yellow in colour in all cases due to involvement of Pitham.

MALAM (stools)
In Manjal Kamalai, due to involvement of Pitham and Vatham, the malam was constipated. The stools was pale yellow in color.

MOOTHRAM (urine)

In all the cases the Moothiram was dark yellow in colour, due to the increased Pitham.

NEIKKURI

Among 20 cases, the neikkuri was just like the ring. It was defined that in the Pitha patient’s urine will show ring shape. Hence this Manjal Kamalai is also a pitha type of disease it produced the ring shape.

In infective hepatitis, bile salts and bile pigments were present in all 20 cases. Urobilinogen was increased in all cases. In obstructive Jaundice there is absence of urobilinogen. Hence 20 cases were diagnosed as infective hepatitis. There are many liver function tests described in modern medicine. But they were not carried out due to lack of laboratory facilities.

UDAL VANMAI

In Manjal Kamalai, Iyarkai vanmai and Kaalavanmai are affected all patients.

CLASSIFICATION

IN BALAVAGADAM, Kamalai is classified into 3 types. Manjal Kamalai is one among the 3 types.

In Yugivaidya Chithamani Kamalai classified into 13 varieties, among 13 types, Manjal Kamalai is the commonly presented entity.

SIGNS AND SYMPTOMS OF MANJAL KAMALAI

i) Loss of appetite.
ii) Yellowish discoloration of the conjunctiva, sclera, urine under surface of tongue, buccal mucosa, and skin which are visible in natural daylight.
iii) Constipation or Diarrhoea. Mental depression.
iv) Body pain

Besides the common signs and symptoms of kamalai, the above mentioned signs and symptoms are noted in all 20 cases. Hence it was diagnosed as Manjal Kamalai

TREATMENT

In all 20 cases, mookirattai ver kudineer 30 ml (1-6 age group) and 60ml (7-12 age group) in every 4th hourly jeeraga chooranam. 1gm 3 times a day with hot water or butter milk.
Regarding this Manjal Kamalai, among these vital forces, Pitham is affected first, and then the other vital forces are deranged. So the main principle in treatment aspects is to make the deranged Pitham to be in their normal places, and carry out normal functions by giving the medicines. Nausea and vomiting are among of the symptoms of Manjal Kamalai. So no need to give medicine for vomiting.

The trial medicine of Mookirattai ver kudineer and jeeraga chooranam having the tastes namely kaippu ;inippu and thuvarppu and these are said to be useful in correcting the deranged pitham and kabam. It is described in Kannusamiyam as follows.

Mookirattai Ver kudineer is bitter is taste thuvarpu),and jeeraga chooranam, inippu,and kaippu so it is very useful in pitha type of diseases.

The bio-chemical investigations have revealed the following facts. The trial medicine of Mookirattai ver kudineer contains tannic acid, starch, reducing sugar and unsaturated compound. But there is no minerals like calcium, phosphate, sulphate, chloride and Iron. All the so patients were advised to take rice sugar cane juice and tender coconut water. Liver plays an important role in metabolism of carbohydrate, protein and fat. Excessive carbohydrate is convert into fat and glycogen. When liver function is affected the synthesis and storage of glycogen is not possible similarly in case of Jaundice the absorption of fat from the intestine is also affected.

Therefore, the body needs under these circumstances perennial supply of reducing sugar for its various metabolic activities. Brain can utilize only the glucose. Sugar can juice is mainly a disaccharide consisting of glucose and fructose. In the intestine the sugar is broken down into constituent by the enzyme invertase present in the surface of epithelium of microvili. It is easily digestend even in jaundice condition. Hence the importance has been attached to the intake of sugar cane juice during the onset of Jaundice.

In the clinical side rice caunji with fat free boiled milk is the food to the patients who suffer from Manjal Kamalai.

When the medicine has been administered in the form of mookirattai ver Kudineer,and jeeragachooranam the patients responded very well. So the
medicine has given a good clinical cure. Nobody shows signs of complications like gastro-intestinal bleeding, encephalopathy and ascites.

Despite the common belief that an uncomplicated cases of viral hepatitis may recover even by complete rest with a prescribed diet regimen, the uniform recovery with remarkable clinical and bio-chemical improvement in above cases had helped to arrive at the view that jeeragachooranam and mookirattai ver kudineer possessing properties capable to normalizes Pitham with a particular hepato-protective effect.

SUMMARY AND CONCLUSION

Manjal kamalai a pitha type of disorder with considerable involvement of liver and gall bladder and wide constitutional features was taken for this study.

