NATIONAL INSTITUTE OF SIDDHA

Tambaram Sanatorium, Chennai – 600 047

AFFILIATED TO THE

TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI – 600 032

A STUDY ON

PANDU NOI

(DISSERTATION SUBJECT)

For the partial fulfillment of

Requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)

BRANCH IV – DEPARTMENT OF KUZHANDHAI MARUTHUVAM

APRIL – 2013

ACKNOWLEDGEMENT

First of all, I have to thank Almighty to do the dissertation work with complete satisfaction.

I express my sincere thanks to VICE - CHANCELLOR, The Tamilnadu Dr.M.G.R. Medical University, Chennai-32.

It is with immense gratitude that I acknowledge our Director PROF. DR.K.MANICKAVASAKAM,M.D.(S), Head of the Department Incharge Department of Kuzhanthai Maruthuvam National Institute of Siddha, Chennai, for granting permission to undertake a study in this dissertation topic and patronizing the work by providing all the necessary facilities.

I express my sincere thanks to Prof. Dr. R. S. Ramaswamy, M.D. (S) Former Hospital Superintendent, National Institute of Siddha, Chennai, for his moral support provided me during this study

It gives me great pleasure in acknowledging the support and help of Prof. Dr. G. Ganapathy.M.D. (S), Former Head of the department, Kuzhandhai maruthuvam, National Institute of Siddha, Chennai – 47, who has attitude and he continually and convincingly conveyed a spirit of adventure in regard to dissertation.

I express my sincere thanks to Dr. M. Meenakshi Sundaram M.D. (S) Associate Professor, Department of Kuzhandhai Maruthuvam National Institute of Siddha, Chennai, for his moral support provided me during this study

I express my heartful thanks to Dr. K. Suresh, M.D. (S), Dr. P. Arulmozhi, M.D.(S), Dr. A. M. Amala Hazel, M.D (S), Dr. K. Vennila, M.D. (S), Lecturers, Department of Kuzhandhai Maruthuvam, National Institute of Siddha, for stimulating suggestions and encouragement helped me in all the time and valuable guidance given to me for this successful completion of work.

My special thanks to Mr. M. Subramanian, Senior Research Officer, (Statistics) National Institute of Siddha for his valuable stastical guidance.

I express my sincere thanks to Dr. D. Aravindan, Asst Professor, Medicinal Botany, NIS, Chennai-47, for helping the identification of raw drug.

I express my sincere thanks to Dr.A.Muthuvel, Asst Professor, Bio Chemistry NIS, Chennai – 47, for helping Bio Chemical Analysis, Dr.V.Suba, Asst. Professor, Pharmacology for Pharmacological study.

Last but not least I express a sense of gratitude and love to my family and my brother for their manual support, strength, and help for everything.

I take this opportunity to express my thanks to my friends and colleagues for their help and co-operation during the entire course of my work.

CONTENTS

INTRODUCTION

The word Siddha comes from the word **Siddhi** which means an object to attain perfection or heaven. The Siddha system is of Dravidian origin and has its entire literature in Tamil language. This system of medicine was developed by the 18 ancient saints who were known by the name "**Siddhars**" whose life goal was to attain salvation. The Siddha system of medicine uses a fascinating combination of herbs, minerals, metals and to promote good health and longevity.

"அண்டத்தில் உள்ளதே பிண்டம்
பிண்டத்தில் உள்ளதே அண்டம்
அண்டமும் பிண்டமும் ஒன்றே
அறிந்து தான் பார்க்கும்போதே"

-சட்டமுனி ஞானம்.

Siddha science considers nature and man as essentially one. "**NATURE IS MAN AND MAN IS NATURE**". Man is said to be the microcosm and Universe is the macrocosm because what exists in the world exists in man. Man is nothing but a miniature world containing the five elements of the various principles which constitute the, vegetables and the animal kingdom.

"தலங்காட்டியிருந்தச் சடமான வைம்பூதம்
நிலங்காட்டி நீர்காட்டி நின்றிருந் தீகாட்டி
வலங்காட்டி வாயுவால் வளர்ந்தே யிருந்த
குலங்காட்டி வானிற் குடியாயிருந்ததே
இருந்திடுமிவ் வைந்தாலெடுத்த சடமிது"

திருமூலர் நாடி

"நிலந் தீ நீர் வளி விசும்போடைந்தும்
கலந்த மயக்கம் உலகமாதலின்"

தொல்காப்பியம்.

According to Siddha medical science, the Universe originally consisted of atoms which contributed to the five basic elements, viz., earth, water, fire, air and sky which correspond to the organs of the human body and they were the fundamentals of all the corporeal things in the world.

“உடம்பார் அழியின் உயிரார் அழிவர்
திடம்பட மெய்ஞானம் சேரவும் மாட்டார்
உடம்பை வளர்க்கும் உபாயம் அறிந்தே
உடம்பை வளர்த்தேன் உயிர்வளர்த் தேனே “

திருமந்திரம்.

It worked on the basic concept that a healthy soul can only be developed through a healthy body. According to Siddha system, the human body is composed of 96 principles,(thaththuvas) 72,000 blood vessels(naadikal) and 1,300 nerves (Nerves). Besides these, there are 10 Nadi (main arteries),10 Vayu (vital pranas) and 14 Vegangal (natural functions).

The food is considered to be basic building material of human body, which gets processed into blood, tissues and wastes. The equilibrium of blood, body tissues and waste products is considered as health and its disturbance or imbalance leads to disease or pathologic state. So the siddhars developed methods and medication that are believed to strengthen their physical body and thereby their souls. The system not only deals with medicine, but with spirituality, righteous way of living, rejuvenation and its main aim, Attainment of Perfection.

The main benefits of siddha system includes,

It treats the disease to its roots, without leaving any traces for it to reappear.

The medicines are made from natural materials.

It is considerably a cheaper form of treatment.

The treatment has to be individualized with far less chances of committing mistakes in diagnosis of treatment.

Pandu Noi (Iron deficiency anemia (IDA)) is the single most common disorder affecting mankind. It is estimated that more than **2 billion** people suffer from IDA worldwide. It is seen in all parts of the world - developed as well as developing countries. In India nearly **70%** of children are estimated to be anemic patients due to iron deficiency. During the past two decades great advances have been made in the knowledge of iron deficiency anemia and the relation of dietary deficiencies in the formation of blood.

There are many types of anemia. Iron deficiency anemia is a decrease in the number of red blood cells in the blood due to lack of iron. The risk of iron deficiency anemia is high during the second year of life because of increased iron requirements related to rapid growth. Premature and low birth weight infants and infants with history of prolonged stay in the neonatal unit are at particularly high risk of developing iron deficiency anemia before 1 year of age.

Iron deficiency anemia affects neurological development by decreasing learning ability, altering motor functions, and permanently reducing the number of dopamine receptors and serotonin levels. Iron deficiency during development can lead to reduced myelination of the spinal cord, as well as a change in myelin composition.

Every age group is vulnerable to iron-deficiency anaemia. In children, anaemia can cause a 5-10 point deficiency in IQ and hamper growth and language development.

"Anaemia is causing red alert for Indian women and children and we need to address it. In India, besides focussing on prevention, The author need to talk of the disease Pandu noi (IDA) and the treatment of rampant anaemia to address health, education and economic consequences due to anaemia,"

So this dissertation focuses the treatment for pandu noi (Iron deficiency Anaemia (IDA)) that is, a safe and simple drug **TIRUDHARATCHATHA CHOORANAM(TC)** by Agasthiyar vaithiya rathinachurukkam, which gone through a thorough literature review of siddha texts books about Biochemical, Pharmacological and Preclinical study, Clinical trials were carried out and the result obtained were discussed in the end.

AIM AND OBJECTIVES

AIM:

The aim of this study on pandu noi is to ensure a new approach in diagnosis for the disease and find out a safe and simple drug. It is essential to find out a safe and simple drug to over come Pandu noi. The drug should be easily available, economic, easily administered and also easily palatable.

OBJECTIVES :

1. To collect various school of thoughts regarding pandu noi mentioned in various siddha literatures.
2. To know the extend of correlation of etiology, signs and symptoms of pandu noi with anaemia.
3. To analyse and review the disease pandu on the basis of its affection on various organ systems.
4. To utilize the diagnostic methods mentioned by siddhars.
5. To correlate the relation between pandu noi and seasonal variations.
6. To study the age-sex ratio, socio economic status for the disease.
7. To study about the predisposing factors contributing to this disease.
8. To conduct a pilot clinical trial with **TIRUDHARATCHATHA CHOORANAM(TC)**
9. To study pharmacological and Bio-chemical analysis of the trial drug.
10. To utilize the modern parameters of investigation for the confirmation of the disease and to follow the prognosis.
11. To make an awareness among the parents about the prevention of the disease in children.

REVIEW OF LITERATURE

SIDDHA ASPECTS

Siddha literatures deal with classification of diseases mainly by mukkutra theory that is Vaatham, Pitham and Kabam.

Disease is defined as cause and effect due to the alterations made by food and activities performed in Sookshma and Sthoola bodies.

'உயிர் தாது, உடல் தாது ஆகியவற்றில் உணவாதி செயல்களால் ஏற்படும் மாற்றங்களின் காரண காரியமே நோய் 'எனப்படும்.

So these texts provide us with a line of treatment of both Sookshma and Sthoola bodies.

Pandu noi is caused by derangement of Pitham. Hence the basic details regarding Pitham is briefly explained before going into the study about Pandu noi.

Mukkutra Theory of Pitham :

Pitham (Azhal) is one of the three vital phenomena (Vaatham, Pitham, Kabam). Among the panchaboodhas, it is formed by the Theyu bootham. In healthy individuals, the existance of the three humours are found in the ratio of 1 : ½ : ¼ respectively. This is explained as

"மெய்யளவு வாதமொன்று மேல் பித்தமோரரையம்
ஐயங் காலென்றே அறி"

கண்ணுசாமியம்

This ratio is altered when there is disturbance to Pitha dhosham, which leads to alteration of Pitham leading to Pitha diseases.

Location of Pitham in the Body

- Pingalai
- Praanavayu
- Neerpai
- Moolakkini
- Irudhayam
- Thalai
- Kopuzh
- Undhi
- Iraippai

- Viyarvai
- Naavil oorukindra neer
- Senner
- Saaram
- Kan
- Thol

More over,

As per **Thirumoolar's** thought,

"பிரிந்திடும் பித்தம் பேராஞ்சலத்தினில்"

As per **Yugimuni's** thought

"போமென்ற பித்தத்துக்கிருப்பிடமே கேளாய்
பேரான கண்டத்தின் கீழதாகும்."

