A STUDY ON
RAKTHA MOOLAM

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INTRODUCTION

The siddha system of medicine is on today enhancing the unique importance as well as genuine significance with full understanding of a part of Saiva Siddhanta and has a heritage of 12000 years.

Siddhars who attained Siddhi are those eternal people who had been well versed in siddha medicine. They believed and probed that siddha medicine is the one which prevents the body and mind from a diseased condition by curing the present disease and by working also as a rejuvenating process for further prevention of the same by treating the whole human body as a whole.

"ஏசீப்போடு ஒரு கருப்பு மக்களின் கையில் கொண்டு உள்ளது
ஏசீப்போடு ஏரிக்கும் பெருமளவு கூறும்
ஏசீப்போடு கூறுகை எனத்தை நாட்டிக்கோள்க
ஏசீப்போடு காட்சியை எனத்தை நாட்டிக்கோள்க"

- ரங்காசலன் சான்மாரியமில்

“Sound mind in a sound body is a sound maxim” and it is a well known proverb regarding good maintenance of one’s good health.

The siddha system of medicine is based on the Tridhoshic theory namely Vali, Azhal and Iyam. It affirms that along with the gross material body there is a subtle body. Any disease affecting an individual normally by nature involve first of all which is unavoidable and pierce this subtle envelope if it is quiet weak. The Tridosha concept is based on
The three vital humours namely Vali, Azhal and Iyam which again are found in the five vital natural elements namely Prithvi, Appu, Theyu, Vayu and Aakayam.

The three humours form the connecting link between the microcosm of man and macrocosm of universe. These Vali, Azhal and Iyam are things commonly contributed by the intake of all the nutritive fluids derived from the solid, liquid, gaseous food materials by the individuals. This theory is applicable to all the walks of medical field but it is a well known principle of the fundamentals of Indian medicine only. Any alteration occurring in the macrocosm will reflect in the microcosm, and the mukkutram will cause disease in the human body.

This is illustrated by Thriuvalluvar in Thirukkural,

"பெரிய கலாயாயின் வர்த்தம் வலியும் காவலாக
பாதி முதியோ சாரங்களில் அலம்"

According to the manuscripts as well as the evidences found out, disease may occur due to the derangement of the either the external factors or internal factors.

The internal factors include constitutional rhogas occurring due to the disturbance of the three humours, the seven thathus, the three Gunas and the Malas the secretive and excretive wastes.
Raktha Moolam

The external factors include seasonal changes or climatic variations or unlikable things like drug abusing and external stress ending in psycho somatic disorders.

Coming to the examination and investigation points of view, application of pulse reading in the diagnosis of a disease is a top most aspect of the siddha system of medicine which formed through the disturbed thathu either individually or in combination with one or two of the remaining thathus.

There are also additional test methods to be adopted and they are, body as a whole, voice, tongue, eye. excreta, urine, external appearance of the patient which are collectively known as “Enn vagai thervugal” which is also a very compulsory procedure for confirmation of above diagnosis. Further tests are also employed on the principles of which is called as “Neerkuri” and “Neikuri” for further more urine analytical study in siddha method of confirmation of the diagnosis.
**SIDDHA PHYSIOLOGY**

The Eastern physiology (Siddha physiology) is purely associated with religion and psychology unlike the western one, which is based on anatomy, which is said to determine the physiological principles involved there in.

“Man is said to be microcosm

And the world is macrocosm”.

According to the above quoted lines our human body is nothing but the miniature of the solar system.

Siddhars explained the human physiology with the unique subtle knowledge.

It involves,

96 Thathuvas – 96 Basic elements

7 Udal Kattugal – 7 Somatic compounds

14 Vegangal – 14 Reflexial functions

6 Suvaigal – 6 Tastes

4 Udal Thee – 4 Body fires

3 Udal Vanmai – 3 Immunities
The living and non-living, which are present both in the microcosm and macrocosm has the 96 basic elements. These elements are responsible for the,

1. Creation
2. Protection and
3. Destruction of life

All of which is mediated through the pancha poothic and mukkutra theory.

96 Basic elements

These are the constituent principles in nature. The enumeration of these thathuvas begins from the lowest and the grossest which is the Earth.

Those Ninety six thathuvas are,

(1) The five elements – I

There are five elements in nature. They are the original basis of all the corporeal things which when die out (or) destroyed resolve themselves again into elements.

All these five elements are found in all bodies in a transformed conditions by a process of transmutation and unions.
They are,

Table – 1

<table>
<thead>
<tr>
<th>Earth</th>
<th>Bone, flesh nerves, skin, hair.</th>
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</thead>
<tbody>
<tr>
<td>Water</td>
<td>Bile, Blood, semen, secretion and sweat</td>
</tr>
<tr>
<td>Fire</td>
<td>Hunger, thirst, sleep, beauty and indolence</td>
</tr>
<tr>
<td>Vayu</td>
<td>Contraction, expansion and Motion</td>
</tr>
<tr>
<td>Akasa</td>
<td>Interspaces of the stomach, heart, neck and the head.</td>
</tr>
</tbody>
</table>

Every element will be found mixed up with the other five elements. One element cannot be viewed dissociated from the other elements also present.

The process of combination of each of these parts with the retained half in the other is known as “**Five fold combination**”.

(2) Five sense organs:

- Mei
- Vai
- Kan
- Mooku
- Sevi
(3) The five object of sense
- Sparisam
- Suvai
- Parvai
- Vasanaï
- Osai

(4) The five organs of action
- Vai
- Kaal
- Kai
- Karuvai
- Eruvai

(5) The five organs of perception
- Vasanam
- Kamanam
- Thanam
- Visarkam
- Anandam

(6) The four intellectual faculties
- Manam
- Puthi
- Sitham
- Agankaram
(7) The ten nerves

- **Idakalai** – From right big toe runs opposite side to the left nostril.
- **Pinkala** – From left big toe runs opposite side to the right nostril.
- **Sulumunai** – Passes through both nostrils.
- **Sikuvai** – For swallowing of food and water in the uvula.
- **Puruden** – It locates at right eye.
- **Kanthari** – It locates at left eye.
- **Atthi** – It locates at right ear.
- **Alambudai** – It locates at left ear.
- **Sangini** – Vagina or tip of the penis.
- **kugu** – It locates anus.

(8) The five status of the soul

- **Nanavu**
- **Kanavu**
- **Urakkam**
- **Perurakkam**
- **Uyirpadakkam**

(9) The three principles of moral exit

- **Malam**
- **Siruneer**
- **Viyarvai**
(10) **The three cosmic qualities**

- Sathuva Gunam
- Raso Gunam
- Thamo Gunam

(11) **The three Humours**

- Vali
- Azhal
- Iyam

(12) **The three regions**

- Gnayiru Mandalam
- Thingal Mandalam
- Akkini Mandalam

(13) **The eight predominant passions**

- Kamam
- Kurotham
- Ulopam
- Moham
- Matham
- Marchariam
- Idumbai
- Ahankaram
The six station of the soul

- Moolatharam
- Suvathitanam
- Manipooragam
- Anagatham
- Visuthi
- Akkianai

The ten vital airs

- Pranan – Regulates the respiratory system.
- Abanan – Helps excretions from the lower organs, evacuation and generation.
- Vyanan – Principle of circulation of energy throughout the entire nervous system.
- Udanan – Regulates the functions of higher organs of the brain.
- Samanan – The principle of digestion and assimilation.
- Nagan – Helps in expanding and contracting the body And also in speaking.
- Kurman – Causes horripilation and winkling.
- Kirukaran – Assists the digestive process and causes anger, sneezing, etc.
- Devadattan – Is responsible for sitting, standing talking, running, perspiring, yawning etc.
Raktha Moolam

- Dhananjayan – Remains after death causing the body to swell, to separate the sutures of the skull etc.

17) The five cases of sheaths of the soul

- Annamaya kosam
- Piranamaya kosam
- Manomaya kosam
- Vinganamaya kosam
- Aanandamaya kosam

Udal kattugal

According to thirumanthiram three are seven udal kattugal, this is explained as

"இருவுருவிலிருந்து ஒருந்து விளக்கு
நூற்றில் முன்னில் பார்த்த பார்த்தும்
பார்த்து சரியில் பார்த்து பார்த்து
அதில் பார்த்துத் தெளிவுக்கொண்டே"
These are responsible for the entire structure of the body and maintain the functions of different organs and vital parts of the body. They play a very important role in the development and nourishment of the body.

14. Vegangal

They were nothing but the reflexes and protecting mechanism of the body. They are abanavayu, Thummal, Siruneer, Malam, Kottaavi, Pasi, Neervetkai, Irummal, Ilaippu, Thookam, Vandhi, Kanner, Sukkilam.

Aru suvaigal

It is the peculiar sensation caused by the contact of soluble substances with the tongue. These must be taken in a correct proportion for healthy living since these have impact over the humours of the body.
The combination of two pootha forms a suvai and also a Uyir thathu.

**Table – 2**

<table>
<thead>
<tr>
<th>Suvai</th>
<th>Pootham</th>
<th>Increases Uyirthathu</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inippu</td>
<td>Mann + Neeir</td>
<td>Iyam increased</td>
</tr>
<tr>
<td>Pulippu</td>
<td>Mann + thee</td>
<td>Azhal, Iyam increased</td>
</tr>
<tr>
<td>Kaippu</td>
<td>Neer + thee</td>
<td>Azhal, Iyam increased</td>
</tr>
<tr>
<td>Uppu</td>
<td>Katru + Vin</td>
<td>Vali increased</td>
</tr>
<tr>
<td>Karppu</td>
<td>Katru + thee</td>
<td>Azhal, Vali increased</td>
</tr>
<tr>
<td>Thuvarpu</td>
<td>Mann + Katru</td>
<td>Vali, Iyam increased</td>
</tr>
</tbody>
</table>

Thus we can alter the level of three humours by giving the particular suvai.

**Udal vanmai – 3**

It speaks about one’s reason for having healthy contour of the body. They were,

1) **Iyarkai Vanmai – Native immunity**

It is the resistance to infections which an individual possesses by virtue of his genetic and constitutional make – up.
2) **Seiyarkai vanmai – Acquired immunity**

   It is of two types

   a. **Active immunity** – It is the resistance developed by an individual as a result of an antigenic stimulus.

   b. **Passive immunity** – The resistance that is transmitted passively to a recipient in a ready made form.

4) **Kala vanmai – Herd immunity**

   Developing the immunity and stamina according to the season and environment. This refers to the overall level of immunity in a community. Eradication of communicable disease depends on the development of the high level of herd immunity.

**Udal thee – 4**

   These are the main anabolic activation to supply energy to the demands of the body by the process of digestion and various cellular activities.

   They are

   ➢ Samaakini

   ➢ Vishamaakini

   ➢ Thekshakini

   ➢ Manthakini
Body constitution

The constitution and character of the individual is determined by the two main factors.

The humour which is determined by (i) diet and (2) The environmental factor.

This body constitution is determined in the womb itself. According to this body constitution dehgi is classified as

Typical -3 Mixed – (6)

Vadha deghi Vatha Pitha dehgi
Pitha deghi Vatha Kaba dehgi
Kaba deghi Pitha Vadha dehgi

Pitha Kaba dehgi
Kaba Vadha dehgi
Kaba Pitha dehgi

When the above explained physiological aspects undergo any change in our body, they exhibit as pathology and its disease status.
Dividing mechanism of jeeva thathu into Vali, Azhal and Iyam

In our body there are several supports to the soul for the existence and continuation of life called pranan. This pranan stimulates the low very active centers viz; the brain and the heart.

Hypothalamus which is called Aakkini the most important cell stations which finally control viscera and other autonomic activity.

1. It controls autonomic nervous system
2. It regulates the secretion of anterior and posterior pituitary
3. It also related to the control of pineal gland

Pathways descend from the hypothalamus to control the sympathetic and parasympathetic activity. The principle descending pathway carrying sympathetic fibers from the hypothalamus is uncrossed traversing the lateral segmentum of the brainstem and the lateral formation.

But some fibers synapse in the brainstem in reticular formation in close proximity to the nucleus of the tractus solitorius of the medulla oblongata which is the primary site of interaction with in the CNS of afferents which influence in autonomic control of cardio – respiratory system. This is called as sushumna
All the positive matter flows along the vertebral column are gathered up here in and it stimulates the spinal cord called with all its ramification.

The other two nadis are called Idakala and pingala of which idakalai coiling round sushumna enters the right nostril, and the other pingala in like manner enters the left nostril.

Thus pingala – is the channel for the current which work in the right half of the body through the right sympathetic trunk.

Idakala is the channel for the current working in the left half of the body (or) the left sympathetic trunk.

Thus, these two right and left sympathetic trunk lying close to the vertebral column on either side consist of several ganglia.

The three nadies Idakala, pingala and sushumna meet in six different places known as shadadharam the six nerve plexues.

Each of these plexues is round like a wheel and hence they are called chakkaras.
Raktha Moolam

Brain

Hyothalamus (Central station for controlling ANS and endocrine glands)

Meet at six different places Called Aadharams

- Superior cervical ganglion
- Cardiac plexus
- Thoracic splanchnic plexus

Medulla oblongata

- Right sympathetic trunk (Pingala)
- Left sympathetic trunk (Idakala)

Shushumna + Samanam

Pingala + paranam

Right sympathetic trunk

Idakala

Pingala

Shushumna

Splanchnic plexus

Superior cervical ganglion

Cardiac plexus

Thoracic splanchnic plexus

Lumbar splanchnic plexus

Pelvic splanchnic plexus

Pranam

Pingala + paranam

Idakala

Splanchnic plexus

Superior cervical ganglion

Cardiac plexus

Thoracic splanchnic plexus

Lumbar splanchnic plexus

Pelvic splanchnic plexus

Pranam
**SIDDHA PATHOLOGY**

Pathology is a scientific study of structure and function of the body in disease. The discipline of pathology forms a vital bridge between initial learning phase of pre clinical sciences and the final phase of clinical subjects.

"அவஸ்தால் பனிர வியாசம் நம்பூ
இலைக் கன்னி மலரம் முற்றிலம்
சுருக்கம் பிற்பகுதியில்லை நரம்பூ
சுருக்கம் பிற்பகுதியில்லை குறிப்பிட்டு
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சுருக்கம் பிற்பகுதியில்லை

The siddhar’s school fully recognize, these 96 thathuvangal and further add that the human body is composed of 72,000 blood vessels, 13,000 nerves, 10 main arteries, 10 vital airs (prana) all together in the form of a network and it is owing to the derangement of the three humours becomes liable to 4,448 diseases.

Siddha pathology explained that, all disease are caused by the mixture of the three cardinal humours (or) Uyir Thathu. Vali, Azhal, Iyam and that the relative proportion of these humours are responsible for a person’s, physical and mental qualities and dispositions.
Siddha pathology deals with mukkutram and piniari muraimai.