The clinical features of the manjal kamalai resembles the clinical entity of infective hepatitis, various literatures collected in siddha and modern side which are dealing the disease.
In this respect well known siddha medical literature’s like bala vagadam, yugi chindamani, Agushtiyar-2000 Siddha maruthuvam, Noi Nadal part II and in modern science. Text book of pediatirics – Nelson, Essential pediatrics- O.P Ghai, current paediatric diagnosis and treatment – lange’s were availed for sources related to Manjal kamalai and its counter part of the infective hepatitis.

20 cases were admitted for study in the In-patient department. Among the patients 11 Males and 9 female.

The diagnosis of this disease were made on the basis of eight types of clinical investigations, and mukkuttram mentioned in siddha literature with concurrent practical application of modern clinical and laboratorial approach.

A traditional proprietary of siddha medicines tried in the form of chooranam and kudineer, which is prepared by dried plants of manjal karisalai, chukku, Jeeragam and sugar. And dried roots of mookirattai.

All the patients were responding well from the beginning with remarkable signs of improvement.

The important observation during this study has been shown that no patients were developed or reported any features of further complications like coma, Intestinal hemorrhage etc.

The occurrence of this disease was marked in kaar, Koothir and mudhuvenil kalam.

All the patients were corresponded to report any untoward developments after their discharge.

The common belief of the public is that the siddha medicines and diet prescribed are more effective in the treatment of manjal kamalai is once again established by the clinical study.

The cost of the medicine used in the treatment of jaundice is very low, that is very cheap when compared with the cost of other medicines used in the treatment of jaundice. The plants, manjal karisalai and Jeeragam, Chukku, sugar and Mookirattai available and cultivated.

The adverse side effects are nil in the treatment of jaundice by the Siddha medicine.
GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI – THRUNELVELI – 627 002.

Br. IV KUZHANTHAI MARUTHUVAM POST GRADUATE
DISSERTATION STUDY ON “MANJAL KAMALAI”

<table>
<thead>
<tr>
<th>Ward</th>
<th>:</th>
<th>Place of birth</th>
<th>:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ip.No</td>
<td>:</td>
<td>Religion</td>
<td>:</td>
</tr>
</tbody>
</table>
Bed.No : Nationality :
Name : Admitted on time :
Age : Discharge on time :
Sex : Diagnosis :
Father’s Name : Medical Officer :
Occupation :
Income :
Address :
Informant :

Complaints and duration
History of present illness
History of previous illness

Birth History:
1. Antenatal history :
2. Prenatal history :
3. New natal history :

Development history :
Nutritional history :
Immunization :
Family history :
Environmental and social history :

SIDDHA ASPECT

GENERAL CONDITION ON ADMISSION
GENERAL APPEARANCE
CONDITION OF 7 UDAL KATTUGAL:
1. Saram
2. Sennear
3. Oon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam / Suronitham

**ENNVAGAI THERVUGAL**
1. Naa
2. Niram
3. Mozhi
4. Vizhi
5. Malam
6. Moothiram
7. Sparism
8. Nadi

**MUKKUTTRANGAL**

**I. Vatham:**
1. Piranan
2. Abanan
3. Viyanan
4. Udhanan
5. Samanan
6. Nagan
7. Koorman
8. Kirugaran
9. Devathanathan
10. Dhananjeyan

**I. PITHAM**
1. Anilam
2. Ranjagam
3. Sathagam
4. Prasagam
5. Alosagam

**II. KABAM**
1. Avalambagam
2. Kilethagam
3. Pothagam
4. Tharpagam
5. Santhigam

KANMAENTHIRIYANGAL:

1. Kai
2. Kal
3. Vai
4. Eruvai
5. Karuvai

PORI PULANGAL:

1. Sound (Ear)
2. Light (Eye)
3. Sensation (Skin)
4. Taste (Tongue)
5. Smell (Nose)

GUNAM:

1. Sathuva Gunam
2. Raso Gunam
3. Thamo Gunam

MALANGAL

1. Malam
2. Moothiram
3. Viyarvai

NILANGAL

1. Kurinji
2. Mullai
3. Marutham
4. Neithal
5. Palai

PARUVA KALANGAL
1. Kaar
2. Koothir
3. Munpani
4. Pinpani
5. Illavenil
6. Mudhuvenil

Neer kuri

Nei Kuri

MODERN ASPECTS

GENERAL EXAMINATIONS:
Height:
Weight:
Head circumference
Mid arm circumference
General appearance
State of nutrition
Skin colour
Cyanosis
Jaundice
Pallor
Erythema
Lymphadenopathy
Clubbing
Pedal oedema