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

Own qualities of Pitham – 6			**Opposite qualities of Pitham – 6**		
Hot	-	அக்கினி	Cold	-	குளிர்ச்சி
Acidic	-	புளிப்பு	Sweet	-	இனிப்பு
Mobile	-	ஊருந்தன்மை	Immobile	-	நிலைத்திருத்தல்
Liquid	-	சலரூபம்	Solid	-	கெட்டி
Acute	-	குரூரம்	Mild or Harmless	-	சாந்தம்
Pungent	-	காரம்	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.

Some illustrations of Pitham:

Gross body	- Chest and abdomen
Systems	- Digestive, circulatory
Functions	- Vital
Gunaas	- Rajasic
Cell	- Protoplasm
Humours	- Gastric Juice
Excretions	- Perspiration, Urine
Expulsion of dhosha	- Watery or yellowish discharge
Feeling of dhosha	- Heat
Classification	- Inflammation
Causes	- Low vitality
Treatment	- Stimulation
Nature	- Sun
Shape	- Liquid
Profession	- Police

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.

Day 1	-	Saaram (Chyle)
Day 2	-	Senneer (Blood)
Day 3	-	Oon (Muscle)
Day 4	-	Kozhuppu (Fat)
Day 5	-	Enbu (Bone)
Day 6	-	Moolai (Bone marrow)
Day 7	-	Sukkilam (Sperm)/ Suronitham (Ovum)

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 followed by affection of other thathus.

In Pandu noi, saaram and senneer thathu are mainly affected. Saaram (Rasam) is affected and is noticed by the symptoms like excessive thirst, dryness of skin, tongue, throat and loss of body weight.

Senneer is affected and is noted by the symptoms like pallor of the skin, excessive intake of foodstuffs having sour and acrid taste and results in general debility.

Physiological aspects of Pitham :

Our body is made up of seven udal thathus namely saaram, senneer, oon, kozhuppu, enbu, moolai, sukkilam / suronitham. These seven thathus constitute the body in normal condition. Senneer has the characteristics of pitham 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 form of the body steady and gives vigour and stimulation. Pitham represents gastric juice, bile, energy, heat, inflammation, anger, irritation etc. Physiological representation and types of pitham in the body are given below.

Relationship of pitham with taste :

Salt	-	Water + Fire
Sour	-	Earth + Fire
Pungent	-	Air + Fire

Salt, sour and pungent increases pitham since they are formed by fire (heat) . So they Possess Veppa Veeriyam.

"புளிதுவர் விஞ்சுங்கறி யார்பூரிக்கும் வாதம்
ஒளியுவர் கைப்பேறில் பித்துச்சீறும் கிளிமொழியே
கார்ப்பினிப்பு 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	-	Sky + Air

"பித்தமதி கரிப்பின் பேசும் பரிகாரம்
சுத்தத் துவரோடு சொல்லினிப்புச் சத்தாகும்
கைப்புச் சுவையே கருதுவதன் வீறு
எய்ப்பபடையு மென்றுரைத்தா ரிங்கு. "

- கண்ணுசாமியம்.

Aggravation of pitham in daily routine

Pitham in raised at the time of 10 a.m to 2 p.m and 10 p.m to 2 a.m

"காலையில் வாதநாடி கடிகையில் பத்தாகும்
பாலையில் பித்தநாடி பகருச்சி பத்தாகும்
மாலையாம் சேத்தும நாடி மதிப்புடன் பத்தாகும்
வாலையா மனோன்மணிக்கு வகுத்துமே தொகுத்ததாமே. "

வைத்திய சார சங்கிரகம்.

Aggravation of pitham in week days

If pitha gets aggravated at morning hours of

Sunday

Tuesday

Saturday and

Krishna patcham Thursday,

the vigour and vitality of body is maintained.

Physiologically the types of alterations of Pitham are,

Thannilai Valarchi

Definition

Provoked kutram in its own locations is called Thannilai Valarchi.

Limitations

Hatefulness of the things causing Thannilai Valarchi and likeliness of the things having opposite properties.

Duration

Pitham gets Thannilai Valarchi during Kaarkaalam (Avani – Purattasi)

Vetrunilai Valarchi

Definition

Provoked kutram to other locations is called vetrunilai valarchi.

Limitations

Signs and symptoms of the affected kutram and the pathological conditions of the udal thathukkal give the detail of limitations.

Duration

Pitham gets vetrunilai valarchi during Koothirkaalam (Ayppasi, Kaarthigai)

Thannilai Adaithal

Provoked kutram neutralizing its own property is called Thannilai adaithal.

Duration

Provoked pitham neutralizes during Munpani kaalam (Margazhi, Thai)

Dietic Factors which alter Pitham

1. When cold foods are mixed with pitham, it gets Thannilai Valarchi

2. When hot foods are mixed with pitham, pitham gets vetrunilai valarchi.

3. When cold and indigestible foods are taken with pitham, it neutralizes its own property i.e. returns to healthy conditions.

This is illustrated by

"மந்திரிப் பண்புடன் குளிர்ச்சி தரும்
வஸ்த்துக்கள் கொண்டிட லழலிடத்தே
வந்திடும் வன்பிணி அவைதனிய
மற்றிடத்தும் நலியுற் றிடுமே
மந்தமுந் தன்மையு மிணைந் திடிலா
மற்றொரு நோயில்லை மனிதருக்கே
இந்திள நெற்றியி னெழிலணங்கே
இந்திள சித்தரின் மொழியிதுவே. "

சித்த மருத்துவாங்கச் சுருக்கம்.

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.

"தேகத்தின் இரத்தத்தில் உள்ள சிவந்த அணுக்கள் குறைந்து தேகம் வெளுத்துவிடுவதற்கு பாண்டு நோய் என்று பெயர். "

- அகத்தியர் வைத்திய பிள்ளைத்தமிழ்

The great Siddhar Agathiyar defined pandu in the following verses.

"கழிவாகுந்த தேகமப்பா காணத்தச வத்தாய்
வற்றிவிடு மன்னவாசல் கேட்கில்
பழிகாரர் முகத்தில் முழியார் போலே பாண்டமெல்லாம்
வெளுக்கடித்தது இரத்தம். "

அகத்தியர் வைத்திய காவியம்

“தேகத்தில் இரத்தம் வற்றித்

தீங்கான விந்த நோய் காணுமப்பா. ”

அகத்தியர் குணவாகடம்.

“உடல் பசிய நிறமாய் பளபளத்து, முகங்குளுப்பையிட்டு இருவிழியும் நீல நிறமாய், இடுப்புக் குத்தோடு, நீரும் பொன்னிறமாய் இறங்குவதே பாண்டின் குணமாகும். ”

உயிர் காக்கும் சித்த மருத்துவம்.

According to Siddha Maruthuvam (Gurusamy Mudaliyar)

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

NOI VARUM VAZHI (ETIOLOGY) :

According to Balavagadam, the etiology of Pandu noi is given as below

“பிறந்தநாள் பிள்ளைக்குத் தானே நன்றாய்

பிதாவாலே பிணியுடலின் மேலே தோன்றும் .....

கனலது மெத்தக் காணும் கண்ணது வெளுக்குந் தானே

ணிவீயுனூதீங்கிக்ஷீ முதடு தானும் .....

Pandu noi may be inherited from the parents. It may be due to genetic factors.

According to Yugimuni the cause of Pandu are as follows,

“அறிந்துமே உற்பத்தி சொல்லக் கேளாய்

அதிசார மலமிளகி யெந்நே ரந்தான்

பிறிந்துமே புளியுப்பு பெருத்தலாலும்

பெத்தமா மக்கினி யிருத்தலாலும்

.....

பறிந்துமே பகல் நித்திரையே செய்தாலும்

பீகிறூக்ஷீ வந்து பாரிலுள்ளோர் படும் பாடே. ”

யூகிவைத்திய சிந்தாமணி.

From the above mentioned lines, it is clear that frequent attacks of diarrhoea, excessive intake of salt and sour food, living in hot surroundings, excessive chewing of pan and nuts excessive in take of alcohol, sleeping in day time are some of the behaviors causing Pandu noi.

According to Agasthiyar Gunavaagadam,

“கொள்ளடா அபக்குவ போசனத் தினாலும்

.....கிராணியாலும்

அளவற்றவி சாரந்தான டையும் போதும்

தெள்ளவே தேகத்தில் இரத்தம் கெட்டு

தெளிவான பாண்டதுவு முண்டாம் பாரே.”

அகத்தியர் குணவாகடம்.

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

According to Theraiyar vagadam

“ தாக முறும் பசியால் தாமுண்ணா ராமாகில்
கோதருநீர் தாகத்தாற் கொள்ளாராய் போத
மிரத்தந் தனைக்குடிக்கும் மின்பவிடாய் போக்கும்
பருத்த விழிகுருடாம் பார்”

Negligence of food and water causes pandu noi.

According to Thanvathiri vaithyam,

“திருந்திடும் **பீகிறூக்ஷீ** ரோகஞ் சேர்ந்திடுங் குணத்தைக் கேளாய்
இருந்திடும் வாதபித்தச் சிலேற்பன மிவைதான் மாறும்
புரிந்துதா னொன்றோடொன்று பொருந்துவதாலு மண்ணோ
டருந்துவதாலும் **பீகிறூக்ஷீ** வணைந்திடு மென்னலாமே. ”

“ஆகிய மூலந் தன்னிலணைந்த வுட்டணத்தினாலுந்
தோகையர் மேகத்தாலும் துயர்மிகு ரோகத்தாலும்
தேக போஷணை யுள்ளோர்க்குத் தரித்திரஞ் சேர்தலாலும்
வேகமாந் திரிதோஷங்கள் மிஞ்சிய பாண்டு வாமே. ”

- தன்வந்திரி வைத்தியம்.

Imbalace between the three thathus, vatham, pitham, kabam, perversion of appetite such as eating mud (PICA), excessive heat accumulation due to altered Abana vayu, excessive sorrow, psychosocial factors are some of the causes of pandu.

Due to worm infestation :

According to Gurunaadi,

“வயல் தனிலே பூநாக மண்ணைத்தானே

வருந்தியது புத்துப்போல வத்தையாகும்

பயல்மொழீ யீர்தேகத்தில் கிருமிதானே. ”

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 anaemia.