The changes takes place in uyir thathu caused by:

- Altered Diet
- Environmental changes
- Habits
- Immoral activities
- Seasonal changes
- Self suppression of vegankal
- Kanma vinaigal

Affect

Udal kattugal

Affect

UYIR THATHU

Vali     Azhal     Iyam
Enbu     Senneer   Saaram
Oon

Kozhuppu
Moolai
Sukkilam
(or)
Suronitham
Noi Nilai
These factors cause disease in man and the symptoms are depicted according to the humour affected.

**Diet:**

Food the constituent of energy is responsible for our activities.

According to Siddha system, the daily food comes under 6 main tastes. The tastes altering the humours are due to the ignorance of the dietary adaptations.

"பந்தை தம்பழ்த்தை விட்டம் கருப்பன் பற்களையும் விட்டம்
நல்ப பந்தை தம்பழ்த்தை விட்டம் முட்டை - கைவாரியானாக
காட்சிபை விட்டம் கல் விட்டம்
என்று கூறும் பந்தை விட்டம் விளக்கம்

- **Inippu** is responsible for obesity, indigestion, diabetes, cervical adenitis. Increased kabham and it’s diseases.
- **Pulippu** is responsible for body weakness, dull vision, giddiness, anaemia, dropsy, feverishness, dryness of the tongue, herpes, scabies, and blisters.
- **Uppu** is responsible for aging, falling of hair progressive weakness of the body.
- **Kaippu** is related to Vali, disorders of physical constituents.
- **Kaarppu** related to excessive dryness of the tongue, defect in spermatogenesis, general malaise, lassitude’s, tremors, back pain.
Raktha Moolam

- **Thuvarppu** related to abdominal discomfort, heart disease, tiredness, vascular constriction and constipation.

**Environment:**

Dwelling places also play a vital role in determining diseases.

- **Kurinji** - Fever, anaemia, liver enlargement, iya diseases etc.
- **Mullai** - Vali & azhal diseases are predominant.
- **Marudham** - Favorable places for dwelling.
- **Neidhal** - Vali diseases, elephantiasis, Hepatomegaly.
- **Palai** - Abode of all ailments.

It is inferred that the environment along with its humidity, fertility, nature of soil, its capacity, population, plays a vital role in man’s internal environment, immunity, the defense and also for the organisms causing diseases in endemic and epidemic situations.

**Habits:**

These are deeds involving ones mental and physical activity. Good deeds and their consequences keep the individual fresh and healthy whereas the notorious deeds lead them to continued stress, reduced immunity and make them embodiment of diseases.
**Immoral activities**

In this modern world, there is a wide opportunity for the human to derail from the disciplined path. It is caused dreadful diseases to enter into the man.

**Season**

Seasonal variations are depicted in 6 major & 6 minor forms. The relation between them and the humours are.

Table – 3

<table>
<thead>
<tr>
<th>Season</th>
<th>Period</th>
<th>Mukkutra Nilaigal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaar</td>
<td>Aavani + Puratasi</td>
<td>Vali↑↑↑, Azhal ↑</td>
</tr>
<tr>
<td>Kudhir</td>
<td>Aippasi + Kaarthigai</td>
<td>Vali -, Iyam↑↑</td>
</tr>
<tr>
<td>Munpani</td>
<td>Maarghazhi + Thai</td>
<td>Vali -, Azhal-, Iyam-</td>
</tr>
<tr>
<td>Pinpani</td>
<td>Maasi + Panguni</td>
<td>Iyam↑</td>
</tr>
<tr>
<td>Ilavenil</td>
<td>Chittirai + Vaigasi</td>
<td>Iyam↑</td>
</tr>
<tr>
<td>Muduvenil</td>
<td>Aaani + Aadi</td>
<td>Vali↑, Iyam↑</td>
</tr>
</tbody>
</table>

↑ - Thannilai Valarchi

↑↑ - Vetrunikai Valarchi

_ - Thannilai Adaithal

Normally food adaptations save people from these changes. If their fail to adapt these humoural changes cause diseases.
Inhibition of vegangal:

These are the urges must be needed. If they are inhibited from their normal physiological pathway they cause.

**Vadham (Abana Vayu)** - Chest pain, peptic ulcer, abdominal pain, body ache, constipation, oliguria and indigestion.

**Thummal** - Head ache, facial pain, back pain, pain in the sense organs etc.

**Siruneer** - Ulcers in the urethral orifice, joints pain, urinary tract infection etc.

**Malam** - Calf muscle pain, head ache, general debility, flatulence and other disease.

**Kottaavi** - Indigestion, contractures in the face.

**Pasi, Neervetkai** - Constitution of the body is totally disturbed, emaciation.

**Kasam** - Chest disorders supervene.

**Ilaippu** - Ulcer and other mega diseases.

**Nithirai** - Heaviness of the head, eye pain, deafness, speech disturbances.

**Vaanthi** - Utricaria, skin diseases, toxic manifestations, anaemia, eye diseases.
Kanneer - Eye diseases, head ache, sinusitis and heart diseases.

Sukkilam - joints pain, fever, chest pain, difficulty in micturition.

Swaasam - Cough, abdominal discomfort, anorexia.

Diagnostic methods

Diagnosis is the mandatory process in the treatment of a patient. Envagai Thervugal which is the unique and special method having a broad and important role in diagnosing a particular disease. It is based upon the principles of:

⇒ Poriyaal arithal
⇒ Pulanaal arithal
⇒ Vinaathal

Poriyaal arithal

It means understanding by the five organs of perception, nose, tongue, eyes, skin and the ears.

Pulanaal arithal

It means understanding by the sense objects smell, taste, vision, somatic sense and sound.

Vinaathal

It means interrogating the patient, learning the history, and symptoms of the disease by asking questions to the patients.
Envagai Thervugal

"நிலையில் நீர் போனது யார், நன்மையாள் என்பது"

- மூத்ரை

- Mei – (Sparisam)
- Niram
- Thoni
- Vizhi
- Naa
- Malam
- Moothiram
- Kaikuri (Naadi)

1. Meikuri (Sensation)

Reveals that the structural and sensational changes present through the body.

Eg. Skin temperature (heat or cold)

Sweat

Dryness

Tenderness

Swellings

Nourishment, etc.,
2. **Niram (Colour)**

   Reveals that any change in the colour of the skin, nails, hairs, conjunctiva, teeth, mucous membrane, etc.,

3. **Thoni (Sound and Speech variation)**

   Reveals that means the quality of sound, the mode of speech and intelligence is to be assessed.

4. **Vizhi (Eye)**

   It reveals that the systemic changes in the organs, by changes in Its colour, structure of the eye and any discharge in the eye. It also reveals the function of the eye.

5. **Naa (Tongue)**

   It reveals that structural changes, colour changes in the tongue, any ulceration, deviation etc.,

6. **Malam (Faeces)**

   The colour, amount and consistency of the faeces will reflects the pathological condition of the body.

7. **Kaikuri (Signs in hand pulse)**

   Is nothing but the vital energy that sustains the life in our body. It has been considered for assessing the prognosis and diagnosis of the disease. Any variation that occurs in the three humours is reflected in the naadi. It serves as a good indicator of all ill health. It can be perceived by feeling it at the appropriate sites.
The diagnostic value of urine is observed by Neerkuri and Neikuri.

Physical findings of urine are said as:

"நூற்று திரிகால் மரல் அல்லது காச்சி இல்லையால் அதிகுடையனூறு பாதிக்கும் முறையால்

Colour, quantity, odour, frothy appearance, constituents, specific
gravity of urine are physical findings.

A drop of gingili oil is dropped into the center surface of upper
surface of the urine, if the oil spreads like snake indicates Vali, spreads
Raktha Moolam

like a ring indicates Azhal, remains floating as a pearl indicates Iyam mixed reaction of any two indicates thontham.

Basically siddha aims to maintain the equilibrium between the five elements despite our constant interaction with the outer world. The five elements, which work as 3 vital forces in the body and perform all physical and mental functions are constantly affected by time, space and nutrition. The humanity suffers more and more from Vali ailments sleep disorders, aches, and pain, constipation, hemorrhoids, nervous disorder, hypertension, etc. It is regard a sound knowledge of noi naadal is essential to formulate therapeutic measures for various ailments.
AIM AND OBJECTIVES

Now-a-days Moolam is an common ailment prevalent in the world, affecting the Mankind because of their improper life style and diet.

According to Yugi there are 21 types of Moolam, each and every type of Moolam has different causes for their occurrence.

Through this dissertation more an attempt is made to evaluate the proper cause for Raktha Moolam.

Raktha Moolam is one of those diseases which cause severe bleeding through the anus. It affects the patients both physically and mentally.

The topic is discussed with following sub topics,

➢ To study the clinical features of the disease.
➢ To explore udal thathuvam.
➢ To review the altered Mukkutram.
➢ To make a clinical observation about incidence of the disease in relation to age sex, occupation, food and other habits
Paruvakkalam and Nilam.
➢ To highlight the Envagai thervu
➢ To make out a proper pathology of Raktha moolam by co-relating with modern aspect medicine and to expose the efficiency of siddhars philosophy.
Pain in and around the umbilicus and spurting of blood from rectum and anal canal which looks like splash in the pan.

Pain may be due to any cause in and around the umbilicus such as obstruction (or) inflammation eg (Gall stones, pancreatitis) which may indirectly initiate the cirrhosis of liver.
Bleeding is due to rupture of Varicosities. A reverse flow of blood due to portal obstruction leads to varicosities of the plexuses which bulges and ruptures when the pressure in the portal vein raises, leading to spurting of blood.

"செருமான் மூள் மேற்குத்துறவுக்கு
சிறு சதுரவம் உறையது இரவாகாது".

Weight loss, Anaemia easy fatigability. All these are due to reduced oxygen carrying capacity of blood results from a deficiency of red cells which occurs due to massive bleeding from rectum.

Oedema – A fall in the total plasma protein level results in lowering of plasma oncotic pressure and increased capillary hydrostatic pressure further it results in increased outward movement of fluid from the capillary wall and decreased in-ward movement of fluid from the interstitial space causing oedema and ascitis.

"பொருள்வேலாங்களை கணிமைலைக் கவர்கின
பெங்கல்கள் முதலில் தொங்கு புருதமு".

Throbbing chest pain - The lowered oxygen content of the circulating blood leads to myocardial hypoxia which manifest itself as chest pain.
Headache and Drowsiness - Further in central Nervous system hypoxia may be evidenced by headache dimness, vision faintness, giddiness and drowsiness.

Yellowishness of the eyes.

In addition to the above signs and symptoms, mild jaundice is also present which is manifested as yellowishness of sclerae in the eyes. It is due to cirrhotic of liver.
Many siddhars have dealt about Raktha moolam. Among them the author has taken Raktha Moolam for dissertation study from YUGI VAIDYA SINTHAMANI. As the name indicates the main symptom of the diseases is bleeding per rectum.

Eyal

Literally Raktha means blood and Moolam means the ano - rectal region. To say it correctly Raktha Moolam is a disease characterized by bleeding per rectum.

Noi Varum Vazhi(Aetiology)

Yugi munivar elaborately describes the various causes for all Moola Noigal although the text does not mention causes separately for each type collectively within two versus. It deals with psychological, Karmic, intrinsic and extrinsic factors of aetiology for all Moola noigal with this and other siddha text we can say the causes of the diseases as,

1. Karmic and psychological causes.
2. Due to wrong diet and act.
3. Maintaing wrong positions in yogasanas.
Raktha Moolam

Etiology:

"குறுநிறமச் செய்யும் கருப்பாலும்
அரியத் வைத்தியால் கருப்பாலும்,
புல்லாலும் விளக்கும் வெப்பாலும்
பச்சைக்குருக்கு வெப்பாலும்,தம்பட்டியடிக்கு வெப்பாலும்,
நோய்க்குருக்கு வெப்பாலும் வெப்பாலும்,நோய்க்குருக்கு வெப்பாலும்,நோய்க்குருக்கு வெப்பாலும்,நோய்க்குருக்கு வெப்பாலும்,நோய்க்குருக்கு வெப்பாலும்".

- புத்த சத்ருந்திவாசி சிக்காராதினர்
"மன்ற முன்னைய வெள்ளியக் கோணத்தில்

குரட்டு மலர் நியாயசங்களில்

வருமாறு வனவரலர் காணத்தக்கு

கையாளி என்று மாற்றம் வர்த்தக வெளியில்

- அவத்தில் 2000

"குறுக்கு வெளியை காண்பதற்கு

நிதியில் ஆராய்ச்சியும் நிறுத்தப்படும்

நேரக்குறியான வெளியை வாகன் காண்பதில்

மாட்டுக்குள் வெளியை காண்பது நடந்து வெளியை

காண்பது என்று பீரை மன்ற நிழல்

தூட்டு வெளியை வாகனமாக நண்பு பின்னர்

புனித விகாரம் பயில் விளைவங்கள் காண வெளியில்

- அவத்தில் காண்பதற்கு
Raktha Moolam

SIDDHA VIEW - சித்தா விளக்கம்

என்று 25 நிலைநிகழ்க்கட்டு கிக்கமருத்து அம்பலாக்கணம் நிறந்து வடிவம் வழிகுறைடு. இது 96 குற்றவாளிகளுடன் தூக்கும் மருத்துவத்தை காணாமல் பார்க்க புரிகுற்றாக்கத்தை அளிக்க, அப்படி தவிர்ப்பட்டது,
24 குற்றவாளிகளிடையே பெருமான் வைக்கப்படுகின்றது. இவ்விதமான நிலை
இன்றும் பெருமான் புரிகுற்றாக்கத்தை அளித்து தவிர்ப்பட்ட பாரிக்கப்படுகின்றது.
தீர்த்தமும் விளக்கங்கள் கிட்டுக் குறிப்பிட்டது.

"அனில் விக்க விளக்கநிகழ்க்கட்டு வரலை விளக்கம்"

- என்று

திண்டு அரியல் மாணிக்கும், பிரிந்து சிவார்த்தத்திற்கும் துறுதக்கியே.

