

A STUDY ON PANDU NOI

Dissertation submitted to

**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
Chennai-32**

For the partial fulfillment of the requirements to the Degree of

**DOCTOR OF MEDICINE (SIDDHA)
(Branch IV - Kuzhanthai Maruthuvam)**



**DEPARTMENT OF KUZHANTHAI MARUTHUVAM
GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI – 627 002.**

MARCH - 2009

ACKNOWLEDGEMENT

First, I have to thank the almighty God for giving the loving parents and express my above and colossal gratitude to them for their valuable support and encouragement in my educational career from the very beginning.

I wish to express my gratitude and acknowledgement to the **Vice Chancellor** Tamil Nadu Dr. M.G.R. Medical University, Chennai and the **Special commissioner**, Commissionerate of Indian Medicine and Homeopathy and **Joint Director** of Indian Medicine and Homeopathy, Chennai, for providing an opportunity to persue this dissertation study.

I also wish to convey my deep gratitude to our Principal **Dr. R. Devarajan M.D(S).**, and Vice Principal **Dr. S.Soundararajan M.D(S).**, Govt. Siddha Medical College, Palayamkottai for providing all the necessary facilities.

I am grateful to our professor **Dr. N. Chandra Mohan Doss M.D(S).**, Head of the Department, Post Graduate Kuzhanthai Maruthuvam Department, Government Siddha Medical College, Palayamkottai for his valuable guidance and encouragement for this study.

With a deep sense of my gratitude I thank to **Dr. R.Patturayan M.D(S).**, HOD. Department of Kuzhanthai Maruthuvam, National Institute of Siddha, Chennai.

I express my whole hearted thanks to **Dr. D. K. Soundararajan M.D(S).**, Lecturer and **Dr. K. Shyamala M.D(S).**, Assistant Lecturer of Post Graduate Kuzhanthai Maruthuvam Department for their suggestion and guidance to this work.

I also thank to **Dr. Kathir Subramaniyam M.D., D.C.H H.O.D** Department of Pediatrics, Tirunelveli medical college.

It is my pleasure and privilege to record my deep gratitude to **Dr. M. Mathivanan M.D., D.C.H.** Assistant Professor the Department of Pediatrics, Tirunelveli Medical College for his suggestion and guidance in this work.

My heartfelt thanks to **Mr. M. Kalaivanan M.Sc.**, Lecturer and his staff of Department of Pharmacology Govt. Siddha Medical College, Palayamkottai for their guidance and help in conducting Pharmacological studies associated with this dissertation.

I am thankful to **Mrs. N.Nagaprema M.Sc.**, Head of the Department of Bio Chemistry and all staff members of the Department of biochemistry, Govt. Siddha Medical College, Palayamkottai for their support in conducting biochemical analysis involved in this study.

I express my sincere thanks to librarian **Mrs. T.Poonkodi M.A., MLIS**, Govt. Siddha Medical College, Palayamkottai and her staffs for their co-operation during this study.

I thank to **Dr. S.Bagirathi, M.B.B.S.**, and all staffs of the Department of Clinical Pathology for all their helps in the clinical investigation and discussion.

My heartfelt thanks to **Dr. Padma M.B.B.S D.M.R.D.** Department of Radiology, Government Siddha Medical College, Palayamkottai.

I gladly acknowledge my husband **Mr.K.Raja** for his timely help in doing this work.

I express my heartfelt thanks to my colleagues in Kuzhanthai Maruthuvam for their support in completing this work.

Finally, I thank **Broad Band Net Café**, Tirunelveli and staffs for their excellent Lithographic work.

INTRODUCTION

Siddha system of medicine is being practiced from the birth of our Tamil language. It has its own lot of specialities comparing other medical systems.

Siddha system of medical practice is considered as divine art as it lays its emphasis on inner soul in addition to that of external body. This system was successfully practiced by divine and spiritual scientists of ancient time who were known as siddhars and they were associated with religion and philosophy. According to this system, man and nature are inseparable and interdependent.

This system has the unique features like removal of the root cause of the disease and perfect remedies for body, mind and soul.

Siddhars proposed various theories out of which the basic and important ones are.

1. Pancha Bootham Theory
2. Three Humoral Theory

1. **Pancha Bootham Theory**

Tolkappiyam, says that the universe is formed by five elements viz, earth, water, fire, air and Ether.

“நிலம் நீர் தீவளி விசும்பேரடைந்தும்
கலந்த மயக்கம் உலகமறதலின்.”

Siddha system teams the above five elements as pancha bootham as every living organism is formed of it in definite proportions. When there occurs change in the proportion, it gives rise to disease.

2. The Three humoral theory

In siddha system of medicine, the three dhosas namely vata, pitta, kapa are the essential constituents of living body which are responsible for regulating all the body functions.

“உயிர்க்காதாரம் உயிர்தொடுதனவும்
முப்பிரிவாகி முக்குணமனுகி
உடலையும் உயிரையு மேம்பிக்காத்து
வருமென முதுமறை வகுக்குந் துணிபே”

- நோய் நாடல் நோய் முதனாடல் திரட்டு

The three humours circulate in the body in different proportions, help in the digestion of foods and maintain the vitality of the body. When there is provocation in the ratio of humours it will disturb the normal condition and will result in dryness (Vatham), heat (Pitham) and Cold (Kapam).

In general, the siddhars contributed wonderful medicines which are in consonance with the composition of the body.

Kuzhanthai maruthuvam is a specialized branch in siddha medicine which deals with the treatment of the diseases of children upto 12 yrs.

The disease that occurs in children can be broadly divided into two viz, karuvil thondrum Noigal or (Ahakaarana Noigal), diseases that occur during the intrauterine period. (Purakaarana Noigal), diseases that occur after the birth of the child, i.e., due to extrinsic causes.

Pandu Noi is a very common disease in children due to malnutrition, ignorance, poverty and poor socio-economic conditions and also among the affluent and well fed children due to unbalanced diet.

Medicine is not only a science but also an art. The science of medicine is vital to man's well-being and survival.

The author selected "Pandu Noi", which can be correlated with the clinical condition called iron deficiency anemia for this dissertation work. To treat this, a safe and common drug is necessary and so Bringaraja Chooranam and Madhulai Manappagu has been chosen as trial drug.

AIM AND OBJECTIVES

AIM

The author selected “Pandu Noi” for this dissertation study because.

Even though our country is developing one, even now some of our people are in poverty line, under poor socio economic status. They have been suffering from various diseases.

“Pandu Noi” affects all from paediatrics to geriatrics, male and female despite wealth and poverty. The multiple aetiology for pandu also impress the author to approach with a view to correlate with that of iron deficiency anemia.

OBJECTIVES

1. To take authentic measures and review the ideas of pandu noi as indicated in siddha literatures and to know the efficacy of the trial medicine.
2. To have an idea about the prevalence of pandu noi with reference to age, sex, socio-economic status, poverty, seasonal variations, land etc.,

3. To know the extent of correlation of aetiology, classification, symptomatology diagnostic methods and line of treatment compared with iron deficiency anemia.
4. To know the alteration of the disease under the topics, mukkutram, udal kattukal, poripulungal, envagai thervugal, neerkuri, neikuri.
5. To make a clinical trial on patients with the trial medicines viz, Bringaraja Chooranam and Madhulai Manappagu in the treatment of pandu noi.
6. To make use of modern parameters in the investigation side to confirm the diagnosis and to follow the progress of the patients.
7. To elicit biochemical analysis and pharmacological action of the trial medicines.

REVIEW OF LITERATURE

SIDDHA ASPECT

Siddha literatures deal with classification of diseases mainly by mukkutra theory i.e., vatham, pitham and kabam. These texts provide us with a line of treatment both for sthoola and sookkuma bodies.

Pandu noi is caused due to derangement of pitham. Hence the basic details regarding Pitham are briefly explained before going into the study about Pandu Noi.

Mukkutra Theory: Pitham

Pitham (Azhai) is one of the three vital phenomena (Vatham, Pitham and Kabam). Among the panchaboothas, it is formed by Theyu bootham. In healthy individuals, the existence of the three humours is found in the ratio of 1:1/2:1/4 respectively. This ratio is altered when there is disturbance to pitha dhosham by the dietetic habits, environmental factors etc, which leads to alteration of pitham leading to pitha diseases.

Location of Pitham in the body:

Pingalai, Piranavayu, Neerpai, Moolaakkini, Irudhayam, Thalai, Koppul, Undhi, Iraippai, Viyarvai, Naavil Oorukintraner, Senneer, Saaram, Kan, Thol.

General Characteristics of Pitham:

- Veppam (Heat)
- Koormai (Sharpness)
- Neippu (Lubricative)
- Nekizhchi (Elastic)

Pitham gets the properties of the substance to which it combines.

Natural Properties of Pitham:

Seripithal (Digestion)	Neervetkai (Thirst)
Vanmai (Strong)	Suvai (Taste)
Vemmai (Heat)	Oli (Light)
Menmai (Soft)	Ninaippu (Thinking)
Paarvai (Sight)	Arivu (Knowledge)
Pasi (Hunger)	

Own qualities of Pitham - 6

Hot	- அக்கினி
Acidic	- புளிப்பு
Mobile	- ஊறுந்தன்மை
Liquid	- சலரூபம்
Acute	- குரூரம்
Pungent	- காரம்

Opposite qualities of Pitham - 6

Cold	- குளிர்ச்சி
Sweet	- இனிப்பு
Immobile	- நிலைத்திருத்தல்
Solid	- கெட்டி
Mild or harmless	- சாந்தம்
Bitter	- கசப்பு

Functions of Pitham:

1. Raising the body's temperature
2. Giving red or yellow colour to the body
3. Raising the body temperature during digestion and assimilation.
4. Produces perspiration, giddiness
5. Raising the volume of blood and its expulsion
6. Gives yellow stain to eye, motion and urine
7. Anger, irresponsible, immobile, thoughtfulness, excitement, thinness.
8. Feeling of irritation
9. All tastes like sour, bitter

Formation of Senneer:

During the process of digestion in our body, Saaram or Rasa thathu (Chyle) is formed on the first day. From saaram, Senneer (blood) is formed on the second day. From senneer, Oon (Muscle) is formed. From oon, Kozhuppu (Fat) is formed. Enbu (Bone) is formed from kozhuppu. From enbu, Moolai (Bone Marrow) is formed. From moolai, Sukkilam (Sperm) or Suronitham (Ovum) is formed on the third, fourth, fifth, sixth and seventh day respectively. The nutrients absorbed after digestion is responsible for the metabolic function of blood.

It is to be noted that the nutrients absorbed after digestion are responsible for the formation of muscular, adipose and nervous tissues and calcification of bones. As saaram and senneer are the primary important thathus of the body, they get deranged themselves and followed by derangement of other thathus.

In Pandu noi, saaram and senneer thathu are mainly affected.

Physiological aspects of Pitham:

Our body is made up of seven udal thathus namely saaram, senner, oon, kozhuppu, enbu, moolai, sukkilam / suronitham. These seven thathus constitute the body in normal condition. Senneer has the characters of pitha and it gives life to each cell and tissue of the body. Blood is the only vehicle, which is concerned with anabolic and catabolic functions of the body.

Among the seven thathus, senneer is considered as pitham, which has the character of Thee (Theyu). Circulation and digestion represent thee in the body. It makes the body steady and gives vigour and

stimulation. Pitham represents gastric juice, bile, energy, heat, inflammation, anger, irritation etc.

Relationship of Pitham with taste:

Salt - Water+ Agni

Sour - Earth + Agni

Pungent - Air + Agni

Salt, sour and pungent increases pitham since they are formed by Agni. So they possess Veppa Veeriyam.

“புனிதுவர் விஞ்சுங்கறி யாற்பூரிக்கும் வாதம்
ஒளியுவர் கைப்பேறில் பித்துச்சீறும் - கிளிமொழியே
காற்பிணிப்பு விஞ்சிற் கபம்விஞ்சு ஞ்சட்டிரதச்
சேரப் புணர் நோயணுகாதே”.

- கண்ணுசாமியம்.

Astringent, sweet and bitter tastes neutralize pitham since these tastes do not contain Agni. Hence they possess Seedha Veeriyam.

Astringent - Earth + Air

Sweet - Earth + Water

Bitter - Akash + Air

“பித்தமதி கரிப்பின் பேசும் பரிகாரம்
சுத்தத் துவரோடு சொல்லிணிப்புச் சத்தாகும்
கைப்புச் சுவையே கருதவதன் வீறு
எய்ப்படையு மென்றுரைத்தா ரிங்கு”.

- கண்ணுசாமியம்.

PANDU NOI

VERU PEYARKAL (SYNONYMS):

Veluppu Noi, Venmai Noi, Venpaandam

IYAL (DEFINITION):

Pandu noi is a disease of Raththa thathu, characterized by pallor of skin, nails, conjunctiva and tongue.

பெயர் காரணம்:

As per siddha tradition the term pandu is derived from the character of “Pandu” pandu has its historical importance in the “MAHABARATHAM” The father of the five heroes ‘Pancha pandavar’ is pandu. It is said that this man when born was very pale and anemic and hence this condition was named after him as pandu.

Noi Varum Vazhi (Etiology):

According to Noi Naadal Part II

தீக்குற்றம் மிகுந்து குருதியின் நிறத்தையும் எடையையும் கெடுத்து உடலுக்கு வேண்டிய ஊட்டத்தையும் கொடாமல் உடலை வெளுக்கச் செய்யும் நோய்.

According to Yugimuni, the causes of Pandu are as follows.

“அறிந்துமே உற்பத்தி சொல்லக் கேளாய்

அதிசார மலமிளகி எந்தே ரந்தான்

பிறிந்துமே புளியுப்பு பெருத்தலாலும்

பெத்தமர மக்கினி யிருத்தலாலும்

பறிந்துமே பகல் நித்திரையே செய்தாலும்

பாண்டு வந்து பாரிலுள்ளோர் படும் பாடே”.

- யூகி சிந்தாமணி.

From the above mentioned lines, it is clear that frequent attacks of diarrhoea, excessive intake of salt and sour foods, living in hot surroundings, are some of the behaviours causing Pandu noi.

According to Agasthiyar Gunavaagam,

“கொள்ளடா அபக்குவ போசனத் தினாலும்
குடிக்கெடுத்த பெரும்பாடு கிராணியாலும்
அளவற்றவி சாரந்தான டையும் போதும்
தெள்ளவே தேகத்தில் இரத்தம் கெட்டு
தெளிவான பண்டதூவு முண்டாம் பரே”.

- அகத்தியர் குணவாகடம்.

Inadequate cooking of foods, negligence in the treatment of diarrhoea, profuse bleeding, excessive sorrows leads to Pandu noi.

According to thanvanthiri vaithyam,

“திருந்திடும் பாண்டு ரோகஞ் சேர்ந்திடுங் குணத்தைக் கேளாய்
இருந்திடும் வாதபித்தச் சிலேற்பன மிவைதான் மாறிப்
புரிந்துதர னொன்றோடொன்று பொருந்துவதாலு மண்ணோ
டருந்துவதாலும் பாண்டு வணைந்திடு மென்னலாமே”

“ஆகிய மூலந் தன்னிலணைந்த வுட்டணத்தினாலுந்
தோகையர் மேககத்தாலுந் துயர்மிகு ரோகத்தாலுந்
தேக போஷணை யுள்ளார்க்குத் தரித்திரஞ் சேர்தலாலும்
வேகமாந் திரிதோஷங்கள் மிஞ்சியே பாண்டு வாமே.”

- தன்வந்திரி வைத்தியம்.

Imbalance between the three thatus, vatham, pitham, kabam, perversion of appetite such as eating mud (PICA), excessive heat accumulation due to altered Abaana vayu, excessive sorrow, psychosocial factors are some of the causes of Pandu.

According to Agasthiyar Vaithyam,

“குயல்வாய் குஷ்டம் சயங்குன்ம நீரிழிவு சுரக்கிராணி
நீரடைப்பு பாண்டு மூல வாய்வு
கயல்வாய் வருங்கண்ணில் குந்தாய் கடினந் தசவாய்வு
காணவாக முன்செய்த உயிர்களும் வினைதானே.”

- அகத்தியர் வைத்தியம்.

Pandu noi is also considered to be one among the ‘kanma noi’

According to Gurunaadi,

“சொல்லாத விஷக்கடிகள் விஷ குன்மம் பாண்டு
தீராத கன்மவினை செய்த பாவம்”

Gurunaadi also says that Pandu is one of the kanma noi.

According to Gurunaadi,

“வயல் தனிலே பூநாக மண்ணைத்தானே
வருந்தியது புத்துப்போல வத்தையாகும்
பயல்மொழி யீந்தேகத்தில் கிருமிதானே”

- குருநாடி நூல்

Pathologically, blood loss occurs due to several causes. One among them is worm infestation, which leads to chronic blood loss from the intestines thus leading to pandu.

According to Theraiyar Vaagadam.

“கருதிய மீனின் முள்ளும் கலந்துமி நெய்யில் வாலும்
மருவிய எலும்புங் கல்லும் மங்கையர் மருந்தீடும்
பருகிய பழஞ்சேற்றாலும் பழமல நிறைகையாலும்
மருகிய மயிர்களாலும் வந்திடும் நோயிதாமே.
புறவரை யுண்கையாலும் போக நீருண்கையாலும்
சுரளவே முடக்கிக் கொண்டு முறக்கிடக்கையாலும்
மருளவே மேடுதன்னில் மனமுறக் கிடக்கையாலும்
பெருக வெண்டொடியினாலும் பிறக்க நோயென்று காணே”.

- தேரையர் வாகடம்.

Theraiyar vaagadam says that thorns of fish, paady bran, bone, stones, old rice, hair in food are the dietary factors causing Pandu.

Then severe constipation, drinking polluted water, sleeping in an abnormal posture are all the causes that bring out Pandu noi.

NOI ENN (CLASSIFICATION):

Classification of Pandu noi based on various siddha books.

YUGI CHINTHAMANI – 800

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Mukkutra pandu
5. Vida pandu
6. Mannun pandu

ROGA NIRNAYA SAARAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Mukkutra pandu
5. Vida pandu

AGASTHIAR GUNAVAAGADAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Vida pandu
5. Miruthika Pandu

T.V.SAMBASIVAMPILLAI

1. Vatha Pandu
2. Pitha pandu
3. Kaba pandu
4. Mukkutra pandu
5. Oothu pandu
6. Neer pandu
7. Eri pandu
8. Vida pandu

VAITHYASARASANKRAHAM

1. Vatha pandu
2. Pitha pandu
3. Moola pandu
4. Moolapitha pandu
5. Vida pandu

THANVANTHIRI VAITHYAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Mukkutra pandu
5. Pitha vatha pandu
6. Sannipatha pandu
7. Paithiya pandu

PARARASASEKARAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Sanni pandu
5. Miruthika pandu

JEEVA RAKSHAMIRTHAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Tridosha pandu
5. Miruthikapuktha pandu

ANUBAVA VAITHYA

DEVA RAGASYAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Mukkutra pandu
5. Miruthikapuktha pandu
6. Vida pandu

MADAVA NIGANDAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Sanni pandu
5. Mannun pandu

ASHTANGA HRIDAYAM

1. Vatha pandu
2. Pitha pandu
3. Kaba pandu
4. Sannipatha pandu
5. Mannun pandu

Kurikunangal in Pandu noi (Clinical features):

1. Murkurikunangal (premonitory symptoms):

The patient experiences insidious onset of fatiguability, difficulty in breathing on exertion, diminished vision, faintness, palpitation and pallor of the skin.