Imbalace between the three thathus, vatham, pitham, kabam, perversion of appetite such as eating mud (PICA), excessive heat accumulation due to altered Abana vayu, excessive sorrow, psychosocial factors are some of the causes of pandu.

Due to worm infestation :

According to Gurunaadi,

"வயல் தனிலே பூநாக மண்ணைத்தானே
வருந்தியது புத்துப்போல வத்தையாகும்
பயல்மொழீ யீர்தேகத்தில் கிருமிதானே."

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 anaemia.
According to Thanvathiri vaithyam,

"திருந்திடும் பீகிறூக்ஷீ ரோகஞ் சேர்ந்திடுங் குணத்தைக் கேளாய்
இருந்திடும் வாதபித்தச் சிலேற்பன மிவைதான் மாறும்
புரிந்துதா னொன்றோடொன்று பொருந்துவதாலு மண்ணோ
டருந்துவதாலும் பீகிறூக்ஷீ வணைந்திடு மென்னலாமே. "
"ஆகிய மூலந் தன்னிலணைந்த வுட்டணத்தினாலுந்
தோகையர் மேகத்தாலும் துயர்மிகு ரோகத்தாலும்
தேக போஷணை யுள்ளோர்க்குத் தரித்திரஞ் சேர்தலாலும்
வேகமாந் திரிதோஷங்கள் மிஞ்சிய பாண்டு வாமே. "

தன்வந்திரி வைத்தியம்.

Imbalace between the three thathus, vatham, pitham, kabam, perversion of appetite such as eating mud (PICA), excessive heat accumulation due to altered Abana vayu, excessive sorrow, psychosocial factors are some of the causes of pandu.

Due to worm infestation :

According to Gurunaadi,

“வயல் தனிலே பூநாக மண்ணைத்தானே
வருந்தியது புத்துப்போல வத்தையாகும்
பயல்மொழீ யீர்தேகத்தில் கிருமிதானே. ”

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 anaemia.

“கிருமியால் வந்ததோடம் பெருகவுண்டு
கேட்கவதின் பிரிவதனை கிரமமாக
.....
தேகமதில் சோகைக் குட்டங் கிருமியாலே ”

குருநாடி நூல்.

According to this, sogai will occur due to worm infestation, which meant Pandu noi.

Due to Toxicity:

1. “பத்தின யிரத தோடம் **பீகிறூகூஷி**வாம் மேனி யெல்லாம்
.....
பித்தமு மதிக மாகிப் பிரலாப மாகப் பேசும்
பத்திய யிரத தோடக் குணமெனப் பகரலாமே ”

Due to impure mercury pandu noi will occur.

2. Over dose of phosphorous, lead, copper sulphate produce the symptoms of pandu.
3. Chronic use of white arsenic produce the symptoms of pandu.

NOI ENN (CLASSIFICATION):

According to Yugimuni Pandu noi is classified into 5 types

“கூறவே பாண்டு விடப் பெயரைக் கேளாய்
குறிப்பாக வைந்துவித மாகும் பாரு
வாரவே வாதமாம் பாண்டி னோடு
மார்க்கமாம் பித்தத்தின் பாண்டு தானும்
தேறவே சிலேட்டுமாம் பாண்டு தானும்
திரிதோடப் பாண்டோடு விட பாண்டாகும் ”

Thanvanthiri classified pandu into 7 types

"பயித்திய பாண்டு வாதபாண்டுவே சிலேத்தும் பாண்டு
வியத்திரி தோஷப்பாண்டு வெறும் பித்த சிலேத்தும் பாண்டு
பயித்திய வாதபாண்டு பகர் சன்னிவாத பாண்டு
நயப்புறும் பாண்டு வேழின் குணத்தை நான் நவிலலுற்றேன்"

தன்வந்திரி வைத்தியம்.

Classification of Pandu noi based on various Siddha books.

AGASTHIAR GUNAVAAGADAM	**VAITHYASARASANKRAHAM**
1. Vatha pandu	1. Vatha pandu
2. Pitha pandu	2. Pitha pandu
3. Kaba pandu	3. Moola pandu
4. Vida pandu	4. Moolapitha pandu
5. Mukkutra pandu	5. Vida pandu

YUGI CHINTHAMANI-800	**T.V. SAMBASIVAM PILLAI**
	ROGA NIRNAYA SAARAM
1. Vatha pandu	1. Vatha pandu
2. Pitha pandu	2. Pitha pandu
3. Kaba pandu	3. Kaba pandu
4. Mukkutra pandu	4. Mukkutra pandu
5. Vida pandu	5. Oothu pandu
	6. Neer pandu
	7. Eri pandu
	8. Vida pandu

THANVANTHIRI VAITHIYAM	**ANUBA VAITHIYA DEVA RAGASIUM**
1. Vatha pandu	1. Vatha pandu
2. Pitha pandu	2. Pitha pandu
3. Kaba pandu	3. Kaba pandu
4. Mukkutra pandu	4. Mukkutra pandu
5. Pitha vatha pandu	5.Miruthikapuktha pandu
6. Sannipatha pandu	6. Vida pandu
7. Paithiya pandu	

PARARASA SEKARAM	MADAVA NITHANAM
1. Vatha pandu	1. Vatha pandu
2. Pitha pandu	2. Pitha pandu
3. Kaba pandu	3. Kaba pandu
4. Sanni pandu	4. Sanni pandu
5. Miruthika pandu	5. Mann pandu

JEEVARAKSHAMIRTHAM	ASHTANGA HRIDAYAM
1. Vatha pandu	1. Vatha pandu
2. Pitha pandu	2. Pitha pandu
3. Kaba pandu	3. Kaba pandu
4. Tridosha pandu	4. Sanni patha pandu
5. Miruthikapuktha pandu	5. Mannun pandu

KURIKUNANGAL IN PANDU NOI (CLINICAL FEATURES)

1. Murkurikunangal (Premonitory symptoms)

Pandu patients exhibit the following symptoms from their initial stage of development itself. The patient experiences insidious onset of fatiguability, dyspnoea on exertion, diminished vision, faintness, palpitation and pallor of the skin.

Theraiyar Neerkkuri illustrates that,

"இயற்கை நீர் சுருங்கினும் இதுவும் சலப்பொருள்
செயற்கை யாயருந்தினும் சிறுத்த நீரிதுவும்
பாண்டு நோய்ச் சம்பவத்தைத் தருமிதில்"

Oliguria occurring suddenly and oliguria occurring even after excessive intake of water are explained as premonitory symptom of pandu noi.

Manmurugiam Ennum Tamil Maruthuva nool states that

"அடிக்கடி உமிழ்தல் ஆக்கை தளர்தல்
நீரும் கசடும் மஞ்சள் நிறம்படல்
நெஞ்சக் கரிப்பு நெடுந்தோல் வெடித்தல்
கண்ணிமை தடித்தல் உண்டத ருமை
மண்தின் றிடுதல் வியர்த்தல் பிறவும்
வெளிற்று நோயின் முன்னதாகும்"

The above mentioned lines explains the premonitory symptoms of pandu as excessive salivation, tiredness, yellow colour urine, fissured skin, thickening of eyelids, pica i.e., eating mud and excessive sweating.

POTHU KURIGUNANGAL (GENERAL SIGNS AND SYMPTOMS)

Agasthiyar Gunavaagadam states that,

"உண்டாகும் வேளை தன்னில் தேக நேர்மை
உறுதியாய்ச் சொல்லுகிறேன் நன்றாய் பாரு
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.

Agathiyar Vaithiya Rathna Churukkam states that,

"பித்தத்திற் பித்தமாகில் பிதற்றிடும் கிறுகிறுப்பு
சத்திய மதிகமாகும் சரீரத்திலிளைப் புண்டாகும்
அத்தியா யுலருமேனி யாகமும் வேறதாகும்
வத்தியே வெளுத்துக் காயம் வறண்டு பின் வீக்கமுண்டாகும்"

Giddiness, emaciation pallor of the body are mentioned as symptoms of pandu noi.

Vaithiya Vilakkam Ennum Amirthasagaram states,

"அகட்டினில் வாயுவுண்டா மருந்திய வுண்டி நாளும்
மிகச் செரியாமை நிற்கு மீளவும் புரளுமென்க
வெகுத்திடு விதனங் காட்டு மெய் கண் கைதான் வெளுக்கும்
மிகச்சல மலத்தை கட்டு மேனிவே றாகுந் தானே
நரம்பின் வழியதாக நடந்தெங்கும் பரந்து டம்பின்
நிரம்பிய மஞ்சணீர் போ லுட்டண நிகழுஞ் சீதம்
வரம்பில தாகு மன்றி மார்பொடு வயிறு மந்தக்
கரம்புறந்தாள தைக்குங் கதித்திடு மூச்சு மட்டும்
வெய்யபோ சனங்கள் வேண்டு மீண்டவை யுண்ண வொட்டா
மெய் மினு மினுப்புக் கொள்ளும் வேறுஞ் செய் குணங்களுண்டாம்
பையுள் செய் பாண்டு ரோகம் பாற்றிரு மதனுக்கேற்ற
செய்ய நன் மருந்து தன்னைத் திறம்பட வினிது கேண்மின்"

Flatulence, indigestion, pallor of the conjunctiva and skin, anuria, constipation, dyspnoea, loss of appetite, glistening of the skin.

In Siddha maruthuvam, Gurusamy 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.

In females scanty menstruation, sometimes menorrhagia may occur.

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 pain in lower extremities.

According to **Sarabendrar Vaithya Muraigal – Karbini Balaroga Chikitchai**

வயிற்றிலே ஒரே சமனான வீக்கம், கண்கள், நகம் முதலியன வெளுத்தல், கண்களில் வீக்கம், பசியின்மை முதலியன காணப்படும்.