புள்ளியும் பாது திருப்பியும் புளியும் சிவார்த்தத்திற்கு வரலை தவிர்ப்பட்டு
வுரைப்பட்டு 21 வரலைப்புடா வரலை குறிப்பிட்டுக்

அனைத்திற்கும்:

"சதிபுராசர் சுயார்த்திகளின் நுழைவு நிகழ்த்த反应

சதுரங்கத்தில் நிறந்து நிகழ்த்த வரை

சதிபுராசர் நுழைவு நிகழ்த்த வரை

சதுரங்கத்தில் வருமானாக நிகழ்த்த வரை

சதிபுராசர் நுழைவு நிகழ்த்த வரை

சதுரங்கத்தில் வருமானாக நிகழ்த்த வரை

சதிபுராசர் நுழைவு நிகழ்த்த வரை

வரலைப்புடா வரலை புளியும் வரலை

வாய்ந்த விளக்கநிகழ்க்கட்டு வரலை
"வாசமாது சுருக்கத்தின் குறிப்பிட்டு
மானியக் கிலோவர்ட் வேல் மும்பி,
பாசை கன்று கிளோவர்ட் குறிப்பிட்டு
பாசைக் குளிராது கீழ்வாழ்வது
பாசைக் குடும்பத்து நெப்பிங்கை,
பாசைக் குட்டிகள் குன்றல் மன்னகத்து, கண்டறியாதே நெப்பிங்கை
நெப்பிங்கையால் கருதும் காலனித்தவர்.”
- புத்தி மானியா சிற்றமுனை

அற்றல்படி: 
1. முத்தம்
2. சோடை மும்பி
3. புரோட்டு மும்பி
4. பிரியல் மும்பி
5. குராம் மும்பி
6. நாக மும்பி
7. சுத்தம்
8. அதிர்வை கும்பம்
9. கிளோவர்ட் கும்பம்
10. பாசை கும்பம்
11. வெள்ளக்கும்பம்
12. கன்று கும்பம்
13. சுருக்கத் துணை
14. தீடுவன மும்பி
15. ஈவித்துச் சுட்டு
16. பாசுபத்தி சுட்டு
17. உரூசி சுட்டு
18. உள் சுட்டு
19. பும் சுட்டு
20. குருவை சுட்டு
21. அம்பை சுட்டு

குறிப்பிட்டு கருதலாம் செய்ய விருத்தவழி செய்யலாம் காரணமாக ப செய்யும் கருதலாயமான அடுத்திட்டுக்காத ஆன்கிளங்காத குறிக்கை பாதிக்கும் சூடை எண்ணத்தை நோக்காக்கி இருக்கிறது. அனைத்தும் கணக்கம் குறிப்பிட்டு அடுத்து இற்றுண்டா எடுக்கும் பாதுகாப்பு, மிகுது சிற்றிருத்துச் செய்யும் பாதுகாப்பிற்கு நேரடியாக வேளாண்ட பாதுகாப்பு காரணியாக

“சுத்திரங்க கிளங்காவல்சுழற்சி நந்தி நீக்கி

முனைகளில் பிரிக்கப் போகும்

நூல்கள் குறிப்பிட்டு கொண்டுச் செய்ய

முக்கியமான எள்ளத்தடி தன்னுள் மாற்றம்

மாற்றங்களுக்கு குறிக்கை வேன்கிறது என்னற்ற

மாற்றங்கள் மிகுதியான கூடுதல் பாதுகாப்பு

நூல்கள் கூறையிலானென்னற்ற பொருள்கள்

சிறுமியர் குறிப்பிட்டு பாதுகாப்பு குறியான்.”

- புஞ்சி தண்டிக்கோம் கி. அரசாசி

- வேதியியல் வாழ்
- உரூசி மாங்கல் திருநூற்றாண்டு பிரித்தானிட் நூல்கள்
- பாதுகாப்பு
Raktha Moolam

- ரக்தம் விவசாயக் காலவரம்
- தக, குறு அசி
- தக, குறு பரவுதல் ரக்தம் விளக்கம்
- பாருபுல் பிளாக் பிளாக் அமை
- கோது அமை
- பெப்ஷாம்
- கரை பெப்ஷாணிகளின் காலவரம்

பெப்ஷாணியின் புரோமியா புகைக் பிளாக் அசாரிய நிர்வுகம் நிலையில் இருக்கும் கால காலவரம் காயவில் விளக்கம் பெப்ஷாணியின் புரோமியா பிளாக் அசாரிய நிர்வுகம் நிலையில் இருக்கும் கால காலவரம் காயவில் விளக்கம் காயப்பட்டர்.
Pathogenesis of the disease

1. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

2. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

3. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

4. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

5. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

6. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

7. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

8. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

9. Various factors such as climate, environment, and lifestyle are known to trigger the disease.

10. Various factors such as climate, environment, and lifestyle are known to trigger the disease.
Raktha Moolam

(Back pressure in the portal vein)

- பிரசாரங்கள் பிறகு புலம், குரை சூர்யக் குருக்களில் புற உரைக்கல்லு சோகம் 2-லில் புற பழந்து கண்காட்டுவது போன்றே காணப்படும் சில காற்றராணங்களுடன் முற்படியே குறிப்பிடுவது வேண்டாம். என்றாலும், என்பது வெளியில் அட்சம் குறிப்பிட்டிருக்கும் நான் தோன்ற வேலா ஆற்றம்பயிற்று வழக்கமும் வைக்கும் வழக்கமும் வைக்கும்.

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் பற்பலமான பட்டியல் வாய்ந்து போக்கும் வழக்கத்தின் மூலம் ஒரு வலுவும் வழக்கத்தின் மூலம் ஒரு வலுவும் வழக்கத்தின் மூலம் ஒரு வலுவும் வழக்கத்தின் மூலம் ஒரு வலுவும் வழக்கத்தின் மூலம் ஒரு வலுவும் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் பற்பலமான பட்டியல் வாய்ந்து போக்கும் வழக்கத்தின் மூலம் ஒரு வலுவும் வழக்கத்தின் மூலம் ஒரு வலுவும் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் பற்பலமான பட்டியல் வாய்ந்து போக்கும் வழக்கத்தின் மூலம் ஒரு வலுவும் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் பற்பலமான பட்டியல் வாய்ந்து போக்கும் வழக்கத்தின் மூலம் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் பற்பலமான பட்டியல் வாய்ந்து போக்கும் வழக்கத்தின் மூலம் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள் 

- மேலும் மரி தோற்ற குறிப்பிட்டிருக்கும் புதுப்பிட்டு வெள்ளம் பயிழ்ச்சியான பற்பல அளவிற்குள்
“ராக்த முளையுருள் கோகல்பான்”

- சோந்தாநாதா

அறிக்கையின் செல்வாக்காக வரும் ஏற்கலந்த நம் கோகல்பான்

தனித்துவம் பின்பற்றி அவச் செய்ய வேண்டும் முயற்சியிலக்கிறோம். ஏனெனில் துணைவரத்து நிற்பாகம் அகிலமாக வந்தடையும் நம்பிப்பாம், முன்னர் தொடரும் விளக்கம்.

சிறப்பமான பெரும் கோகல்பான் விளக்கம்

“சிறியமான வைப்பாட்டைக் கோகல்பான்

விளக்கம் பற்றி உரைத்து வந்தேன்”

னாட்டுத் தொடர்பான விளக்குற்றங்கள் வெளியூரையே கோகல்பான்

செய்யும் முயற்சியானது மறியாக வெளியூரையே செய்யும் அரசியல் வைப்பாட்டிலே நல்லாக தொடர்புக்கு கேட்டுக்கோரிய. அவற்றை முற்பாடு செய்யும் வழியாக வைப்பாட்டிலே நல்லாக தொடர்புக்கு கேட்டுக்கோரிய. அதற்குச் சாத்தியவான வைப்பாட்டிலே நல்லாக தொடர்புக்கு கேட்டுக்கோரிய.

நாளும்

“நூறாண்டுகளுக்கு அடுத்து

கோகல்பான் விளக்கம்”

நாளைய கோகல்பான் கேட்டுக்கோரிய
"ராக்ஷாம் வேலூரனுட் செல்வுக்கு புரோகரிக்க வேண்டும்
பிறக்க நிலையமாக செய்தியுடைய
சார்பு மற்றும் ஫லம் தருகிக் கொள்ளும்வரது ஒட்டையை
மாற்றும் நிலையை வழங்கும் விளைவுகளை"
Raktha Moolam

என்றும் வெள்ளியனூறுவின் பெற்றிகளை காண்டு, முதல் வர்த்தகத்தில் 2 குறிப்பிட்டன மூலம், சிறையில், கான்மூலம், புதுக்கிறப்பு விசாக்கித்து, காத்தியின் வெள்ளியனூறு பெற்றிகளை ஆன்புறையும்.

சொல்லி சார் யுடை சூழ்ந்தால் மூர்த்த வெள்ளியனூறு முகமூலம் மட்டுமே குற்றின "நூறுண்டு லாஷ்டசிர்வித்து மூர்த்த வெள்ளியோல்." சொல்லி ஆபாசானது என்று புகை ஓரால்பாறு ரக பாத்தியம் குறிப்பிட்டு எச்சினல்களிலும்.

முற்பினால் இனி பாத்தியப்பணுச்சுத்த லாஷ்டசிர்வித்து மூர்த்த 2 குற்றினாகக் காணும்.

பாதுகாப்பில் முற்பின 2 குற்றின் லாஷ்டசிர்வித்து மூர்த்தக்கைத். இங்கு லாஷ்டசிர்வித்து இல் விலைநிலை மிக்கானது மூர்த்தக் லாஷ்டசிர்வித்து பாத்தியப்பணுச்சுத்த.

1. பாத்திய
2. அபாசான
3. இயற்பை
4. சாகான

- பாத்திய பாத்தியப்பணு லாஷ்டசிர்வித்துக் கொள்ள, அழகு, அசெரை, கழுகையில், பாதுகாப்பில்.
- அபாசான பாத்தியப்பணு லாஷ்டசிர்வித்து - நூறுண்டு மூடி முற்பினிக்கும் திறக்கப்பட்டர் விளைநிலை பாதுகாப்பில்.

இனி விலைநிலை மூன்றுப்போலம் பாத்தியப்பணு அம்மன் கொள்கிற்கைத்து.

1. அம்மன் பிரிக்கும்
2. புதுக்கிற பிரிக்கும்
3. சாத்து பிரிக்கும்
4. அலுவலக பிரிக்கும்
5. பரிபட்டிகைகள்

- முருகச்சி பிரதான பாரிபலவ் பிறப்பப் போக்கட்டை.
- பரிபட்டிகை பாரிபலவ் பிறப்பப் போக்கட்டை.
- முருகச்சி பிரதான பாரிபலவ் பிறப்பப் போக்கட்டை.
- கவுண்டிக் பிரதான பாரிபலவ் பிறப்பப் போக்கட்டை.
- முருகச்சி பிரதான பாரிபலவ் பிறப்பப் போக்கட்டை.

அவ்விடைகள் பாரிபலவ் போக்கட்டை மற்றும் பாரிபலவ் பிறப்பப் போக்கட்டை உள்ளே அவ்விடைகள் பாரிபலவ் பிறப்பப் போக்கட்டை பாரிபலவ் பிறப்பப் போக்கட்டை.

- அவ்விடைகளைச் செய்து கால்வாய் அவ்விடைகள் பாரிபலவ் போக்கட்டை பாரிபலவ் பிறப்பப் போக்கட்டை.
- அவ்விடைகளைச் செய்து கால்வாய் அவ்விடைகள் பாரிபலவ் பிறப்பப் போக்கட்டை.
- அவ்விடைகளைச் செய்து கால்வாய் அவ்விடைகள் பாரிபலவ் பிறப்பப் போக்கட்டை.
Raktha Moolam

2.2 காதல்கருத்துகள்:

இருவரிடையே பாரிப்பருடாள் இருந்து கருத்துக்குரிய காதல்கருத்துகள்

- பாரிப்பருட விளக்காகத்து இரு முறையும் முறையும்

- பாரிப்பருட விளக்காகத்து இருமுறை பொய்யும்

- பாரிப்பருட விளக்காகத்து இருமுறை முறக்கமும்

- பாரிப்பருட விளக்காகத்து இருமுறை முறக்கமும் முறக்கமும் இருமுறை வெளிப்பகுதி
MODERN PATHOLOGICAL VIEW

The Stanza reveals the pathogenesis of the disease explained by four stages

1. The umbilical pain and increased portal pressure
2. Ruputre of the portal vessels and recurrent bleeding from the anus
3. Complications due to heavy blood loss
4. Manifestations of the cirrhotic liver.

The umbilical pain and increased portal pressure

The pain produced here is usually the radiating pain of the surrounding area of the umbilicus and not especially produced in the umbilicus itself
The cirrhotic liver produces the increased portal pressure in the portal vein due to constrictions of replaced fibrous tissue around the blood vessels and greatly impeding the flow of portal blood vessels causing portal hypertension.
Rupture of the portal vessels

“When the Raised portal pressure prefers the lower tract, the backward pressure in the portal vein causes the rectal veins in the anus to undergo varices.

Along with this when the patients have the congenital weakness of the venous wall with weak submucous connective of the rectum it easily predisposes to the formation of rectal varices.

Recurrent bleeding from the rectal varices

Thus when ever there is increased in pressure (especially the portal pressure is raised after ingestion of food), these fragile blood vessels are in danger of breaking and bleeding and the pressure in the vein produces the bleeding as if “spurting from the anus”.

Complications due to heavy blood loss

“There is heavy blood loss due

1. Recurrent bleeding
2. Insufficiency of coagulation factors due to cirrhotic liver and
3. Spleenomegaly
All these contribute to mass reduction in the Red blood cells and reduced oxygen transport to tissues which produces certain changes throughout the body and manifest as anaemia, they are

- Paleness of the skin and usually the skin becomes thin and inelastic as the epidermis and dermis undergo atrophy.

"ஹை காலனாவனைகள் பொருளாகும்"

The other manifestations of anaemia are

- Weakness
- Malaise
- Easy fatigability
- Oedema

**Oedema**

It is the result of an increase in the forces that tend to move fluids from the intravascular compartment into the interstitial fluid.

When there is heavy blood loss it produces a fall in the total plasma protein level, which results in lowering of plasma oncotic pressure.

This results in increased outward movement of fluid from the capillary wall and decreased inward movement of fluid from the interstitial space causing oedema and ascites.
Cells that are particularly vulnerable to hypoxia may undergo fatty changes (or) even ischemic necrosis such damage is most frequently encountered in

- Muscle cells of the myocardium
- The sensitive ganglion cells of the cortex and basal ganglion

All these manifest as

- The respiratory difficulty as the throbbing chest pain

In the central nervous system hypoxia may be evidenced by headache dimness of vision drowsiness and mental confusion.

In addition to this accumulation of ammonia due to inability of the liver to detoxify is characterized by the disturbances in consciousness and behaviour and later coma – Hepatic encephalopathy.

**Manifestation of cirrhotic liver**

Yellowishness of the eyes – indicates the presence of jaundice. Bilirubin pigment has high affinity for elastic tissues and hence jaundice is particularly noticable in tissues rich in elastin content such as skin and sclerae.
Jaundice is largely due to failure of the liver cells to metabolize bilirubin and the severity of the jaundice varies according to the type of liver cell failure.

- **In acute failure** - Jaundice parallels the extent of the liver cell damage

- **In cirrhosis** - Jaundice may be mild because this is due to balance achieved between the hepatic necrosis and regeneration. But in later stage its manifestation is very severe.
According to Gunavaagadam the symptoms are:

- Pain in the umbilicus
- Bleeding per rectum
- Protrusion of the pile mass
- Emaciation.