**VITAL SIGNS:**

- Temperature
- Pulse Rate
- Respiratory Rate
- Blood pressure
- Chest
- CVS
- RS

**SYSTEMIC EXAMINATION**

1. Examination of the abdomen
   a) Inspection: size, shape, distended veins, position of umbilicus
   b) Palpation: Liver, Spleen, Gallbladder and tenderness.
   c) Percussion: Fluid Hrill, shifting dullness, stony dullness
   d) Auscultation: Bruits or Bowel sings

**EXAMINATION OF OTHER SYSTEM:**

**SPECIAL INVESTIGATIONS**

Total W.B.C Count:
Differential W.B.C count:
Erythrocyte sedimentation Rate:
Hemoglobin percentage
Total serum bilirubin
Direct:
Indirect:
Total serum protein
Australian Antigen (HBs Ag)

**LIVER FUNCTION TEST**

SGOT
SGPT
SAP

x-ray:
plain abdomen
USG: Abdomen

URINE:
Albumin
Sugar
Bile salts
Bile pigments
Urobilinogen

MOTION:
Color
Ova
Cyst
Occult blood

TREATMENT:

ADVICE

PROGRESS OF THE PATIENT:

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL
PALAYAMKOTTAI – THRUNELVELI – 627 002.

Br. IV KUZHANTHAI MARUTHUVAM
POST GRADUATE
DISCHARGE SHEET FOR “MANJAL KAMALAI”

Name : Ward :
Age / Sex : Ip.No :
Occupation / Income : Bed. No :
Date of Admission : 
Date of Discharge : 
Diagnosis : 
Signs and Symptoms During Admission During Discharge
Fever
Nausea
Vomiting
Loss of appetite
Yellow colored urine
Yellow colored conjunctiva
Pale colored motion
General weakness
Abdominal pain
Tender and palpable liver
Constipation
Anemia
Pruritus
Diarrhoea

ANNEXURE – II
PREPARATION AND PROPERTIES OF TRIAL MEDICINE

Name of the trial medicine: “JEERAGA CHOORANAM”

INGREDIENTS: MANJAL KARISALAI – 2 part
Jeeragam – 2 part
Chukku – 1 part
Sugar – 1 part

PREPARATION OF THE TRIAL MEDICINE:
The raw drugs and manjal karisalai juice are dried and powdered well and Filtered is a cloth to get a fine powder and preserved in an air tight container.

**DOSAGE:**

1 – 2 gram
3 times a day, After food, 7 days.
Adjuvant : Butter milk, Hot water
Indication : Manjal kamalai
Duration : 3 months
Reference book : Gunapadam mooligai vaguppu

**PROPERTIES OF THE INGREDIENTS**

<table>
<thead>
<tr>
<th>1. ஹேரோகம்</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botanical Name : Cuminum Cyminum</td>
</tr>
<tr>
<td>Family : Apiaceae</td>
</tr>
<tr>
<td>Suvai : Inippu</td>
</tr>
<tr>
<td>Thanmai : Thatptham</td>
</tr>
<tr>
<td>Pirivu : Inippu</td>
</tr>
<tr>
<td>Part used : Seeds</td>
</tr>
</tbody>
</table>

**ACTIONS:**

Carminative
Stimulant
Stomachic
Astringent
Antimicrobial

**CONSTITUENTS:**

Thymine – Essential oil

\[
\text{Cuminol, Cymene} \quad \text{\{ Therphenes} \]


Henricia contanal, 14 – hepta consanol and stigmastorol Isolated from roots.


Whole plant is tonic and deobstrucent is hepatic and splenic enlargements and is emetic. Plant juice is combination with aromatics is administered for catarrh and Jaundice.

- Chopra et. At. 1956

- Indian material medica by Dr. Nadkarni 1992,472.

**General properties.**
**Botanical Name:** Wedelia Calendulacea  
**Family:** Compositae  
**Suvai:** Kaippu  
**Thanmai:** Veppam  
**Pirivu:** Karppu  
**Part used:** Whole plant

**ACTIONS:**  
Hepato tonic  
Alternative  
Hematinic  
Diuretic  
Cholagogue  
Tonic  
Febrifuse

**CONSTITUENTS:**

Reducing sugars in the alcoholic extract of leaves and whole plant. Presence of sterols was confirmed in petroleum ether extract of seeds. Wedelactone was obtained from the leaves and stem. The alcoholic solution of the crystals gave dark green colour with Ferric chloride. The crystals were soluble in warm pyridine.