SYMPTOMS OF VARIOUS TYPES OF PANDU :

1. Vatha pandu :

"கொள்ளவே வாத பாண்டு ரோகங் கேளாய்
குடல் புரட்டியடி வயிறுதான் வலிக்குந்
தள்ளவே தாகமொடு பசியுமில்லை
தழலான சரசரப் பாகத்தானும்
நள்ளவே நரம்பெல்லாங் கறுப்புமாகும்
நடுக்கலொடு கண் சிவப்பு மல பந்தந்தான்
விள்ளவே தலைவலிக்கு மேனி வீங்கும்
வெளுப்பாகும் வாதத்தின் பாண்டுதானே "

- யூகிமுனி

The symptoms of Vatha pandu 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 :

"வாமென்ற மேனியெல்லா மஞ்சளித்து
மகாவெளுப்பு உண்டாகி மந்தக் கண்ணாந்
தாமென்ற தாகமொடு மூர்ச்சை யாகுந்
தனி வாயில் மிளகு போற்றா னுறைக்கும்
நேமென்ற நெஞ்ச முள்தானு முண்டாய்
நெருக்கியே மூச்சு முட்டதுவே யாகுங்
கோமென்ற கிறுகிறுத்துவாய் கைப்பாகுங்
கிளர்ப்பித்த பாண்டுவெனக் கூறலாமே"

- யூகிமுனி

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 :

"கூறியதோர் நரம்பு தோல் மிக வெளுப்பு
கிளர் நாவு உப்புறைக்கும் மயிர் கூச்சாகும்
வாறியதோர் வாந்தியாங் குரலுங் கம்மும்
மகத்தான தும்மலுடன் கோழையாகும்
ஈறியதோர் இருமலொடு மயக்கமுண்டாம்
இருப்பசதி யிந்திரிய நட்டமாகும்
சீறியதோர் சோபமொடு தாபமாகுஞ்
சிலேட்டுமத் தின்பாண்டெனச் செப்பலாமே"

யூகிமுனி

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

4. Mukkutra pandu :

“செப்பவே யருசியொடு சோபதாகஞ்
செயலான சுவாச மொடு இளைப்புமாகும்
வெப்பவே மேகனத்தில் சிறுநீர்தான் வீழும்
மிடுக்கான பெல வீனமார் பிடித்தல்
துப்பவே சூட்டோடு தியக்கமாகுந்

தும்மலாயுடம் பெங்குமூதிக் காணுந்
திப்பவே தேகமெங்கு மசதியாகும்
திரிதோடப் பாண்டென்னச் செப்பும் நூலே”

யூகிமுனி

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

5. Vida pandu :

“நூலாக நகத் தோடேயுடல் வெளுக்கும்
நோய் நரம்பு சூடாகும் தாகமாகும்
ஆலாகயருசியொடு சத்தி விக்கல்
அதட்டியே இருமலுடனதி சுவாசம்
வாலாக வயிற்றிரைச் சலதிசார தோடம்
மாசுரமுமுண்டாதல் மார்கனத்தல்
வேலாகமேனியெங்கு மிகவே ஊதல்
விடபாண்டு அசாத்தியமென்றே விளம்பலாம். ”

யூகிமுனி

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

According to **Thanvanthiri Vaithyam,**

1. Vatha pandu :

"ஆன கண் மலசலங்கள் அணிநகங் கறுப்பதாகுந்
தாகமா மங்கமெங்குந் தடியடி படுகை போலே
மீனமாய் நடுக்கமுண்டா மிசைந்த துர்பலமுமுண்டா
மானநீர் வாதபாண்டு வென்ன வகுக்கலாமே. "

Eyes, face, urine, motion and nails are black in colour, thirst, tremor, fatigue.

2. Pitha pandu :

"உற்றதோர் அன்னபேதம் அராசக முதர மந்தம்
முற்றி மார்ப்பு நோய் மூத்திரம் பொன்னின் வன்மை
வேற்றி சேர் புறங்கால்கை கண் வீங்குடல் வெளுத்தல் வேர்த்தல்
பற்றி தொக்கிற்காய் பணித்திய பாண்டுவாமே."

Anorexia, flatulence, chest complaints, high coloured urine, oedema of the dorsum of feet and hand, perspiration and pallor of skin.

THODAR 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 and sexual intercourse lead to kamalai (jaundice). This is stated by Yugimuni as follows.

" விளம்பவே பாண்டு முற்றிருக்கும் போது
மீறியே பித்தவஸ்துதனைப் புசித்தால்
புளம்பவே மங்கையுடன் புணர்ச்சி செய்தால்
பூண்டிடுமே காமாலை யெனும் ரோகம்"

யூகி முனி.

MUKKUTRA VERUPAADUGAL (PATHOLOGY)

Udal vanmai is affected due to excessive intake of salt and sour foods, which cause indigestion and loss of appetite. This affects rasam and raththa thathus which lead to increase in pitham and do not give nutrition to the body affecting ranjagam leading to pallor of the skin. The increased pitham affects both vatham and kabam increasing the pallor of the skin. Further kabam increases producing generalized swelling of the body.

PINIYARI MURAIMAI (DIAGNOSIS) :

Pini means the disease, which affects the body.

Ari means indentify

Muraimai means rules.

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

1. Porialarithal (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 sense organs of perception namely Skin, Tongue, Eye, Nose and Ear.

Pulangal are five objects of senses, which are Tactile 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 thervugal is a unique method of diagnosing the disease, which was developed by siddhars.

"நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்."
நோய் நாடல் நோய் முதல் நாடல்

"மெய்க்குறி நிறம் தொனி விழிநா இருமலம் கைக்குறி."

தேரையர்

PANDU IN RELATION WITH ENNVAGAI THERVUKAL :

1. Naadi (Pulse)

உடலில் உயிர் தரித்திருப்பதற்கு காரணமான சக்தி எதுவோ அதுவே தாது அல்லது நாடி எனப்படும்.

நோய் நாடல் நோய் முதல் நாடல்.

Naadi is responsible for the existence of life. It is a suitable diagnostic tool used by Siddhars. It is recognised as one of the principle means of diagnosis and prognosis of the disease from time immemorial.

NAADI NADAI IN PANDU NOI :

1. Pitha Naadi :

‘‘ ஆமேதான் யத்தி சுரம் **பாண்டு** சோகை

.....

நாமேதான் சொன்னோமே பித்தக்கூறு

நவின்றிட்டார் வாசமுனி நவின்றிட்டாரே’’

அகத்தியர்

2. Kaba Naadi :

‘‘ தானமுள்ள சேத்துமந்தானிளகில் வெப்பு

.....

ஏன முறுங் காமலை **பாண்டு** சோபை

ஏழு சுரங்கள் பலதுக்கம் விட முண்டாமே’’

சதகநாடி

3. Vatha Kaba Naadi :

‘‘ வாதத்தில் சேத்துமமாகில் வலியொடு வீக்கமுண்டாம்

.....

தீதுற்று **மெய்வெளுத்துத்** திடமுடனசனஞ் செல்லா’’

அகத்தியர் நாடி

4. Kaba Vatha Naadi :

‘‘ கண்டாயோ சிலேற்பனத்தில் வாதநாடி

.....

விண்டாலே யிளைப்பிருமல் சோபை பாண்டு’’

சதகநாடி

5. Kaba Pitha Naadi :

“ இடமான சேத்துமத்தில் பித்த நாடி

.....

வெகு சுரமும் நாவறட்சி **பாண்டு** ரோகம்”

சதகநாடி

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 buds 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, conjuctiva 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) :

Hyperemia, ulceration, response of pupil, pallor, protrusion, sunken eyes, sharpness of vision, excessive lacrimation, accumulation of secretion at the angle of eyes, visual disturbance and any specific diseases of the eyes should be noted.

In Pandu noi, pallor of conjuctiva is seen.

7. Malam (Faeces) :

Colour, consistency, quality, smell, frequency, constipation/diarrhoea, presence of mucous, blood and undigested food particles in the stool should be studied. In Pandu noi, the following changes may be noted.

Vatha Pandu	-	Constipation
Pitha Pandu	-	Diarrhoea
Mannun Pandu	-	Worms

8. Moothiram (Urine) :

Neer IIakkanam (Method of collection of urine) :

“ அறிந்துமாறிரதமும் அவிரோதமதாய்
அஃகல் அலர்தல் அகாலவூன் தவர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காது பெய்
தொரு முகூர்த்தக் கலைக் குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”

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.

Neerkkuri :

“ வந்த நீர்க்கரியெடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவை யறைகுது முறையே”

சித்த மருத்துவாங்க சுருக்கம்

Urine has the following five characters,

1. Niram - Colour of the Urine
2. Edai - Specific gravity of the Urine
3. Manam - Smell of the Urine
4. Nurai - Frothy nature of the Urine
5. Enjal - Quantity (Increased or decreased amount) of Urine voided

Neerkkuri in Pandu noi :

‘‘ இயற்கை நீர் சுருங்கினும் இதுவும் சலப்பொருள்
செயற்கை யாயருந்தினும் சிறுத்த நீரிதுவும்
பாண்டு நோய் சம்பவத்தைத் தருமிதில்
தூண்டு றாய் பேதியும் சோர்வும் பிறக்குமே’’

நோய் நாடல் நோய் முதல் நாடல் திரட்டு

Neikkuri :

‘‘ நிறக்குறிக் குரைத்த நிருமான நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவிடுத்
தென்றுறத் திறந்தொலி யேகா தமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுஞ் செய்தியை யுணரே’’

நோய் நாடல் நோய் முதல் நாடல்

The specimen collected for neikkuri 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 from its position and spreading of the oil drop should be noted.

‘‘ அரவென நீண்டினஃதே வாதம்’’
‘‘ ஆழிபோற் பரவின் அஃதே பித்தம்’’
‘‘ முத்தொத்து நிற்கின் மொழிவ தென் கபமே’’

Oil spreads like a snake	- Vatha neer
Oil spreads like a ring	- Pitha neer
Oil remaining and floating like a pearl	- Kaba neer

Neikkuri in pandu noi :

‘‘ விரைவுடன் கதிர்போல் நீண்டு வேற்றுமைக் குணங்கள் கண்டால்
குருதிதான் கெட்டு நாசம் குன்றி குணமதென்னே’’

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

ROLE OF MUKKUTRAM IN *PANDU*

Vatham :

Its mathirai alavu is 1 mathirai. *(1 cc)*

Location of vatham in the body :

Vatham is located in the abanan, faeces, idakalai, 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 dysnoea is present.

2. Abanan (Keezh nokkukaal) :

It is located in the lower abdomen and extremities. It is responsible for excretion of urine, stools, ejaculation of 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 is resides in the eyes and responsible for opening and closing of the eyelids and intelligence.

7. Koorman :

It is located in the eyes. It causes winking of the eyelids, yawning and closure of mouth. It gives strength and helps to visualize things and causes lacrimal secretion.

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 mathirai *(½ cc)*

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. Ranjagam :

It gives colour to the blood.
In Pandu it is affected due to pallor of conjunctiva and skin.

3. Saathagam :

It controls the entire body functions responsible for the activities of the body
In Pandu, it is affected due to inability to do the works properly and sluggishness.

4. Alosagam :

This gives brightness to the eyes.

5. Pirasagam :

It gives complexion to the skin.