According to Aavi Alikkum Amutha murai churukkam

The Symptoms are

- Loss of appetite.
- Lower abdominal discomfort
- Constipation.
- Pain around umbilicus
- Bleeding per rectum
- Drowsiness
- Anaemia and breath less ness
According to Cegarasa Sekaram

- Presence of pile mass
- Swelling of pile mass
- Bleeding per rectum.

This is given in the verse follows.

"இரங்கின் பிளையும் விளக்கம் ஸ்வாதொல்ல விளக்கம்
மும்பை வளர்வுக் கலனம் மாறாத ரோஜா விளக்கம்
மும்பையின் கனவின் மறைவு காண்கு
யாவ் குன்று கனவின் மறைவு காண்கு மெய்தோ"

According to Aathma ratchaamirtham Entra Vaidhya Saara Sangiragam.

The symptoms are

Consuming food stuffs with bitter and sour taste.

- Body becoming warmer
- Loss of appetite
- Lower abdominal discomfort
- Constipation
- Pain around the umbilicus
- Bleeding per rectum
- Protrusion of the pile mass
- Irritation and burning sensation in the anus
- Anaemia
Raktha Moolam

According to Thirumoolar Karukkidai Vaidhyam - 600

The symptoms are

- Pile mass.
- Bleeding per rectum.

According to “Veerama Munivar” aruli cheitha “Nasakanda venba”

- Pain in the umbilicus
- Spurting out of blood during defaecation
- Emaciation
- Pallor
- Weakness in limbs
- Sobai
- Chest pain
- Head ache
- Giddiness
ANATOMY

As Raktha moolam has cirrhosis of liver as cause of its occurrence, here in this dissertation anatomy of both Anal canal and liver are described.

Anatomy of Rectum and the anal canal

Rectum is the distal portion of the large gut placed between the sigmoid colon above and the anal canal below in front of last three pieces of sacrum and coccyx.

It is about 12cm long and 2.5 cm in diameter.

Anal canal is the terminal part of the large intestine. It extends from the ano - rectal junction to the anal opening at perineum.

It is about 4cm long and 1.25 cm in diameter.

Ano - rectal ring

This is a muscular ring present at the ano – rectal junction. It is formed by the fusion of puborectalis, deep external sphincter and internal sphincter.

It is easily felt by the finger in the anal canal.
Sphincters of anal canal

Internal sphincter

- It is the downward extension of circular muscle of rectum.
- It is involuntary in nature
- It surrounds the upper three-fourths of the anal canal

External sphincter

- It is made up of striated muscle
- It is under voluntary in control
- It surrounds the whole length of the anal canal

Pectan or white line of Hilton

It is a broad line of 1.25 cm width. It is an important surgical landmark, because this line is referred to as the inter-sphincteric groove as it is dividing line between the external and the internal sphincter.

It is the dividing line between the superior and inferior rectal vessels.

It is the important site of portocaval communication. Other importance of this Hilton's lines are,
### Table - 4

<table>
<thead>
<tr>
<th>Below the line</th>
<th>Above the line</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Histology</strong></td>
<td><strong>Lined by stratified squamous epithelium</strong></td>
</tr>
<tr>
<td><strong>2. Supply of nerve, skin</strong></td>
<td><strong>Inferior haemorrhoidal nerve and is pain sensitive</strong></td>
</tr>
<tr>
<td><strong>3. Haemorrhoids</strong></td>
<td><strong>External haemorrhoids occur here</strong></td>
</tr>
<tr>
<td><strong>4. Lymphatics</strong></td>
<td><strong>End in the superficial inguinal nodes</strong></td>
</tr>
</tbody>
</table>

**Arterial supply**

The anal canal is supplied by the inferior rectal branches of the internal pudendal arteries

**Venous drainage of rectum and anal canal**

There are two plexuses of veins draining the rectum and the anal canal

1. **Submucosal plexus of veins**

   **Above the Hiltons line** – They are drained by superior rectal vein and thus reach port system there are no valves in superior rectal veins.
**Below the Hiltons line** – they are drained by inferior rectal vein and this into the caval system.

- There are valves in the inferior rectal veins. Which permit the blood flow away from the rectal veins.
- Thus there is connection between the portal and the systemic veins in the submucosal plexus.
- A reverse flow of blood due to portal obstruction leads to varicosities of those plexuses which bulge and form piles or haemorrhoids.

2. **Perimuscular plexus of veins**

Lies on the surface of the muscle coat, especially in the lower part of the rectum and the anal canal. They are drained by superior rectal veins above and the middle rectal veins at the sides.
ANATOMY OF THE LIVER

Liver is the largest gland in the body situated in the right upper quadrant of the abdominal cavity.

- Reddish brown in colour
- 1.5 kg weight
- It has 2 anatomical lobes right and the left
- Right lobe being 6 times the size of the left

Internal lobes and segments

Liver consist of many lobes. Each lobe consist of large number of lobules, each lobe is a honey comb like structure.

Each lobule consist of a mass of hepatic cells arranged in irregular radiating column between which the blood channels called sinusoids present.

Each lobule has

- Hepatic cells
- Blood vessels
- Bile ducts and
- The secreating glands

Hepatic cells

The liver cells comprise about 60% of the liver. In between the hepatocytes blood spaces are present called sinusoids. The sinusoids are lined by sinusoidal lining cells
Sinusoidal lining cells are

- **Endothelial cells** - These are in contact in lumen of the sinusoids. Fenestrae of this cells determine the exchange of fluids and other particulate matter.

- **Kupffer cells** - They are the phagocytic cells derived from blood monocytes

- **Fat storing cells** - These stellate sessile cells lie within the space of disse. They may contain they store vitamin A and other retinoids.

- **Pit cells** - they are highly mobile natural killer lymphocyte. They show spontaneous action against virus infected hepatocytes.

  All these cells are very important because their damages contribute to portal hypertension.

Blood supply

Liver has dual blood supply

- **The portal vein** - it brings venous blood from the intestine and spleen

- **The hepatic artery** – coming from the celiac artery supplies the liver with arterial blood.

These vessels enter the liver through a fissure, the porta hepatis, which lies in the inferior surface of the right lobe. Inside the porta, the portal vein and Hepatic artery divided into branches to the right and left lobes.
Portal traid

- It is an channel through which the smallest radicle of
- Portal vein
- Hepatic artery and the
- Bile duct are distributed

All these are bound together by a delicate connective tissue called Gilson capsule.

Portal vein

The portal vein about 8 cm in length formed at the the level of second lumber vertebrae behind the neck of pancreas by the junction of;

- Superior mesentric vein and
- The splenic vein
Liver is both secretary and excreatory organ. Liver is included one among the organs of the alimentary system because it produces a secretion the Bile which plays an important role in fat digestion.

And it is the main organ which receives nutrients and other organic materials from the digested food and re-distributes to other organs. This is mainly done by the portal system.

PORTAL SYSTEM

Any portal system is defined by the fact that the blood drains from one venous system to another without having arteries interposed between the two.
The ingested food substance are digested and absorbed into the venous side of the Gastro intestinal tract. This blood is high in nutrients and is also potentially high in toxins and needs to the filtered of unwanted material beyond being distributed to the rest of the body.

Thus portal vein brings de-oxygenated blood from stomach, intestine, Spleen and pancreas. Which is rich in monosaccharides, amino acids and hormones of Gastro intestinal tract.

The portal vein which enters the liver again becomes smaller and smaller in the substance of the liver until the second venous capillary bed is reached sinusoids of liver.

The blood from hepatic artery and portal vein gets mixed up in the hepatic sinusoids. The hepatic cell gets oxygen and nutrients from the sinusoids. The hepatocytes lining the sinusoids filter detoxify and add extract substance to and from the blood at this stage and once filtered the blood passes into the central vein.

This flow of blood from intestines to liver through portal vein is known as Enterohepatic circulation

The central Vein is the beginning of second venous system. The venous system is filled with progressively larger and large veins known as hepatic veins. The hepatic veins eventually drain the filtered blood into inferior venecava.
MODERN PATHOLOGY

HAEMORRHOIDS

“If Bile (or) phlegm be determined to the veins in the Rectum, it heats the blood in the veins and these veins becoming heated attract blood from the nearest veins, and being gorged the inside of the gut swells outwardly, and the heads of the veins are raised up, the faces passing out, and injured by the blood collected in them, they squirt blood, most frequently along with the faeces, but sometimes without faeces”.

Hippocrates (460-375 BC)

Definition:

Haemorrhoids (or) piles are the dilated veins within the anal canal in the sub-epithelial regions formed by radicals of superior, middle and inferior rectal veins.

Classification:

They are classified into two types

➢ Internal haemorrhoids

➢ External haemorrhoids
## Internal haemorrhoids vs External haemorrhoids

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Internal haemorrhoids</th>
<th>External haemorrhoids</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>They occur above the Hilton’s line</td>
<td>They occur below the Hilton’s line</td>
</tr>
<tr>
<td>2.</td>
<td>Due to varicosities developed by superior Rectal veins</td>
<td>Due to varicosities developed by inferior rectal veins</td>
</tr>
<tr>
<td>3.</td>
<td>Bright red (or) purple in colour, because they are covered with mucous membrane</td>
<td>It is situated outside the anal orifice and is covered by the skin</td>
</tr>
<tr>
<td>4.</td>
<td>They are painless as they do not have pain receptors</td>
<td>They are painful</td>
</tr>
<tr>
<td>5.</td>
<td>They bleed during straining</td>
<td>They do not bleed</td>
</tr>
</tbody>
</table>

Internal haemorrhoids is again divided into 2 types according to their cause of occurrence

1. **Primary haemorrhoids**

   Located at 3, 7, 11o’clock positions, related to the branches of the superior haemorrhidal vessels

2. **Secondary haemorrhoids**

   One which occurs between the primary sites.
**Degree of haemorrhoids**

<table>
<thead>
<tr>
<th>Degree</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Piles within that may bleed but do not prolapsed</td>
</tr>
<tr>
<td>Second</td>
<td>Piles that prolapsed during defecation but return back without manual help</td>
</tr>
<tr>
<td>Third</td>
<td>Piles that are prolapsed during defecation not but need to be replaced by manual help</td>
</tr>
<tr>
<td>Fourth</td>
<td>Piles that are permanently prolapsed</td>
</tr>
</tbody>
</table>

**Etiology**

- **Hereditary**
- **Morphological**
  - Absence of valves in the superior rectal veins
  - Radicals of superior rectal veins lie unsupported in loose connective tissue of the rectum
  - Contraction of veins which enter above the muscular layer increases the venous congestions below.
- **Occupational**
  - Heavy manual labours as porters prolonged as in drivers, Traffic police man.
- **Obesity**
- **Sedentary life style**
Raktha Moolam

- Low fiber diet
- Excess alcohol intake
  - Causes cirrhotic liver which leads to portal hypertension.
- Caffeine It can cause General hypertension

Secondary causes

- Chronic constipation
- Portal hypertension
- Venous stasis of pregnancy
- Uterine Tumours
- Tumours of the rectum

As this dissertation topic Raktha Moolam has the portal hypertension as their root cause for their occurrence. A short description about the portal hypertension is also discussed here

PORTAL HYPERTENSION

Definition

A sustained elevation of the portal venous pressure above 12mm Hg normally (8-12 mmHg).
Incidence

The most common cause of portal hypertension is cirrhosis of liver. The major cause for cirrhosis in Indian culture are,

- Alcohol
- Hepatitis B and C infections

Causes of Portal Hypertension

Table - 5

<table>
<thead>
<tr>
<th>Prehepatic</th>
<th>Hepatic(80%)</th>
<th>Post- hepatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Portal vein or splenic vein thrombosis</td>
<td>Portal pyaemia</td>
<td>Budd - chiari syndrome</td>
</tr>
<tr>
<td>Hypercoagulable Status</td>
<td>Alcoholic Cirrhosis</td>
<td>Constrictive pericarditis</td>
</tr>
<tr>
<td>Periportal Inflammation Trauma</td>
<td>Idiopathic Portal Hypertension</td>
<td>Congestive cardiac failure</td>
</tr>
<tr>
<td>Extrinsic Compression from pancreas, stomach</td>
<td>Primary Biliary Cirrhosis</td>
<td>Veno - Occlusive Disease</td>
</tr>
<tr>
<td>Neonatal Umbilicus Sepsis</td>
<td>Schistosomasis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hepatitis B, C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nodular regenerative hyperplasia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wilson’s disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Haemochromatosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Congenital Hepatic fibrosis</td>
<td></td>
</tr>
</tbody>
</table>
PATHOGENESIS OF PORTAL HYPERTENSION

Portal hypertension is the build up of pressure in the veins connecting the intestines and the liver, normally the pressure is low compared with the Arterial pressure.

Increase in portal pressure is primarily related to progressive hepatic Fibrosis.

The two major pathways leading to portal hypertension are

- Increased interhepatic vascular resistance (left)
- Increased inflow from the dilated hyper dynamic both the factors play an important role which to leads splanchnic circulations (Right)

When the liver parenchymal cells are destroyed due to inflammation and damage they have considerable powers of regeneration to restore the normal architecture.

However if the loss of liver cells is recurrent the following changes occur in hepatocytes.

- There is striking reduction in the number of fenestrate in the endothelial cells.
- Change of Fat storing cells into myofibroblast which secrete collagen I, II & IV.
Impaired synthesis of the vasodilator, nitric oxide and increased release of vasoconstrictor, endothelin.

This leads to collagenization of the space of tissue and the liver microcirculation may be altered.

They contribute to deprivation of nutrients intended for the hepatocytes and impaired hepatic removal of toxins which leads to loss of power of regeneration of liver called Cirrhosis, In turn the replaced fibrous tissue eventually contracts around the blood vessels. The hepatic flow of blood comes from both the hepatic artery 20% and the portal vein 80%.

As the resistance increases there by greatly impending the flow of portal blood vessels resulting in portal hypertension.
**Collateral circulation**

As the portal pressure increases in Normal 100% of the portal venous blood flow can be recovered where as in cirrhosis only 13% is obtained. The remainder tries to find another root to lower the pressure in venecava system. At the expense of pressure gradient it produces collaterals.
**Anastomoses between the portal and systemic venous system**

Collaterals form in area where capillaries and the venous systems are contiguous and often had been joined during embryonic stage.