2. Pothu Kurikunangal (General signs and symptoms):

Agasthiyar Gunavaagadam states that,

“உண்டாகும் வேளை தன்னில் தேக நேர்மை
உறுதியாய்ச் சொல்லுகிறேன் நன்றாய் பரு
குண்டான முகம் கண்கள் உதடு நாக்கு
குறிப்பான வாய் வேகும் தேக முற்றும்
வெண்டாக வேயுலர்ந்து வெண்மை யாகி
விரல் கால்கள் முழுவதிலும் ரத்தம் வற்றி
கண்டான கால்கள் தான் தணிந்து நிற்கும்
கருவான நாடியது மெதுவாய்ப் போமே.
போமே தான் தீபனங்கள் மட்டுப்பட்டு
பொலிவான கண்விழிகள் பெருத்துத் தோன்றும்
ஆமே தான் அசக்தியு மாயாசங் கண்டு
அவர் நடையும் தளர்ந்து பெருமூச்சு கண்டு
மூமேதான் மூர்ச்சையுடன் மார்துடித்து
முடிவான கணுக்காலில் வீக்கமுண்டாய்
தாமே தானிருதயத்தின் வதனந் தன்னிற்
துருத்தி நிகர் சத்தமது கேட்கும் பாரே”.

- அகத்தியர் குணவாகடம்

Stomatitis, dryness of the skin, pallor of the face, eyes, lips, tongue and nails, lassitude, tiredness, low volume pulse, anorexia, swelling of the

eyelids, dyspnoea on exertion, palpitation, oedema of the ankle joint, added heart sounds in the precordium are mentioned as the signs and symptoms of Pandu noi.

In Siddha Maruthuvam, Kuppusamy Mudaliar states,

Inability to walk, headache, palpitation, blurring of vision, giddiness, syncope, dyspnoea, anorexia, vomiting, paleness of the skin, nailbeds become swollen and pallor, fissured tongue, glossitis, hoarseness of voice.

If it occurs in pitha thegi, anorexia, indigestion, burning sensation, pallor of skin, glossitis, dysphagia, vomiting with bile, bitter taste and diarrhoea occurs. If the symptoms persist for longer duration it results in jaundice.

According to Vaithya Sarasankraham,

Loss of appetite, thirst, pallor of the skin, lips, eyes and tongue, face becomes dry due to excessive heat, flatulence, swelling and back pain in lower extremities.

According to Jeeva Rakshamirtham,

Dryness of the body, palpitation, high coloured urine, loss of appetite, lassitude, perspiration, emaciation.

According to Sarabendrar Vaithya Muraigal – Karbini Balaroga Chikitchai.

Abdominal swelling, pallor of the eyes and nail beds, oedema of the eyes and loss of appetite.

Symptoms of various types of Pandu:

According to Kuppusamy Mudaliar,

1. Vatha pandu:

The symptoms of Vatha paandu are lower abdominal pain, thirst, loss of appetite, dryness of the skin, visible veins due to pallor of the skin, Redness of the eyes, constipation, headache, anasarca and pallor of the skin.

2. Pitha pandu:

Yellowish colouration and pallor of the skin, diminished vision, thirst, fainting, pungent taste like pepper, chest pain, dyspnoea, giddiness and bitter taste.

3. Kaba Pandu:

Pallor of the skin, salty taste, flushing of the skin, vomiting, husky voice, sneezing, cough with expectoration, fainting, lassitude, anasarca and thirst.

4. Mukkutra Pandu:

Anorexia, thirst, dyspnoea, anasarca, chest pain, lassitude, sneezing, warmness of the skin, weakness.

5. Vida Pandu:

Pallor of the skin, excessive thirst, anorexia, vomiting, hiccough, cough, dyspnoea, flatulence, diarrhoea, fever, heaviness of the chest and anasarca.

6. Mannun Pandu:

Most common in children and pregnant women who have perverse taste to have mud, sand, ash, brick powder, camphor etc. Symptoms arise

according to their habit like abdominal distension, indigestion, vomiting, diarrhoea, fever and worm infestation. As a result emaciation of body, pallor, oedema and palpitation will occur.

According to Anubhava vaithya Devaragasiyam,

Miruthika Puktha Pandu:

Due to excessive intake of mud, rasam and raththa thathu becomes weak and results in pandu noi. It is characterized by constipation, swelling around the umbilicus, face, leg and genital organs, worms in the faeces and haematemesis.

According to Agasthiyar Gunavaagam,

Miruthika Pandu:

“பாரேநீ மல மூத்திரம் யாவு மய்யா
பண்பாக மஞ்சள் நிறந் திறக்குமப்பா
கூறே நீ மிருத்திகா பாண்டின் நேர்மை
குண முடனே சொல்லுகிறேன் குறிப்பாய்க் கேளு
தேரே நீ தித்திப்பு மண்ணுங் கூட
திரமன துவர்மண்ணு சவுட்டு மண்ணும்
தீராகத் தினந்தினமுந் தின்று வந்தால்
திரமன மிருத்திகா பாண்டுண்டாமே

- அகத்தியர் குணவாகடம்

Faeces and urine turns yellow in this condition. This condition is caused mainly by eating soil frequently.

Thoder noi of pandu noi (complications)

When the disease progresses kabam increases resulting in sobai (oedema). Moreover when pandu noi is severe excessive intake of pitha diets lead to kamalai (jaundice). This is stated by Yugimuni as follows.

“விளம்பவே பாண்டு முற்றிருக்கும் போது
மீறியே பித்தவஸ்துதனைப் புசித்தால்
பூண்டிடுமே காமாலை யென்னும் ரோகம்”.

- யூகிமுனி.

Mukutra verupaadugal (Siddha Pathology):

According to Siddha system, body is constituted by 96 thathuvas. So if any derangement in thathuvam leads to pathological changes in the body. The first change occurs in the panchabootham level. This is followed by changes in vatham, pitham and kabam leading to changes in other thathuvas. Then changes occur in thathukkal and malams and develop various symptomatologies.

“கொள்ளவே அபக்குவ போஷணத்தினாலும்”

- அகஸ்தியர் குணவாகடம்

In pandu noi impairment of pitha dhosha due to changes in the bootham followed by loss of appetite. This leads to changes in rasa thathu and raththa thathu followed by pallor of the body, lassitude, weight loss etc.

Due to Nutritional defect, i.e., low iron diet leads to the derangement in Anal Pitham and Ranjaka Pitham.

Then viyanan one of the types of vatham also affected.

These two dhoshas are increased and affects kabam followed by dropsy.

Piniyari Muraimai (Diagnosis):

Pini means the disease, which affects the body

Ari means identify

Muraimai means rules

Piniyari muraimai is the method of determination of diseases. It is based upon three main principles. They are

1. Poriyalarithal (Inspection)
2. Pulanalarithal (Palpation)
3. Vinathal (Interrogation)

Physicians Pori and pulan are used as tools for examining the Pori and Pulan of the patient. The above principles correspond to the methodology of Inspection, Palpation and Interrogation in modern medicine, helping the physician to arrive at a clinical diagnosis of the disease.

Pori is considered as the five senses of perception namely Skin, Tongue, Eye, Nose and Ear.

Pulangal are five objects of senses, which are Sensation, Taste, Sight, Smell and Sound.

Vinathal is asking informations regarding the history of the disease, its clinical features from the patient or his close relatives who are taking care of the patient, when the patient is not in a position to speak or if the patient is a child.

Ennvagai Thervukal (Eight tools of diagnosis):

Ennvagai thervukal is a unique method of diagnosing the disease, which was developed by siddhars.

“நாடிப்பரிசம் நாநிறம் மொழிவிழி

மலம் மூத்திரமிவை மருத்துவராயுதம்”.

- நோய் நாடல் நோய் முதல் நாடல்

Hence the diagnosis is made by the following:

Naadi (Pulse), Sparisam (Sensation), Naa (Tongue), Niram (Colour), Mozhi (Sound), Vizhi (Eyes), Malam (Faces), Moothiram (Urine).

Pandu in relation with Ennvagai Thervukal:

1. Naadi (Pulse):

Naadi is responsible for the existence of life. It is a suitable diagnostic tool used by Siddhars from the unknown part. It is recognized as one of the principal means of diagnosis and prognosis of the disease.

The powers of Naadi manifest in the body as three vital forces namely vatham, pitham and kabam. They normally exist in the ratio 1:1/2:1/4 respectively. Derangements in these ratios lead to various disease entities.

“மெய்யளவு வாதமொன்று
மேல் பித்தமோரையாம்
ஐயங்கொலன்றே அறி”.

- கண்ணுசாமியம்.

Naadi Nadai:

“அண்டிடவே தரித்திரர்கள் விருத்தர் பாலர்
அன்பாகத் தண்ணீரில் மூழ்கினோர்கள்
கொண்டிடவே இவர்களது உறுப்பின் தரது
கூறவே முடியாது எவர்க்குக் கிட்டும்”

- பதார்த்த குண சிந்தாமணி

From the above lines, it is difficult to find Naadi in Children, but in Noi Naadal Noi Mudal Naadal Thiratu the Naadi for Pandu Noi - Kaba pitha naadi, Kaba vatha naadi, vatha kaba naadi.

2. Sparisam (Palpation):

The warmth, chillness, dryness, roughness of the skin, oozing, sweating, tenderness, fissures, depigmented changes in the skin, swelling, ulcer and hepatosplenomegaly may be noted.

3. Naa (Tongue):

The colour, dry or wet, coating, excessive salivation, redness, ulceration, fissure, pallor, any malignant growth, predominant taste in the tongue, speech, movement and deviation of the tongue along with the conditions of the teeth and gums should be noted.

In Pandu noi, pallor of the tongue and loss of taste are seen.

4. Niram (Colour):

Changes in the colour of the skin, teeth, eyes, nail, lips due to vatham, pitham, kabam and mukkutram, hypo and hyper pigmentation are noted.

In Pandu pallor of skin, conjunctiva and nail beds are noted.

5. Mozhi (Sound):

This includes clarity of speech, any disturbances, high or low-pitched voice, slurring and incoherent speech and hoarseness of voice.

6. Vizhi (Eyes):

Any colour change indicating vatham, pitham, kabam, and mukkutram, hyperemia, ulceration, response of pupil, pallor, protrusion, sunken eyes, sharpness of vision, excessive lacrimation, accumulation of secretion at the angle of eye. Visual disturbance and any specific diseases of the eyes should be noted.

In Pandu noi, pallor of conjunctiva is seen.

7. Malam (Faeces):

Colour, consistency, quality, small, frequency, constipation / diarrhoea, presence of mucous, blood and undigested food particles in the stool should be studied.

In Pandu noi Constipation, Diarrhoea, may be noted.

8. Moothiram (Urine):

Neerkuri and Neikuri:

Neerilakkanam (Method of collection of urine):

“அருந்துமறிரதமும் அவிரோதமதாய்
அ.:கல் அலர்தல் அகரலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தரவியே கரது பெய்
தொரு முகூர்த்தக் கலைக் குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”.

- தேரையர்

Prior to the day of urine examination the patient is advised to take balanced diet and the quantity of food must be proportionate to his appetite. The patient should sleep well. After waking up in the morning the first voided urine is collected in a wide mouthed glass container and is subjected to the analysis within one and half hours.

Neerkuri:

“வந்த நீர்க்கரிஎடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவவை யறைகுது முறையே”

- தேரையர்

Urine has the following five characters

Niram, Edai, Manam, Nurai, Enjal.

Neerkuri in pandu noi:

In Pandu noi, decreased amount of urine is voided even after excessive intake of water.

Neikuri:

“நிறக்குறிக் குரைத்த நிருமண நீரிற்
சிறக்க வெண்ணையோர் சிறுதுளி நடுவிடுத்
தென்றுறத் திறந்தொளி ஏகா தமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுஞ் செய்தியை யுணரே”.

- நோய் நாடல் நோய் முதல் நாடல்

The specimen collected for neerkuri is kept open in a glass dish being exposed well to the sunlight. Add one drop of gingelly oil without shaking. It should not be disturbed by the position and spreading of the oil drop.

“அரவென நீண்டின. :தே வாதம்”
“ஆழிபோற் பரவின் அ. :தே பித்தம்”
“முத்தொத்து நிற்கின் மொழிவ தென் கபமே”

- தேரையர்

Oil spreads like a snake - Vatha neer

Oil spreads like a ring - Pitha neer

Oil remaining and floating like a pearl - Kaba neer

Neikuri in pandu noi:

“விரைவுடன் கதிர்போல் நீண்டு வேற்றுமைக் குணங்கள் கண்டால்
குருதிதான் கெட்டு நரசம் குன்றிய குணமெதன்னே”.

- தேரையர்

If the oil spreads like a kathir (ray), it indicates Pandu noi.

Mukkutram:

Vatham, Pitham and kabam are the three forms of mukkutram. These are mentioned for the normal physiological conditions of the body. If any one of these factors increase or decrease it leads to some pathological changes in the body and thus produce the disease.

Vatham:

Its mathirai alavu is 1.

Location of vatham in the body:

Vatham is located in the abanan, faeces, ida kalai, spermatic cord, pelvic bone, skin, nerves, joints, hair and muscles.

Vatham has ten forms:**1. Piranan (Uyirkaal):**

It resides in the heart and legs to nose and controls knowledge mind and five objects of sense useful for breathing.

In Pandu noi, it is affected when dyspnoea is present.

2. Abanan (Keezh nokkukaal):

It is located in the lower abdomen and extremities. It is responsible for passing urine, stools, sperm and menstrual flow.

In Pandu noi, it is affected when diarrhoea and oliguria are present.

3. Viyanan (Paravukaal):

It resides mainly at the heart and responsible for movements of the body and sensation.

In Pandu noi, it is affected when swelling of the body, pallor of eyes and lips are present.

4. Samanan (Nadukkaal):

It is located in the stomach and helps for proper digestion

In Pandu noi, it is affected if anorexia is present.

5. Uthanan (Melnokkukaal):

It is located on the chest and responsible for vomiting, cough and sneezing.

In Pandu noi, it is affected when excessive thirst is present.

6. Naagan:

It resides in the eyes and responsible for opening and closing of the eyes and intelligence.

7. Koorman:

It is located in the eyes and responsible for vision and yawning.

8. Kirukaran:

It is located in the throat and responsible for salivation, nasal secretion and appetite.

In Pandu, it is affected if anorexia is present.

9. Devathathan:

Its location is at eruvai and karuvai. It is responsible for laziness, sleep and anger.

In Pandu, it is affected if sluggishness and insomnia are present.

10. Dhananjeyan:

It resides in the nose and escapes on the third day after death by bursting the cranium.

PITHAM:

Its mathirai alavu is ½.

Location of pitham in the body:

Pitham is located in pirana vaayu, pingalai, bladder, moolaakini, heart, umbilical region, abdomen, stomach, sweat, blood, eyes and skin.

Five forms of pitham:**1. Anala pitham:**

This gives appetite and helps for digestion

In Pandu, it is affected if loss of appetite is present.

2. Pirasagam:

It given complexion to the skin

In Pandu, it is affected due to pallor of conjunctiva and skin altered lusture.

3. Ranjagam:

It gives colour to the blood

In Pandu it is affected due to pallor of conjunctiva and skin.

4. Alosagam:

This brightens the eyes

5. Saathagam:

It controls the entire body functions responsible for activities of the body.

In Pandu, it is affected due to inability to do the works properly and sluggishness.

KABAM:

Its mathirai alavu is $\frac{1}{4}$.

Location of Kabam in the body:

Kabam is located in samanavayu, sperm, head, tongue, fat, bone marrow, blood, nose, chest, nerve, bone, brain, eyes and joints.

Five Forms of kabam:

1) Avalambagam:

It Controls heart, lungs and other forms of kabam.

It is affected in Pandu, due to dyspnoea

2) Kilethagam :

It makes the food wet and helps for digestion

In pandu, it is affected due to anorexia.

3) Pothagam :

It is responsible for taste.

In pandu it is affected due to anorexia and tastelessness.

4) Tharpagam :

It keeps the eyes cool

5) Santhigam:

It is responsible for the lubrication and aids for movement of joints.

Paruvakaalam (Season)

The whole year is constituted by six seasons. They are follows.

1. Kaar Kaalam - Avani and Puratasi - August and September
2. Koothir Kaalam - Aippasi and Karthigai - Octobar and November
3. Munpani Kaalam - Markazhi and Thai - December and January
4. Pinpani Kaalam - Maasi and Panguni - February and March
5. Elavenil Kaalam - Chiththirai and Vaigasi - April and May
6. Muduvenil Kaalam - Aani and Aadi - June and July.

In every season, changes will occur in the land, water, plants, animals and human beings, which modify the physiology and make them susceptible to certain specific diseases which are common in that season.

The occurrence of pandu commonly seen during the kaarkalam and koothirkalam because of pitham thannilai valarchi adaithal and vetrunilai valarchi adarthal occurs.

Kuttram	Thannilai Valarchi	Vetrunilai Valarchi	Thannilai Adaithal
Vatham	Muduvenil Kaalam	Kaar Kaalam	Koothir Kaalam
Pitham	Kaar Kaalam	Koothir Kaalam	Munpani Kaalam
Kabam	Pinpani Kaalam	Elavenil Kaalam	Muduvenil kaalam

Nilam:

- 1. Kurinji - Hill regions and surroundings
- 2. Mullai - Forest regions and surroundings
- 3. Marutham - Cultivating regions and surroundings
- 4. Neithal - Coastal regions and surroundings.
- 5. Palai - Desert regions and surroundings

People living in Kurinji, Neithal and Palai may have increased chance to acquire Pandu Noi.

UDAL KATTUGAL:

Our Body consists of seven udal kattugal. It Gives strength to the body.

- 1. Saaram - It gives strength to the body and mind.
- 2. Senneer - It is responsible for knowledge, strength, boldness and healthy complexion

3. Oon - Gives structure and shape to the body and is responsible for the movement of the body.
4. Kozhuppu - Lubricates the organs and proceeds on its own works.
5. Enbu - Protects vital organs and is useful for movements.
6. Moolai - Present inside the bones and it gives strength and maintains the normal conditions of the bone.
7. Sukkilam/Suronitham - Responsible for the propagation of species.

In Pandu, Saaram is affected which leads to sluggishness, dyspnoea and tiredness. Seneer is affected which leads to pallor of skin and conjunctiva.

Prognosis of Pandu:

Curable and Incurable Types:

It is said that Vida Pandu had poor prognosis. Even though other types are curable, symptoms like vomiting, diarrhoea, oedema of body, excessive thirst, hiccough, diabetes mellitus, tuberculosis occurs it leads to complications.