Juice of the leaves of yellow flowered variety is administered in teaspoonful doses in jaundice and fever. Traditionally the plant is extensively used against jaundice.

The plant is also found to be effective in the treatment of infective hepatitis. And effective against liver injury and inflammation having anti hepatotoxic effect. The isolated constituents of this plant exhibited strong activity against liver damage induced by chemicals.

Wedelia has been largely used in the treatment of derangement of liver and gall bladder. The plant contained wedelolactone and desmethyl wedelolactone besides sulphur containing peptides.

The alcoholic extract of the entire plant has been reported to have anti – viral – activity.
GENERAL PROPERTIES:

Botanical Name : Zingiber officinale
Family : Zingiberaceal
Suvai : Karppu
Thanmai : Veppam
Pirivu : Karppu
Part used Rhizome

ACTIONS:
Stimulant
Carminative
Dyspepsia and colic
Digestive
Appetizer
Rubifacient
Stomachic

CONSTITUENTS:
The characteristic odor and flavor of ginger root is caused by a mixture of zingerone, shoagoles and gingerols. In laboratory animals the gingerols increase the motility of the gastrointestinal tract and have Analgesic, sedative. Anripyretic and antibacterial properties.

Ginger is may also decrease joint pain from arthritis and may have blood thinning and cholesterol lowering properties. And useful treating heart disease.

DIARRHOEA:
Zingerone is likely to be the active constituent against enterotoxigenic Escherichia coli heat – labile enterotoxin – Induced diarrhea.

Nausea:
Ginger has multiple studies for treating nausea.
Botanical Name : Saccharum officinarum  
Family : Poaceae  
Suvai : Inippu  
Thanmai : Seetham  
Pirivu : Inippu  
Part used : Saccharum juice, Sugar

ACTIONS:  
   Antiseptic  
   Demulcent

CONSTITUENTS:  

Sachearine, matter, water, mucilage, resin Fat, albumin and Vit C.

It is a good remedy in cough, hiccup, aphthae and hoarseness and locally in granulations of the eyelids and cornea. It is chiefly used as a vehicle to disguise the taste of various nauseous medicines and chiefly of medicines given to children.

It is largely used as a preservative and antiseptic to protect active ingredient and certain iron preparations against oxidation and putrefaction but not against fermentation.

GENERAL PROPERTIES:

BOTANICAL NAME :   Boerhaavia Diffusa  
Family : Nyctaginaceae

Common Trade Name : Pigweed  
Suvai : Kaippu  
Thanmai : Veppam  
Pirivu : Karppu
**ACTIONS:**

<table>
<thead>
<tr>
<th>Main actions</th>
<th>Other Actions</th>
<th>Standard Dosage</th>
<th>Decoction:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protects liver</td>
<td>Detoxifies</td>
<td>Leaves, Root</td>
<td>1 cup 1-3 times daily</td>
</tr>
<tr>
<td>Supports liver</td>
<td>Increases bile</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduces inflammation</td>
<td>Cleanses blood</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relieves pain</td>
<td>Stops convulsions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supports kidney</td>
<td>Kills bacteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increases urination</td>
<td>kills amebas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stops’ bleeding</td>
<td>kills viruses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lowers blood pressure</td>
<td>Stimulates milk flow</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Mildly laxative**

Brazil’s leading medical herbalists reports Erva tostao is a plant medicine of great importance extra ordinarily beneficial in the treatment of liver disorders.

It is employed in Brazilian herbal medicine to stimulate the emptying of the gall bladder, as a diuretic for all types of liver disorders [Including jaundice and hepatitis] gallbladder pain and stones urinary tract disorders, renal disorders.

In Ayurvedic herbal medicine systems in India the roots are employed as a diuretic, Digestive laxative, internal inflammation of all kinds, oedema Jaundice, menstrual problems, anemia and liver, gallbladder and kidney disorders.

Throughout the tropics, Erva tostae is considered an excellent natural remedy for guinea worms.

The novel alcaloids found in Erva tostao have been documented with immune modulating effects.

In one study the alkaloid fraction of the root evidenced a dramatic effect in reducing an elevation cortisol levels under stressful conditions [cortisol is an inflammatory chemical produced in the body in an immune response].

Boerhaavic acid, Borhavine, heptadecyclic acid, histidine, punarnavine, sitosterols, stigmasterol are present.