**Table - 6**

<table>
<thead>
<tr>
<th>Site of anastomosis</th>
<th>Portal vessel</th>
<th>Systemic vessel</th>
<th>Signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Plexus around the lower end of oesophagus</td>
<td>Oesophageal branch of left gastric and short gastric vein</td>
<td>Lower systemic oesophageal vein</td>
<td>Haemetemisis (or) malena</td>
</tr>
<tr>
<td>2. Around umbilicus</td>
<td>Paraumbilical veins</td>
<td>Superior veins of the anterior abdominal wall</td>
<td>Caput medusae</td>
</tr>
<tr>
<td>3. Plexus around the lower third of rectum and anal canal</td>
<td>Superior haemorrhoidal vein</td>
<td>middle and inferior haemorrhoidal vein</td>
<td>Rectal varices (heavy bleeding through the anus)</td>
</tr>
<tr>
<td>4. Extraperitoneal surfaces of abdominal organ</td>
<td>Tributaries of superior and inferior mesenteric veins</td>
<td>Sub diaphragmatic and Retroperitoneal veins</td>
<td>Silent</td>
</tr>
</tbody>
</table>
Pathogenesis of Haemorrhoids in Portal hypertension

In the chronic liver disease due to any cause, scar tissue is formed which interrupts normal blood flow causing to sustained elevation in the blood pressure.

This sustained elevation of blood pressure leads to the visceral congestion and it causes the large amount of blood to be pooled in the portal system. It is analogous to water build up behind a dammed up stream. This occurs slowly as liver disease develops, allowing the body compensate by formation of more blood.

When a stream is dammed up, water accumulates until the dam overflows. Like that, there is increase in the portal pressure up to a maximum limit later it drains the remaining blood by opening the collateral connections due to \textbf{backward pressure of portal system.}

Connections from the portal system to the inferior venecava include.

- Haemorrhoidal veins
- Splenorenal collaterals and
- Some abdominal and retroperitoneal collaterals
Connections from the portal system to the superior venecave include

- Gastric verices
- Oesophageal varices

The re-opened umbilical veins through abdominal wall collaterals to the inter mammary system.

When the backward pressure prefers the inferior venecaval system, this deviation of blood into these channels leads to varicosities at the lower end of the anus called haemorrhoides.

These fragile blood vessels known as varices which become stretched swollen and twisted and in danger of breaking and bleeding.

Predisposing causes of internal hemorrhoids.

- Familial congenital weakness of the venous wall
- The weak submucous connective tissue of rectum
- The rectal venous plexuses which are most dependent on the portal circulation
- The venous return interrupted by the muscular contraction of rectum during defecation.

When a patients has above explained predisposing causes along with portal hypertension the occurrence of piles is very easily precipitated by cirrhosis of the liver.
Other clinical manifestations of portal Hypertension

Jaundice

In acute failure – Jaundice parallels the extent of liver cell damage

In cirrhosis - Jaundice is very mild.

Ascites

This is due to

- Lowering of plasma oncotic pressure
- Increased capillary hydrostatic pressure
- Increased lymph production in the space of tissue due to increased sinusoidal pressure.

Hepatic encephalopathy

Cirrhosis of liver also decreases hepatic clearance of substances from the systemic circulation this increases the portal input of substance such as ammonia and other toxic substance from Gastro intestinal tract, which by passes liver and the enters the brain producing the serious effect.

Spleenomegally

Increase in portal hypertension causes splenic congestion, the engorged spleen more effectively destroys blood cellular elements, contributing to anemia, leucopenia and Thrombocytopenia.
CONSEQUENCES OF CIRCULATORY CHANGES IN CIRRHOSIS

INTRAHEPATIC VASCULAR RESISTANCE

PORTAL HYPERTENSION

SPLENOMEGALY, PANCYTOPENIA

PORTO-SYSTEMIC SHUNTS

BLEEDING VARICES

TRANSLOCATION FROM GUT OF BACTERIA ENDOTOXINS

ASCITES

HEPATIC ENCEPHALOPATHY

SPONTANEOUS BACTERIAL PERITONITIS

HEPATOURENAL RENAL SYNDROME

SODIUM & WATER RETENTION

SYSTEMIC HYPOTENSION

ACTIVATION OF NEURO HUMORAL FACTORS

PORTAL SYSTEMIC TRANSLOCATION SHUNTS FROM GUT OF ACTIVATION OF BACTERIA NEURO HUMORAL ENDOOTOXINS

HEPATIC SPONTANEOUS RENAL HEATORENAL SYNDROME

BACTERIAL PERITONITIS

BLEEDING VARICES

ASCITES

HEPATIC ENCEPHALOPATHY
CLINICAL FEATURES OF BLEEDING PILES

Constipation

Longstanding constipation leads to the development of piles states and hence it is the commonest and prominent in the clinical history. History of constipation is not usual in the bleeding, but it occurs in later stage.

Pain and Tenuesmus

This is marked feature in most of the piles cases, but in bleeding piles is less prominent. This occurs during the defaecation process in long standing cases it becomes trouble some while maintaining day to day activities.

Discharge

Mucous discharge is present in selected cases during the bowel movements than that of fistula cases, well the watery discharge is a prominent feature through out the day. But this is not a phenomenon in bleeding piles.

Bleeding

Bleeding is a remarkable prominent symptom which brings the patient to physician’s table only when the notable amount of fresh blood is present during defaecation process which is often refered as “splashing of the pan”.

Protrusion of pile mass

In the longstanding cases of internal hemorrhoids protrusion becomes a common aspect which sometimes need manual corrections.

BLEEDING PER ANUS

Bleeding per anus can be classified into two groups of which one is painful and other is painless condition.

Bleeding per anus with pain

- Fissure in anus
- Fistula in ano
- Carcinoma of the anal canal
- Ruptured perianal haematoma
- Ruptured anorectal abscess
- Injury etc.

Bleeding per anus without pain

- Blood alone = polyp, villous adenoma and diverticular disease, complicated portal hypertension.
- Blood during defecation – haemorrhoid
- Blood with mucous: ulcerative colitis, crohn’s disease
  Intussusception, ischemic colon etc
- Blood mixed with stool – carcinoma of the colon
- Blood streaked on stool – carcinoma of the rectum, fissure in ano
Bleeding per anum other than haemorrhoides

Classification

➢ Defects of blood vessels
  a. The vascular purpurea
  b. Hereditary haemorrhagic telangiectasis

➢ Disorders of blood platelets

➢ Defects of the clotting mechanism
  a. Hereditary
  b. Acquired

The above mentioned are less common hemorrhage disorders due to defects in the clotting mechanism.

Differential Diagnosis

Fissure in ano

An enlarged abscess in the long axis of the lower anal canal. Acute anal fissure is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory induration or oedema of its edges. There is accompanying spasm of the anal sphincter muscles. Chronic anal fissure is characterised by inflamed indurated margins and base consisting of either scar tissue or the lower border of the internal sphincter muscles. Sharp, agonizing pain starting during defeacation, often overwhelming in intensity and lasting an hour or more. Bleeding is
Raktha Moolam

usually slight and consists of bright streaks on the stools. A slight discharge may present in fully established cases.

**Ano rectal abscess**

**These are three types**

- **Perianal**
  
- **Ischio – rectal and**
  
- **Pelvi rectal abscess**

  *Perianal abscess* an acutely tender rounded cystic lump will be seen by the side of the anal verge below the hilton’s line, palpated by the index finger and the abscess is less painful.

  *Ischio – rectal patient* will complain of excruciating throbbing pain by the side of anal canal on inspection browny oedematous swelling is felt on the side of the rectum.

  *Pelvi – rectal abscess* it mimics pelvis abscess. This abscess lies above the levator ani but below the pelvic peritoneum.

**External haemorrhoids: (Perianal haematomes)**

It is covered by the skin very painful and on examination a tense tender swelling which resembles a semi – ripe black current is seen. These episodes result in a tender blue swelling at the anal verge due to thrombosis of a vein in the external plexus.
**Prolapse of rectum**

The main complaints are something comes out per rectum during defaecation. It may come out spontaneously on standing, walking or coughing. The prolapse may reduce spontaneously or require digital reduction. Other symptoms are anorectal bleeding, mucous discharge anal pain.

**Fistula -in - ano**

A fistula in ano is attracting lined granulation tissue, which connects deeply in the anal canal or rectum and superficially on the skin around or rectum and superficially on the skin around the anus. It usually results from anorectal abscess which burst spontaneously. The fistula continues to discharge because of reinfection and no pain is felt on proctoscopic examination.

**Carcinoma of the rectum:**

The symptoms are bleeding during defaecation proliferative growth in the ampulla, sense of incomplete defaecation and the patient may get up in the morning with drawn urgent urge for defaecation. In case of annular carcinoma affecting the upper part of rectum, the patient complaints of increasing constipation. Liver should always palpated for metastasis. Peritoneum may be studded with secondary deposits. Ascites may be the result and enlarged iliac group of nodes can be felt.
Complications

- Profuse Hemorrhage
- Strangulations
- Thrombosis
- Ulceration
- Gangrene
- Fibrosis
- Suppuration
- Pylephlebitis (portal pyaemia)
EVALUATION OF THE DISSERTATION TOPIC

Materials and Methods

The pathological evaluation on topic “Raktha Moolam” was carried out at the Post Graduate Department of Noi – Naadal branch in Government Siddha Medical College, Palayamkottai.

1. Selection of cases

Bleeding per rectum of 20 cases are selected from the out patient department of Government Siddha Medical College, Tirunelveli Medical College, AVB Gastro care clinic and Velayutham Pillai hospital. In this 2 typical cases of Raktha Moolam was selected and followed by author whose work was under close supervision of the professor and lectures of the post graduate of Noi - Naadal Department.

Evaluation of clinical parameters

A detailed history and clinical features of the patients were taken carefully. The clinical history contains.

1. Detailed history of past and present illness.
2. Family history.
3. Personal history.
4. Occupational history.
5. Dietary habits.
6. Age and Sex difference.
Clinical features of Raktha Moolam

- Pain in (or) around the umbilicus
- Spurting of blood from the anus.
- Anaemia
- Easy Fatigability
- Oedema
- Throbbing chest pain
- Headache
- Drowsiness
- Yellowishness of the eyes.

Study on Siddha Clinical Diagnosis

Modes of investigating the cases are Poriyaal Arithal, Pulanaal Arithal and Vinaathal were adapted to assess the humoral pathology. These modes were carried on the fundamental of Udal Kattugal and Envagai Thervu.

The clinical investigation

For further detailed study about the modern investigatory parameter were used the following laboratory investigation were done in these cases.
Blood

- Total count
- Differential count
- Erythrocyte sedimentation rate.
- Haemoglobin estimation.

Urine

- Albumin
- Sugar
- Deposits

Motion

- Ova
- Cyst

Specific test

- Ultra sonogram
- Sigmoidoscopy (or) protoscopy
- Liver biopsy
- Liver function test.
**STATISTICAL ANALYSIS**

**RAKTHA MOOLAM**

**Statistical Analysis**

The study subjects are analysed by the statistic of mean, median, standard deviation and percentages. The inferences were arrived by the Test of significance students ‘t’ test. The risk factors are inferred by the odds ratio.

**Observation and results**

**Table 7: Age and sex wise classification of the study trials.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Sex</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>Total</th>
<th>Mean</th>
<th>S. d</th>
<th>‘t’</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>14</td>
<td>45.7</td>
<td>12.7</td>
<td>0.357</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>2</td>
<td>Female</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>6</td>
<td>46.7</td>
<td>13.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>2</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>3</td>
<td>20</td>
<td>46.0</td>
<td>12.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean ages of male and female or 45.7 ± 12.7 and 46.7 ± 13.3 respectively. The difference between the means is not statistically significant. The both sexes are having the same age.
Etiological factors

Table 8: The Etiological factors distributed with percentage.

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Aetiological factor</th>
<th>n</th>
<th>Cases affected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>1.</td>
<td>Alcohol</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>2.</td>
<td>Cirrhosis of liver</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>3.</td>
<td>Chronic constipation</td>
<td>20</td>
<td>16</td>
</tr>
<tr>
<td>4.</td>
<td>High fatty diet</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>5.</td>
<td>Prolonged sitting</td>
<td>20</td>
<td>9</td>
</tr>
</tbody>
</table>

Chronic constipation scored eighty percentage to the affected cases next, prolonged sitting scored 45%. Alcohol and heavy fatty diet scored 30% each. From the above table, cirrhosis of liver is also a cause for Raktha Moolam as it is a rare manifestation it scored only 10%.
**Risk factor**

Some habits of the study subjects are analysed. They are attributed to the risk factor of the diseases. The risk factors are enumerated below.

**Table 9: Attributed risk factors with diseases.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Risk factor</th>
<th>n</th>
<th>Odds ratio</th>
<th>Significance</th>
<th>Risk told</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Alcohol with Heavy fatty diet</td>
<td>6</td>
<td>3.7</td>
<td>Significance</td>
<td>3.7 times</td>
</tr>
<tr>
<td>2.</td>
<td>Prolonged sitting with chronic constipation</td>
<td>8</td>
<td>3</td>
<td>Significance</td>
<td>3 times</td>
</tr>
<tr>
<td>3.</td>
<td>Exposure to excessive heat with chronic constipation</td>
<td>5</td>
<td>4.3</td>
<td>Significance</td>
<td>4.3 times</td>
</tr>
</tbody>
</table>

The above table shows that the risk of alcohol with heavy fatty diet intake is 3.7 times greater than the risk of without heavy fatty diet. Prolonged sitting with constipation risk is 3 times greater than the prolonged sitting without constipation. Similarly Excess heat exposure of chronic constipation risk is 4.3 times greater than without excess heat exposure.
### Table 10: Percentage distribution of Mukkutra Nilaigal.

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Component</th>
<th>n</th>
<th>Types of Mukkutra Nilai</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Vali</td>
<td>20</td>
<td>Pranan</td>
<td>9</td>
<td>45.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Abanan</td>
<td>20</td>
<td>100.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Viyanan</td>
<td>4</td>
<td>20.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Uthanam</td>
<td>10</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Samanar</td>
<td>10</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Koorman</td>
<td>2</td>
<td>10.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Kirukaran</td>
<td>12</td>
<td>60.0</td>
</tr>
<tr>
<td></td>
<td>Devabathan</td>
<td>20</td>
<td></td>
<td>12</td>
<td>60.0</td>
</tr>
<tr>
<td>2.</td>
<td>Azhal</td>
<td>20</td>
<td>Anal pitham</td>
<td>12</td>
<td>60.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Ranjaga pitham</td>
<td>13</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Sadhaga pitham</td>
<td>5</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Prasaga pitham</td>
<td>13</td>
<td>65.0</td>
</tr>
<tr>
<td>3.</td>
<td>Iyam</td>
<td>20</td>
<td>Avalampagam</td>
<td>13</td>
<td>65.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Kilathagam</td>
<td>13</td>
<td>65.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Bothagam</td>
<td>11</td>
<td>15.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Tharpagam</td>
<td>2</td>
<td>10.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>Santhigam</td>
<td>1</td>
<td>5.0</td>
</tr>
</tbody>
</table>
Udal Thathukkal:

**Table 11: Distribution of Udal Thathukkal with percentage.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Udal thathukkal</th>
<th>n</th>
<th>affected No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Saaram</td>
<td>20</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Senneer</td>
<td>20</td>
<td>14</td>
<td>70.0</td>
</tr>
<tr>
<td>3.</td>
<td>Oon</td>
<td>20</td>
<td>4</td>
<td>20.0</td>
</tr>
<tr>
<td>4.</td>
<td>Kozhuppu</td>
<td>20</td>
<td>4</td>
<td>20.0</td>
</tr>
<tr>
<td>5.</td>
<td>Enbu</td>
<td>20</td>
<td>1</td>
<td>5.0</td>
</tr>
</tbody>
</table>

Cent percentages of the cases are affected by saaram. 70% of the study subjects are affected by senneer. The oon and kozhuppu are affected in 20% of the cases each. Enbu is affected only in 5% of the subjects.
Table 12: Study subjects are classified according to the Viralkadai Alavu.