According to Sarabendrarr Vidhy Muraigal:

அதிக நாளான பாண்டு ரோகம் சிகிச்சைக்கு வசப்படாது. புதிதானாலும் உடல் வீக்கத்தில் மஞ்சள் நிறம் காணப்பட்டால் குணம் ஏற்படாது. மலச்சிக்கலோ அல்லது பச்சை நிறமான அதிசாரமோ ஏற்பட்டால் அசாத்தியம், பலவீனம், வாந்தி, மூர்ச்சை நாவறட்சி, இரத்த குறைவினால் உடல் வெளுப்பு முதலியவைகளுடன் கூடிய ரோகியும் குணமடைவது சிரமம்.

பற்கள், நகம், கண் இவைகள் அதிகம் வெளுத்தாலும் எல்லாவற்றையும் வெண்ணிறமாக பார்த்தாலும் அந்த ரோகம் அசாத்தியமாகும். அசாத்திய ரோகத்தை முற்றிலும் குணப் படுத்த முடியாவிட்டாலும் சிறிது குறிகுணங்களை குறைத்து ஆயுளையும் சிலகாலம் நீடிக்க செய்யலாம்.

கைகள், கால்கள், தலை முதலான இடங்களில் வீக்கம் ஏற்பட்டு இளைத்து கைகால்களும் இளைத்து வயிறு பெருத்தும் உள்ள பாண்டு ரோகியையும், ஆண்குறி, தொடையிடுக்கு ஆகிய இடங்களில் வீக்கம், அடிக்கடி மயக்கம், அதிசாரம், சுரம் கண்டால் தீராது.

Asaathiya Pandu:

பாண்டு ரோகிக்கு வீக்கம், சோம்பல், தாகம், அரோசகம், வாந்தி, விக்கல், இருமல், பேதி என்னும் இக்குணங்கள் உண்டாகி எந்த வஸ்துவை பார்த்தாலும் மஞ்சள் நிறமுண்டாகில் அசாத்தியம்.

- அகத்தியர் வைத்தியப் பிள்ளைத் தமிழ்.

Fate of the disease:

Kannusamiyam States that,

“வெப்புப் பிணியதனில் வெம் மேத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் - செப்பும்
கிரணியிற் பண்டில் கிளர் நீர்சுருங்கிற்
பிரணன் பிடியெனப் பேசு.”

- கண்ணுசாமியம்

“பாண்டு பிரமேகம் பன்வாத சூலை குன்மம்
வேண்டா சயஞ்சன்னி வெண்ணேசைபை - நீண்ட
அதிநீரே காமலை யானபிணி தம்மு
ளதி சாரமக காதறி”

- கண்ணுசாமியம்

Noi Neekkam (Treatment):

The speciality of siddha treatment emphasis not only for complete healings but also for the prevention and rejuvenation. This is said as follows,

Kappu (Prevention)

Neekkam (Treatment)

Niraivu (Restoration)

Siddha system stated that even during the time of conception, some defects creep into the fertilized embryo. These defects form the basis of the manifestation of certain constitutional disease later on during the existence of the individual.

Diseases are produced by the unequilibrium of three thathus, which may be due to various causes like diet, life style pattern, mental and physical activities.

When treating the diseases the following principles must be noted.

“நோய்நாடி நோய்முதல் நாடியது தணிக்கும்
வாய்நாடி வாய்ப்பச் செயல்.”

“உற்றானளவும் பிணியளவும் காலமும்
கற்றான் கருதிச் செயல்.”

- திருக்குறள்

So it is essential to know the disease and the cause for the onset of disease, the nature of the patient, the severity of the illness, the season and the time of occurrence of the disease must be observed.

Line of Treatment of Pandu:

The aim is to normalize the vitiated mukkutram, vayus and the affected Raththa thathu. As this disease is caused by the deterioration of Ranjaga pitham, effective medicinal preparations have to be administered in the beginning itself to raise the Raththa thathu, to achieve the normal function of it.

Before starting the actual treatment, the presence or absence of toxins in the body produced due to derangement of three thathus should be controlled. This is explained as follows:

“சத்தியால் பித்தந் தரமும்
பேதியால் வாதந் தரமும்
அஞ்சனத்தால் கபந் தரமும்”

Usually for pitha diseases, emetics are given to alter the deranged pitham. But there are some exceptions to this rule. For instance, in Pandu noi, since the patient is already weak and drowsy, the administration of emetic medicine is excluded from the line of treatment.

1. To bring out the Tridosha to its normal physiological state, laxative is to be administered.
2. To improve haemoglobin content of blood, iron preparations are used.
3. Removal of the causative factors.
4. Pathiyam ie, diet and other restrictions to normalize the affected thathu and to maintain a longer drug action.
5. Intake of rich nutritious food is also a part of treatment.

The author took Bringaraja Choornanam, Madhulai Manappagu as a trial drug for Pandu because herbal drugs are potent and safer than metallic drugs.

Diet:

“மறறுபாடில்லா உண்டி மறுத்துண்ணின்
ஊறுபா டில்லையு யிர்க்கு”

- திருக்குறள்

Diet should be of strengthening the body and rejuvenating the blood. For Pandu noi, the following food items are recommended.

Greens:

Karaisalai, Ponnangani, Arukeerai, Sirukeerai, Murungaikeerai, Manathakkalikeerai.

Vegetables:

Kathiri pinju, Avarai pinju, Murungai pinju, Vazhai kachal may be given.

Fruits:

Dates, Orange, Grapes, Apple, Fig, Gooseberry and Pomegranate.

Easily digestible foods like porridge, mutton soup, bone soup must be given in acute states of Pandu noi. Soup prepared from the liver of goats may be given to rejuvenate the blood and strengthen the heart.

After the normal appetite is restored properly, prepared meat of Kaadai, Kowthari and Udumbu can also be given. They tone up the system debilitated and also help in rejuvenation.

MODERN ASPECT

INTRODUCTION

The blood is the most precious fluid in the body a fact expressed in such common terms as “the life blood”. Blood is one of the extracellular body fluids, which circulates in a closed system of blood vessels. It is an essential component of the internal environment. It’s physical and chemical constituents also remain constant within physiological limits. The constant nature of the blood is one of the important haemostatic conditions of the body.

Properties of blood

Blood is a type of tissue and following are the physical properties.

- 1. Colour:** Opaque fluid and red in colour.
- 2. Volume:** 5 litres in adult.
- 3. Reaction and pH:** Slightly alkaline pH - 7.4.
- 4. Specific gravity:** Total blood - 1.05 – 1.061,
Blood cells – 1.092 - 1.101, Plasma – 1.022 – 1.026.
- 5. Viscosity:** Blood is five times more viscous than water.

Composition of Blood:

Blood consist of 45 % of solid portion (RBC, WBC, and Platelets) and 55 % of fluid portion (Plasma).

Functions of Blood:

1. Nutrient Function:

Nutritive substances like glucose, amino acids, lipids, and vitamins derived from digested food are absorbed from gastro intestinal tract and carried by blood to different parts of the body for growth and production of energy.

2. Respiratory Function:

Transport of respiratory gases is done by the blood. Blood conveys oxygen from the lungs to the tissues oxidation of food and production of energy and eliminates carbon-dioxide from the tissues.

3. Excretory Function:

Waste products formed during various metabolic reactions in the tissues are removed by the blood and carried to the excretory organs like kidney, skin, liver etc.

4. Transport of hormones and enzymes:

The hormones and some of the enzymes are carried by blood to different parts of the body from the source of secretion.

5. Regulations of body temperature:

The human being is a homeothermic animal and the body temperature has to be kept constant within a narrow limit. Blood transfers heat from the warmer to the cooler parts of the body.

6. Regulation of water balance:

Blood maintains the water content of the tissues and helps in the regulation of fluid in different compartments of the body.

7. Regulation of acid – base balance:

The plasma proteins and haemoglobin act as buffers and help in the regulation of acid – base balance.

8. Defensive Function:

Blood has a dual function in the defense mechanism. The white blood cells and especially the polymorphonuclear leucocytes have a phagocytic action and surround and attack the disease germs entering the

human body. In fact pus is the debris of dead white cells killed in such encounters. The plasma proteins specifically i.e., the gamma globulins produce antibodies against the antigens present in foreign bodies and germs. Blood also transports antibodies, antitoxins, and lysins, which are protective substances against the bacteria and other injurious substances entering the body.

9. Regulation of osmotic pressure:

The plasma proteins play the major role in regulating the osmotic pressure of tissue fluids.

10. Storage function:

Water and some important substances like protein, glucose, sodium and potassium are constantly required by the tissue. Blood serves as a readymade source for these substances and is taken from the blood during conditions like starvation, fluid loss, and electrolyte loss.

The Red Blood cells or Erythrocytes:

Erythrocytes or Red Blood Cells (RBC) are the non-nucleated formed elements in the blood. The red colours of these cells are due to the presence of colouring matter – haemoglobin in these cells. The erythros means red.

Morphology:

Circular, non-nucleated, biconcave discs, around 7.8μ in size with 2.2μ thickness at the periphery and 1μ at the center.

Production of Erythrocytes:

Areas of the body that produce erythrocyte cells.

1. In the early few weeks of embryonic life - yolk sac
2. During the middle trimester of gestation - Liver, spleen, thymus,
and lymphnodes.
3. Later part of gestation and after birth - Red bone marrow
4. UP to the age of 5 - Red marrow of all the bones.
5. After the age of 5 and adult - Red marrow of proximal end
of long bones and flat bones such
as ribs, Vertebrae, pelvis, sternum,
and iliac bone.

Sometimes under conditions of exchanged stimuli, reticuloendothelial system also takes up the embryonic function and yellow marrow shall be transformed into the red marrow. Even in these bones, the marrow becomes less productive as age increases.

Genesis of Red Blood Corpuscles:

In the bone marrow there are cells called pluripotential Haemopoietic Stem Cells (PHSC) from which all the cells in the circulating blood are derived. The large portion of reproduced stem cells differentiates to form the other cells. The early offspring still cannot be recognized as the different types of blood cells, even though they have already become committed to a particular line of cells and are called committed stem cells.

The different committed stem cells will produce colonies of specific types of blood cells. There, a committed stem cell that produces

colony – forming unit blast (CFU – B) and then erythrocytes produced from these are called colony forming unit – erythrocytes (CFU-E).

Growth and reproduction of the different stem cells are controlled by multiple proteins called Growth inducers. The another set of proteins are called differentiation inducers whose function is differentiation of the cells.

Stage of differentiation of Red Blood Corpuscles.

Colony forming unit erythrocyte (CFU-E) [primordial stem multipotential].



Proerythroblast [First cell that belonging the RBC series – unipotential]



Basophil erythroblast [Begins synthesis of haemoglobin]



Poly chromatophil erythroblast [contains basophilic cytoplasm and haemoglobin]



Normoblast [with small nucleus and more haemoglobin-orthochromic erythroblast]

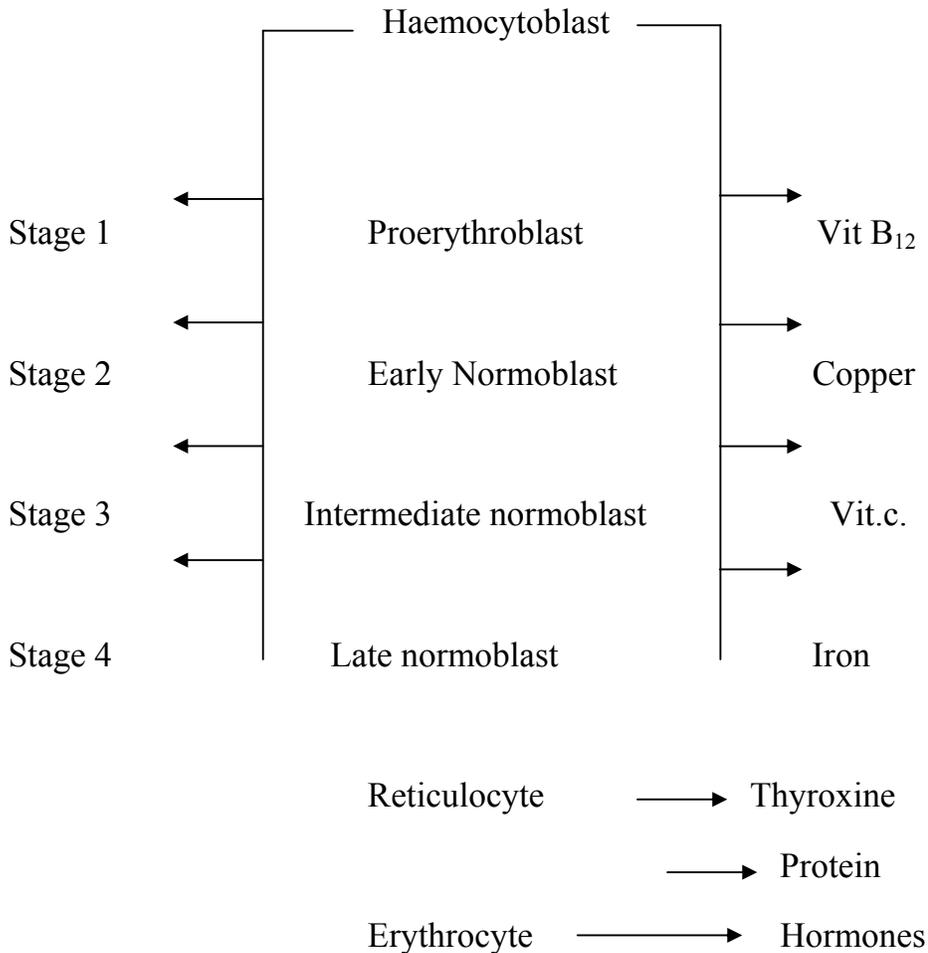


Reticulocytes [small amount of basophilic reticulum is present]



Erythrocytes [Matured Red Blood Corpuscles]

The stage of maturation of the RBC are given below



Stage I – Pro erythroblast (Megaloblast)

This early cell is large (15-20) μ and contains no haemoglobin. The cytoplasm is basophilic. The nucleus is about 12 μ and occupies about three quarters of the cell volume and the chromatin forms a fine stippled reticulum.

Stage II – Early Normoblast (Early erythroblast)

This cell is smaller than pro erythroblast and shows active mitosis. The nucleoli disappeared and cytoplasm is basophilic.

Stage III – Intermediate Normoblast (Late erythroblast)

This cell is smaller (10-15) μ and shows active mitosis. Haemoglobin begins to appear and its eosinophilic staining gives the cytoplasm a polychromatic appearance.

Stage IV-Late Normoblast (Normoblast)

Mitosis has now ceased and the diameter of the cell is 7 – 10 μ . The nucleus is smaller and the condensed chromatin assumes a “*cart wheel*” appearance and finally becomes deeply stained in a uniform manner. This appearance is called pyknosis and is a stage in the degeneration of the nucleus, which breaks up and finally disappears owing to the extrusion or lysis and a young RBC (reticulocyte) is formed. The maximum level of haemoglobin is attained and the cytoplasm gives an eosinophilic reaction.

Maturation of erythroblasts thus involves a decrease in the size of the cell, increased condensation and finally pyknosis of the nucleus. There is accumulation of haemoglobin and a change in the staining reaction of the cytoplasm from the basophilic to eosinophilic, viz polychromatophil.

Substances necessary for the formation of Erythrocytes Corpuscles:

Protein, Iron, Copper, Manganese, Vitamins (B12, C and Folic acid), Internal Secretions (Thyroxine), **Hormones** (erythropoietin, androgens and thyroxine).

The significant functions of Red Blood Corpuscles:

1. Transport of oxygen from the lungs to tissues and CO₂ from tissues to lungs.
2. Carbonic anhydrase, the enzyme present in the blood catalyses the reaction between carbon-di-oxide and water, thereby transporting them from the tissue to the lungs in the form of the bicarbonate ion (HCO₃).
3. Erythrocyte cells take part in main metabolic activities.

Life Span And Fate of Red Blood Cells

Average life span of red blood cell is about 120 days.

Daily 10% of red blood cells, which are senile, get destroyed in normal young healthy adults. This causes release of about 0.6g% of haemoglobin into the plasma. From this 0.9 to 1.5mg% bilirubin is formed.

Normal values of Erythrocytes

Infants	-	4 - 4.5 million/cu.mm
2 - 6 years	-	4.5 million/cu.mm
6 - 14 years	-	4.5 - 4.8 million/cu.mm.

HAEMOGLOBIN

Haemoglobin is the colouring matter of erythrocytes. The respiratory function of the blood is carried out by haemoglobin. Haemoglobin is a conjugated protein consisting of iron containing pigment portion called Haem (4%) and a protein of the histone class called globin (96%). Haem is an iron containing porphyrin known as iron protoporphyrin IX (metallo porphyrin). Therefore haemoglobin is an iron + porphyrin + globin compound.

Four haem molecules are attached to the globin molecules to form one molecule of haemoglobin. The molecular weight of haemoglobin is 68,000.

Varieties of haemoglobin:

Haemoglobin is of two types namely.

1. Adult haemoglobin – HbA (2 alpha chains and 2 Beta chains)
2. Fetal haemoglobin – HbF (2 alpha chains and 2 gamma chains)

Formation of haemoglobin

1. 2 succinyl co-A + 2 glycine \longrightarrow 4pyrrole.
2. 4 Pyrole \longrightarrow Protoporphyrin IX
3. Prtoporphyrin IX Fe⁺⁺ \longrightarrow Haem
4. Haem + polypeptide \longrightarrow Haemoglobin chain (Alpha or Beta)
5. 2 Alpha chains + 2 Beta chains \longrightarrow Haemoglobin A.

Metabolisum of Haemoglobin:

This section deals with three aspects of haemoglobin

- I. Synthesis of haemoglobin
- II. Catabolism of haemoglobin
- III. Conversion of haemoglobin to bile pigments.

I. Synthesis of haemoglobin

Haemoglobin is haem + globin. In adults synthesis of haemoglobin takes place in the red bone marrow from 3 sources namely, protoporphyrin, Iron and globin. Certain co-factors are required to facilitate the synthesis.