Erva tostae has long been used in traditional medicine systems as a diuretic [To increase Urination] In Traditional preparation for a general liver tonic.
BIO-CHEMICAL ANALYSIS OF SEERAGA CHOORANAM

PREPARATION OF THE EXTRACT

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

QUALITATIVE ANALYSIS

<table>
<thead>
<tr>
<th>S.NO</th>
<th>EXPERIMENT</th>
<th>OBSERVATION</th>
<th>INFERENCE</th>
</tr>
</thead>
</table>

1. **TEST FOR CALCIUM**  
2ml of the above prepared extract is taken in a clean test tube. 2 ml of 4% Ammonium oxalate solution is added to it.  
No white precipitate is formed  
Absence of calcium

2. **TEST FOR SULPHATE:**  
2ml of the extract is added to 5% barium chloride solution.  
No white precipitate is formed  
Absence of sulphate

3. **TEST FOR CHLORIDE**  
The extract is treated with silver nitrate solution.  
A white precipitate is formed  
Indicates the presence of chloride

4. **TEST FOR CARBONATE**  
The substance is treated with concentrated HCL.  
No brisk effervescence is formed  
Absence of carbonate

5. **TEST FOR STARCH**  
The extract is added with weak iodine solution.  
Blue color is formed  
Indicates the presence of starch

6. **TEST FOR IRON-FERRIC**  
The extract is treated with concentrated Glacial acetic acid and potassium Ferro cyanide.  
No blue color is formed  
Absence of ferric Iron.

7. **TEST OF IRON FERROUS:**  
The extract is treated with concentrated Nitric acid and ammonium thio cyanaate.  
Blood red color is formed  
Indicates the presence of Ferrous Iron

<table>
<thead>
<tr>
<th>S.NO</th>
<th>EXPERIMENT</th>
</tr>
</thead>
</table>
| 8.   | TEST FOR PHOSPHATE  
The extract is treated with ammonium Molybdate and concentrated nitric acid. | No yellow precipitate is formed | Absence of Phosphate |
9. **TEST FOR ALBUMIN**
The extract is treated with Esbach’s reagent.  
No Yellow precipitate is formed  
Absence of Albumin

10. **TEST FOR UNSATURATION**
Potassium permanganate solution is added to the extract.  
No blue black precipitate is formed  
Absence of Tannic acid

11. **TEST FOR THE REDUCING SUGAR**
5ML of Benedict’s qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.  
It gets decolorized  
Indicates the presence of unsaturated compound

12. **TEST FOR THE REDUCING SUGAR**
5 ML of Benedict’s qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.  
No colors change Occurs  
Absence of Reducing Sugar

13. **TEST FOR AMINO ACID:**
One or two drips of the extract is placed on a filter paper and dried it well. After drying, 1% Ninhydrin is sprayed over the same and dried it well.  
Violet colors is formed  
Indicates the presence of Amino acid

---

**BIO-CHEMICAL ANALYSIS OF MOOKIRATTAI VER KUDINEER**

**PREPARATION OF THE EXTRACT**

5gms of **MOOKIRATTAI VER KUDINEER** was weighed accurately and placed in a 250ml clean beaker. Then 50ml distilled water was added and dissolved well. Then it was boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is made up to 100ml with distilled water. This fluid was taken for analysis.
# QUALITATIVE ANALYSIS