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Viralkadai Alavu</th>
<th>n</th>
<th>Affected</th>
<th>No of cases affected</th>
<th>% of cases affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>120</td>
<td>12</td>
<td>60%</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>7 ½</td>
<td>20</td>
<td>6</td>
<td>30%</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>20</td>
<td>2</td>
<td>10%</td>
<td></td>
</tr>
</tbody>
</table>

The above table gives the Viralkadai Alavu of the Raktha Moolam subjects. From this Viralkadai Alavu 7 is noted for 60% cases then 7 ½ is noted for 30% cases and for Viralkadai Alavu 8 is noted only in 10% cases.
Ennvagai Thervugal

The study subjects were observed by the Siddha diagnosis. The observed diagnosed results are enumerated in the table.

**Table 13: Study subjects classified according to Ennvagai Thervugal.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Ennvagai thervugal</th>
<th>n</th>
<th>Types</th>
<th>Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>1.</td>
<td>Naadi</td>
<td>20</td>
<td>Azhal iyam</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Iya vali</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Vali Azhal</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Azhal Vali</td>
<td>4</td>
</tr>
<tr>
<td>2.</td>
<td>Sparisam</td>
<td>20</td>
<td></td>
<td>16</td>
</tr>
<tr>
<td>3.</td>
<td>Naa</td>
<td>20</td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>4.</td>
<td>Niram</td>
<td>20</td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>5.</td>
<td>Vizhi</td>
<td>20</td>
<td></td>
<td>14</td>
</tr>
<tr>
<td>6.</td>
<td>Malam</td>
<td>20</td>
<td></td>
<td>16</td>
</tr>
</tbody>
</table>

The above table shows the selection and observation of the cases. From this the Typical Raktha Moolam patients shows Iya Vali and Iya Azhal Naadi. In the other study subjects 20% of the cases are affected by Azhal Iyam and Azhal Vali each. 50% of the cases are affected by Vali Azhal. Sparisam is affected in 80% cases Vizhi is affected in 70% cases. The Naa and Niram are affected in 65% cases.
**Clinical features:**

**Table 14: Clinical findings of the study subjects.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Clinical features</th>
<th>n</th>
<th>Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>1</td>
<td>Pain in (or) around the umbilicus</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Bleeding per rectum</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>Anaemia</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>Easy fatigability</td>
<td>20</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>Oedema</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>Throbbing chest pain</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>Headache</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>Drowsiness</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>Yellowishness of eyes</td>
<td>20</td>
<td>2</td>
</tr>
</tbody>
</table>

The above analysis shows that cent percentage of the cases are having bleeding per rectum.

*Anaemia is seen in 60% cases. Easy fatigability and edemas are seen 55% and 25% respectively 10% cases are affected with Throbbing chest pain, Head ache, Drowsiness and Yellowish ness of Eye.*

*Thus, only the 2 typical cases satisfy all the clinical feature mentioned by the disease Raktha Moolam.*
**Laboratory Investigation:**

**Table 15: Analysis of the clinical Hematological trials.**

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Sex</th>
<th>n</th>
<th>%</th>
<th>Population mean</th>
<th>Space mean</th>
<th>S.D</th>
<th>‘t’</th>
<th>Significant</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>14</td>
<td>5</td>
<td>35.7</td>
<td>15 mgs</td>
<td>10.7</td>
<td>3.2</td>
<td>5.0</td>
</tr>
<tr>
<td>2</td>
<td>Female</td>
<td>6</td>
<td>2</td>
<td>33.3</td>
<td>13.25 mgs</td>
<td>9.8</td>
<td>2.0</td>
<td>4.2</td>
</tr>
</tbody>
</table>

From the above interpretations the Hb level in male is $10.7 \pm 3.2$ mgs%. Which is very less than 15mgs of normal mean value. The differences between the two mean values is highly statistically significant.

Similarly, the female sex is also having much lesser Hemoglobin level than the Normal value. This difference is also highly statistically significant.

In respect to Serum bilirubin The 2 typical cases alone has increased value compared to the Normal level.
DISCUSSION

Interpretation of Parameters

Patients with bleeding complaints from the anus were interrogated thoroughly and their history, aliments characters of signs and symptoms were noted in materials and method.

The inference obtained from this is explained as follows.

Age and Sex distribution

In this male were predominantly affected and the incidence is high in the age group above 40 years. This is due to their habits of alcoholism.

Family history

Genetic history present only in the general bleeding piles cases. However, in this play, it is an important predisposing character which easily contributes rupture of the veins and gets bleeding.

Personal habits

The incidence of this disease is high in peoples who take high fatty diet and alcoholic.

Alcoholic for more than 10 years.
Residential area

There is no direct relationship with the residential area. Irrespective of their residing place, the food habit and heavy alcoholism will lead to the disease soon.

Paruvakalangal

There is no direct relationship with seasonal variation.

Siddha parameters

Mukkutram

Vali, Azhal and Iyam constitutes the Mukkutrangal and the affected thathus are

Derangement in Vali

Table - 16

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types</th>
<th>Changes</th>
<th>Character</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pranan</td>
<td>Affected</td>
<td>Weakness of the limbs, headache, chest pain.</td>
</tr>
<tr>
<td>2</td>
<td>Abanan</td>
<td>Affected</td>
<td>Umbilicus pain, spurting of blood from the rectum.</td>
</tr>
<tr>
<td>3</td>
<td>Uthanan</td>
<td>Affected</td>
<td>Tiredness</td>
</tr>
<tr>
<td>4</td>
<td>Viyanan</td>
<td>Affected</td>
<td>Disturbed absorption and distribution of digested food substance.</td>
</tr>
<tr>
<td>5</td>
<td>Samanan</td>
<td>Affected</td>
<td>Disturbed circulation in portal vein. Indigestion.</td>
</tr>
<tr>
<td>6</td>
<td>Kooraman</td>
<td>Affected</td>
<td>Increased sleep.</td>
</tr>
<tr>
<td>7</td>
<td>Kirukaran</td>
<td>Affected</td>
<td>Loss of appetite.</td>
</tr>
<tr>
<td>8</td>
<td>Thevathathan</td>
<td>Affected</td>
<td>Tiredness, anxiety.</td>
</tr>
</tbody>
</table>
**Derangement of Azhal**

*Table – 17*

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types</th>
<th>Changes</th>
<th>Character</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anal pitham</td>
<td>Affected</td>
<td>Loss of appetite.</td>
</tr>
<tr>
<td>2</td>
<td>Ranjagam</td>
<td>Affected</td>
<td>Anaemia</td>
</tr>
<tr>
<td>3</td>
<td>Sathagam</td>
<td>Affected</td>
<td>Giddiness and emaciation</td>
</tr>
<tr>
<td>4</td>
<td>Prasagam</td>
<td>Affected</td>
<td>Paleness of the skin.</td>
</tr>
</tbody>
</table>

**Derangement of Kabam**

*Table – 18*

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types</th>
<th>Changes</th>
<th>Character</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Avalambagam</td>
<td>Affected</td>
<td>Dyspnoea</td>
</tr>
<tr>
<td>2</td>
<td>Kilethagam</td>
<td>Affected</td>
<td>Indigestion</td>
</tr>
<tr>
<td>3</td>
<td>Pothagam</td>
<td>Affected</td>
<td>Nausea and hesitating foods.</td>
</tr>
</tbody>
</table>
### Table – 19

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types</th>
<th>Changes</th>
<th>Character</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Saaram</td>
<td>Affected</td>
<td>Loss of weight, lassitude and diminished activity of the sense organs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Decreased)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Seneer</td>
<td>Affected</td>
<td>Anaemia and tiredness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Decreased)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Oon</td>
<td>Affected</td>
<td>Weight loss</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Decreased)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Kozhuppu</td>
<td>Affected</td>
<td>Emaciation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Decreased)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Enbu</td>
<td>Affected</td>
<td>Splitting of hairs and nails</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Decreased)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Mollai</td>
<td>Not affected</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Sukkilam / Suronitham</td>
<td>Not affected</td>
<td></td>
</tr>
</tbody>
</table>
Interpretation Of Ennvagai Thervu

Naa

If the disease process takes a long course then the Naa becomes coated and pale. Except this all other qualities of Naa are usually normal.

Niram

The colour of the body is pale in colour. The colour change is due to veluppu noi. Which ensues after a long course of Raktha Moolam.

Vizhi

Vel Vizhi has a pale or yellow colouration due to cirrhotic liver.

Sparisam

Body is said as Ushnam (or) hyperthermic. Other features of sparisam are usually normal.

Malam

The colour is usually reddish yellow due to bleeding from rectal varices or dark brown due to gastric varices.

Constipation is encountered in later stage.
Moothiram

➢ Neerkuri:

The colour is usually light reddish yellow. The amount is usually normal. But when veluppu and sobai develop the amount is reduced. There is no froth.

➢ Neikuri:

Salladai kan pol sitharal – typical case

Aravil Mothiram and mellana paraval – due to internal piles.

Naadi:

The Kai Naadai in Raktha moolam is “Pitha Vatham” and “Vatha Pitham” in internal piles.

Aya azhal in typical case.
Interpretation of Modern parameters

After examination of clinical features by envagai thervugal, the patient was subject to essential laboratory an investigation which includes the following.

Haematological examination

Haemoglobin is lowered

Urine examinations

are normal

Stool examination

It is positive for occult blood

Liver function test

Bilirubin level is raised

SGOT level is raised

SGPT level is raised

Alkaline phosphatase level is raised

Ultra Sono Gram of Abdomen

It shows Hepatomegaly patently cirrhosis of liver.

Colour Doppler study

It shows evidence of portal hypertension

Liver biopsy

It also shows the evidence of post necrotic cirrhosis of liver.
This Raktha moolam comes under Moola Noigal -21 in Yugi vaithya sinthamani which is explained by Yugi Munivar.

For any type of Moolam Vali humour is affected followed by alteration in other humors. But in Raktha moolam, Pitham is primarily vitiated this is followed by alteration in other humours.

The Raktha moolam is characterized by the presence of pain in the umbilicus, bleeding in the rectum, followed by anaemia, oedema, headache, chest pain and drowsiness which are the symptoms of internal piles, in addition to this yellowishness of the eyes is also present which indicates the manifestation of cirrhotic liver that makes the Raktha moolam, a rare disease.

The underlying pathogenesis for the Raktha moolam is any chronic liver disease resulting in cirrhosis leading to backward pressure in the portal vein and varicosities at the lower end of the anus.
CONCLUSION

The study on Raktha Moolam was carried out in this dissertation giving importance to the clinical features written in the poem and the changes, which are depicted in the Uyir Thathukkal and Udal Thathukkal.

All 1 to 20 cases were having the clinical features of bleeding per rectum and some of the cases have Anaemia, according to the severity of bleeding.

But only the 2 typical cases satisfy all the clinical features of the Raktha Moolam including yellowishness of the eyes.

On further investigating 1 to 20 typical cases with the help of modern parameters like, Liver function test, Liver biopsy, routine Blood and Urine investigations, Ultra sonogram and Colour Doppler study.

The study concludes that Raktha Moolam is an bleeding disorder in the anus which has the cirrhotic liver as the root cause for their occurrence followed by increased portal hypertension and varicosities in the anus.
BIBLIOGRAPHY

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3. Thirumoolar Karukkidai Vaithiyam – 600
4. Eswara maignana naadi
5. Agasthiyar – 2000
6. Agasthiyar Kanma Kaandam
7. Theriyar Gunavaagadam
8. Thirumoolar Ennyaram
9. Aaavi Alikkum Amuthamurai Churukkam
10. Aatham Rathchaamirtham – Kandasamy mudhaliyar
11. Siddha maruthuvam – Dr. Kuppusamy Mudhaliyar H.P.I.M.,
12. Udal Thathuval – Dr. P.M. Venugopal H.P.I.M.,
13. Noilla Neri – Dr. Durairasan H.P.I.M.,
14. Siddha Maruthuvanga Churukkam
   - Dr. K.S. Uthamarayan – H.P.I.M.,
15. Thottrakirama Aaraichiyam Siddha maruthva Varalarum
   - Dr. K.S. Uthamarayan – H.P.I.M.,
   - Dr. M. Shanmugavelu H.P.I.M.,
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22. Short Practice of surgery – Bailey & Love

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25. Disease of the Liver and Biliary System - Sheila Sherlock.