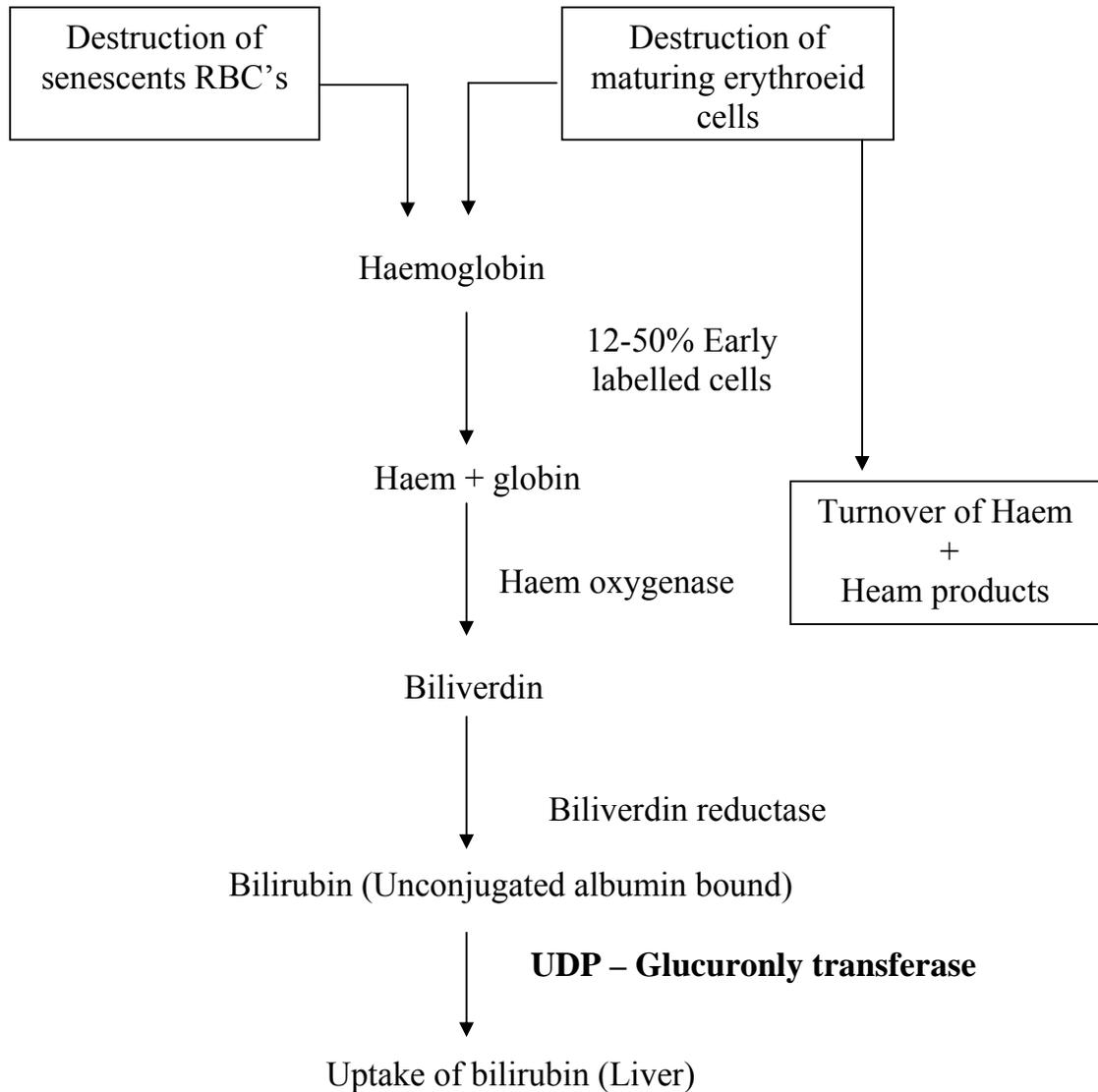
1. Vitamin B₁₂ (extrinsic factor)
2. Intrinsic factor
3. Folic acid group of vitamins
4. Copper.

Synthesis of haemoglobin and maturation of the erythrocytes proceeds simultaneously. The immature erythrocytes contain free porphyrin. As the cells mature, the porphyrin content decreases and is replaced by haemoglobin. Thus the circulating red blood cells, which are rich in haemoglobin, contain only traces of porphyrin.

II. Catabolism of haemoglobin:

Erythrocytes at the end of their life span of 120 days are broken down. Simultaneously the haemoglobin is degraded. Daily about 8gms of haemoglobin are broken down in the body and this corresponds to the formation of about 300mg of bile pigments per day. The normal sites of haemoglobin degradation are the reticulo endothelial cells of the spleen, bone marrow and liver. The globin which is the protein portion may be reutilized as such or may break down further into its constituent amino acids and enter to the amino acid "pool" for reutilization. The haem portion breaks down resulting in the formation of bile pigments.

III. Conversion of Haemoglobin to bile pigments:



The haemoglobin released from the red cells is phagocytosed by macrophages in the liver, spleen and bonemarrow. During the next few hours to days, the macrophages release the haemoglobin back into the blood for production of new red blood cells or to the liver and other tissues for storage in the form of ferritin. The porphyrin portion of haemoglobin molecule is converted by the macrophage through a series of stages into bile pigment bilirubin, which is released into the blood and later secreted by the liver into the bile.

Normal values of haemoglobin in different age groups

	Mean	Range
Cord blood	17.1	13.7-20.5
7 days	18.8	14.6-23.0
20 days	15.9	11.3-20.5
45 days	12.7	9.5-15.9
75 days	11.4	9.6-13.2
120 days	11.9	9.9-13.9

1 year	12.2	10.0-13
5 year	12.5	12-13
10 year	13.5	13-14
Older	15	14-16

Normal Values

Packed cell Volume (Haematocrit value) – (P.C.V.)

1-13days	:	54.0 ± 10.0%
14-60days	:	42.0 ± 7.0%
3 months -10years	:	36.0 ± 5.0%
11-15years	:	39.0 ± 5.0%

Mean Corpuscular Volume (M.C.V)

1-13 days	:	106-98fl
14-60 days	:	90 fl
3 month-10years	:	80fl
11-15 years	:	82 fl

Mean Corpuscular Haemoglobin (M.C.H)

1-13 days	:	38-33 picograms
14-60 days	:	30 picograms
3 month-10years	:	27 picograms
11-15 years	:	28 picograms

Mean corpuscular Haemoglobin concentration (M.C.H.C)

1-13 days	:	36 – 34g/dl
14-60 days	:	33g/dl
3 month-10years	:	34g/dl
11-15 years	:	34g/dl

Mean corpuscular diameter (M.C.D.)

1-13 days	:	8.6 μ m
14-60 days	:	8.1 μ m
3 month-10years	:	7.7 μ m
11-15 years	:	7.6 μ m

Reticulocytes

Cord blood	:	5.0%
2 week	:	1.0%
3 months	:	1.0%
6months-6years	:	1.0%
7-12 years	:	1.0%
Adult	:	1.6%

ANEMIA

A. DEFINITION

A Greek word anaemia/anemia meaning without blood. It is defined as a qualitative or quantitative deficiency of hemoglobin, a molecule found inside red blood cells.

WHO criteria for diagnosis of Anemia

Children 6 months – 6 years : Less than 11

Children 6 months – 14 years : Less than 12.

- IAP Text book of paediatrics 2nd edition.

Grading of Anaemia

WHO grades anemia according to haemoglobin level as follows,

HB between 10gm and cut off point for age : Mild

Hb between 7 to 10gm : Moderate

Hb under 7 gm : Severe

B. ETIOLOGY OF ANEMIA:

a. New Born

1. Haemolytic disease (Rh or ABO incompatibility)
2. Result of blood loss (ante natal, natal, post natal)

b. Young Infants (3 months to 18 months)

1. Physiological anemia (Normal variation of Hb and RBC and not a true anemia).
2. Iron deficiency anemia especially in prematures (with or without protein deficiency).
3. Megalaoblastic anemia of Infancy.
4. Infections, dysenteries and diarrhoea.

C. Older babies and Children.

Common Causes	Less Common Casues
1. Malnutrition and iron deficiency	1. Leukemia
2. Infections	2. Inherited defects of RBC Haemoglobinopathies, or congenital spherocytosis.
3. Nephritis, Nephrosis.	3. Bleeding disorders i. Haemophilias. ii. Thrombocytopenic purpura and petechial bleeding.
4. Ankylostomiasis	4. Rare causes – aplastic anemia, pernicious anemia.

C. ETIO – PATHOGENESIS:

1. Anemia due to defects in haemoglobin synthesis.

When there is deficiency of Iron, Vitamin B₁₂, Vitamin C, Folic Acid, Pyridoxine, Thyroxine, Proteins and Copper, there is decreased haemoglobin synthesis.

2. Anemia due to immaturation of Red Blood cells

In megaloblastic anemia large nucleated red blood cells are seen in the red marrow of the bones. This immaturation is due to non-availability of Vitamin B₁₂, Folic Acid.

3. Anemia due to Red Blood cell defects:

The life span of matured red blood cells is about 120 days. Some times they may die within their usual lifetime. This leads to anemia.

PATHOPHYSIOLOGY OF ANEMIA:

Subnormal level of haemoglobin causes lowered oxygen carrying capacity of the blood which leads to hypoxia in organs.

- Increased release of oxygen from haemoglobin
- Increased blood flow to tissues
- Maintenance of the blood volume
- Redistribution of blood flow to maintain the cerebral blood supply.

SYMPTOMS AND SIGNS OF ANEMIA:

Symptoms:

Lassitude, easy fatiguability, breathlessness on exertion, palpitation, tinnitus, throbbing in head and ears, generalized muscular weakness, dizziness, headache, hair loss, insomnia, angina, dimness of vision, paraesthesia in fingers and toes.

Signs

Pallor (Pale skin, mucosal linings and nail beds) Cheilosis, Koilonychia, Systolic flow murmurs, Oedema, Cardiac dilatation and tachycardia.

CLASSIFICATION OF ANEMIA:

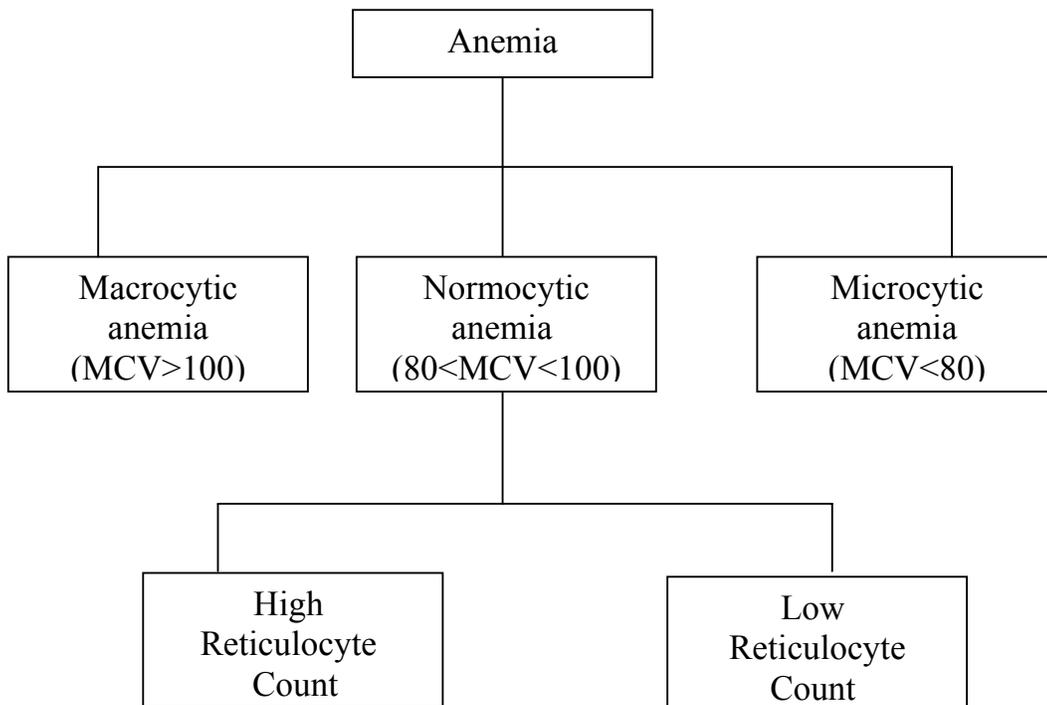
Morphologic classification:

Based on the red cell size, haemoglobin content and red cell indices anemias are classified as follows.

Based on erythrocyte morphology

1. Microcytic Hypochromic anemia : Iron deficiency, Thalassemia, Haemoglobinopathies and Haemolytic anemia.

2. Normocytic normochromic anemia: Aplastic anemia
3. Macrocytic normochromic anemia : Folate and vitamin B₁₂
deficiency hypothyroidism.
4. Macrocytic hypochromic anemia : Combined deficiency of Iron
and folate or vitamin B₁₂



Microcytic Anemia:

It is a result of haemoglobin synthesis failure / Insufficiency. The sizes of red cells are smaller than normal.

Causes:

Heme synthesis defect

- Iron deficiency anemia
- Anemia of chronic disease

Globin Synthesis defect

- Alpha – and beta – thalassemia
- HbE and HbC syndrome
- Sideroblastic defect
- Hereditary sideroblastic anemia
- Acquired sideroblastic anemia including lead toxicity.
- Reversible sideroblastic anemia.

Macrocytic anemia

The red cells are bigger than normal.

Causes:

- Deficiency of vit B₁₂ and folic acid due to inadequate intake or insufficient absorption (during gastric bypass surgery).
- Hypothyroidism
- Alcoholism and liver disease cause macrocytosis.
- Methotrexate, Zidovudine and other drugs that inhibit DNA replication.

Normocytic Anemia:

The size of red blood cells remains normal but haemoglobin levels are always decreased.

Causes:

- Acute blood loss
- Anemia of chronic disease
- Aplastic anemia (bone marrow failure)
- Haemolytic anemia.

Dimorphic Anemia:

When two causes of anemia act simultaneously eg. Macrocytic hypochromic anemia or following blood transfusion (more than one abnormality of red cell indices seen).

Heinz Body Anemia:

Heinz bodies are an abnormality that forms on the cells by taking certain medications, eg. Acetaminophen.

Complications:

- Hypoxemia worsen the cardio-pulmonary status of patient.
- Brittle or rigid fingernails
- Cold intolerance in iron deficiency anemia
- Behavioral disturbances in children.

Anemia during pregnancy:

Problems for the fetus include growth retardation, prematurity, intrauterine death, rupture of the amnion and infection.

Based on Etiopathogenesis:

- | | |
|-------------------------------|---|
| 1. Nutritional Anemias | PEM, Iron, Folic acid, vitamin B ₁₂ Vitamin C, Pyridoxine, or thyroxine deficiency. |
| 2. Haemolytic Anemias | |
| Congenital | Thalassemia, Sickle cell anemia, Hereditary spherocytosis, G-6-PD deficiency |
| Acquired | Certain infections like malaria and Kala azar, Rh or ABO incompatibility, autoimmune, drugs like primaquine, Furazolidine and phenacetin. |

3. Haemorrhagic

Actue	Trauma, Epistaxis, Circumcision, Bleeding diathesis (leukemias purpura, haemophilia) Haemorrhagic disease of newborn, scurvy.
Chronic	Hookworm, Chronic dysentery, Oesophageal varices.

4. Bone Marrow

Depression

Primary	Hypoplasia or Aplasia, Fanconi's anemia Infections, Irradiation, Chronic illness like Nephritis, leukemia and other neoplastic diseases. Drugs like chloramphenicol, sulfas etc.
Secondary	

5. Infection

Acute	Fulminating osteomyelitis Septicaemia Tuberculosis Rhemumatic fever
Chronic	Sub acute bacterial endocarditis Wound infections Congenital syphilis.

6. Other Miscellaneous

Conditions

Cretinism
Chronic amoebic dysentery
Repeated bouts of diarrhoea.

IRON

Iron is an essential constituent of haemoglobin, myoglobin, cytochromes and other components of respiratory enzymes like cytochrome oxidase, catalase and peroxidase. The main functions of iron are,

1. Transport of oxygen to the tissues
2. Participation in cellular oxidation mechanism.

Distribution of Iron in the body:

Iron is distributed in the body as follows,

1. Haemoglobin – present in red cells, contains most of the body iron (65 %)
2. Myoglobin – comprises a small amount of iron in the muscles (4%)
3. Haem and non-haem enzymes – eg cytochrome catalase, peroxidase, succinic dehydrogenase and flavoproteins constitute a fraction of total body iron (0.5%)
4. Transferrin bound iron – circulates in the plasma and constitutes another fraction of total body iron (0.5%)

All these forms of iron are in functional form.

5. Ferritin and Haemosiderin – are the storage forms of excess iron (30 %). They are stored in the mononuclear phagocyte cells of the spleen, liver and bone marrow and in parenchymal cells of the liver.

Daily iron requirements in different age groups:

Pregnant females	-	30 mg / day
Females 11 years to 30 years	-	15 mg / day
Adult males	-	10 mg / day
Males 11 years to 17 years	-	12 mg / day
Upto 10 years (M/F)	-	10 mg / day
Full term infants	-	1 mg /kg/day from 4 months of age
LBW Babies	-	2 mg/kg/day from 2 months of age
Babies 1000 to 1500 grams	-	3 mg/kg/day from 2 months of age
Less than 1000 grams	-	4 mg/kg/day from 2 months of age

IRON METABOLISM:**Absorption:**

Absorption of iron takes place in all parts of the small intestine by the following mechanism. A substance called apotransferrin secreted by the liver flows into the duodenum. There it binds with free iron and iron compounds haemoglobin and myoglobin to form transferrin. Transferrin binds with receptors of intestinal epithelial cells. Now transferrin molecule carrying iron is absorbed into the epithelial cells and released in the form of plasma transferrin. Ascorbic acid, citric acid, amino acids and sugars in the diet enhance absorption of iron.

Transport:

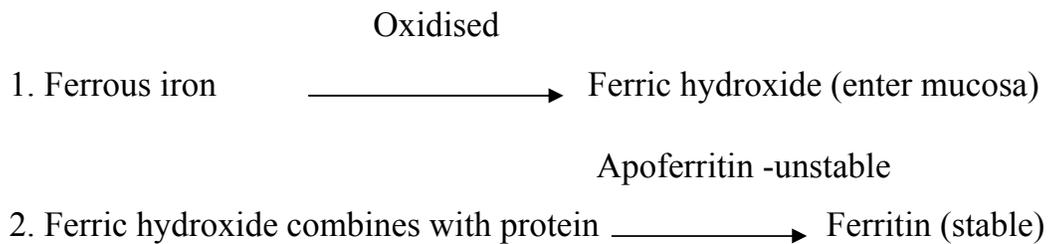
Iron is transported in plasma bound to β – globulin transferrin, synthesized in the liver. Transferrin bound iron is made available to the marrow where the immature red cell precursors utilize the iron for haemoglobin synthesis. Transferrin is reutilized after iron is released

from it. A small amount of transferrin is delivered to other sites such as parenchymal cells of the liver. Normally transferrin is about one third saturated. But in conditions where transferrin – iron saturation is increased, parenchymal iron uptake is increased. Virtually no iron is deposited in the mononuclear phagocyte cells (RE cells) from the plasma transferrin – iron but instead these cells derive most of their iron from phagocytosis of senescent red cells. Storage form of iron (ferritin and haemosiderin) in RE cells is normally not functional but can be readily mobilised in response to increased demands for erythropoiesis. However conditions such as malignancy infection and inflammation interfere with the release of iron from iron stores causing ineffective erythropoiesis.

Storage:

Storage of excessive iron in the blood is deposited in all cells especially in the liver hepatocytes. A smaller amount being stored in reticulo endothelial cells of the bone marrow. In the cell cytoplasm, it combines with apoferritin to form ferritin. This iron stored as ferritin is called storage iron. Some iron is stored in an insoluble form haemosiderin.

The subsequent stages of Fe (iron) absorption are outlined below.



Normally the total body iron is divided into functional and storage compartments. Approximately 80% of the functional iron is found in haemoglobin.