In pandu, it is affected due to altered skin lusture.

Kabam :

Its mathirai alavu is ¼ mathirai. *(¼cc)*

Location of Kabam in the body :

Kabam is located in samanavayu, sperm, head, tongue, vulva, 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

4. Tharpagam :

It keeps the eyes cool.

5. Santhigam :

It is responsible for the lubrication and aids free movements of joints.

Paruvakaalam (Season) :

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

1. Kaar kaalam	- Avani and Puratasi	- Aug 16 to Oct 15
2. Koothir kaalam	- Aippasi and Karthigai	- Oct 16 to Dec 15
3. Munpani kaalam	- Markazhi and Thai	- Dec 16 to Feb 15
4. Pinpani kaalam	- Maasi and Panguni	- Feb 16 to Apr 15
5. Ilavenil kaalam	- Chithirai and Vaigasi	- Apr 16 to June15
6. Mudhuvenil kaalam	- Aani and Aadi	- June 16 toAug15

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

Physiologically the types of alterations of Mukkutram are,

	Thannilai Valarchi	Vetrunilai Valarchi	Thannilai Adaithal
Vatham	Muthuvenil kaalam	Kaar kaalam	Koothir kaalam
Pitham	Kaar kaalam	Koothir kaalam	Munpani kaalam
Kabam	Pin panai kaalam	Ilavenil kaalam	Muthuvenil 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, Mullai, Neithal and Palai may have in 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. Venneer - Responsible for the propagation of species.

In Pandu ,

Saaram is affected which leads to sluggishness, dyspnoea and tiredness.

Senneer is affected which leads to pallor of skin and conjuntiva.

PROGNOSIS OF PANDU :

Curable and Incurable Types **:**

நஞ்சு வெளுப்பு நோய் தீருவது அரிது. மற்ற வெளுப்பு நோய்கள் தக்க மருந்துகளால் எளிதில் குணமாகுமெனினும், அந்நோய்களில் வாந்தி, கழிச்சல், வீக்கம் (ஊதல் நோய்), நீர்வேட்கை, விக்கல், மதுமேகம், இளைப்பிருமல் ஆகிய குறிகுணங்களிலேதேனும் ஒன்று அல்லது பல அந்நோய்களில் துணை நோயாகித் துன்பங்களை அதிகமாக விளைவிக்கின் எளிதில் தீராது.

சித்த மருத்துவம்.

According to Sarabendrar Vaidhya Muraigal :

அதிக நாளான பாண்டு ரோகம் சிகிச்சைக்கு வசப்படாது. புதிதானாலும் உடல் வீக்கத்தில் மஞ்சள் நிறம் காணப்பட்டால் குணம் ஏற்படாது. மலச்சிக்கலோ அல்லது பச்சை நிறமான அதிசாரமோ ஏற்பட்டால் அசாத்தியம்.

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 உள்ள பாண்டு ரோகியையும், ஆண்குறி, தொடையிடுக்கு ஆகிய இடங்களில் வீக்கம், அடிக்கடி மயக்கம், அதிசாரம் சுரம் கண்டால் தீராது.

Asathiya Pandu :

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

அகத்தியர் வைத்தியப் பிள்ளைத் தமிழ்.

Kannusamiyam states that**,**

"வெப்பு பிணியதனில் வெம் மேகத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் செப்பும்
கிராணியிற் பாண்டில்கிளர் நீர்சுருங்கிற்
பிராணன் பிரியுமெனப் பேசு"

"சொல்லு பித்தத்திற் சோபை சோபைதனில் வாயு தொந்தம்
வல்லவதிற் பாண்டுவன் பாண்டில் நல்ல
வயிற்றுக் கடுப்பு வளர் கடுப்பிற் சீதம்
பயிலிற் கெடுதி யெனப்பன"
"பாண்டு பிரமேகம் பன்வாத சூலை குன்மம்
வேண்டா சயஞ்சன்னி வெண்சோபை நீண்ட
அதிநீரே காமாலை யானபிணி தம்மு
ளதி சாரமா காதறி"

According to Sadhaga Naadi,

"தானான பிரமேகம் வாதசூலை
சார்வான நீரிழிவு குன்மரோகம்
மானான சயரோகஞ் சன்னிதோடம்
யடுத்த விடங்காமாலை பாண்டு சோகை
மானே கேள் கபரோக மந்திர வியாதி
மஞ்சள் நோய் குலைநோவு பயித்தியரோகம்
ஊனாகும் வருமிடத்தில் யதிசாரங்கள்
உண்டாகில்ய சாத்தியாமா முறுதி தானே"

NOI NEEKKAM (TREATMENT)

The speciality of Siddha treatment emphasise 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 has 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 unequillibrium of three thathus, which may be due to various causes like diet, life style pattern, mental and physical activities.

When treating for cure of the disease the following principles must be noted.

"நோய்நாடி நோய்முதல் நாடியது தணிக்கும்
வாய்நாடி வாய்ப்பச் செயல்"
"உற்றா னளவும் 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.

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 பித்தத் தாழும்
பேதியால் வாதத் தாழும்
அஞ்சனத்தால் கபந் தாழும்

Usually for pitha diseases, emetics are to be given to restore 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 improve haemoglobin content of blood, iron preparations are used.
2. Removal of the causative factors.
3. Pathiyam ie, diet and other restrictioins to normalize the affected thathu and to maintain a longer drug action.
4. Intake of rich nutritious food is also a part of treatment.

The author took **TIRUDHARATCHATHA CHOORANAM(TC)** as a trial drug for Pandu.

Diet :

"மாறுபா டில்லா உண்டி மறுத்துண்ணின்
ஊறுபா டில்லையு யிர்க்கு. "
திருக்குறள்
"மருந்தே உணவு, உணவே மருந்து"
திருமூலர்.

Diet regimen for Pandu Noi :

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

Greens :

Karaisalai, Ponnanganni, Arukerai, 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 stages of Pandu noi. Soup prepared from the liver or heart 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 deblitated system and also help in rejuvenation.

REVIEW OF LITERATURE
MODERN ASPECTS

The commonest nutritional deficiency disorder present throughout the world is iron deficiency but its prevalence is higher in the developing countries. This is due to poor hygienic status of people in these countries.

The blood is the most precious fluid in the body a fact that expressed in such common terms as "the life blood". It is also considered as fluid of growth, fluid of health. Blood is one of the extracellular body fluids, which circulate 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 with in physiological limits. The constant nature of the blood is one of the important haemostatic conditions of the body.

Blood contains iron in the form of Haemoglobin and also as cytochromes etc. Any form of iron deficiency cause anaemia.

Blood

Definition:

Blood is a complex fluid which circulates rapidly in closed system of blood vessels.

Properties of Blood:

1. **Colour**

   Blood is an opaque fluid and it is red in colour

2. **Volume**

   The Volume of blood in a normal adult is 5 litres

3. **Reaction and PH**

   Blood is slightly alkaline and its pH in normal condition is 7.4

4. **Specific gravity**

   The specific gravity of total blood - 1.052 – 1.061

   The specific gravity of blood cells - 1.092 – 1.101

   The specific gravity of plasma - 1.022 – 1.026

5. **Viscosity**

Blood is five times more viscous than water. It is mainly due to red blood cells and plasma proteins

Composition of Blood:

Blood consist of a solid portion and a fluid portion. The solid portion constitutes the blood cells namely RBC, WBC, and platelets and the fluid portion is plasma. The cells form 45% and the plasma forms 55% of the total volume of the blood.

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 alveoli of lungs to the tissues for the oxidation of food and production of energy. The carbon-di-oxide formed in the tissues as a result of this process is carried to the lungs, where it is exhaled.

3. **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.

4. **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

5. **Regulation of body temperature**

Because of high specific heat of blood, it is responsible for maintaining the thermoregulatory mechanism in the body. i.e. the balance between heat loss and heat gain in the body

6. **Regulation of acid – base balance**

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

7. **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

8. Regulation of osmotic pressure

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

9. Defensive Function

Blood has WBCs , Gamma globulins which have phagocytic action. They also transport protective subtances such has anti-bodies, anti-toxins and lysins.

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 these substances are taken from the blood during conditions like starvation, fluid loss, and electrolyte loss.

The Red Blood cells or Erythrocytes:

The erythrocytes of most of the higher animals including man are circular, non-nucleated, biconcave discs.

Diameter : 7.2 μ (6.9 – 7.4 μ)

Thickness : At the periphery it is thicker with 2.2 μ and at the it center is thinner with 1 μ

Because of this, the outer edge appears as a rim around a central depression, and when seen edge-wise, it has approximate appearance of dumb-bell.

Surface Area : 120 square μ

Volume : 85 – 90 Cubic μ

Properties of RBC

1. Rouleaux formation

When blood is taken out, the red blood cells pile up one above another like the pile of coins. This property of red blood cell is called *rouleaux* formation.

2. Specific Gravity

The specific gravity of red blood cell is 1.092 to 1.101.

3. Packed Cell volume

When the blood is collected in a centrifuge tube along with proper anticoagulant and centrifuged for a period of 30 minutes at a speed of 3000 rpm the red blood cells settle at the bottom of the tube leaving the clear plasma at the top. The red blood cells form 45% of the total blood. This is called *the packed cell volume or hematocrit*.

4. Suspension Stability

During circulation the red blood cells remain suspended uniformly in the blood. This property of the red blood cells is called the suspension stability

Erythropoiesis

Erythropoiesis is the process by which the origin, development and maturation of erythrocytes occur.

In the bone marrow, there are cells called uncommitted pluripotent hemopoietic stem cells because these cells are not designed to form a particular type of blood cell. When these cells are designed to form a particular type, the stem cells are called committed pluripotent hemopoietic stem cells.

The different committed stem cells will produce colonies of specific types of blood cells. Then a committed stem cell that produces colony forming unit blast (CFU – B) and then erythrocytes are produced from these CFU – B and are called colony forming unit – erythrocytes (CFU-E).

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

Sites of Erythropoiesis

1. In the early few weeks of embryonic life - yolk sac
2. During the middle trimester of gestation - Liver, Spleen, lymphoid organ
3. Later part of gestation and after birth - Red bone marrow and liver
4. Up to the age of 5 - Red bone marrow of all the bones
5. After the age of 5 to Adult - Red bone marrow of long bones and flat bones

Stages of Erythropoiesis

Stage I – Pro erythroblast (Megaloblast)

This early cell is large (15-20) μ. The cytoplasm is basophilic staining with a deep violet blue and there is a pale-staining crescent near the nucleus and the cell contains no haemoglobin. The nucleus is about 12μ 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 diameter 15µ. It shows active mitosis. The nucleoli have disappeared. Condensation of chromatin network occurs. The cytoplasm is basophilic. So this cell is also called **basophilic erythroblast**.