26. Principles of internal medicine – Harrison’s

27. Davidson’s Principle and practice of medicine.
A Study to Diagnose Raktha Moolam through Siddha Diagnostic Methodology

SELECTION PROFORMA


11. Address:
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………

12. Complaints and duration:
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………

13. History of present illness:
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………

14. Past history:
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………

15. Family History:
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
   …………………………………………………………………………………………………
<table>
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<tr>
<th>Habits</th>
<th>1.Yes</th>
<th>2.No</th>
</tr>
</thead>
<tbody>
<tr>
<td>16. Betelnut chewer :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Tea :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Coffee :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Prolonged sitting in a place:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. Smoking :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21. Excess Alcohol in take :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. High Fatty diet :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Low fiber diet :</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24. Intake of hot foods:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25. Food habits : V NV M</td>
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**GENERAL ETIOLOGY FOR RAKTHA MOOLAM**

<table>
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<tr>
<th>Habits</th>
<th>1.Yes</th>
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<tbody>
<tr>
<td>26. Exposure to Excessive cold</td>
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<td></td>
</tr>
<tr>
<td>27. Exposure to Excessive heat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28. Heavy intake of Tubers</td>
<td></td>
<td></td>
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<tr>
<td>29. Increased intake of salt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30. Heavy intake of spicy food</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31. Increased anger</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32. Indulging excessively in sexual act</td>
<td></td>
<td></td>
</tr>
<tr>
<td>33. Chronic Constipation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>34. Maintaining wrong Yogic posture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35. Hereditary Predisposition</td>
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<td></td>
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<tr>
<td>36. History of Kiranthi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>37. Venous stasis of Pregnancy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>38. P/H of Hepatitis B. infection</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
39. Portal hypertension

40. Tumours of the Rectum

GENERAL EXAMINATION

41. Weight (kg)

42. Temperature (°F)

43. Pulse rate/minute

44. Heart rate/minute

45. Respiratory rate/minute

46. Blood pressure (mmHg)

47. Pallor

48. Jaundice

49. Cyanosis

50. Lymphadenopathy

51. Pedal edema

52. Clubbing

53. Jugular venous pulsation

VITAL ORGANS EXAMINATION

54. Heart

55. Lungs

56. Brain

57. Liver

58. Kidney

59. Spleen

60. Stomach
# Siddha System of Examination

**Ennvagai Thervukal**

## Naa

61. *Maad Padinthiruthal*
   1. Present  
   2. Absent  

62. *Niram*
   1. Karuppu  
   2. Manjal  
   3. Velluppu  

63. *Suvai*
   1. Pulippu  
   2. Kaippu  
   3. Inippu  

64. *Vedippu*
   1. Present  
   2. Absent  

65. *Vai neer ooral*
   1. Normal  
   2. Increased  
   3. Reduced  

## Niram

67. *Niram*
   1. Karuppu  
   2. Manjal  
   3. Velluppu  

## Mozhi

68. *Mozhi*
   1. Sama oli  
   2. Urattha oli  
   3. Thazhntha oli  

## Vizhi

69. *Niram*
   1. Karuppu  
   2. Manjal  
   3. Sivappu  
   4. Velluppu  

70. *Kanneer*
   1. Present  
   2. Absent  

71. *Erichchal*
   1. Present  
   2. Absent  

72. *Peelai seruthal*
   1. Present  
   2. Absent  

**MEI KURI**

73. Veppam
   1. Mitham  
   2. Migu     
   3. Thatpam  

74. Viyarvai
   1. Normal   
   2. Increased 
   3. Reduced  

75. Thodu vali
   1. Present  
   2. Absent   

**MALAM**

76. Niram
   1. Karuppu  
   2. Manjal   
   3. Sivappu  
   4. Velluppu 

77. Sikkal
   1. Present  
   2. Absent   

78. Sirutthal
   1. Present  
   2. Absent   

79. Kalichchal
   1. Present  
   2. Absent   

80. Seetham
   1. Present  
   2. Absent   

81. Vemmai
   1. Present  
   2. Absent   

**MOOTHIRAM**

**NEER KURI**

82. Niram
   1. Venmai   
   2. Manjal   
   3. Crystal clear 

83. Manam
   1. Present  
   2. Absent   
84. **Nurai**

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>1. Nil</td>
<td>2. Increased</td>
<td>3. Reduced</td>
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85. **Edai (Ganam)**

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<tbody>
<tr>
<td>1. Normal</td>
<td>2. Increased</td>
<td>3. Reduced</td>
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86. **Enjal (Alavu)**

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<tbody>
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<td>1. Normal</td>
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87. **NEI KURI**

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<tbody>
<tr>
<td>1. Aravam</td>
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</tr>
<tr>
<td>3. Muthu</td>
<td>4. Aravil Mothiram</td>
</tr>
<tr>
<td>5. Aravil Muthu</td>
<td>6. Mothirathil Aravam</td>
</tr>
<tr>
<td>7. Mothirathil Muthu</td>
<td>8. Muthil Aravam</td>
</tr>
<tr>
<td>9. Muthil Mothiram</td>
<td>10. Asathiyam</td>
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<td>11. Mellena paraval</td>
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**NAADI (KAI KURI)**

I. **Naadi Nithanam**

88. **Kaalam**

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<table>
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<tr>
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<tbody>
<tr>
<td>1. Kaarkaalam</td>
<td>2. Koothirkaalam</td>
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<tr>
<td>3. Munpanikaalam</td>
<td>4. Pinpanikaalam</td>
</tr>
<tr>
<td>5. Ilavenirkaalam</td>
<td>6. Muthuvenirkaalam</td>
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89. **Desam**

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<tr>
<td>1. Kulir</td>
<td>2. Veppam</td>
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90. **Vayathu**

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<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>1. 1-33yrs</td>
<td>2. 34-66yrs</td>
<td>3. 67-100yrs</td>
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91. **Udal Vanmai**

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<tbody>
<tr>
<td>1. Iyalbu</td>
<td>2. Valivu</td>
<td>3. Melivu</td>
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92. **Vanmai**

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<tbody>
<tr>
<td>1. Vanmai</td>
<td>2. Menmai</td>
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93. Panbu

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<tr>
<td>1. Thannadai</td>
<td>2. Puranadai</td>
<td>3. Illaitthal</td>
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<td>13. Pakkanokku</td>
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94. Naadi nadai

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<tbody>
<tr>
<td>1 Vali</td>
<td>2. Azhal</td>
<td>3. Iyam</td>
</tr>
<tr>
<td>7. Azhal Iyam</td>
<td>8. Iya vali</td>
<td>9. Iya Azhal</td>
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95. MANIKKADAI NOOL (Viral Kadai Alavu)

IYMPORIGAL / IYMPULANGAL

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>1. Normal</td>
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<td></td>
</tr>
<tr>
<td>96. Mei</td>
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<td></td>
</tr>
<tr>
<td>97. Vaai</td>
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<td></td>
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<tr>
<td>98. Kan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>99. Mookku</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100. Sevi</td>
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KANMENTHIRIYANGAL / KANMAVIDAYANGAL

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<tbody>
<tr>
<td>1. Normal</td>
<td>2. Affected</td>
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</tr>
<tr>
<td>101. Kai</td>
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<td></td>
</tr>
<tr>
<td>102. Kaal</td>
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<td></td>
</tr>
<tr>
<td>103. Vaai</td>
<td></td>
<td></td>
</tr>
<tr>
<td>104. Eruvaai</td>
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<td></td>
</tr>
<tr>
<td>105. Karuvaai</td>
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### 106. YAAKAI

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### 107. GUNAM

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### UYIR THATHUKKAL

#### 1. Vali

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<tbody>
<tr>
<td>108. Uyirkkaal (Praanan)</td>
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<td></td>
</tr>
<tr>
<td>109. Keelnokkukkaal (Abaanan)</td>
<td></td>
<td></td>
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<tr>
<td>110. Paravukaal (Viyaanan)</td>
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<td></td>
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<tr>
<td>111. Melnokkukkaal (Udhaanan)</td>
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<tr>
<td>112. Nadukkaal (Samaanan)</td>
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<tr>
<td>113. VaanthikKaal (Naahan)</td>
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<tr>
<td>114. Vizhikkaal (Koorman)</td>
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<tr>
<td>115. Thummikkaal (Kirukaran)</td>
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<td></td>
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<tr>
<td>116. Kottavikkaal (Devathathan)</td>
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<tr>
<td>117. Veengukkaal (Dhananjeyan)</td>
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#### 11. Azhal

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<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>118. Aakkanal (Anal pitham)</td>
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<td></td>
</tr>
<tr>
<td>119. Ollolithe (Prasaka pitham)</td>
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<td></td>
</tr>
<tr>
<td>120. Vannaeri (Ranjaka pitham)</td>
<td></td>
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<tr>
<td>121. Nokkazhal (Aalosaka pitham)</td>
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<tr>
<td>122. Aatralangi (Saathaka pitham)</td>
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### III. Iyam

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<tbody>
<tr>
<td>123. Aliiyyam (Avalambagam)</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>124. Neerppiiyam (Kilethagam)</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>125. Suvaikaaniyam (Pothagam)</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>126. Niraivuiyam (Tharpagam)</td>
<td>☐</td>
<td>☐</td>
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<td>127. Ondriiyam (Santhigam)</td>
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### UDAL THATHUKKAL

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<tbody>
<tr>
<td>128. Saaram</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>129. Senneer</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>130. Oon</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>131. Kozhuppu</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>132. Enbu</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>133. Moolai</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>134. Suronitham/ Sukkilam</td>
<td>☐</td>
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### MUKKUTRA MIGU GUNAM

<table>
<thead>
<tr>
<th></th>
<th>1. Present</th>
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<tbody>
<tr>
<td>135. Emaciation</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>136. Blackish colouration of the body</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>137. Desire to take hot food</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>138. Tremors</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>139. Abdominal distension</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>140. Insomnia</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Weakness</td>
<td>1. Present</td>
<td>2. Absent</td>
</tr>
<tr>
<td>--------------------------------------------</td>
<td>------------</td>
<td>-----------</td>
</tr>
<tr>
<td>Weakness of sense organs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Giddiness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sluggishness</td>
<td></td>
<td></td>
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<tr>
<td>Constipation</td>
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<table>
<thead>
<tr>
<th>II. Azhal Migu Gunam</th>
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<tbody>
<tr>
<td>Yellowish discolouration of the skin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yellowish discolouration of the eye</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yellowish discolouration of urine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yellowish discolouration of faeces</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased appetite</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Burning sensation in the body</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III. Iyam Migu Gunam</th>
<th>1. Present</th>
<th>2. Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excessive salivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eraippu (dyspnoea)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heaviness of the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whiteness of the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chillness of the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduced appetite</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sluggishness</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
162. NOI UTRA KAALAM
1. Kaarkaalam
2. Koothirkaalam
3. Munpanikaalam
4. Pinpanikaalam
5. Ilavenirkaalam
6. Muthuvenirkaalam

163. NOI UTRA NILAM
1. Kurinji
2. Mullai
3. Marutham
4. Neithal
5. Paalai

164. Date of Birth

165. Time of Birth

166. Place of Birth

167. NATCHATHIRAM
1. Aswini
2. Barani
3. Karthikai
4. Rohini
5. Mirugaseeridam
6. Thiruvathirai
7. Punarpoosam
8. Poosam
9. Aavilyam
10. Makam
11. Pooram
12. Uthiram
13. Astham
14. Chithirai
15. Swathi
16. Visakam
17. Anusam
18. Kettai
19. Moolam
20. Pooradam
21. Uthiradam
22. Thiruvonam
23. Avittam
24. Sadayam
25. Poorattathi
26. Uthirattathi
27. Revathi
00. Not known
168. RAAASI

00. Not known ☐

RECTAL EXAMINATION

169. Inspection

1. Place :
   ........................................................................
   ........................................................................

2. Shape :
   ........................................................................
   ........................................................................

3. Colour :
   ........................................................................
   ........................................................................

4. Base :
   ........................................................................
   ........................................................................

5. Discharge :
   ........................................................................
   ........................................................................

170. Palpation :
   ........................................................................
   ........................................................................

171. Bimannual Examination:
   ........................................................................
   ........................................................................
   ........................................................................
INVESTIGATION

BLOOD

172. TC (Cells/cumm) : [ ] [ ] [ ] [ ]


174. Hb (gms%) : [ ] [ ]

175. E.S.R. (mm/hr) : 1.1/2hr [ ] 2.1hr [ ]

176. Blood Sugar (R) (mgs%) : [ ] [ ]

URINE

177. Albumin : 0. Nil [ ] 1.Trace [ ] 2.+ [ ] 3.++ [ ] 4.+++ [ ]

178. Sugar : 0. Nil [ ] 1.Trace [ ] 2.+ [ ] 3.++ [ ] 4.+++ [ ]

DEPOSITS

1. Yes 2. No

179. Pus cells [ ] [ ]

180. Epithelial cells [ ] [ ]

181. RBCs [ ] [ ]

182. Crystals [ ] [ ]

MOTION TEST

1. Yes 2. No

183. Ova [ ] [ ]

184. Cyst [ ] [ ]

185. Occult blood [ ] [ ]
186. Liver Function Test:
…………………………………………………………………………………………
…………………………………………………………………………………………

187. USG - Abdomen:
…………………………………………………………………………………………
…………………………………………………………………………………………

188. Proctoscopy:
…………………………………………………………………………………………
…………………………………………………………………………………………

189. Sigmoidoscopy:
…………………………………………………………………………………………
…………………………………………………………………………………………

CLINICAL SYMPTOMS OF RATHA MOOLAM:

190. Pain in and around the umbilicus  □  1. Present  □  2. Absent
191. Bleeding per Rectum          □  □
192. Anaemia                      □  □
193. Easy Fatigability            □  □
194. Oedema                       □  □
195. Throbbing chest pain         □  □
196. Head ache                    □  □
197. Drowsiness                   □  □
198. Yellowishness of Eyes        □  □
PROTOCOL

A STUDY TO DIAGNOSE KURUTHI MOOLAM THROUGH SIDDHA DIAGNOSTIC METHODOLOGY

By
Dr. G. SUBATHRA,
P.G. STUDENT,
DEPARTMENT OF NOI NAADAL,
G.S.M.C, PALAYAMKOTTAI.

1. BACKGROUND

RAKTHA MOOLAM:-

It denotes the massive bleeding from the Anus. The Massive bleeding is caused due to rupture of the rectal varices in the rectal canal as a complication of portal hypertension.

In Yugi Vaithiya Sinthamani, Yugi classified 21 types of Moolam, Raktha moolam is one among the moolam classifications.

YUGI EXPLAINED “ Raktha moolam “ as mentioned below

துற்று வியாயம் தோற்றம் குள குளம் வியாயம்
மஞ்சலில் ஓட்டம் லேகோ புரூகம்
செரியில் செரியில் லேகோ புரூகம்
மிகுகவுன் லேகோ புரூகம் செரியில்
எனில் மாவு பிளைட்டில் கீழே இடையில்
மாவுள் செரியில் லேகோ புரூகம்
நாவும் துற்று வியாயம் தோற்றம் குளம் வியாயம்
மஞ்சலில் ஓட்டம் லேகோ புரூகம்
செரியில் செரியில் லேகோ புரூகம்
மிகுகவுன் லேகோ புரூகம் செரியில்.
In Raktha moolam, Pitha humour is altered. As a result of this the following symptoms will occur,

Pain around the umblicus, Massive bleeding from the rectum, analcanal, weight loss, oedema, chest pain, Head ache, Faintness, Yellowishness of eyes.

2. AIM

a) PRIMARY AIM

To Diagnose Raktha moolam through envagai thervu, manikadai nool.

b) SECONDARY AIM

To co-relate Raktha moolam with Nilam, Kaalam, Sothidam.

3. POPULATION AND SAMPLE

Raktha moolam (as explained above under the song). Patients satisfying the inclusion and exclusion criteria mentioned below.

The samples of Raktha moolam patients are selected from O.P. and I.P. departemtns of Govt. Siddha medical college, Palayamkottai, Tirunelveli medical colleges High ground, under the guidance of Faculties and Head of the department of post graduate. NOI NAADAL DEPARTMENT
4. SAMPLE SIZE
A sample of 20 patients will be taken for detailed study.