Loss of iron from the body:

- Mainly iron is lost from the body by desquamation
- Excessive sweating
- About 1mg of iron is excreted through faeces each day.
- Whenever bleeding occurs, additional quantity of iron is lost.
- In women about 20 mg iron per period is lost during menstrual cycle.

Regulation of total body iron:

Absorption and excretion of iron are maintained almost equally under normal physiological conditions. When the iron storage is saturated in the body, it automatically reduces further absorption of iron from the gastrointestinal tract by feed back mechanism. The factors, which reduce absorption of iron are,

1. Stoppage of apotransferrin formation in the liver, so that the iron could not be absorbed from the intestine.
2. Reduction in the release of iron from the transferrin so that transferrin is completely saturated with iron and further absorption is prevented. This type of regulation is known as feedback mechanism.

IRON DEFICIENCY ANEMIA (IDA)

Iron deficiency anemia is currently the most widespread micronutrient deficiency and affects infants, preschool children, adolescents and women of child bearing age. One third of the world's population suffers from iron deficiency anemia. The major factors influence the prevalence of anemia include socio-economic status, dietary patterns, the degree of urbanization, educational background, accessibility to health care facilities, prophylaxis programs and the prevalence of worm infestation in the population.

Structures of the Red Corpuscles in IDA:

In iron deficiency anemia, the red blood corpuscles are decreased or normal in number and the haemoglobin content of the red blood corpuscles is reduced. In the blood smear, the red cells appear pale with a large central pale area and many of the red blood cells appear to be smaller than the normal. This type of anemia is called "Hypochromic and Microcytic anemia".

Etiology:

The etiology varies with the age, sex, and country of residence of the patient.

Etiological factors in Iron Deficiency Anemia:

1. Increased Physiologic requirements

- Rapid Growth : Infants, Preadolescence
- Menstruation
- Pregnancy.

2. Decreased iron stores

- Preterms
- Small for dates
- Twins.

3. Decreased iron assimilation

- Iron poor diet
- Iron malabsorption
- Sprue, non tropical sprue
- Pica
- GI surgery
- Chronic diarrhoea
- Delayed weaning
- Malnutrition

4. Blood loss

- Gastro intestinal bleeding
- Milk induced enteropathy
- Peptic ulcer disease
- Inflammatory bowel disease
- Meckel's diverticulum
- Drugs – Salicylates
- Hook worm infestation
- Fetal Maternal transfusion
- Haemoglobinuria – prosthetic Heart valve
- Iatrogenic
- Idiopathic pulmonary hemosiderosis

- Intense exercise
- Bleeding diasthesis
- Repeated venous sampling

5. Increased demands.

- Prematurity
- Low birth weight
- Recovery from PEM
- Adolescence

Pathogenesis:

Iron deficiency anemia develops when the supply of iron to the bone marrow is insufficient for the requirements of haemoglobin synthesis.

It has been stated that the body is normally in a state of positive iron balance. When a negative iron balance occurs due to either blood loss, increased requirements or impaired absorption, the deficit is made good by iron mobilized from the tissue stores and an adequate supply of iron for haemoglobin formation is maintained. It is only when the tissue stores are exhausted that the supply of iron to the marrow for haemoglobin synthesis becomes inadequate and hypochromic anemia develops.

Thus iron deficiency may be regarded as developing in two stages.

1. The progressive depletion and cultivate exhaustion of the available tissue iron stores.
2. Iron deficiency state, which may be divided into three distinct stage of severity,

Stage	Manifestation
Early State	Storage iron depletion
Second Stage	Iron limited erythropoiesis
Third Stage	Iron deficiency anemia.

Stages of Iron Deficiency Anemia:

1. Storage Iron Depletion:

Iron reserve is small or absent and is characterized by reduced serum ferritin or reduced iron concentration in marrow and liver tissue. Haemoglobin and serum iron, Transferritin concentration and saturation are within normal limits.

2. Iron Limited Erythropoiesis:

Haemoglobin (Hb) may still be normal but serum iron is low and TIBC increased with a low serum ferritin and raised free erythrocyte protoporphyrin (FEP).

3. Iron Deficiency Anemia:

The flow of iron to erythroid marrow is impaired to cause reduction in haemoglobin concentration with a progressive microcytic hypochromic anemia associated with reduced serum iron, transferrin saturation and serum ferritin level.

CLINICAL FEATURES:

Symptoms	Signs
Weakness	Pallor of the skin, mucous membrane,
Headache	palms, nails and conjunctiva.
Bodyache	Smooth, pale, glossy tongue.
Giddiness	Angular stomatitis.
Fatigue	Cheilosis
Lassitude	Hepato Splenomegaly
Breathlessness on exertion	Koilonychia
Dimness of vision	Pica
Dizziness	Tachycardia
Insomnia	Cardiomegaly
Inability to concentrate	High volume pulse
Tinnitus	Heamic murmur
Anginal pain	Oedema.
Paraesthesia in fingers and toes	
Palpitation	
Loss of appetite	
Mental apathy	
Constipation	
Abdominal distension	
Hair loss	
Exercise intolerance	

Role of iron deficiency anemia in various systems

Cardiovascular System:

Dyspnoea and palpitation are common symptoms while on exertion but in very severe anemia the patient may get cardiac failure and there may be dyspnoea at rest. Haemic murmurs are commonly heard in anemic patients. The murmurs are most often mild systolic murmurs heard at the mitral area.

Systolic bruits over the carotid arteries in the neck are sometimes present in anemia usually they are bilateral and occur in the absence of an aortic systolic bruit and disappear following correction of the anemia. Jugular venous pressure increase in severe anemia due to the high pulse pressure with a capillary pulsation. Oedema of the legs occasionally occurs in ambulant patient with severe anemia as the result of venous capillary pressure on exertion and increased capillary permeability.

Central Nervous System:

Symptoms include faintness, giddiness, headache, roaring and banging in the ears, tinnitus, spots before the eyes, lack of concentration and drowsiness and with sever anemia clouding of consciousness, numbness, coldness and sometimes tingling of the hands and feet.

Reproductive System:

Menstrual disturbances are commonly associated with anemia.

Renal System:

Slight proteinuria may be present with severe anemia. Anemia may further reduce renal function at which nitrogen retention develops; correction of anemia in such patient is usually followed by a fall in blood urea.

Gastro Intestinal System:

Anorexia is the commonest symptom, nausea, flatulence and constipation may also occur. Slight to moderate smooth hepatomegaly is common in severe anemia and when congestive heart failure develops the liver may become tender. In certain cases of iron deficiency anemia, spleen may be enlarged.

Pyrexia:

Mild pyrexia may occur with severe anemia but marked fever is due to either the causative disorder or due to some complicating factor.

Complications of Iron Deficiency anemia:

- Iron deficiency anemia may be the present finding in gastro intestinal cancer.
- In patients with heart disease severe anemia may precipitate angina pectoris or congestive heart failure.
- Infections are more common in Iron deficiency anemia, especially those of respiratory, gastrointestinal and urinary tracts.
- Chronic iron deficiency anemia reduces the efficiency in work and study.

Investigations required for Iron deficiency anemia:**1. Blood investigations:**

- Total Red Blood cell count
- Differential count
- Erythrocyte sedimentation rate
- Mean corpuscular volume

- Mean corpuscular haemoglobin concentration
- Packed cell volume
- Peripheral blood smear
- Red cell survival
- Serum iron
- Serum Ferritin concentration
- Serum protein
- Serum creatinine.

2. Urine investigations:

- Urine sugar
- Urine albumin
- Deposits
- Red blood cells
- Pus cells

3. Stool investigations:

- Occult blood
- Organisms
- Ova
- Cyst
- Red blood cells
- Pus cells

Special investigations occasionally required:

- X-ray barium meal, X-ray Barium enema, X-ray Chest
- Endoscopy, Colonoscopy, Sigmoidoscopy,
Gastroduodenoscopy

- Isotope Studies.
 - a. Determination of life span of red cells using ^{51}Cr labeled erythrocytes.
 - b. Determination of absorption, utilization, and disposal of iron using ^{58}Fe .
- Skeletal survey for multiple myeloma and secondary deposits
- Bone marrow examination
- Liver Function Test (LFT)
- Jejunal biopsy, urography, selective angiography
- Ultrasonography.

Differential Diagnosis:

Iron deficiency anemia must be differentiated from other hypochromic anemias.

1. Anemia of Infection:

Chronic infections such as rheumatic fever, rheumatoid arthritis, tuberculosis and malaria may have associated with mild to moderate anemia, which is normochromic or slightly hypochromic. Serum iron is low, total iron binding capacity is also decreased. Bone marrow haemosiderin is present.

2. Pyridoxine (Vit B6) Deficiency Anemia:

It is characterised by severe microcytic hypochromic anemia, often early in infancy and progressive hepatosplenomegaly. There is elevation of serum iron, marrow shows erythroid hyperplasia with nucleated normoblasts containing iron inclusions, the so-called “sideroblasts” in abundance. There are abnormalities of tryptophan metabolism.

3. Some Haemoglobinopathies:

In haemoglobin abnormalities like thalassemia, the red cells are microcytic and hypochromic. Thalassemia minor is distinguished by normal serum iron, normal total iron binding capacity, decreased mean corpuscular volume, normal serum ferritin and transferrin iron saturation.

4. Sideroblastic Anemia:

Most of the red cells are hypochromic and microcytic, serum iron is high and iron deposits in the marrow, liver and spleen are excessive. Many erythrocytes and erythroblasts contain non haemoglobin iron (ringed sideroblasts) in their mitochondria. The spleen is usually enlarged.

5. Anemia of Lead Poisoning:

Anemia of lead poisoning is hypochromic and microcytic and may be moderate to severe. Basophilic stippling of red cells, which helps to differentiate it from iron – deficiency anemia, pronounced increase of aminolevulinic acid and coproporphyrin in the urine is characteristic of lead poisoning. Increased levels of lead in urine and blood are required for definite diagnosis.

DIAGNOSIS:

Following criteria are essential to diagnose Iron deficiency anemia.

- History of inadequate intake of dietary iron and blood loss if any
- Typical symptoms and signs like easy fatigability, pallor, pica, koilonychia, smooth tongue, cheilosis, and dysphagia associated with general considerations.

- Hypochromic and microcytic structure of red blood cells.
- Low serum iron, increased total iron binding capacity.
- Bone marrow haemosiderin absent
- Blood loss usually occult
- Platelet count is either normal or raised
- Haemoglobin estimation variably reduced
- Reduced mean cell volume
- Erythrocyte count may be normal or reduced
- Serum ferritin level is reduced.

MANAGEMENT:

This can be considered under three headings.

1. Correction of anemic state:

Over all correction of nutrition with articles rich in iron is important. Iron deficiency is corrected by intake of rich iron content diet and administration of medicinal iron.

2. Replenishment of iron stores

3. Elimination of the cause.

Prophylaxis:

The main principles in the prevention of nutritional Iron Deficiency anemia are,

1. The regular consumption of a well balanced diet containing an adequate quantity of iron.
2. The periodic administration of iron as drug during increased physiological needs such as rapid growth during infancy and preadolescence, pregnancy, lactation, and menstruation.

3. Maintenance of a normal haemoglobin level in the mother for the prevention of iron deficiency anemia in infants. Premature and unduly small infants should be given prophylactic iron as a routine therapy. Iron rich sources should be added in the infants diet from the third or fourth month and thereafter be progressively increased. Following control of infection iron should be given to all infants if the haemoglobin level is decreased.

Prevention of IDA:

The basic approaches to the prevention of IDA include.

1. Protection and promotion of breast – feeding for as long as possible along with timely weaning is effective in preventing IDA. Low birth weight infants need iron supplementation from the age of 2 months.
2. Dietary modification and consumption of larger amounts of habitual foods increases total iron consumption by 25-30 percent. Processes like germination (sprouting of green gram) consumption and green leafy vegetables would be additional long-term methods for prevention of IDA.
3. Periodic deworming with anti-helminthic drugs for hookworm infestation and schistosomiasis should be considered in endemic areas.
4. Supplementation with medicinal iron is considered necessary to reduce the extent of anemia in developing countries.

5. Food and salt fortification with iron are evolving rapidly and would be one of the most effective ways to control IDA. Salt fortification gives an iron content of 1mg per gram of salt in the preparation.

Diet:

Haem iron sources:

- Muscle meat (red more than white)
- Organ meat (e.g. Liver)
- Fish and shellfish
- Poultry

Non – haem iron sources:

- Oatmeal, legumes (peas, beans)
- Nuts
- Pulses
- Dried fruit
- Whole meat
- Bread, eggs
- Green leafy vegetables
- Iron fortified cereal foods, chocolate
- Jaggery and yeast
- Foods rich in vitamin C enhance iron absorption.

Self care procedures for Iron deficiency anemia:

1. Eat more foods that are good sources of iron
2. Concentrate on green leafy vegetables, red meat, beef liver, poultry, fish, wheat germ, oysters, dried fruit, and fortified cereals.

3. Foods high in vitamin C like citrus fruits, tomatoes, and strawberries help the body absorbing iron from food.
4. Red meat not only supplies a good amount of iron, it also increases absorption of iron from other food sources.
5. Take an iron supplement. Consult your physician for proper dosage.
6. While iron is best absorbed when taken on an empty stomach, it can upset your stomach, taking iron with meals is less upsetting the stomach.
7. Avoid antacids, phosphates (which are found in soft drinks, beer, ice cream, candy bars, etc) and the food additive EDTA. These block iron absorption.
8. Increase dietary fibre to prevent constipation
9. Avoid aspirin and products with aspirin.
10. To make the best use of folic acid, eat good sources of folic acid daily.
11. These include vegetables like asparagus, Brussels, sprouts, spinach, romaine, lettuce, collard greens and broccoli.
12. Black – eyed peas, cantaloupe orange juice, oatmeal, whole grain cereals, wheat germ, liver and other organs are excellent sources.
13. Eat fresh uncooked fruits and vegetables often. Don't overcook food that destroys folic acid.

MATERIALS AND METHODS

The study on the clinical evaluation of the disease Pandu noi was carried out in the Postgraduate Kuzhanthai Maruthuvam Department in Government Siddha Medical College, Palayamkottai. Twenty patients of both male and female children were selected for the study and admitted in the Postgraduate, Kuzhanthai Maruthuvam In-patients ward. After discharge all of them were advised to come to out-patients ward for further follow-up. Another 60 patients were also treated with trial drug in the Out-patients ward.

Selection of Patients:

The present study covers both male and female children of varying age groups. All cases were carefully and thoroughly examined before admission. Those who fulfilled the criteria for Pandu noi (Iron deficiency anemia) according to the patho-physiology of Siddha and Modern reviews were selected. The opinion of professor and lecturer was obtained and detailed history was recorded in the proforma of casesheet attached in annexure-1.

Study of Clinical Diagnosis:

A case sheet was prepared on the basis of Siddha methodology and modern methodology to diagnose the disease. An individual case sheet was maintained for each and every patient.

A complete history of patient was taken. Name, Age, Sex, History of present and past illness, ante-natal, neonatal, post-natal history, personal and dietetic history, family history, socioeconomic status were noted.

Siddha diagnosis was made on the basis of Ennvagai thervukal, mukkutram and Ezhu udalkattugal.

Modern diagnostic methods were adopted with the consultation of paediatric professor.

Investigations:

The Modern diagnostic tests such as blood test for TC, DC, ESR, HB, PCV, MCV, MCH, MCHC, TIBC, peripheral blood smear etc., urine analysis for albumin, sugar, deposits etc. and stool examination for ova, cyst, occult blood to rule out any existing illness.

Haemoglobin values and PCV were estimated before and after therapy.

Administration of Trial Medicine:

The trial drug was prepared carefully. Madhulai manappagu and Bringaraja chooranam with the adjuvant of honey was given to all 20 patients two times a day during their treatment period.

The Biochemical analysis of trial drug was carried out in the Biochemical laboratory and Haematinic affect and laxative action of the drug was tested in the Pharmacological laboratory and the results are attached in Annexure III&IV.

Analysis of observations made from the 20 patients with signs and symptoms of the disease were recorded. In addition to medicine the patients were advised to take iron-rich diet and to attend the Out-patients ward for followup.

RESULTS AND OBSERVATIONS

For this clinical study 20 cases were selected and treated in the In-patient ward and Out-patient ward of P.G Department of Kuzhanthai Maruthuvam, Govt. Siddha Medical College, Palayamkottai. Results were observed with respect to the following criteria.

1. Sex distribution
2. Age distribution
3. Religion distribution
4. Family distribution
5. Soci-economic status
6. Dietary habits
7. Seasonal reference
8. Reference to Thinai
9. Reference to Mukkutra kaalam
10. Reference to Etiological factors
11. Reference to Mukkutram
 - a. Affected vatham
 - b. Affected pitham
 - c. Affected kabham
12. Reference to Ezhu Udal kattugal
13. Reference to Envagai thervukal
14. Signs and symptoms of Pandu noi during admission and discharge
15. Results after treatment.

The observations recorded with the above said criteria were given in the tabular column form.

1. Sex Distribution:

S. No.	Sex	No. of cases	Percentage
1.	Male children	13	65
2.	Female Children	7	35

Among the 20 patients selected, 65% of patients were male children and 35% of patients were female children.

2. Age distribution:

S. No.	Age	No. of cases	Percentage
1	1-6 months (Kaappu Paruvam)	-	-
2.	6-12 months (Senkeerai paruvam)	-	-
3.	1-3 years (Thalattu, Sappani, Mutha and Varugai paruvams)	-	-
4.	3-6 years (Ampuli Sitril, Siruparai, Siruthaer viduthal – male child. Ammanai, Neeraduthal, Oonjal – female child)	6	30
5	6-12 years (Siruparuvam – male child. Paethai and pethumbai – female child)	14	70

Among the 20 patients treated, 14 cases (70%) belonged to 6-12years and 6 cases (30%) belonged to 3-6 years. The percentage is more in the age group of 6-12years.

3. Religion Distribution:

S. No.	Religion	No. of cases	Percentage
1	Hindu	17	85
2.	Christian	2	10
3.	Muslim	1	5

Out of 20 cases 85% were Hindus, 10% were Christians and 5% were Muslims.

4. Family History:

S. No.	Family History	No. of cases	Percentage
1	Positive	-	-
2.	Negative	20	100

All the 20 patients had negative family history.

5: Socio- Economic status of the patient:

S. No.	Socio-Economic Status	No. of cases	Percentage
1	Poor	15	75
2.	Middle	5	25
3.	Rich	-	-

Out of the 15 patients, 75% of cases were poor and 25% were middle class people.

6. Dietary habits:

S. No.	Diet	No. of Cases	Percentage
1.	Vegetarian	3	15
3.	Mixed	17	85

85% of cases have mixed diet and 15% of cases have vegetarian diet.

7. Seasonal Reference

S. No.	Paruva Kaalam	No. of cases	Percentage
1.	Kaar kaalam (Aavani& Purattasi)	13	65
2.	Koothir kaalam (Iyppasi & Karthigai)	4	20
3.	Munpani kaalam (Markazhi & Thai)	-	-
4.	Pinpani kaalam (Masi & Panguni)	-	-
5.	Elavenil kaalam (Chithirai & Vaigasi)	1	5
6.	Muthuvenil kaalam (Aani & Aadi)	2	10

Among the 20 cases selected, 65% of cases were admitted in Kaar kaalam, 20% of cases were admitted in Koothir kaalam, 5% of cases were admitted in Elavenil kaalam and 10% of cases were admitted in Muthuvenil kaalam.