Stage III – Intermediate Normoblast (Late erythroblast)

This cell is smaller (10-15) µ and shows active mitosis. Nucleus is still present. Chromatin network shows further condensation. Haemoglobin begins to appear and its eosinophilic staining give the cytoplasm a polychromatic appearance.

Stage IV – Late Normoblast (Normoblast)

Mitosis has now ceased and the diameter of the cell is 8 – 10 µ. The nucleus becomes very smaller and the condensed chromatin assumes a ***"cart wheel"*** **appearance or ink spot** and finally becomes deeply stained in a uniform manner. Quantity of haemoglobin increases. Cytoplasm becomes almost acidophilic. So the cell is called **Orthochromic erythroblast.** In this cell, the nucleus disintegrates and disappears. The process is called **pyknosis.**

Stage V – Reticulocyte

It is slightly larger than matured red blood cells. Cytoplasm contains reticular network. It is basophilic in nature. During this stage, the cells enter the blood through the capillary membrane by means of a process called **diapedesis.**

Matured Erythrocyte

Reticular network disappears. Matured red blood cell is biconcave; smaller in size; diameter of 7.2 microns. It is with haemoglobin and without nucleus.

It takes 5 days for the development of reticulocyte from proerythroblast. The reticulocyte take two more days to become matured red blood cells.

Changes during Erythropoiesis

Four important stages occurring are,

1. Reduction in size of the cell
2. Disappearance of nucleoli and nucleus
3. Appearance of Haemoglobin
4. Change in the staining properties of the cytoplasm

Regulation of Erythropoiesis

1. Tissue oxygenation is the basic regulator of red blood cell production. Any condition that causes the quantity of oxygen transported to the tissues to decrease (Hypoxia) ordinarily increases the rate of red blood cell production. Hypoxia occurs in conditions like very high altitudes, anaemia, prolonged cardiac failure, cyanotic heart disease and lung disease.
2. Erythropoietin is a circulatory hormone, formed mainly in kidneys, increase mainly during hypoxia. It stimulates the production of proerythroblast from hempoietic cells in the bone marrow.
3. Epinephrine, norepinephrine and several of the prostaglandins stimulate erythropoietic production.

Life Span and fate of Red Blood Cells

Average life span of red blood cell is about 120 days. The senile red blood cells are destroyed in reticulo endothelial system.

When the cells become older, the cell membrane become more and more fragile. The diameter of the capillaries is less or equal to that of red blood cells. The younger red blood cells can pass through the capillaries easily; however the older cells become fragile. So these cells are destroyed while trying to squeeze through the capillaries. The destruction occurs mostly in the capillaries of spleen because the splenic capillaries have a thin lumen. So the spleen is usually called ***'grave yard'*** of red blood cells. Daily 10% of red blood cells, which are senile, get destroyed in normal young healthy adults.

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 a conjugated protein consisting of iron containing pigment protein 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. This high molecular weight and consequently great size of the molecules are the cause of the colloidal nature when they are dispersed in water. It is a chromoprotein, forming 95% of dry weight of red blood cell and 30 to 34 % of wet weight.

Varieties of haemoglobin

Haemoglobin is of two types namely.

1. Adult haemoglobin – HbA
2. Fetal haemoglobin – HbF

There are some structural differences between these two types of haemoglobin. In adult haemoglobin the globin contains 2 alpha chains and 2 beta chains. In fetal haemoglobin there are two alpha chains and two gamma chains instead of beta chains.

Formation of haemoglobin:

1. 2 Succinyl co-A + 2 glycine → 4 Pyrrole
2. 4 Pyrrole → Protoporphyrin IX
3. Protoporphyrin IX Fe^{++} → Haeme
4. Haeme + polypeptide → Haemoglobin chain (Alpha or Beta)
5. 2 Alpha chains + 2 Beta chains → Haemoglobin A

Properties of haemoglobin:

1. Buffering Capacity:

Haemoglobin is an effective buffer. Its isoelectric point is 6.8. The buffering capacity of haemoglobin is due to the presence of large number of histidine residues in the globin fraction. It is estimated that a molecule of haemoglobin has 35 histidine residues

2. Transport of oxygen:

The physiological importance of haemoglobin is due to its capacity to combine reversibly with oxygen. It combines with haemoglobin to form oxyhaemoglobin readily, at high pressure as existing in the lungs. Oxyhaemoglobin readily dissociates, at low partial pressure as prevailing in the tissues. This property of haemoglobin provides an effective and excellent system for the transport of oxygen from the atmosphere (lungs) to the cells of the body.

3. Haem-Haem interaction:

The haem groups in haemoglobin combine with oxygen in such a way that each haem group combine with one molecule of oxygen.

4. Combination with Carbon monoxide:

Haemoglobin combines with carbon monoxide to form carboxy haemoglobin with an affinity, two hundred times more than with oxygen.

5. Formation of carbamino compound:

A small amount of CO_2 carried in the blood is in combination with the free amino acids of the proteins of haemoglobin to form carbamino compounds

($Hb.NH_2 + CO_2 \rightarrow Hb.NHCOOH$)

6. Oxidation- Reduction properties of haemoglobin:

Under normal conditions, iron exists in ferrous form in the haemoglobin. It can be converted to ferric form by oxidation with ferricyanide and this result in the formation of Methemoglobin.

7. Action of weak acids and alkalies:

Weak acids and alkalies act on haemoglobin by separating the haem from globin. In the presence of oxygen, haem gets oxidized to haematin in which iron is in the ferric form. Haematin can combine with the chloride ion to form haematin chloride, which is also known as haemin.

8. With strong acids

A more vigorous change takes place with strong acids. In addition to the separation of globin from haemoglobin, the iron from the haem is removed, resulting in the formation of an iron free derivative.

Metabolism of Haemoglobin

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

I. Synthesis of haemoglobin

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 B_{12} (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 erythrocyte contains free porphyrin. As the cells mature the porphyrin content decrease 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 8 gms of haemoglobin are broken down in the body and this corresponds to the formation of about 300 mg of bile pigments per day. The normal sites of haemoglobin degradation are mainly the reticuloendothelial 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 be amino acid "pool" for reutilization. The haem portion breaks down resulting in the formation of bile pigments.

Normal values of haemoglobin of different age groups

	Mean(gm/dl)	**Range(gm/dl)**
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 – 13.0
5 year	12.5	12 - 13
10 year	13.5	13 - 14
Older	15	14 - 16

Packed cell volume and blood indices

Packed cell volume (PCV) is the haematocrit value expressed as the percentage of cellular elements with that of whole blood.

Significance of determining PCV

Determination of PCV helps in

1. Diagnosis and treatment of anaemia
2. Diagnosis and treatment of polycythemia
3. Determination of extent of dehydration and recovery from dehydration after treatment
4. Decision of blood transfusion

Blood Indices

Blood indices are specifically meant for erythrocytes. The number, shape, volume and colour of the red blood cells indicate the quality of blood. So these features are named as blood indices.

IMPORTANCE OF BLOOD INDICES

Blood indices have got diagnostic value in determining the type of anaemia

Different Blood Indices

Following are the various blood indices

1. Mean corpuscular Volume (MCV)
2. Mean corpuscular haemoglobin (MCH)
3. Mean corpuscular haemoglobin concentration (MCHC)
4. Colour Index (CI)

1. Mean corpuscular Volume (MCV)

Mean corpuscular Volume is the average volume of single red blood cells and it is expressed in cubic microns (cu.μ).

$$MCV = \frac{\text{PCV in 1000 ml or 100 ml x 10}}{\text{RBC count in millions / cu.mm}}$$

2. Mean corpuscular haemoglobin (MCH)

Mean corpuscular haemoglobin is the quantity or amount of haemoglobin present in one red blood cell. It is expressed in micro gram or pico gram (pg).

$$MCH = \frac{\text{Haemoglobin in grams per 1000 ml of blood or 100 ml x 10}}{\text{RBC count in millions / cu.mm}}$$

3. Mean corpuscular haemoglobin concentration (MCHC)

This is the concentration of haemoglobin in one red blood cell. It is the amount of haemoglobin expressed in relation to volume of one red blood cell. So the unit of expression is percentage.

$$MCHC = \frac{\text{Haemoglobin in grams per 1000 ml of blood x 10}}{\text{PCV in 100 ml of blood}}$$

4.Colour Index (CI)

This is the ratio between the percentage of the haemoglobin and the percentage of red blood cells in the blood.

All the above mentioned blood indices are reduced in iron deficiency anaemia

Normal values:

$$CI = \frac{\text{Haemoglobin \%}}{\text{RBC \%}}$$

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

1 – 13 days	-	54 ±10.0%
14 – 60 days	-	42.0 ±7.0%
3 months – 10 years	-	36.0 ±5.0%
11 – 15 years	-	39.0 ±5.0%

Mean Corpuscular Volume (M.C.V)

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

Mean Corpuscular Haemoglobin (M.C.H)

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

Mean Corpuscular Haemoglobin Concentration (M.C.H.C)

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

Mean corpuscular diameter (M.C.D)

1 – 13 days	-	8.6 µm
14 – 60 days	-	8.1 µm
3 months – 10 years	-	7.7 µm
11 – 15 years	-	7.6 µm

Reticulocytes

Cord blood	-	5.0%
2 weeks	-	1.0%
3 months	-	1.0%
6 months – 6 years	-	1.0%
7 – 12 years	-	1.0%
Adult	-	1.6%

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. Iron is necessary for electron transport chain, oxidative phosphorylation

3. Peroxidase, lysosomal enzyme, is required for phagocytosis and killing of bacteria by neutrophils
4. Iron is associated with effective immune competence of the body.

Daily Iron Requirements in different age groups:

Pregnant and lactating females	-	40 mg/day
Females 11 years to 30 years	-	18 mg/day
Adults 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 sources

Rich Sources

- Muscle meat (Red more than white)
- Organ meat (Liver, heart, kidney)
- Beef liver

Red meat not only supplies a good amount of iron it also increases absorption of iron from other food sources

Good Sources

- Greens
- Leafy vegetables
- Nuts
- Cereals
- Wheat germs
- Fish
- Shellfish
- Poultry
- Egg

- Iron fortified cereals and foods
- Apples and dry fruits
- Jaggery
- Yeast
- Molasses
- Oysters

Poor Sources

- Wheat and Polished rice

Distribution of iron in the body

Total quantity of iron in the body averages 4 - 5 gm of total body weight.