5. INCLUSION CRITERIA
1. Age – above 40 years
2. Complaints – insidious onset of intermittent bleeding from anus.
3. Willing to give blood and urine specimen for investigations whenever required.
4. Willing to co-operate manual examinations – rectal

6. EXCLUSION CRITERIA
1. Bleeding piles due to General constipation & straining at stool
2. Bleeding piles due to pregnancy
3. Bleeding piles due to Tumours of the rectum
4. Bleeding piles due to Hereditary predisposition

7. CONDUCT:
Raktha Moolam patients satisfying the inclusion and exclusion criteria will be included in this study.

Siddha diagnostic procedure such as Envagai thervu, Manikadai nool, Nilam, Kalam and Sothidam of the patients will be noted.

8. FORM:-
FORM – Diagnostic proforma for Raktha moolam
Formation of Portal Vein

Fig 1
Tributaries of Portal Vein

Fig 2
Portal Venous System

Fig 3
Locations of Haemorrhoids

Fig 4
Intra Hepatic Causes of Cirrhosis

Fig 5
Pathogenesis of Portal Hypertension

Blood backing up into the portal vein leading to varices

Fig 6
Rectal Varices due to Portal Hypertension

Fig 7
Direct view of Haemorrhoid seen on Sigmoidoscopy

Fig 8
# Clinical Laboratory Report

**Name:** Mr. Shanmugasundram 45 M  
**Referred by:** Dr. G. Badari Narayanan M.D. D.M. MACG (USA)  
**Date:** 11-6-2003

## Blood

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>8.8 Grams %</td>
</tr>
<tr>
<td>T.W.B.C. Count</td>
<td>7,600 cells/cmm</td>
</tr>
<tr>
<td>Differential Count</td>
<td></td>
</tr>
<tr>
<td>RBC</td>
<td>4.5</td>
</tr>
<tr>
<td>WBC</td>
<td>5,000 m/cmm</td>
</tr>
<tr>
<td>Platelets</td>
<td>2.5</td>
</tr>
<tr>
<td>E.S.R. 1/2 Hour</td>
<td>60 m/m</td>
</tr>
<tr>
<td>E.S.R. 1 Hour</td>
<td>125 m/m</td>
</tr>
<tr>
<td>Urea</td>
<td>23 mg/dl</td>
</tr>
<tr>
<td>Sugar</td>
<td>110 mg/dl</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>118 mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.2 mg/dl</td>
</tr>
<tr>
<td>Amylase</td>
<td>26 U/L</td>
</tr>
<tr>
<td>SGOT</td>
<td>77 U/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>25 U/L</td>
</tr>
<tr>
<td>ALK Phosphatase</td>
<td>510 mu/ml</td>
</tr>
<tr>
<td>Gamma GT</td>
<td>98 U/L</td>
</tr>
<tr>
<td>BILIRUBIN</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1.2 mg/dl</td>
</tr>
<tr>
<td>Direct</td>
<td>0.7 mg/dl</td>
</tr>
<tr>
<td>Indirect</td>
<td>0.5 mg/dl</td>
</tr>
<tr>
<td>PROTHROMBIN TIME</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>15 Seconds</td>
</tr>
<tr>
<td>Test</td>
<td>19 Seconds</td>
</tr>
<tr>
<td>PROTEIN</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>7.0 g/dl</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.7 g/dl</td>
</tr>
<tr>
<td>Globulin</td>
<td>3.3 g/dl</td>
</tr>
</tbody>
</table>

## Urine

- Light yellow colour, trace clear sample.
- Bile salts: Negative
- Bile pigments: Negative
- Urobilinogen: Positive 1 in 4 dil
- Deposits: Pus cells 3-5 and also seen/HPF.
- Blood: HBS Ag: Negative.
- HCV: Negative.

---

**Working Hours:** 9.00 A.M. to 9.00 P.M. Sunday Holiday

---

*Lab Technologist*
Thank you for the reference.
Name : Mr. Shanmuga Sundaram 45/M
Date : 16/05/2003.
Ref.by:Dr : Executive Health Check up.

ULTRASONOGRAM OF ABDOMEN:

Liver is moderately enlarged measuring 15.9 cms in the long axis.
Parenchyma shows coarse patchy hypoechoic areas. Surface is irregular. Biliary passages are not dilated.
Portal vein measures 1.37 cms in diameter.

Gall bladder normal configuration. No intra-luminal densities.
Walls are uniformly thick.

Pancreas normal contour and echogenicity.

Spleen is slightly enlarged, measuring 13.2 cms in the long axis.

Kidneys are of normal size & architecture. Right kidney measures 10.5 X 3.8 cm and the left 10.3 X 4.1 cm.
The collecting system is not dilated.

Urinary bladder normal configuration. Trans – Sonic.

IMPRESSION:

1. Hepatomegaly with coarse patchy hypoechoic regions suggestive of cirrhosis liver.
2. Gallbladder shows thick walls.
3. Splenomegaly.
4. Kidneys are normal.
Thank you for the reference.
Name : Mr. Shanmuga Sundaram 45/M
Date : 11/06/2003.
Ref.by:Dr : G. Badarinarayanan., MD,DM,(GAS),

ULTRASONOGRAM OF ABDOMEN & COLOUR DOPPLER:
The portal vein measures 1.41 cms in diameter at the porta hepatis.

Flow is hepatopetal. Spontaneous signals with nophasic variations noted.

Hepatic veins are of normal caliber.

Dilated veins are seen in the region of the lesser omentum.

No recanalisation of the para umbilical vein.

Spleenic vein shows spontaneous phasic signals.

IMPRESSION:

1. Colour Doppler shows evidence of portal hypertension.

Dr. Clara Jeyakumar, MBBS,
**SHAKTHI LABORATORIES**

**SHAKTHI DIAGNOSTIC CENTRE**

10/5, "Harsha Complex", Vannarpettal, TIRUNELVELI - 627 003. ☏️: 2500843, 2501136

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>66</th>
<th>Visit Date:</th>
<th>31-Mar-08</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient Name</td>
<td>Mr. Shunmuga Sudaram</td>
<td>Age/Sex</td>
<td>50/M</td>
</tr>
<tr>
<td>Ref By</td>
<td>Dr. G. Badri Narayanan MD., DM.,</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**COMPLETE HAEMOGRAM**

( by ABX Micros-60, Haematology Analyser)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>RESULTS</th>
<th>UNITS</th>
<th>REFERENCE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAEMOGLOBIN</td>
<td>3.8 grams/dl</td>
<td></td>
<td>Male: 13.5 to 17 12 to 15.5</td>
</tr>
<tr>
<td>TOTAL WBC COUNT</td>
<td>5,200 cells/cumm</td>
<td></td>
<td>Female: 4,000 to 10,000</td>
</tr>
<tr>
<td>DC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>POLYMORPHS</td>
<td>66 %</td>
<td></td>
<td>Male: 40 to 65</td>
</tr>
<tr>
<td>LYMPHOCYTES</td>
<td>25 %</td>
<td></td>
<td>Female: 30 to 50</td>
</tr>
<tr>
<td>EOSINOPHILS</td>
<td>9 %</td>
<td></td>
<td>Male: 2 to 8</td>
</tr>
<tr>
<td>MONOCYTES</td>
<td>0 %</td>
<td></td>
<td>Female: 2 to 4</td>
</tr>
<tr>
<td>BASOPHILS</td>
<td>0 %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.S.R</td>
<td></td>
<td></td>
<td>Male: Less than 1</td>
</tr>
<tr>
<td>1/2 HOUR</td>
<td>5 mm</td>
<td></td>
<td>Female: 5 to 20</td>
</tr>
<tr>
<td>1 HOUR</td>
<td>14.0 mm</td>
<td></td>
<td>Male: 40 to 52</td>
</tr>
<tr>
<td>HAEMATOCRIT (P.C.V.)</td>
<td></td>
<td></td>
<td>Female: 38 to 45</td>
</tr>
<tr>
<td>PLATELET COUNT</td>
<td>1,27,000 cells/cumm</td>
<td></td>
<td>Male: 1,50,000 to 4,00,000</td>
</tr>
<tr>
<td>TOTAL RBC COUNT</td>
<td>1.50 millions/cumm</td>
<td></td>
<td>Female: 4.6 to 6.0</td>
</tr>
<tr>
<td>MCV (Mean Cell Volume)</td>
<td>33 fl</td>
<td></td>
<td>Male: 4.2 to 5.4</td>
</tr>
<tr>
<td>MCH (Mean Cell Haemoglobin)</td>
<td>25 pg</td>
<td></td>
<td>Female: 76 to 96</td>
</tr>
<tr>
<td>M.C.H.C.</td>
<td>27 grams/dl</td>
<td></td>
<td>Male: 27 to 31</td>
</tr>
<tr>
<td>RDW (Red cell Distribution Width)</td>
<td>11.3 %</td>
<td></td>
<td>Female: 32 to 36</td>
</tr>
<tr>
<td>TOTAL NEUTROPHILS</td>
<td>3,432 cells/cumm</td>
<td></td>
<td>Male: 11.5 to 14.5</td>
</tr>
<tr>
<td>TOTAL LYMPHOCYTES</td>
<td>1,300 cells/cumm</td>
<td></td>
<td>Female: 2000 to 8000</td>
</tr>
<tr>
<td>TOTAL EOSINOPHILS</td>
<td>468 cells/cumm</td>
<td></td>
<td>Male: 40 to 440</td>
</tr>
<tr>
<td>TOTAL MONOCYTES</td>
<td>0 cells/cumm</td>
<td></td>
<td>Female: Upto 300</td>
</tr>
<tr>
<td>TOTAL BASOPHILS</td>
<td>0 cells/cumm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.P.V (Mean Platelet Volume)</td>
<td>7.6 fl</td>
<td></td>
<td>Male: Upto 40</td>
</tr>
<tr>
<td>P.D.W. (Platelet Distribution Width)</td>
<td>12.5 %</td>
<td></td>
<td>Female: 6.5 to 11.0</td>
</tr>
</tbody>
</table>

**PATHOLOGIST/BIOCHEMIST**

FACILITIES AVAILABLE: Haemology analyser, Bio-Chemistry analyser, ELISA Reader, Blood gas analyser, Electrolyte Analyser, Microbiology Lab, Hormone assay Lab.
Mr. S. Shanmuga sundaram 50/M 2.4.08

Ref. By Dr. G. Badarinarayanan M.D., D.M., (Gastro)

MOTION ANALYSIS

Appearance: Brown colour, Blood stained sample.

Microscopic: Ova - Nil  Cyst - Nil

Fat Globules: Negative

Occult Blood: POSITIVE

Lab - Technician
**AVB** *8471*

**Name**: Mr. S. Shanmuga Sundaram  
**Age**: 50 / Male

**Ref By**: Dr. G. Badarinarayanan M.D., D.M., FACG (USA)

**Date**: 31-03-08

### LIVER FUNCTION TEST

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin - Total</td>
<td>2.7 mg/dl</td>
</tr>
<tr>
<td>Direct</td>
<td>2.0 mg/dl</td>
</tr>
<tr>
<td>Indirect</td>
<td>0.7 mg/dl</td>
</tr>
<tr>
<td>S.G.O.T</td>
<td>15 U/L</td>
</tr>
<tr>
<td>S.G.P.T</td>
<td>288 U/L</td>
</tr>
<tr>
<td>ALK. PHOSPHATASE</td>
<td>160 U/L</td>
</tr>
<tr>
<td>Gamma GT</td>
<td>60 U/L</td>
</tr>
<tr>
<td>Protein - Total</td>
<td>6.2 g/dl</td>
</tr>
<tr>
<td>Albumin</td>
<td>2.8 g/dl</td>
</tr>
<tr>
<td>Globulin</td>
<td>3.4 g/dl</td>
</tr>
<tr>
<td>Prothrombin Time - Control Test</td>
<td>15 Secs</td>
</tr>
<tr>
<td></td>
<td>17 Secs</td>
</tr>
<tr>
<td>Plasma Ammonia</td>
<td>90 mmol/L</td>
</tr>
<tr>
<td>HBs Ag.</td>
<td>NEGATIVE</td>
</tr>
</tbody>
</table>
**Upper GI Endoscopy Report**

1. **IInd Part of Duodenum**
2. **Pyloric Orifice**
3. **Healthy Gastric Mucosa**
4. **Gr.I Oesophageal Varices (Magnified)**
5. **Diffuse Oesophageal Candidiasis**

**Impression**

Gr.IV. Oesophageal Varices / Diffuse Oesophageal Candidiasis.
SARANYA ENDOSCOPIC SERVICES

AVB Gastro Care Clinic
121, Thiruvananthapuram Road,
(Opp. Vaikaalpalam Bus Stop)
Palayamkottai - 627 002.

Phone : 2581630
Fax : 91-462-2573213
E-mail : badaritvl@dataone.in

CONSULTATION BY APPOINTMENT
GASTRO - ENTEROLOGY WING

SIGMOIDOSCOPY REPORT

Name : Mr. S. Shanmugasundaram  Age : 50  Sex : M  Date : 2-4-2008

RECTAL EXAMINATION :
- Sphincter Tone
- Anal Verge
- Mucose
- Finger Stall
- Pile - Mass
- Mass - lesion
- Miscellaneous

Extensive Internal Pile mass present.

PROCTO - SIGMOIDOSCOPE :

- Passed upto
- Mucosa
- Blood
- Mucus
- Pile - mass
- Mass lesion
- Faeces
- Foreign body
- Miscellaneous
- BIOPSY

25cms.

Extensive Internal Pile Mass with Bleeding tendency.

EXTENSIVE INTERNAL PILE MASS WITH BLEEDING TENDENCY.

Dr. G. Badarinarayanan
M.D.D.M.F.A.C.G. M.O.M.G.E. (Gastro enterology)
Member Indian Society of Gastro enterology
Member, Society of Gastro Intestinal Endoscopy of India
Fellow, American College of Gastro enterology
Member of Organisation Mondiale De Gastro enterology
Formerly Gastro Enterologist, Endoscopist
Tirunelveli Medical College Hospital
Consultant Gastro enterologist
Referee by Dr.

Dr. G. Badarinarayanan MD DM MACG

Thank you for the reference

Ref. No.: 2599/2003

SURGICAL PATHOLOGY REPORT

Name of Patient: Mr. S. Shanmugasundaram
Age & Sex: 45/M
Date of accessioning: 23.5.2003
Date of Report: 27.5.2003
Specimen accessioned: Biopsy Liver

Macroscopic Description
Specimen of tiny linear fragments of grey white tissue 0.3cms

Microscopic Description
Section studied shows liver tissue with loss of lobular architecture with clusters of hepatocytes separated by bands of fibrocollagenous with portal triads surrounded by chronic inflammatory cell infiltration.

Diagnosis
SUGGESTIVE OF A POST NECROTIC CIRRHOSIS OF LIVER

Dr. K. Shantaraman M.D.
Consultant Pathologist

Dr. K. Swaminathan M.D.
Consultant Pathologist

Dr. J. Suresh Durai B.Sc., M.D.
Consultant Pathologist