<table>
<thead>
<tr>
<th>S.NO</th>
<th>EXPERIMENT</th>
<th>OBSERVATION</th>
<th>INFERENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>TEST FOR CALCIUM</td>
<td>No white precipitate is formed</td>
<td>Absence of calcium</td>
</tr>
<tr>
<td></td>
<td>2ml of the above prepared extract is taken in a clean test tube. 2 ml of 4% Ammonium oxalate solution is added to it.</td>
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</tr>
<tr>
<td>2.</td>
<td>TEST FOR SULPHATE:</td>
<td>No white precipitate is formed</td>
<td>Indicates the presence of sulphate</td>
</tr>
<tr>
<td></td>
<td>2ml of the extract is added to 5% barium chloride solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>TEST FOR CHLORIDE</td>
<td>A white precipitate is formed</td>
<td>Indicates the presence of chloride</td>
</tr>
<tr>
<td></td>
<td>The extract is treated with silver nitrate solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>TEST FOR CARBONATE</td>
<td>No brisk effervescence is formed</td>
<td>Absence of carbonate</td>
</tr>
<tr>
<td></td>
<td>The substance is treated with concentrated HCL.</td>
<td></td>
<td></td>
</tr>
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<td>5.</td>
<td>TEST FOR STARCH</td>
<td>Blue color is formed</td>
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<td>6.</td>
<td>TEST FOR IRON-FERRIC</td>
<td>No blue color is formed</td>
<td>Absence of ferric Iron.</td>
</tr>
<tr>
<td></td>
<td>The extract is treated with concentrated Glacial acetic acid and potassium Ferro cyanide.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>TEST OF IRON FERROUS:</td>
<td>Blood red color is formed</td>
<td>Indicates the presence of Ferrous Iron</td>
</tr>
<tr>
<td></td>
<td>The extract is treated with concentrated Nitric acid and ammonium thio cyanate.</td>
<td></td>
<td></td>
</tr>
</tbody>
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<td>The extract is treated with ammonium Molybdate and concentrated nitric acid.</td>
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<td>No Yellow precipitate is formed</td>
<td>Absence of Albumin</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>---</td>
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</tr>
<tr>
<td><strong>10.</strong></td>
<td><strong>THST FOR UNSATURATION</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Potassium permanganate solution is added to the extract.</td>
<td>No blue black precipitate is formed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Absence of Tannic acid</td>
<td></td>
</tr>
<tr>
<td><strong>11.</strong></td>
<td><strong>TEST FOR THE REDUCING SUGAR</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5ml of Benedict’s qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.</td>
<td>It gets decolorized</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indicates the presence of unsaturated compound</td>
<td></td>
</tr>
<tr>
<td><strong>12.</strong></td>
<td><strong>TEST FOR THE REDUCING SUGAR</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5ml of Benedict’s qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.</td>
<td>No colors change Occurs</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Absence of Reducing Sugar</td>
<td></td>
</tr>
<tr>
<td><strong>13.</strong></td>
<td><strong>TEST FOR AMINO ACID:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One or two drips of the extract is placed on a filter paper and dried it well. After drying, 1% Ninhydrin is sprayed over the same and dried it well.</td>
<td>No Violet colors is formed</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Absence of of Amino acid</td>
<td></td>
</tr>
</tbody>
</table>

**ANNEXURE – IV**

**PHARMACOLOGICAL ANALYSIS**

**HEPATO – PROTECTIVE STUDY OF JEERAGA CHOORANAM**
AIM: To analyse the hepato-protective study of

1. JEERAGA CHOORANAM

Materials and Methods

Two groups of albino rats each consisting of 3 rats were used in this study. One group (A) of Albino rats received CCL4 0.2 ml/100mg body weight subcutaneously. The second group (B) received CCL4 0.2 ml /100 gm body weight with trial drug JEERAGA CHOORANAM for 8 days continuously.

On the ninth day the animals were sacrificed and liver lobes excised for histopathological studies.

HISTOPATHOLOGICAL STUDIES

1. In the CCL4 given group there was marked liver damage. Both the sample liver shows hepatocytes with lobular disarray. There is ballooning of the cytoplasm with plenty of fat vacuoles. In focal areas they coalesce to form fatty cysts. Few of the hepatocytes are completely replaced by fat vacuoles. Minimal fibrosis was seen extending form portal tract.

2. In group that received CCL4 and drug an over all protection from CCL4 damage was observed. Both the liver section shows normal hepatocytes arranged around central vein with mild dilatation. Sinuses appear dilated with prominent kupffer cells. Portal tracts appear expanded.

3. These two groups of liver section were compared to the normal histological appearance of the rat liver.

IMPRESSION

1. Group A: Moderate to severe fatty changes.
2. Group B: Non specific changes.

CONCLUSION:
The test drug have hepato-protective action against CCL4 induced liver damage.

ANTI- PYRETIC STUDY OF MOOKIRATTAI VER KUDINEER

AIM: To study anti-pyretic activity of Mookirattai ver kudineer.

PROCEDURE:

Group of six Albino rats were selected and divided equally in to 2 groups all the rats were made hyperthermic by subcutaneous injection of 12% suspension of
yeast at a dose of 1 ml/100 ml of body weight. 10 Hors later one group of animals was given the test drug by gastric tube at a dose of 20 mg/ml and the second group received only distilled water at a dose of 2 ml.

The mean rectal temperature for the two groups were recorded at 0 hr, 1 1/2 hrs, 3 hrs and 4 1/2 hrs after the drug administration. The difference between the mean temperature of the control group and that of the other group was measured.