8: Thinai Reference

S. No.	Thinai	No. of cases	Percentage
1.	Kurinji (Hill area)	3	15
2.	Mullai (Forest area)	-	-
3.	Marutham (Fertile area)	17	85
4.	Neithal (Coastal area)	-	-
5.	Paalai (Desert area)	-	-

Among 20 cases, 85% belonged to Marutham and 15% belonged to Kurinji Nilam.

9. Mukkutra Kaalam:

S. No.	Kaalam	No. of cases	Percentage
1	Vatham	20	100
2.	Pitham	-	-
3.	Kapham	-	-

In this study, all the 20 patients were in Vatha Kaalam since all the patients belong to children.

10. Aetiological factors:

S. No.	Aetiological factors	No. of Cases	Percentage
1.	Nutritional deficiency	20	100
2.	Worm infestation	-	-

Out of 20 cases treated, the etiological factor was found to be Nutritional deficiency in 100% cases.

11. Reference to Mukkutran

A. Affected Vatham

S. No.	Vatham	No. of cases	Percentage
1.	Pranan	7	35
2.	Abaanan	-	-
3.	Viyaanan	20	100
4.	Uthaanan	-	-
5.	Samaanan	20	100
6.	Naagan	-	-
7.	Koorman	-	-
8.	Kirukaran	20	100
9.	Devathathan	20	100
10.	Thananjeyan	-	-

Among 10 types of Vatham Viyanan, Samanan, Piranan, Kirukaran and Devathathan were affected.

B. Affected Pitham:

S. No.	Pitham	No. of cases	Percentage
1.	Anar Pitham	20	100
2.	Ranjagam	20	100
3.	Sathagam	20	100
4.	Prasagam	20	100
5.	Alosagam	-	-

Among 5 types of pitham all were affected expect Alosagam in all patients.

C. Affected Kabam:

S. No.	Kabam	No. of cases	Percentage
1.	Avalambagam	7	35
2.	Kilethagam	20	100
3.	Pothagam	20	100
4.	Tharpagam	-	-
5.	Santhigam	-	-

Among the twenty cases, Avalmbagam, Kilethagam and Pothagam were affected.

12. Reference to Udalkattugal:

S. No.	Udalkattugal	No. of cases	Percentage
1.	Saaram	20	100
2.	Senneer	20	100
3.	Oon	-	-
4.	Kozhuppu	-	-
5.	Enbu	-	-
6.	Moolai	-	-
7.	Sukkilam/Suronitham	Not applicable	Not applicable

Regarding seven Udalkattugal, Saram, and Senneer were affected in all 20 patients.

13. Reference to Envagai Thervugal:

S. No.	Envagai Thervugal	No. of cases	Percentage
1.	Naa	20	100
2.	Niram	20	100
3.	Mozhi	-	-
4.	Vizhi	20	100
5.	Malam	-	-
6.	Moothiram	-	-
7.	Naadi -Vathakabham	8	40
	Kabhavatham	7	35
	Kabhapitham	5	25
8.	Sparisam	-	-

Among the Envagai Thervugal Naa, Niram and Vizhi were affected in all 20 cases.

14. Reference to signs and symptoms:

S. No.	Signs and Symptoms	During Admission No of cases	During discharge No of Cases
1	Pallor of conjunctiva and nail beds	20	5
2.	Anorexia	20	-
3.	Ulceration of mouth	10	-
4.	Diarrhoea	-	-
5.	Lassitude	20	2
6.	Emaciation	6	3
7	Palpitation	20	4
8	Dyspnoea on exertion	20	4
9	Worm infestation	-	-

15. Among the 20 cases studied the results were observed as follows.

S. No.	Gradation	No. of Cases	Percentage
1.	Good	11	55
2.	Fair	6	30
3.	Poor	3	15

Among the 20 patients selected, 55% of cases showed good response, 30% of cases showed Fair response and 15% of cases showed Poor response.

IN THE PATIENTS WARD CASE REPORT

S. No.	IP. No.	Name	Age/Sex	D.O.A.	Signs and symptoms	D.O.D.	No of days treated	
							IP	OP
1	1356	Rajagopal	8/MC	28.5.08	Pallor of conjunctiva and nailbeds anorexia, lassitude, dyspnoea on exertion, Palpitation	02.06.08	6	30
2	1686	Shanmugavel	8/MC	28.06.08	Pallor of conjunctiva and nail beds, anorexia, ulceration of mouth lassitude, dyspnoea on exertion, palpitation	10.07.08	13	27
3	1867	Mariappan	12/MC	18.07.08	Pallor of conjunctiva and nailbeds, anorexia, ulceration of mouth, lassitude	01.08.08	15	25
4	2049	Anand	8/MC	07.08.08	Pallor of conjunctiva and nailbeds, anorexia, lassitude, dyspnoea on exertion, palpitation	16.08.08	10	30
5	2028	Suresh	12/MC	05.08.08	Pallor of Conjunctiva and nailbeds, anorexia, ulceration of mouth, lassitude, dyspnoea on exertion, palpitation	11.08.08	7	25
6	2111	Vadivel	12/MC	14.08.08	Pallor of conjunctiva and nail bods, anorexia, lassitude, dyspnoea on exertion, palpitation	18.08.08	5	35
7	2077	Vasanth	6/MC	11.08.08	Pallor of conjunctiva and nailbeds anorexia, lassitude, dyspnoea on exertion palpitation	19.08.08	9	25
8	2417	Ebi	5/MC	18.08.05	Pallor of conjunctiva and nailbeds, anorexia, ulceration of mouth, lassitude, palpitation dyspnoea on exertion	25.08.08	8	20
9	2206	Madumathi	8/FC	22.08.08	Pallor of conjunctiva and nailbeds, anorexia, ulceration of mouth, lassitude, palpitation, dyspnoea on exertion	29.08.08	8	25
10	2277	Eswari	5/FC	28.8.08	Pallor of conjunctiva and nailbeds, anorexia, lassitude, palpitation, dyspnoea on exertion	06.09.08	10	30

11	2374	Sanjay	5/MC	08.09.08	Pallor of conjunctiva and nailbeds, anorexia, ulceration of mouth, lassitude, dyspnoea on exertion, palpitation	14.09.08	7	33
12	2344	Balan	5/MC	04.09.08	Pallor of conjunctiva and nailbeds, anorexia, lassitude, palpitation, dyspnoea on exertion	17.09.08	14	26
13	2540	Rani	11/FC	24.09.08	Pallor of conjunctiva and nailbeds, anorexia, lassitude, palpitation, dyspnoea on exertion	28.09.08	5	35
14	2551	Banu	5/MC	25.09.08	Pallor of Conjunctiva and nail beds, anorexia, ulceration of mouth, lassitude, dyspnoea on exertion, palpitation	07.10.08	12	20
15	2575	Muthu	5/MC	27.09.08	Pallor of Conjunctiva and nail beds, anorexia, lassitude, palpitation dyspnoea on exertion,	07.10.08	11	25
16	2579	Naveenkumar	9/MC	28.09.08	Pallor of Conjunctiva and nail beds, anorexia, ulceration of mouth, lassitude, palpitation dyspnoea on exertion,	09.10.08	13	27
17	2757	Kaleeshwari	6/FC	22.10.08	Pallor of Conjunctiva and nail beds, anorexia, lassitude, palpitation, dyspnoea on exertion,	13.11.08	23	15
18	2786	Mani	8/MC	30.10.08	Pallor of Conjunctiva and nail beds, anorexia, ulceration of mouth, lassitude, Palpitation, dyspnoea on exertion,	11.11.08	12	25
19	2992	Maheshwari	8/FC	18.11.08	Pallor of Conjunctiva and nail beds, anorexia, ulceration of mouth, lassitude	10.12.08	23	15
20	3000	Muthulakshmi	12/FC	19.11.08	Pallor of Conjunctiva and nail beds, anorexia, lassitude, Palpitation, dyspnoea on exertion,	28.11.08	10	30

D.O.A. – Date of Admission

IP - Inpatient

D.O.D – Date of Discharge

OP - Outpatient.

Haematological Investigation Results

S.No	I.P.No	Name	Age	Sex	Before Treatment							After Treatment						
					Hb gm%	RBC mill/cumm	PCV %	MCV fl	MCH pg	MCHC gm%	Peripheral Blood smer	Hb gm%	RBC mill/cumm	PCV %	MCV fl	MCH pg	MCHC gm%	Peripheral Blood smer
1	1356	Raja Gopal	8	MC	7.2	2.8	22	78	26	32	H.M	8.3	3.2	28	88	26	30	H.M
2	1686	Shanmugavel	8	FC	8.2	3.1	24	77	26	33	H.M	11	3.7	30	81	30	37	N
3	1867	Mariappan	12	MC	7	2.8	22	78	25	32	H.M	9.3	3.5	30	86	27	31	N
4	2049	Anand	8	MC	6.8	3	22	73	23	31	H.M	9.1	3.7	29	78	25	31	H.M
5	2028	Suresh	12	MC	8.4	3.4	26	76	25	32	H.M	11.2	3.9	34	87	29	33	N
6	2111	Vadivel	12	MC	7.4	3	23	77	25	32	H.M	10	3.6	30	83	28	33	N
7	2077	Vasanth	6	MC	7.5	3.4	24	71	22	31	H.M	8.2	3.5	26	80	23	29	H.M
8	2147	Ebi	5	MC	8.4	3.5	25	71	24	33	H.M	11	3.9	29	74	28	38	N
9	2206	Madumathi	8	FC	8.8	3.6	27	75	24	32	H.M	10.8	4	31	78	27	35	N
10	2277	Eswari	5	FC	9.5	3.8	28	74	25	33	H.M	12	4.2	33	83	29	36	N
11	2374	Sanjay	5	MC	8.3	3.4	26	76	24	31	H.M	11.1	4.0	34	85	28	33	N
12	2344	Balan	5	MC	8	3.3	25	76	24	32	H.M	10.0	3.6	30	83	28	33	N
13	2540	Rani	11	FC	7.2	3.1	23	74	23	31	H.M	10.2	3.7	31	84	28	33	N
14	2551	Banu	5	FC	8	3.3	26	78	24	31	H.M	11	3.8	30	79	29	36	N
15	2575	Muthu	5	MC	8.1	3.3	24	73	25	33	H.M	11.3	3.9	32	82	29	35	N
16	2579	Naveenkumar	9	MC	7.8	3.2	24	75	24	33	H.M	10.8	3.8	32	84	28	34	N
17	2757	Kaleeshwari	6	FC	8.2	3.3	26	78	25	31	H.M	10.4	3.7	31	84	28	34	N
18	2786	Mani	8	MC	8.6	3.9	30	77	22	29	H.M	11	4.2	36	86	26	31	N
19	2992	Maheshwari	8	FC	8.2	3.5	36	74	23	32	H.M	10.8	4.1	35	83	26	31	N
20	3000	Muthu lakshmi	12	FC	7.6	3	23	76	25	33	H.M	10	3.5	32	91	29	31	N

PCV - Packed Cell Volume
 MCV - Mean Corpuscular Volume
 MCH - Mean Corpuscular Haemoglobin

MCHC - Mean Corpuscular Haemoglobin Concentration

Investigation Results

S. No.	I.P. No.	Age	Sex	Blood												Urine						Motion								
				Before Treatment						After treatment						Before Treatment			After Treatment			Before Treatment			After Treatment					
				TC Cells / Cumm	DC			ESR (mm)		TC Cells / Cumm	DC			ESR (mm)		A	S	D	A	S	D	Ova	Cyst	OB	Ova	Cyst	OB			
					P	L	E	1/2 hr	1 hr		P	L	E	1/2 hr	1 hr															
1	1356	8	MC	7,700	68	27	5	4	8	9,200	58	40	2	4	8	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
2	1686	8	FC	8,700	58	35	7	10	22	9,000	55	37	8	7	15	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
3	1867	12	MC	8,300	57	56	7	8	16	8,500	60	38	7	5	10	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
4	2049	8	MC	9,000	62	35	3	9	18	9,200	55	37	3	6	12	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
5	2028	12	MC	9,800	65	30	5	7	14	9,900	63	33	4	4	8	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
6	2111	12	MC	10,400	52	45	3	6	12	9,500	55	39	6	3	6	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
7	2077	6	MC	9,800	60	36	4	9	18	9,700	58	38	4	5	10	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
8	2147	5	MC	9,000	60	36	4	6	12	9,200	65	33	2	2	4	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
9	2206	8	FC	9,200	56	40	4	7	14	9,000	60	38	2	3	6	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
10	2277	5	FC	8,700	60	38	2	6	12	8,800	63	35	2	2	5	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
11	2374	5	MC	8,800	60	36	4	9	18	8,800	62	35	2	5	10	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
12	2344	5	MC	9,200	65	33	2	6	12	9,000	63	35	2	3	7	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
13	2540	11	FC	9,500	60	35	5	8	16	9,600	63	36	2	4	8	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
14	2551	5	FC	9,200	58	38	4	7	14	9,000	60	36	4	3	6	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
15	2575	5	MC	9,000	60	36	4	7	14	9,200	62	35	3	4	8	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
16	2579	9	MC	9,200	62	30	8	6	12	9,000	62	34	4	2	4	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
17	2757	6	FC	9,700	62	35	3	8	16	9,800	64	34	2	5	10	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
18	2786	8	MC	9,000	58	38	4	4	8	9,000	60	37	3	3	7	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
19	2992	8	FC	8,300	57	36	7	5	10	8,200	60	35	5	4	8	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
20	3000	12	FC	10,000	60	38	2	6	12	9,800	65	33	2	3	7	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil

TC - Total count
 DC -Differential count
 ESR - Erythrocyte sedimentation rate

P – Polymorphs
 L- Lymphocytes
 E- Eosinophil

A - Albumin
 S - Sugar
 D - Deposit

OB - Occult Blood
 HM – Hypochromic
 Microcytic cells

DISCUSSION

Pandu is a clinical entity described by Siddhars and it has clinical features such as loss of appetite, lassitude, emaciation, pallor of the mucous membranes, conjunctiva, tongue and nails, dyspnoea on exertion. These features are identical with “Iron deficiency anemia” (Hypochromic microcytic anemia) a clinical entity described in modern literatures. The clinical features of “Pandu Noi” have been furnished by some of the siddha literatures like Balavagadam, Yugi Vaithiya Chinthamani etc.

Sex:

According to the clinical study 65% of the cases were found to be male children and 35% were found to be female children.

Age:

Among the affected children 70% were found to be within the age limit of 6-12 years (Siruparavam – male child and Paethai and Pethumbai – female child) and 15% were found to be within the age limit of 3-6 years (Ampuli, Sitril, Siruparai, Siruthaer Viduthal – male, Ammanai, Neeraduthal and Oonjal – female)

Socio-Economic Status:

Poor Socio economic status is a main predisposing factor in Paandu noi, as nutritious food and hygienic life is not available to them. In this study 75% of the patients belonged to poor socio economic status.

Paruva Kaalam:

From this study 65% of cases developed Pandu noi in Kaarkaalam 5% in Elavenil kaalam and 10% in Mudhuvvenil Kaalam. According to Siddha concept, Thannilai Valarchi of pitham is during Kaar Kaalam and Koothirkaalam.

Nilangal:

Among the 20 cases, 17 cases were from Marutha nilam and 3 cases were from Kurinji nilam. According to the texts, Kurinji nilam may cause disease regarding blood. Marutha nilam is devoid of diseases but due to pollution, this concept gets deviated in this study.

Etiology:

Generally, this disease is due to dietetic factors which cause vitiation of pitham and kabam. History of the patients reveal that this disease was caused by craving for mud, sand and ash, eating of salt, sour and pungent tasted food items and wandering in the hot sun. Siddhars have also stated the same causes described above this disease are due to the derangement of pitham leading to alteration in blood.

Mukkutram:

Among the three vital forces, pitham is mainly affected. Among the five types of pitham, Ranjagam was affected mainly which causes discolouration of mucous membrane. Also other forms of pitham like Analam, Saathagam and Pirasagam were affected in 100% of cases. The derangement of Pitham is followed by derangement of Kabam and Vatham.

Udal Kattugal:

Among seven Udal Kattugal, Saaram and Senneer were affected in 100% of the cases.

Envagai Thervukal:

In this, the changes of Naadi, Sparisam, Naa , Niram, Mozhi, Vizhi, Malam and Moothiram were noted.

a) Naadi:

According to this study, Vathakaba naadi was found in 40% of cases, Kabavatha naadi in 35% of cases and Kabapitha naadi in 25% of cases.

b) Naa:

In all of the cases, the tongue was pallor in colour.

c) Niram:

Due to involvement of pitham, the body was pallor in colour. This condition was noted in almost all cases.

d) Vizhi:

In this, pallor of the conjunctiva was noted in almost all cases.

e) Malam:

The colour of the stool was pale yellow in colour.

f) Moothiram:

In Pandu noi, due to increased pitham, the urine was yellow or dark yellow in colour.

Neikuri:

The neikuri was ring shaped in 10 cases which was defined for pitha disease and pearl shaped in 10 cases.

The diagnosis of the disease was made on the basis of Ennvagai thervukal and available modern investigation methods. In most of the cases, haemoglobin level, total red blood cells, MCV, MCH and MCHC were reduced.

Treatment:

Among the vital forces, pitham is mainly affected in Pandu noi.

Following Pitham, Kabam and Vatham are also deranged. So the principal aim in the treatment aspects is to make the deranged vital forces normal by giving the trial drug.

Before starting the actual treatment efforts are made to normalize the deranged thathus. This is explained in Siddha as follows.

“சத்தியால் பித்தந் தரமும்
பேதியால் வாதந் தரமும்
அஞ்சனத்தால் கபந் தரமும்”

Usually for pitha diseases, emetics are to be given to alter the deranged pitham. But there are some exceptions to this rule. For instance, in Pandu noi since the patient is already weak and drowsy, the administration of emetic medicine is exclude from the line of treatment.

In this study all the 20 cases were treated with Bringuraja Chooranam and Madhuali Manappagu.

The trial medicine having the properties of neutralizing pitham was given to patients to set right the deranged pitham on the basis of Arusuvai and Panchabootham.

“பித்த மதிகரிப்பின் பேசும் பரிகாரம்
சுத்த துவரோடு சொல்லிணிப்புச் சத்தாகும்”.

The selected trial drugs Bringuraja Chooranam and Madhuali Manappagu has astringent and sweet tastes, which normalize the increased pitham. The trial drug also contains iron in the ferrous form, which is

easily absorbed. These helped to arrive at a view that trial drug possesses properties capable to normalize the pitham with a significant haematinic effect.

Iron preparations which form the basis for erythropoiesis come to the consideration first to meet out this iron deficiency anemia. In this measure patients were given Bringaraja Chooranam with the adjuvant of honey and Madhuali Manappagu.