Iron is distributed in the body as follows.

1) Haemoglobin – present in red cells contain 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 form of excess iron (30%). They are stored in the mononuclear phagocytic cells of the spleen, liver and bone marrow and in parenchymal cells of the liver

Iron Metabolism:

The iron required for haemoglobin synthesis is derived from two primary sources – ingestion of food containing iron and recycling of iron from senescent red cells.

Absorption

Iron is mostly found in food in ferric form. In the acidic medium provided by gastric HCl, the Fe^{3+} is released from food. Ascorbic acid (Vitamin C) and cysteine convert ferric form to ferrous form. This ferrous form is soluble and readily absorbable. Absorption of iron takes place from almost all part of the small intestine mainly from duodenum and proximal jejunum. Iron from diet containing haem is better absorbed than non haem iron.

Factors affecting iron Absorption

1) Acidity, ascorbic acid and cysteine enhances iron absorption
2) In Iron deficiency anaemia iron absorption is increased to 2 to 10 times that of normal
3) Small peptides and amino acids favour iron absorption
4) Phytate and oxalate (found in leafy vegetables) interfere with iron absorption.
5) Food additives (EDTA) and antacids reduce iron absorption
6) A diet with high phosphate which are found in soft drinks, beer, ice cream, candy bar decreases iron absorption
7) Smoking and alcohol interferes with iron absorption
8) Impaired absorption of iron is observed in malabsorption syndrome such as steatorrhea
9) In patients with partial or total surgical removal of stomach, iron absorption is severely impaired

Iron in the mucosal cells:

The iron (Fe^{2+}) entering the mucosal cell by absorption is oxidized to ferric form (Fe^{3+}) by the enzyme ferroxidase. Fe^{3+} then combines with apoferritin to form ferritin which is the temporary storage form of iron. Form the mucosal cells, iron may enter the blood stream (which mainly depends on the body needs) or lost when the cells are desquamated.

Transport of iron in the plasma:

The iron liberated from the ferritin of mucosal cells enters the plasma in ferrous state. Here, it is oxidized to ferric form by a copper containing protein, ceruloplasmin which possesses ferroxidase activity. Another cuproprotein ferroxidase II also helps for the conversion of Fe^{2+} to Fe^{3+}.

Ferric iron then binds with a specific iron binding protein, namely **transferrin** or siderophilin. The plasma transferrin can bind with 400 mg of iron/dl plasma. This is known as **total iron binding capacity (TIBC)** of plasma.

Storage of Iron:

Storage of excessive iron in the blood is deposited in all cells especially in the liver hepatocytes. The hepatic cells contain large amounts of a protein called apoferritin, which is capable of combining reversibly with iron. Therefore when iron is available in

the body fluids in extra quantities, it combines with apoferritin to form ferritin and stored. When iron is in the low level, the ferritin releases the iron. Thus, apoferritin – ferritin system of liver acts as **blood iron buffer** as well as iron storage medium. **Hemosiderin** is another iron storage protein and this is insoluble form.

Excretion

The body is unable to regulate its iron content by excretion alone. The amount of iron lost per day is 0.5 – 1.0 mg which is independent of iron intake. This loss is nearly twice more (i.e. 1 – 2 mg per day) in menstruating women. Iron is lost from the body as a result of desquamation of epithelial cells from the gastro intestinal tract, sweat, and loss via hair and nail. Iron excreted in the faeces mainly consists of unabsorbed iron and desquamated mucosal cells.

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 the further absorption of iron from the gastrointestinal tract by feedback 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

ANAEMIA

DEFINITION:

Anaemia is defined as a reduction of the red blood cell volume or haemoglobin concentration below the range of values occurring in healthy persons.

WHO criteria for diagnosis of Anaemia

Children 6 months - 6 years	Less than 11
Children 6 years -14 years	Less than 12

Grading of Anaemia:

WHO grades anaemia according to haemoglobin level as follows,

Hb between 10 gm and cut off point for age	-	Mild
Hb between 7 to 10 gm	-	Moderate
Hb under 7 gm	-	Severe
Hb under 5 gm	-	Very Severe.

CLASSIFICATION

A. Based on Production/destruction of RBC

A useful classification of the anaemia of childhood divides them into 2 broad groups.

I. Decreased or ineffective production of red blood cells or haemoglobin

II. Increased destruction or loss of red blood cells.

Anaemia resulting primarily from inadequate production.

I. Marrow failure

1. Aplastic anaemia

   Congenital, acquired

2. Decreased number of red blood cell precursors in the marrow

   Congenital, acquired

3. Marrow replacement

   Malignancies, osteopetrosis

   Storage disorders

II. Deficiency of Specific Factors

1. Megaloblastic anaemia
   - Folic acid deficiency or malabsorption
   - B_{12} deficiency or malabsorption
2. Microcytic anaemia

Iron deficiency

Copper deficiency

Lead poisoning

III. Impaired Erythropoietin Production

1. Chronic renal disease
2. Hypothyroidism, Hypopituitarism
3. Chronic inflammation, infection
4. Malignancy
5. Protein malnutrition

Anaemia resulting primarily from rapid destruction.

I. Blood Loss

- Acute haemorrhage
- Chronic haemorrhage

II. Haemolytic Anaemia

a. Intrinsic Defects

Intrinsic abnormalities

- Membrane Defects (Membranopathy)

Hereditary spherocytosis, elliptocytosis

Enzyme Defects (Enzymopathy)

Enzyme of glycolytic pathway

Enzyme of the pentose phosphate pathway

- Defects in synthesis of haemoglobin (Haemoglobinopathy)

Hb S, C, D, E

b. Extrinsic abnormalities

i. Immunologic disorders

Rh iso immunization

A (or) B iso immunization

Other minor Blood group incompatibilities

ii. Active antibody formation

B. Morphological classification

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

1. Microcytic Hypochromic anaemia	Iron deficiency, Thalassemia haemoglobinopathies haemolytic anaemia.
2. Normocytic normochromic anaemia	Aplastic anaemia
3. Macrocytic normochromic anaemia	Folate and vitamin B_{12} deficiency hypothyroidism
4. Macrocytic hypochromic anaemia	Combined deficiency of iron and folate or Vitamin B_{12}

Microcytic anaemia:

The size of red cells is smaller than normal and colour index less than one. The mean corpuscular volume is less than 78 cubic microns

Causes of Microcytic anaemia:

a) Inadequate intake of iron, defective absorption of iron, idiopathic hypochromic anaemia, starvation, dietary deficiency, anaemia of milk fed children.
b) Excessive need of iron during growth, pregnancy
c) Chronic haemorrhages
d) Inadequate utilization of haematinics – myxoedema, chronic sepsis, chronic renal diseases.

Macrocytic anaemia:

The red cells are bigger than normal and the colour index is above one. The mean corpuscular volume is more than 94 cubic microns.

Causes of Macrocytic anaemia:

a. Deficiency of the extrinsic factors, nutritional anaemias, pellagra.
b. Absence of intrinsic factor, total gastrectomy
c. Cirrhosis of liver
d. Megaloblastic anaemia, hypoblastic anaemia.

Normocytic Anaemia:

The size of the red cells is more or less than the normal size. The colour index is less than the normal range and the mean corpuscular volume is 78 to 94 cubic microns.

Causes of Normocytic anaemia:

a. Acute hemorrhage
b. Haemolytic anaemias
c. Blood destruction by metals, protozoa, haemolysis
d. Leukemia, Hodgkin's disease, drug poisoning

B. Based on Etiopathogenesis:

1. Nutritional Anaemias	PEM, Iron, Folic acid, Vitamin B12, Vitamin C, pyridoxine, or thyroxine deficiency.
2. Haemolytic Anaemias	
Congenital	Thalassemia
	Sickle cell anaemia
	Hereditary spherocytosis
	G-6-PD deficiency
Acquired	Malaria
	Kala azar
	Rh or ABO incompatibility

3. Haemorrhagic

Acute	Trauma
	Epistaxis
	Haemorrhagic disease of newborn
	Scurvy
Chronic	Hookworm
	Chronic dysentery
	Oesophageal varices

4. Bone Marrow Depression

Primary	Hypoplasia or Aplasia
	Fanconi's Anaemia
Secondary	Infections
	Irradiation

5. Infections

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

6. Other Miscellaneous Conditions

	Chronic amoebic dysentery
	Repeated bouts of diarrhea

CLINICAL FEATURES OF ANAEMIA

The haemoglobin level at which symptoms and signs of anaemia develops depends upon 4 main factors.

1. The Speed of onset of anaemia:

Rapidly progressive anaemia causes more symptoms than anaemia of slow onset, as there is less time for physiological adaptation.

2. The Severity of Anaemia:

Mild anaemia produces no symptoms or sign, but a rapidly developing severe anaemia (haemoglobin below 6 gm) may produce significant clinical features.

3. The age of the patient:

The young patient due to good cardiovascular compensation tolerate anaemia quite well as compared to the elderly

4. The haemoglobin dissociation:

In anaemia, the affinity of haemoglobin for oxygen is depressed. As a result oxy haemoglobin is dissociated more readily to release free oxygen for cellular use.

PATHOLOGICAL RED BLOOD CELLS IN ANAEMIA:

In anaemia, many kinds of abnormal red cells including nucleated forms are seen in the circulation. These abnormal cells are,

I. Anisocytosis (Variation in size of RBC)

a. Macrocytosis:

The size of the cell is 9 to 12 microns. This occurs in pernicious anaemia, plumbism, acute anaemia due to severe haemorrhage and erythroblastosis foetalis

b.Microcytosis:

The size is less than 6 microns. This occurs in Iron deficiency anaemia, chlorosis, chronic bleeding, polycythaemia and anaemias secondary to infections.

c. Normocytosis:

The red cells are in normal size, found mainly in post haemorrhagic anaemias.

II. Poikilocytosis (Variation in shape of RBC)

a. Ovalocytosis:

The oval shaped red cells occur in some human families. Such a condition does not cause ill-health, but a minority may manifest haemolytic phenomena.

b. Spherocytosis:

Spherocytosis may be seen in congenital haemolytic anaemia and in certain other acute haemolytic anaemias. The red cells are very fragile.

c. Sickle cells:

In arterial blood, the red blood cells are normal in shape, but in venous blood some cells assume the shape of sickle.