**STUDY OF ANTI PYRETIC BY YEAST INDUCED METHOD USING THE DRUGS OF MOOKIRATTAI VER KUDINEER**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of Drugs / groups</th>
<th>Dose / 100 gram body weight</th>
<th>Initial Temperature in centigrade</th>
<th>After Drug Administration</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 1/2 hour</td>
<td>3 hours</td>
</tr>
<tr>
<td>1.</td>
<td>Control (Water)</td>
<td>1 ml</td>
<td>36.0 37.0</td>
<td>36.0 37.0</td>
<td>36.0 38.0</td>
</tr>
<tr>
<td>2.</td>
<td>Standard (Paracetamol)</td>
<td>20 mg</td>
<td>37.0 38.0</td>
<td>37.0 37.0</td>
<td>36.5 36.5</td>
</tr>
<tr>
<td>3.</td>
<td>Mookirattai ver Kudineer</td>
<td>2ml</td>
<td>37.0 36.0</td>
<td>37.0 36.0</td>
<td>36.5 35.5</td>
</tr>
</tbody>
</table>

**Significant Action**

**INFERENCE**
Mookirattai ver kudineer has good anti – pyretic action. Significant action.

**STUDY OF ANTI PYRETIC BY YEAST INDUCED METHOD USING THE DRUGS OF JEERAGA CHOORANAM**
<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of Drugs / groups</th>
<th>Dose / 100 gram body weight</th>
<th>Initial Temperature in centigrade</th>
<th>After Drug Administration</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1 ½ hour</td>
<td>3.0 hours</td>
<td>4 ½ hours</td>
</tr>
<tr>
<td>1.</td>
<td>Control (Water)</td>
<td>1 ml</td>
<td>36.0 37.0</td>
<td>36.0 37.0</td>
<td>36.0 38.0</td>
</tr>
<tr>
<td>2.</td>
<td>Standard (Paracetamol)</td>
<td>20 mg</td>
<td>37.0 38.0</td>
<td>37.0 37.0</td>
<td>36.5 36.5</td>
</tr>
<tr>
<td>2.</td>
<td>Jeeraga chooranam</td>
<td>100 mg</td>
<td>36.0 36.0</td>
<td>36.0 36.0</td>
<td>35.5 35.5</td>
</tr>
</tbody>
</table>

Significant Action

**INFERENCE**

JEERAGA CHOORANAM has good anti – pyretic action.significant action.
To study the acute anti-inflammatory effect JEERAGA CHOORANAM & MOOKIRATTAI VER KUDINEER.

PREPARATION OF THE TEST DRUG

50gm of the test drug was dissolved in 250 ml of water, boiled and reduced into 50 ml. A dose of 2 ml was given to each rat.

PROCEDURE:

Nine healthy albino rats weighing 100 – 150 gm were taken and divided into three groups, each consisting of 3 rats.

First group was kept as control by giving distilled water of 2ml/100 gm of body weight. The second group was given ibuprofen at dose of 20 mg /100 gm body weight. The third group received the test drug JEERAGA CHOORANAM & MOOKIRATTAI VER KUDINEER 2ml/100 gm body weight.

Before administration of test drug, the hind-paw volumes of all rats were measured. This was done by dipping the hind-paw up to tibio-tarsal junction into a mercury plethyskography. While dipping the hind-paw, by pulling the syringe piston, the level of mercury in the centre small tube was made to coincide with red marking and reading was noted from the plethysmograph.

Soon after measurement, the drugs were administered orally. One hour later, a sub-cutaneous injection of 0.1 ml of 1% (W/V) carrageenin in water was made into planter surface of both hind-paw of each rat. Three hours after carrageenin injection, the hind-paw volumes were measured once again. The difference between in the initial and final volume was calculated and compared.

The method is more suitable for studying the anti-inflammatory activity in acute inflammation. The values are given in the table.

### STUDY OF ACUTE ANTI-INFLAMMATORY BY HIND PAW METHOD USING PLETHYSMOGRAPH USING THE DRUG ON MOOKIRATTAI VER KUDINEER

<table>
<thead>
<tr>
<th>S.N o</th>
<th>Name of Drug /groups</th>
<th>Dose/10 Gram body weight</th>
<th>Initial Reading average</th>
<th>Final Reading Averag</th>
<th>Mean Difference</th>
<th>Percentage Inflammation</th>
<th>Percentage Inhibition</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.No</td>
<td>Name of Drug /groups</td>
<td>Dose/100 Gram body weight</td>
<td>Initial Reading average</td>
<td>Final Reading Average</td>
<td>Mean Difference</td>
<td>Percentage Inflammation</td>
<td>Percentage Inhibition</td>
<td>Remarks</td>
</tr>
<tr>
<td>------</td>
<td>----------------------</td>
<td>---------------------------</td>
<td>-------------------------</td>
<td>-----------------------</td>
<td>-----------------</td>
<td>-------------------------</td>
<td>-----------------------</td>
<td>---------</td>
</tr>
<tr>
<td>1.</td>
<td>Control(water)</td>
<td>2ml</td>
<td>0.55</td>
<td>1.4</td>
<td>0.85</td>
<td>100</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Standard (IbuBrufen)</td>
<td>20mg</td>
<td>0.55</td>
<td>0.85</td>
<td>0.3</td>
<td>35.2</td>
<td>64.8</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Mookrattai ver Kudineer</td>
<td>2ml</td>
<td>0.7</td>
<td>1.0</td>
<td>0.3</td>
<td>35.2</td>
<td>64.8</td>
<td></td>
</tr>
</tbody>
</table>