The aim of therapy is to increase the amount of hemoglobin. As the trial drug has significant haematinic, stomachic and tonic actions, the aim are attained.

During the treatment, iron rich diet was strictly advised. Along with the trial drug all the patients were advised to take green leafy vegetables, fruits, meat, seafoods, nuts, cereals and eggs, because these are rich in iron content.

SUMMARY

The study of Pandu noi is done to find out a complete relief to those affected, that too with a herbal drugs of simple preparation. Bringaraja chooranam and Madhulai Manappagu.

Various literature evidence relevant to Pandu noi were collected from both Siddha system as well as modern system of medicine.

The efficacy of the drug Bringaraja chooranam and Madhulai Manappagu has been studied and observed.

Twenty patients from both sexes of different age groups were selected and treated in the In-patient ward of PG-Kuzhanthai Maruthuvam Department of GSMC. Among the 20 patients, 13 were male children and 7 were female children. Further follow up of the cases were done in the outpatient ward. Specific investigations and the prognosis of the patient were studied and the proforma was prepared accordingly. Maximum occurrence of this disease was observed in school going children (6 to 12 years) but the other have no exception.

Findings reveal about the impact of the disease in the body. Statistical study of the details in the case sheet was observed and the results have a see through idea about the disease. The drug selected for the study was found to be easy for administration and the children found it easy for intake. No adverse side effects were reported during or after the course of treatment.

The biochemical analysis and pharmacological studies of the drug revealed its efficacy. From the studies, the drug Bringaraja Chooranam and Madhulai Manappagu possesses significant haematinic action.

CONCLUSION

From the above studies, it is clear that Pandu Noi is caused due to derangement of pitham followed by derangement of vatham and kabham. Both symptomatic relief and qualitative improvement were observed in the patients. Routine haematological investigations were undertaken to watch the prognosis.

The drug taken for study **Bringaraja Chooranam and Madhulai Manappagu** has been proved clinically to be potent haematinic and it raises the haemoglobin level in anaemic patients when given regularly not less than for 15 days along with other healthy supplementary diets.

Pathologically affected patients may be restored to normal physiological life by means of drugs. But the restored life shall be maintained only when the patient is given nutritional diet, personal hygiene and a systemic way of life.

ANNEXURE-I

PREPARATION OF THE TRIAL DRUG

BRINGARAJA CHOORANAM

தேவையான சரக்குகள் :

கரிசலாங்கண்ணி சமூலம்	:	100 கிராம்
கடுக்காய் தோல்	:	30 கிராம்
நெல்லிவற்றல்	:	30 கிராம்
தான்றிக்காய் தோல்	:	30 கிராம்
சர்க்கரை	:	200 கிராம்

செய்முறை:

கடைச்சரக்குகளை நன்கு உலர்த்தி தூசுகளை நீக்கி சுத்தி செய்து எடுத்துக் கொண்டேன். சர்க்கரை தவிர மற்ற சரக்குகளை தனித்தனியாக சூரணித்து வஸ்திரகாயம் செய்து, பிறகு சர்க்கரை சேர்த்து அரைத்து எடுத்துக் கொண்டேன்.

அளவு: 500 மி.கி. - 1 கிராம் (வயது, எடை, உடல் வன்மைக்கு தக்கவாறு), இருவேளை உணவிற்கு பின், 40 நாட்கள்.

அனுபானம் : தேன்

தீரும் நோய் : பாண்டு ரோகம்

ஆதாரம் : அனுபவ வைத்திய தேவ ரகசியம்.

MADHULAI MANAPPAGU

தேவையான சரக்குகள்

கற்கண்டு	:	500 மில்லி லிட்டர்
பன்னீர்	:	500 மில்லி லிட்டர்
மாதுளை இரசம்	:	500 மில்லி லிட்டர்
தேன்	:	500 மில்லி லிட்டர்.

செய்முறை :

கற்கண்டை கரைத்துப் பின் வடிகட்டி எடுத்துக் கொண்டேன். மாதுளம் பழத்தை துணியிலிட்டு அதன் இரசத்தை பிழிந்து எடுத்துக் கொண்டேன். அதனை மற்றவைகளுடன் சேர்த்து அடுப்பிலிட்டுக் கொதிக்க வைத்துப் பாகுபதம் வந்தவுடன் இறக்கி எடுத்துக் கொண்டேன்.

அளவு: 10-15 மில்லி லிட்டர், இருவேளை உணவிற்குப் பின்.

தீரும் நோய்கள் : பாண்டு, கைகால் எரிவு, வாந்தி.

ஆதாரம் : சித்த வைத்தியத் திரட்டு

REVIEW OF LITERATURE OF TRIAL DRUGS

கரிசாலை

Botanical Name : Eclipta Prostrata

Family : Asteraceae

Parts used : பூண்டு

வேறுபெயர் :

கரிசனாங்கண்ணி, கரிசாலை, கரியசாலை, கைகேசி, கைவீசி இலை, கையாந்தகரை, பிருங்கராஜம், கரிப்பான், கையான், தேகராஜம்.

சுவை - கைப்பு, தன்மை - வெப்பம், பிரிவு - கார்ப்பு.

செய்கை :

பித்தநீர்ப்பெருக்கி, உரமாக்கி, உடல்தேற்றி, ஈரல்தேற்றி.

சுத்தி முறை:

சமூலத்தை கழுவி மண் நீக்கி நிழலில் உலர்த்தி எடுத்துக் கொண்டேன்.

பொதுகுணம்:

“குரற்கம்மற் காமலை குட்டமொடு சோபை.

யுறற்பாண்டு பன்னோ யொழிய - நிரற்சொன்ன

மெய்யந் தகரையொத்த மீளினு நற்புலத்துக்

கையந் தகரையொத்தக் கால்”

- அகத்தியர் குணவாகடம்

இதனால் குரலுறுப்பு நோய், காமாலை, குட்டம், வீக்கம், பாண்டு, ஆகியவை போம். உடலிற் பொற்சாயலும், ஆளிக்ஞள்ள பலமும் உண்டாகும்.

Constituents:

Nicotine, Ecliptine, Triterphenoid and Flavanoid, Dithienylacetylene ester, Terthienyl Dimethyl Wedeloacetone-7.

கடுக்காய்

Botanical Name	:	Terminalia Chebula
Family	:	Combretaceae
Parts used	:	கடுக்காய் தோல்

வேறுபெயர்: அக்கோடம், அபையன், அமுதம், அம்மை, சேதகி, வரிக்காய்.

சுவை: முக்கிய சுவை துவர்ப்பு, அத்துடன் சிறிது இனிப்பு, புளிப்பு, கார்ப்பு, கைப்பு,

தன்மை - வெப்பம், பிரிவு - இனிப்பு

செய்கை:

மலமிளக்கி, பசித்தீத்தூண்டி, உரமாக்கி, உடற்றேற்றி

சுத்திமுறை:

கழுநீரில் ஊறப்போட்டு மஞ்சள் நீரைப் போக்கிக் கொட்டையை நீக்கி உலர்த்திக் கொண்டேன்.

பொதுகுணம்:

“கடுக்காயுந் தாயுந் கருதிலொன்றென் றாலும்
கடுக்காய்த் தாய்க்கதிகந் காண்நீ - கடுக்காய்நோய்
ஓட்டி யுடற்றேற்றும் உற்றவன்னை யோகவைகள்
ஊட்டியுடற் றேற்று முவந்து”

- அகத்தியர் குணவாகடம்

தாயோ அறுசுவை உணவை ஊட்டி உடலைத் தேற்றுவாள். கடுக்காயோ உடற்பிணிகளை ஓட்டி உடலைத் தேற்றும். ஆதலின் தாய் உணவை மாத்திரம் ஊட்டுந்தன்மை உடையவளாதலால், தாயினும் கடுக்காய் சிறந்தகெனக் கொள்க.

Constituents:

Iron, Copper, Glucose, Sorbitol, Fructose, Sucrose, Eighteen typical Amino acids, Succinic acid, Phosphoric acid, Tannins like Gallic acid, Chebulinic acid, Corilagin.

நெல்லிவற்றல்

Botanical Name : Phyllanthus emblica

Family : Euphorbiaceae

Parts used : வற்றல்

வேறுபெயர்: ஆமலகம், ஆம்பல், தாத்தாரி, கோரங்கம், மிறுதுபலா.

சுவை - புளிப்பு, துவர்ப்பு, இனிப்பு,

தன்மை - தட்பம்

பிரிவு - இனிப்பு

செய்கை:

துவர்ப்பி, குளிர்ச்சியுண்டாக்கி, சிறுநீர்பெருக்கி, மலமிளக்கி.

சுத்திமுறை:

பால் விட்டு வேகவைத்துக் கொட்டையை நீக்கி உலர்த்திக்

கொண்டேன.

பொதுகுணம்

“இல்லா மலக மிரண்டு மயின்றானே

யில்லா மலகமிருக்குமே - இல்லாமல்

வாழைக் கனியும் வடையு மிழுது முண்பான்

வாழைக் கனியுன் வைத்த வன்”

- தேரையர் யமக வெண்பா.

நெல்லியை கற்பமுறைப்படி தக்க பத்தியத்துடன் உபயோகித்தால், வல்லை, மகோதரம், பாண்டு, பெருவயிறு, மூலம், பெரும்பாடு, சோபை முதலிய பிணிகள் தீரும்.

Constituents:

Richest known natural source of Vitamin C, Tannin.

Uses:

Dried fruit is useful in haemorrhage, diarrhoea and dysentery with iron it is a valuable remedy in anemia, jaundice and dyspepsia.

தான்றிக்காய்

Botanical Name	:	Terminalia bellerica
Family	:	Combrefaceae
Parts used	:	தான்றிக்காய் தோல்

வேறுபெயர்: அக்கந்தம், அமுதம், அம்பலத்தி, ளரிகட்பலம், கலித்துருமம்.

சுவை - துவர்ப்பு

தன்மை - வெப்பம்

பிரிவு - இனிப்பு

செய்கை:

துவர்ப்பி, கோழையகற்றி, மலமிளக்கி, உரமாக்கி

சுத்திமுறை:

தாழைவிழுது சாற்றில் ஒரு சாமம் ஊற வைத்து விதையை நீக்கி இரவியிலுலர்த்திக் கொண்டேன.

பொதுகுணம்:

“ஆணின் பென் மேனிக் கழுகும் ஒளியுமிகும்

கோணிக் கொள் வாதபித்தக் கொள்கை போம் - தானிக்காய்

கொண்டவர்க்கு மேகமறும் கூறா அனற்றணியும்

கண்டவர்க்கு வாதம் போம் காண்”

- குணபாடம் மூலிகை

இது உடற்கு அழகையும், ஒளியையும், கொடுத்து முக்குற்றங்களையும் தன்னிலைப்படுத்தும்.

Constituents:

Iron, Copper, Glucose, Galactose, Fructose, Rhamnose, Mannitol, Tannins like, Gallic acid.

சர்க்கரை

Botanical Name	:	Saccharum Officinarum
Family	:	Gramineae
Parts used	:	கருப்பஞ்சாறு, சர்க்கரை, வேர்

வேறுபெயர்: புனற்பூசம், இக்கு, வேய்

சுவை - இனிப்பு

தன்மை - சீதம்

பிரிவு - இனிப்பு

செய்கை:

அழுகலகற்றி, உள்ளழலாற்றி

சுத்திமுறை:

அம்மியில் வைத்துக் கட்டியெல்லாம் நொறுங்கும்படி அரைத்து எடுத்துக் கொண்டேன்.

பொதுகுணம்:

“சீனிச்சர்க்கரைக்குத் தீராத வன்சுரமுங்
கூனிக்கும் வாதத்தின் கூட்டுறவும் - ஏனிற்கும்
வாந்தி யொடுகிருமி மாறாத விக்கலுமே
பேரந்திசையை விட்டுப் புரண்டு”

- அகத்தியர் குணவாகடம்

இதனால் வாதசுரம், வாதநோய், வாந்தி, நுண்புழு, விக்கல் நீங்கும்.

Constituents:

Juice contains saccharine matter (Cane Sugar) water, mucilage, resin, fat, albumin etc. guanine, co-oxalate.

மாதுளை

Botanical Name	:	Punica Granatum
Family	:	Lythraceae
Parts used	:	பழம்

வேறுபெயர்: தாடிமம், பீசுபுரம், மாதுளங்கம், மாதுளம், மாதுளுங்கம்.

சுவை - இனிப்பு, தன்மை - தட்பம், பிரிவு - இனிப்பு

செய்கை:

குளிர்ச்சியுண்டாக்கி, பசித்தீத்தூண்டி

பொதுகுணம்:

“வெடித்துவீழ் பழத்தை வாகங்கி மெல்லிய சீலை கட்டி
கடுக்கெனப் பிழிந்து கொண்டு கண்டசர்க் கரையுங்கூட்டிக்
குடித்திட வெடிப்பு மறையுங் குளிர்ந்திடும் அங்கமெல்லாம்
வடித்தநன் மொழியி னாளே மாதளம் பழத்தின்சாரே.”

- தேரையர் குணவாகடம்

Constituents:

Calcium, Magnesium, Phosphorus, Riboflavin, Vitamins, Oxalic acid, Iron, Sodium, Potassium, Copper, Sulphur, Chlorine, Thiamine, Nicotinic acid.

பன்னீர்

Botanical Name : Rosa centifolia

Family : Rosaceae

Parts used : பூ

வேறுபெயர்: குலாப்பூ, ரோஜாப்பூ, சிற்றாமரை

சுவை - இனிப்பு, துவர்ப்பு, தன்மை - சீதம், பிரிவு - இனிப்பு

செய்கை:

மலமிளக்கி, பசித்தீத்தூண்டி, குளிர்ச்சியுண்டாக்கி, உரமாக்கி.

பொதுகுணம்:

“பன்னீர் மிகக்குளிர்ச்சி பரளய் ககசன்னிக்
கிந்நீருண், ஏகும் இளைப்புமறுஞ் - சன்னிகளும்
வாதபித்த ஐயமொடு மானே வியாகுலமும்
பூதலம்விட் டேகும் புகல்”

- அகத்தியர் குணவாகடம்

இஃது குளிர்ச்சியைத் தரும், இளைப்பைப் போக்கும், வளி ஐயக் குற்றங்களைத் தன்னிலையடையச் செய்யும்.

Constituents:

Geraniol, Citronellol, Nerol, Linalool, Phenyl ethyl alcohol, Farnesol, Paraffin as Stearpoten, Eugenol, Methyl eugenol, Rose Oxide, Damascenone.

கற்கண்டு

Botanical Name : Saccharum officinarum
Family : Gramineae
Parts used : கற்கண்டு

வேறுபெயர் : புனற்பூசம், இக்கு, வேய்

சுவை - இனிப்பு

தன்மை - தட்பம்

பிரிவு - இனிப்பு

செய்கை:

அழுகலகற்றி, உள்ளழலாற்றி

பொதுகுணம்:

“ஈறின் தடிப்பு மிருமலும்பல் வந்திகளுஞ்
சீறுகப முட்டினமுஞ் சேராதே - தேறியநற்
சொற்கண் டளங்குயில்கள் சூழ மடவனமே
கற்கண் டெனவுரைக்குங் கால்.”

- அகத்தியர் குணவாகடம்

இதனால் பல்வரணை, இருமல், வாந்தி, ஐய அழல் தீரும்.

Constituents:

Saccharine matter, water, mucilage, resin, fat, albumin, guanine , co-oxalate.

தேன்

செய்கை:

உள்ளழலாற்றி, மலமிளக்கி, துவர்ப்பி, அழுகலகற்றி,
கோழையகற்றி, போஷணகாரி, பசித்தீத்தூண்டி, தூக்கமுண்டாக்கி

மலைத் தேனின்குணம்:

“ஐயிரும லீளைவிக்க லக்கிப்புண் வெப்புடல்நோய்
பைய வெழியும் பசியுமுறும் - வையகத்தி
வெண்ணுமிசை யாமருந்திற் கேற்ற வனுபான
நண்ணுமலைத் தேனென்றி னால்”.

- குணபாடம் சீவ வகுப்பு

மலைத்தேனினால், கபகாசம், சுவாசம், விக்கல், கண்விரணம், சுரம்,
தேகக்கடுப்பு முதலிய பிணிகள் நீங்கும். பசியும் தொனியும் உண்டாகும். இது
மருந்துகளுக்கு நற்றுணை மருந்தாகும்.

Constituents:

Iron, Copper, Manganese, Zinc, Calcium, Phosphorus, Sodium,
Ascorbic acid, Niacin, Thiamine, Riboflavin, Pantothenic acid.

Uses:

In moderate doses, has beneficial effect on digestion and appetite.

Honey is used in curing rickets, marasmus, malnutrition and scurvy

It is a useful laxative for children.

Babies generally fall asleep after taking honey.

- Indian materia medica -II

ANNEXURE-II

BIO – CHEMICAL ANALYSIS

BIO – CHEMICAL ANALYSIS OF BRINGARAJA CHOORANAM

Preparation of the extract:

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

Qualitative Analysis:

S. No.	Experiment	Observation	Inference
1.	<u>Test for calcium</u> 2ml of the above prepared extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxalate solution.	No white precipitate is formed.	Absence of calcium.
2.	<u>Test for sulphate:</u> 2ml of the extract is added to 5% barium chloride solution.	No white precipitate is formed.	Absence of sulphate.
3.	<u>Test for chloride</u> The extract is treated with silver nitrate solution.	No white precipitate is formed.	Indicates the Absence of chloride.
4.	<u>Test for carbonate</u> The substance is treated with concentrated Hcl.	No brisk effervescence is formed.	Absence of carbonate.

5.	<u>Test for Starch</u> The extract is added with weak iodine solution.	No blue colour is formed	Absence of starch.
6.	<u>Test for iron</u> <u>Ferric</u> The extract is treated with concentrated glacial acetic acid and potassium ferro cyanide.	No blue colour is formed.	Absence of ferric iron.
7.	<u>Test of iron :</u> <u>Ferrous:</u> The extract is treated with concentrated Nitric acid and ammonium thio cynate.	Blood red colour is formed.	Indicates the presence of ferrous iron.
8.	<u>Test for phosphate</u> The extract is treated with ammonium molybdate and concentrated nitric acid.	No yellow precipitate is formed.	Absence of phosphate.
9.	<u>Test for albumin</u> The extract is treated with Esbach's reagent.	No yellow precipitate is formed.	Absence of albumin.
10.	<u>Test for Tannic acid</u> The extract is treated with ferric chloride reagent.	Blue black precipitate is formed.	Indicates the Presence of Tannic acid.
11.	<u>Test for unsaturation</u> Potassium permanganate solution is added to the extract.	It gets decolourised.	Indicates the Presence of unsaturated compound.

12.	<p><u>Test for the reducing sugar</u></p> <p>5ml of benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.</p>	Colour change occurs.	<p>Indicates the Presence of reducing sugar.</p>
13.	<p><u>Test for amino acid:</u></p> <p>One or two drops of the extract is placed on a filter paper and dried it well. After drying, 1% ninhydrin is sprayed over the same and dried it well.</p>	Violet colour is formed.	<p>Indicates the Presence of amino acid.</p>

Inference

The given sample of “**BRINGARAJA CHOORANAM**” contains Ferrous iron, Tannic acid, Reducing Sugar, Amino acid, and unsaturated compound.

BIO – CHEMICAL ANALYSIS

BIO – CHEMICAL ANALYSIS OF MADHULAI MANAPPAGU

Preparation of the extract

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

Qualitative Analysis

S. No.	Experiment	Observation	Inference
1.	<u>Test for calcium</u> 2ml of the above prepared extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxalate solution.	White precipitate is formed.	Indicates the Presence of calcium.
2.	<u>Test for sulphate:</u> 2ml of the extract is added to 5% barium chloride solution.	No white precipitate is formed.	Absence of sulphate.
3.	<u>Test for chloride</u> The extract is treated with silver nitrate solution.	A white precipitate is formed.	Indicates the Presence of chloride.
4.	<u>Test for carbonate</u> The substance is treated with concentrated Hcl.	No brisk effervescence is formed.	Absence of carbonate.

5.	<u>Test for Starch</u> The extract is added with weak iodine solution.	No blue colour is formed	Absence of starch.
6.	<u>Test for iron</u> <u>Ferric</u> The extract is treated with concentrated glacial acetic acid and potassium ferro cyanide.	No blue colour is formed.	Absence of ferric iron.
7.	<u>Test of iron :</u> <u>Ferrous:</u> The extract is treated with concentrated Nitric acid and ammonium thyo cynate.	Blood red colour is formed.	Indicates the trace amount of ferrous iron is present.
8.	<u>Test for phosphate</u> The extract is treated with ammonium molybdate and concentrated nitric acid.	No yellow precipitate is formed.	Absence of phosphate.
9.	<u>Test for albumin</u> The extract is treated with Esbach's reagent.	No yellow precipitate is formed.	Absence of albumin.
10.	<u>Test for Tannic acid</u> The extract is treated with ferric chloride reagent.	No blue black precipitate is formed.	Absence of Tannic acid.

11.	<u>Test for unsaturation</u> Potassium permanganate solution is added to the extract.	It gets decolourised.	Indicates the Presence of unsaturated compound.
12.	<u>Test for the reducing sugar</u> 5ml of benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	colour change occurs.	Indicates the Presence of reducing sugar.
13.	<u>Test for amino acid:</u> One or two drops of the extract is placed on a filter paper and dried it well. After drying, 1% ninhydrin is sprayed over the same and dried it well.	No Violet colour is formed.	Absence of amino acid.

Inference

The given sample of “**MADHULAI MANAPPAGU**” contains chloride, calcium, ferrous iron and unsaturated compound, reducing Sugar

ANNEXURE- III

PHARMACOLOGICAL ANALYSIS OF

BRINGARAJA CHOORANAM

STUDY ON HAEMATINIC EFFECT

Preparation of the trial drug:

Varieties of preparations in Siddha system of medicine are well known for its haematinic effects of which Bringaraja Chooranam is one of the best. To prove the efficacy of this medicine, an attempt was made to study its effect using “Albino rats”. For this purpose, rats were made anaemic by the following procedure.

Artificially Induced Iron deficiency:

The albino rats taken for this experiment were kept in aluminium cages and provided with drinking water and milk, free from iron. The administration of the iron preparation under investigation was started, when the haemoglobin level fell to nearly 6.0gm/100ml. At the beginning of the experiment 40% was determined.

Study on Rats:

Nine albino rats were first divided into three equal groups, with three rats in each group. The first group received water. The second group received honey. The third group received the test drug at a dose of 100mg of Bringaraja Chooranam. All the above procedures were continued for four weeks at the rate of once in a day. The Haemoglobin levels of rats were measured after 1 week, 2 weeks, 3 weeks and 4 weeks. The results observed are tabulated in the following chart.

S.No	Drug	Dose	Before Drug administration				After drug administration			
			Initial gm/dl	I week gm/dl	II week gm/dl	III week gm/dl	IV week gm/dl	V Week gm/dl		
1	Water	2ml	5.8	5.8	5.7	5.5	5.3	5.1	5.2	
		2ml	5.9	5.9	5.8	5.5	5.2	5.0		
		2ml	6.3	6.3	6.2	6.0	5.7	5.3		
		2ml	6.5	6.5	6.2	6.0	5.8	5.5		
		2ml	6.6	6.6	6.3	6.0	5.8	5.5		
		2ml	6.2	6.2	6.0	5.7	5.3	5.0		
			6.3	6.2	6.0	5.7	5.5	5.2		
2.	Bringaraja Chooranam	100mg	6.0	6.0	6.5	7.0	7.7	8.5	8.9	
		100mg	6.2	6.3	6.7	7.2	8.0	9.0		
		100mg	6.0	6.1	6.7	7.2	8.0	8.7		
		100mg	5.8	6.0	6.5	7.1	7.8	8.8		
		100mg	6.0	6.0	6.4	7.0	7.5	8.8		
		100mg	6.5	6.7	7.3	8.0	8.5	9.5		
			6.0	6.1	6.6	7.2	7.9	8.9		

Discussion:

A remarkable raise of Hb above 8.9gms/dl is seen in the group treated with trial drug. From these studies it is clear that the drug Bringaraja Chooranam has significant haematinic action.

PHARMACOLOGICAL ANALYSIS OF
BRINGARAJA CHOORANAM
STUDY ON LAXATIVE ACTION

Drug: Bringaraja Chooranam

Method: Charcoal meal method

Procedure:

Six albino rats of uniform weight and size selected and divided into 2 groups each containing 3 rats. All the rats were fasted for 48 hours before starting the experiment. The first group was treated as control group. The control group received distilled water (1ml) orally. The second group was fed by test drug Bringaraja Chooranam (100mg).

One hour later each animal of the groups was given 0.5ml of an aqueous suspension of 10% charcoal meal, the animals were sacrificed with chloroform. The small intestine from pylorus up to caecum was removed and the distance traveled by charcoal was measured by measuring the distance from pylorus. At the end of the experiment, the distance traveled by the carbon particle gives the extent of laxative action if any comparing with that of control.

S. No.	Name of Drugs / Groups	Dose / 100 gram body weight	Total Length of the intestine	Charcoal meal traveled up to	% of charcoal Travelled	Remarks
1	Control water	2 ml	65cm	65cm	100	Good
2	Bringaraja Chooranam	100mg	65cm	61 cm	93.8	Good

Result:

The trial drug Bringaraja Chooranam shows significant laxative action.

PHARMACOLOGICAL ANALYSIS OF MADHULAI MANAPPAGU STUDY ON HAEMATINIC EFFECT

Preparation of the trial drug:

Variety of preparations in Siddha system of medicine is well known for its haematinic effects of which Madhulai Manappagu is one of the best. To prove the efficacy of this medicine, an attempt was made to study its effect using “Albino rats”. For this purpose, rats were made anaemic by the following procedure.

Artificially Induced Iron deficiency:

The albino rats taken for this experiment were kept in aluminium cages and provided with drinking water and milk, free from iron. The administration of the iron preparation under investigation was started, when the haemoglobin level fell to nearly 6.0gm/100ml. At the beginning of the experiment 40% was determined.

Study on Rats:

Nine albino rats were first divided into three equal groups, with three rats in each group. The first group received water. The second group received honey. The third group received the test drug at a dose of 100mg/1ml of Madhulai Manappagu. All the above procedures were continued for four weeks at the rate of once in a day. The Haemoglobin levels of rats were measured after 1 week, 2 weeks, 3 weeks and 4 weeks. The results observed are tabulated in the following chart.

S. No.	Drug	Dose	Before Drug administration				After drug administration			
			Initial gm/dl	I week gm/dl	II week gm/dl	III week gm/dl	IV week gm/dl	V Week gm/dl		
1	Water	2ml	5.8	5.8	5.7	5.5	5.3	5.1	5.2	
		2ml	5.9	5.9	5.8	5.5	5.2	5.0		
		2ml	6.3	6.3	6.2	6.0	5.7	5.3		
		2ml	6.5	6.5	6.2	6.0	5.8	5.5		
		2ml	6.6	6.6	6.3	6.0	5.8	5.5		
		2ml	6.2	6.2	6.0	5.7	5.3	5.0		
			6.3	6.2	6.0	5.7	5.5	5.2		
2	Madhulai Manappagu	10ml	6.0	6.2	6.7	7.5	8.3	9.0	9.2	
		10ml	6.2	6.5	7.0	7.5	8.5	9.2		
		10ml	6.2	6.5	7.0	7.7	8.2	9.5		
		10ml	6.3	6.5	7.0	7.7	8.5	9.0		
		10ml	6.4	6.6	7.0	7.5	8.2	9.0		
		10ml	6.0	6.3	7.0	7.8	8.7	9.5		
			6.1	6.4	6.9	7.6	8.4	9.2		

Discussion:

A remarkable raise of Hb above 9.2gms/dl is seen in the group treated with trial drug. From these studies it is clear that the drug Madhulai Manappagu has significant haematinic action.

ANNEXURE - IV

GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL

POST GRADUATE RESEARCH CENTRE

BRANCH IV – KUZHANTHAI MARUTHUVAM

PALAYAMKOTTAI – 627 002.

CASE SHEET PROFORMA – PANDU NOI

Name of the Medical unit:	Nationality	:
I.P. No.	Religion	:
Bed. No.	Date of Admission	:
Name	Date of Discharge	:
Age/ Sex	Duration of treatment	:
Occupation (Parents)	Diagnosis	:
Income (Parents)	Medical Officer	:
Informant		:
Address		:
Complaints and duration		:
History of present illness		:
History of past illness		:
Antenatal History		:
Birth and Neonatal History		:
Dietetic and Nutritional History		:
Developmental History		:
Family History		:
Social History		:

Immunization History :

General Examination

1. Consciousness :

2. Decubitus :

3. Anemia :

4. Jaundice :

5. Cyanosis :

6. Clubbing :

7. Pedal oedema :

8. Lymphadenopathy :

9. Nourishment :

10. Skin changes :

Vital Signs

1. Pulse

- Rate :

- Rhythm :

- Volume :

- Character :

2. B.P. :

3. R.R. :

4. Temperature :

Anthropometry

1. Wt – Weight :

2. Ht – Height :

3. Mid arm circumference :

4. Head circumference :
5. Chest :
6. Skin fold thickness :

Siddha System - Clinical Examination:

Poripulangal

- Mei :
Vai :
Khan :
Mookku :
Sevi :

Kanmendriyam – Kanmavidayam

- Kai :
Kaal :
Vaai :
Eruvaai :
Karuvaai :

Gunam

- Sathuvam :
Rajo :
Thamo :

Nilam

- Kurinchi :
Mullai :
Marutham :
Neithal :

Palai :

Paruva Kaalam

Kar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Muthuvenil :

Uthayam – Athakayam

Puyam :

Sayam :

Kaal :

Paatham :

Pira Uruppugalin Nilai

Moolai :

Iruthayam :

Puppusam :

Kalleeral :

Manneeral :

Kudal :

Siruneeragam :

Kuri :

Mummalam

Viyarvai :

Malam :

Moothiram :

Mukkutra Udal

Vaatha thegi :

Piththa thegi :

Kabha thegi :

Kalappu thegi :

Udal Kattugal

Saaram :

Senneer :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam/Suronitham :

Envagai Thervugal

Naadi :

Sparisam :

Naa :

Niram :

Mozhi :

Vizhi :

Malam :

Moothiram :

Vatham

Piranan :

Abaanan :
Uthanan :
Viyaanan :
Samaanan :
Naagan :
Koorman :
Kirugaran :
Devathathan :
Dhananjeyan :

Pitham

Analam :
Ranjagam :
Sathagam :
Alosagam :
Pirasagam :

Kabam

Avalambagam :
Kiletham :
Pothagam :
Tharpagam :
Santhigam :

Neerkuri

Niram :
Manam :
Nurai :
Edai :

Enjal :

Neikuri :

Malakuri

Niram :

Nurai :

Elagal :

Erugal :

Modern Aspects

Systemic examination

Cardiovascular system:

1. Inspection:
2. Palpation:
3. Percussion:
4. Auscultation:

Examination of other systems

Respiratory system:

Abdomen:

Central nervous system:

Excretory system:

Lab Investigations

1. Blood

TC :

DC :

Hb :

ESR :

PCV :
MCV :
MCH :
MCHC :
Total RBC count :
Peripheral blood smear :

2. Urine

Albumin :
Sugar :
Deposits :
Bile salt :
Bile pigments :

3. Motion

Ova :
Cyst :
Occult blood :

DIFFERENTIAL DIAGNOSIS :

PROGNOSIS :

MARUTHUVAMURAI :

ADVICE :

DAILY PROGRESS :

Date	Symptoms	Medicine

Place :

Date :

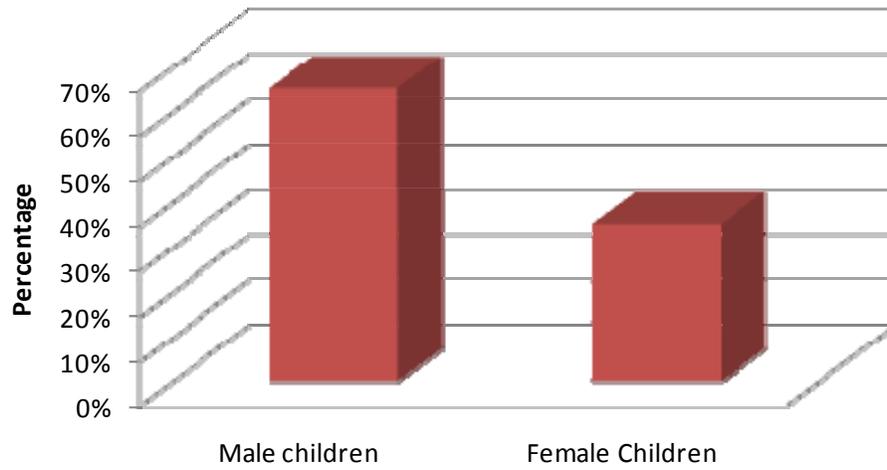
Signature of the Medical Officer,

BIBLIOGRAPHY

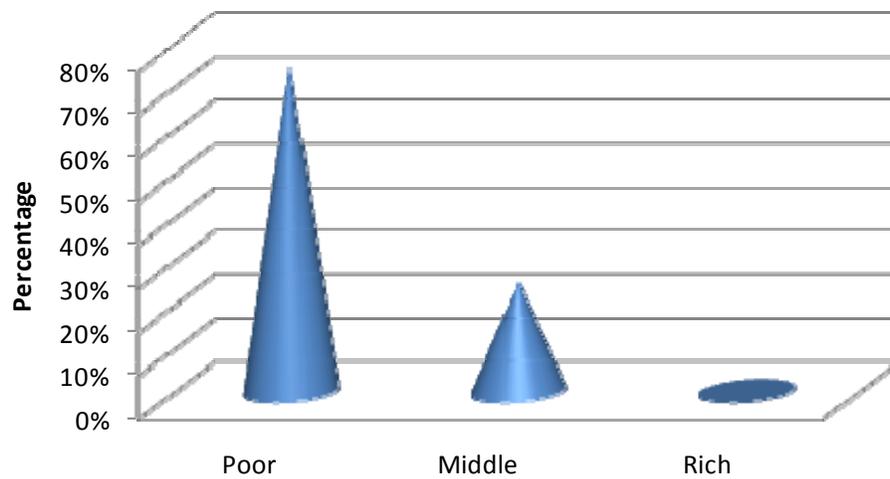
- Balavagadam (2nd edition).
- Siddha Maruthuvam – K.N.Kuppusami Mudhaliar.
- Siddha Maruthuva Noinaadai Noimuthal naadai (Part I & II) –
M. Shanmugavelu.
- Yugivaidya Chinthamani.
- Agasthiar Gunavaagadam.
- Agasthiar Vaidhya Pillai Tamil.
- Thanvanthiri Vaithyam 1st part.
- Sarabendrar Vaidhya Muraigal, Karpini Balaroga Shikitchai.
- Jeevarakshamirtham.
- Theraiyar Vaagadam.
- Agasthiyar Paripooranam.
- Roganiranayasaram.
- Anubava Vaithya Devaragasyam.
- Ashtanga Hridaya (volume-I).
- Gunapadam Mooligai Vahuppu – K.S. Murugesu Mudaliar.
- Gunapadam Thathu Jeeva Vahuppu – Dr.R.Thiyagarajan.
- Siddha Maruthuvanga Churukkam (2nd edition) –
K.S.Uthamarayan.

- Thotrakirama Aaraichiyum Siddha Maruthuva Varalaarum – K.S. Uthamarayan.
- T.V. Sambasivam Pillai Dictionary.
- Essential Pediatrics (5th edition) – O.P. Ghai.
- Text book of Pediatrics (15th edition) – Nelson.
- I.AP Test book of Pediatrics (2nd edition).
- Achar's Text book of Pediatrics (3rd edition).
- The Short text book of Pediatrics – Suraj Gupta.
- Davidson's Principles and practice of Medicine.
- Practical Medicine (12th edition), J.Pradip Mehta.
- Pathologic Basis of disease (5th edition) –Robbins.
- Text book of pathology (4th edition) – Harsh Mohan.
- Text book of Medical Physiology – C. Guyton and Hall.
- Essentials of Medical Physiology (2nd edition) – Sembulingam & Prema Sembulingam.
- Clinical Hematology (8th edition) – Lear Febiger.
- Fundamentals of Biochemistry – Ambika Shanmugam.
- Wealth of India.
- Indian Material Medica (3rd edition) – K.M. Nadkarni.

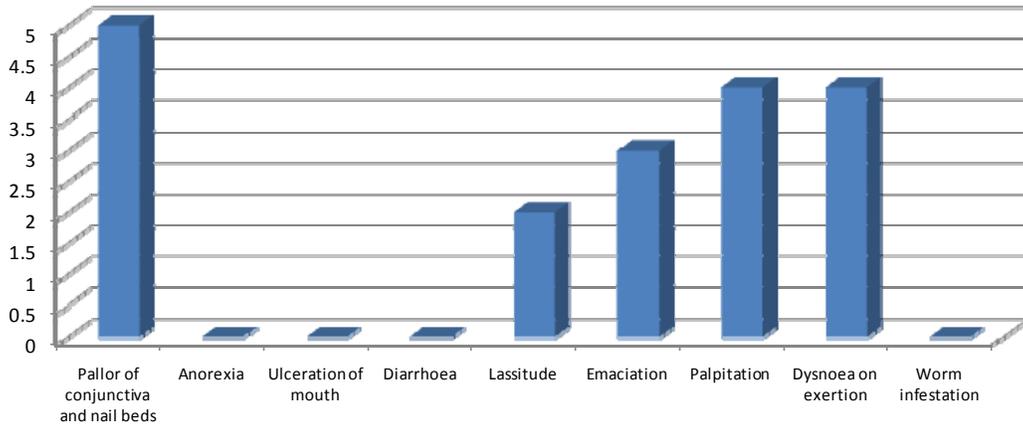
Sex Distribution



Socio-Economic status of the patient



Reference to signs and symptoms



Among the 20 cases studied the results were observed as follows