Significant Action

STUDY OF ACUTE ANTI-INFLAMMATORY BY HIND PAW METHOD USING PLETHYSMOGRAPH USING THE DRUG ON JEERAGA CHOORANAM

STUDY OF CHRONIC ANIT-INFLAMMATORY EFFECT BY COTTON PELLET METHOD USING THE DRUGS OF JEERAGA CHOORANAM
**REFERENCE**

The test drug has significant action in acute anti-inflammatory action.

**STUDY OF CHRONIC ANIT-INFLAMMATORY EFFECT BY COTTON PELLET METHOD USING THE DRUGS OF MOOKIRATTAI VER KUDINEER**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of Drugs/Group</th>
<th>Dose/100 gm body weight</th>
<th>Pellet Weight</th>
<th>Pellet Weight of the Granuloma of drugs</th>
<th>Percentage of inflammation</th>
<th>Percentage of inhibition</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Control (water)</td>
<td>1 ml</td>
<td>10mg</td>
<td>250mg</td>
<td>100</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Standard (IbuBrufen)</td>
<td>20mg</td>
<td>10mg</td>
<td>55 mg</td>
<td>22</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Jeeraga Chooranam</td>
<td>100mg</td>
<td>10mg</td>
<td>100 mg</td>
<td>40</td>
<td>60</td>
<td></td>
</tr>
</tbody>
</table>

**ACKNOWLEDGEMENT**

I am the Alpha and the Omega. The Beginning and the End. The first and the last.
First of all most I thank my almightily who empowered me, with his grace and blessings from the beginning and till the end of my dissertation work.

I express my gratitude and acknowledgement to the vice chancellor, the Tamil Nadu Dr. M.G.R Medical University, Chennai.

I sincerely thank Dr. Devarajan M.D(S), Principal, Govt. Siddha Medical College Palayamkottai. who permitted and initiated this dissertation work.

I also than Dr. Soudharajan M.D(S), Vice Principal, Govt Siddha Medical Colleg Palayamkottai.

I am grateful to our progress Dr. N.Chnadra Mohan Doss M.D(s), H.O.D and Dr. K. Shyamala Asst.Lecturer,PG Kuzhanthai maruthuvam, for this valuable guidance and encouragement for this study. It would not be possible to complete this dissertation without their valuable suggestions. The author is greatly indebted to then for their efforts.

It is my pleasure and privilege to record my pleasure and privilege to record my deep of gratitude to Dr. Mathivanan M.D., DCH., Asst. Prof. Pediatrics Tirunelveli Medical college for his suggestion and guidance in this work.

I thank Mr. Kalaivanan Department of pharmacology. And pharmacology lab. Workers, Govt Siddha Medical college. palayamKottai for helping me in this work by Furnishing pharmacological. Analysis of the trial drug.

I thank Mrs. N. Naga prema M.SC., M.phil and other staffs of the department o biochemistry for furnishing the bio chemical analysis of the trial drug.

I thank to Dr. S. Bagirathi all staffs of the department of clinical pathology, for all their helps in the clinical investigation and discussion.

The another express his gratitude to The librarian G.S.M.C Palayam kottai.

My heartful thanks to my beloved parents Mr. M.P. Chandra Selvan and Mrs. M. Pachaiammal for them blessings and my husband Mr. T. Senthil kumar for valuable guidance and encouragement, notes collecting, and printing in all aspects.

I wish to acknowledge the help and encouragement provided by my friends and colleagues to complete this work successfully.

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5. Yugi vaidhya Chinthamani – Dr. Thiyagarajan
6. Siddha Maruthuvam – Dr. Kuppuswamy mudaliyar
7. Bala vagadam – Dr. Pon. Gurusironemani
9. Agathiyar vaidya chindamani venba – Dr.Prema Saraswathi mahal
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