III. Polychromatophilia (Irregularity in staining)

This indicates an increase in immature red cells in circulation and occurs in the following forms

a) Normoblasts:

Nucleated red cell indicates over activity of bone marrow, commonly seen in severe anaemia.

b) Punctate Basophilia (Basophilic stippling):

It occurs in lead poisoning and severe anaemia, and chronic malaria.

c) Reticulocytes:

Occurs in acute bleeding and in pernicious anaemia.

IRON DEFICIENCY ANAEMIA (IDA)

Iron deficiency is the most common and widespread nutritional disorder in the world. The numbers are staggering: 2 billion people – over 30% of the world's population – are anaemic, many due to iron deficiency. Malaria, HIV/AIDS, hookworm infestation, schistosomiasis, and other infections such as tuberculosis are particularly important factors contributing to the high prevalence of anaemia in some areas.

Iron deficiency and anaemia reduce the work capacity of individuals and entire populations, bringing serious economic consequences and obstacles to national development.

Iron Requirements during childhood

Understanding of iron requirements, intakes and bioavailability is essential to explain the vulnerability of some individuals to develop iron deficiency anaemia.

The iron released from the senescent, red cells during the first 8-12 weeks of life (a period of quiescent erythropoiesis) is stored in the body and helps to maintain erythropoiesis upto 4-6 months in a normal term infant and upto 2-3 months in low birth weight infant. Normal infants at birth have about 75 mg of iron per kg body weight, two thirds of which is present in red blood cells. Infant and children should continue to absorb 0.8 to 1.0 mg of iron daily to reach the adult body stores of 4-5 gms.

Normal body losses of iron are about 20µg/kg/day and most of these losses occur by the shedding of cells from intestinal mucosa. These losses are small and are relatively constant but may increase many folds in the presence of diarrhoea, dysentery, and parasitic infections.

Certain factors protect infants from becoming iron deficient in first few months of life. These include,

1. Preferential delivery of iron to the fetus during the pregnancy particularly during last three months of gestation.
2. Placental transform to the newborn immediately after birth when the cord is allowed to pulsate before being clamped.
3. Exclusive breast feeding for first four to six months of life, due to better bioavailability of iron from the breast milk.

STRUCTURES OF THE RED CORPUSCLES IN IDA:

In iron deficiency anaemia, the red blood corpuscles are decreased or normal in the number and 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 Anaemia:

1. Increased physiological 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

Intense exercise

Bleeding diasthesis

Repeated venous sampling

5. Increased demands

Prematurity

Low birth weight

Recovery from PEM

Adolescence

Growth

Iron deficiency anaemia is more in children between ages of 6 months and 2 years and from 11 to 16 years due to spurts of growth during these periods.

Menstruation

Iron deficiency in post pubescent girls is most commonly caused by the loss of more iron through menstruation.

Pregnancy

During pregnancy, anaemia is almost universal.

IRON POOR DIET

- Dietary inadequacy is present in more than 80 percent of cases especially in the poorer groups. This is still encountered in privileged societies under the following circumstances
- Infants are also at high risk because the diet predominantly milk contains very small amounts of iron. Human milk provides only about 0.3 mg/ litre of iron
- Premature babies have only lesser amount of storage iron in the liver as well as body
- Children especially during the early years of life have a need for dietary iron to accommodate growth and expansion of the blood volume.

IRON MALABSORPTION:

- Iron malabsorbtion is an unusual cause of iron deficiency where malnutrition is rampant however both histologic and functional abnormalities of the intestine are common. Defective iron absorption is
- caused by non-tropical sprue.
- Partial or total gastrectomy impairs iron absorption caused by reduction in gastric acidity and
- acceleration of the food through the upper portion of the small bowel. The absorption of both haem
- iron and non-haem is defective.
- Pica or the habitual ingestion of non-food substances is common in children and pregnant women. It
- markedly inhibit iron absorption.
- Pancreatic enzymes may contribute to the high incidence of iron deficiency in patients with cystic
- fibrosis.

GASTRO INTESTINAL BLEEDING:

- In adult men and postmenopausal women, occult bleeding from the gastrointestinal tract is the most common cause of iron deficiency.
- Gastrointestinal bleeding also is prevalent among iron deficient infants and children.
- Characteristically gastro intestinal bleeding is occult and unsuspected
- Milk induced enteropathy associated with occult Gastrointestinal bleeding has been implicated as the cause of iron deficiency in some infants.
- Peptic ulcer disease is a well-documented cause of occult blood loss.
- Crohn's disease and ulcerative colitis also are commonly associated with iron deficiency.
- During the first year of life, meckel diverticulum is a well-recognized cause of asymptomatic bleeding.
- Corticosteroids, Indomethacin and other non-steroidal anti-inflammatory agents may also induce gastrointestinal tract bleeding
- Hookworm infestation (Ankylostomiasis) is the most important cause of intestinal blood loss worldwide.

The parasites Ankylostoma duodenale and Nectar americanus attach to the proximal portion of the small intestine and suck blood from submucosal vessels. The amount of blood lost is a function of the hook wormload, which in turn is proportional to the number of ova in the stool. Each worm has been in the intestine for months or years, draws 0.2-0.5 ml of blood per day. It has been estimated that the loss of haemoglobin for every twelve worms may be one percent. Fecal ova counts in excess of 5000/g are regularly associated with iron loss of more than 3to 4 mg/ day and a high incidence of iron-deficiency anaemia.

Pathogenesis:

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

It has been pointed out 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 and the supply of iron to the marrow for haemoglobin synthesis becomes inadequate, hypochromic anaemia 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. The development of anaemia.

Iron deficiency state, which may be divided into three functionally distinct stage of severity

Stage of Iron deficiency anaemia:

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 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 anaemia:

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

CLINICAL FEATURES:

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

Epithelial tissue changes

Long standing IDA causes epithelial tissue changes in some patients. The changes occur in the nails (koilonychia or spoon shaped nails), tongue (atrophic glossitis), mouth (angular stomatitis) and oesophagus causing dysphagia from development of thin membranous webs at the post cricoid area (Plummer Vinson syndrome).

ROLE OF IRON DEFICIENCY ANAEMIA IN VARIOUS SYSTEMS

Cardiovascular system:

Dyspnoea and palpitation are common symptoms while on exertion but in very severe anaemia the patient may get cardiac failure and there may be dyspnoea at rest. Haemic murmurs are commonly heard in anaemic 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 anaemia usually they are bilateral and occur in the absence of an aortic systolic bruit and disappear following correction of the anaemia. Jugular venous pressure increase in severe anaemia due to the high pulse pressure with a capillary pulsation. Oedema of the legs occasionally occurs in ambulant patient with severe anaemia as the result of venous and 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 severe anaemia clouding of consciousness, numbness, coldness and sometime tingling of the hands and feet.

Reproductive system:

Menstrual disturbances are commonly associated with anaemia.

Renal system:

Slight proteinuria may be present with severe anaemia. Anaemia may further reduce renal function to the point at which nitrogen retention develops. Correction of anaemia 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 is severe anaemia and when congestive heart failure develops the liver may become tender. In certain cases of iron deficiency anaemia, spleen may be enlarged.

Pyrexia:

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

Dietary Iron:

The dietary iron comes from two sources, Heme and non-heme, the later being the major source of iron in diet and is found in varying degrees in all foods of plant origin. Heme iron is present in meat, fish, and poultry, but the intake of these products is generally low. Heme iron is better absorbed than non-heme iron and is not influenced by dietary factors.

Breast milk even in spite of low levels of iron (0.5 mg/ lit) has a better absorption and bioavailability as compared to cow's milk. Good sources of iron in the diet includes, pulses, dhals, green leafy vegetables, dates, nuts, jaggery, meat and fish. Administration of 50 mg of vitamin C increases iron absorption by two folds.

Complications in Iron Deficiency anaemia:

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

Investigations required for Iron Deficiency anaemia:

1. **Blood Investigations**

    Haemoglobin

    Total Red Blood cell count

    Peripheral blood smear

    Packed cell volume

    Mean corpuscular volume

    Mean corpuscular haemoglobin

    Mean corpuscular haemoglobin concentration

    Total iron binding capacity

        Differential count

        Erythrocyte sedimentation rate

        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**

    Ova

    Cyst

    Occult blood

    Red blood cells

    Pus cells

Special Investigations occasionally required:

- X-ray barium meal, X-ray Barium enema, X-ray chest
- Endoscopy, colonoscopy, sigmoidoscopy, gastro duodenoscopy
- 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

Laboratory Diagnosis:

In Iron deficiency anaemia the haemoglobin is less than 11 gm in children. The red cell count in rarely below 2.5 million/cubic millimeter and the red cells are usually microcytic and hypochromic reticulocytes and platelets are normal or increased. The white cell count is normal. Serum iron is usually below 30µg/100ml. (Normal is 250 - 350µg/100ml). Bone marrow haemosiderin is absent. The PCV, MCV, MCH , MCHC are all reduced.

Differential Diagnosis:

Iron deficiency anaemia must be differentiated from other hypochromic anaemia.

1. **Anaemia of infection:**

Chronic infections such as rheumatic fever, rheumatoid arthritis, tuberculosis and malaria may have associated mild to moderate anaemia, 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 anaemia:**

It is characterized by severe hypochromic microcytic anaemia, often early in infancy and progressive hepatospleenomegaly. 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 anaemia:

Most of the red cells are hypochromic and microcytic, serum iron is high and iron deposit 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. Anaemia of lead poisoning:

Anaemia 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 anaemia, pronounced increase of aminolevulinic acid and coproporphyrin in the urine is characteristic of lead poisoning. Increased levels of lead in blood are required for definite diagnosis.

Diagnosis:

Following criteria are essential to diagnose iron deficiency anaemia

- History of inadequate intake of dietary iron and blood loss if any
- Typical symptoms and signs like easy fatiguability, 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
- 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 less than haemoglobin level would suggest
- Serum ferritin level is reduced

MANAGEMENT

This can be considered under three headings:

1. Correction of anaemic 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 medical iron.
2. Replenishment of iron stores
3. Elimination of the cause

Response to Treatment

1. A positive response to therapy can be defined as a daily increase in haemoglobin concentration of 0.1 gm/dl (0.3 or 1 % increase in haemocrit) from the 4th day onwards.
2. Reticulocytes increase within 3 to 5 days and reach a maximum at 5 to 10 days.
3. Haemoglobin concentration is virtually normal after 2 months of therapy. However food containing iron should be continued for 3 to 6 months to build up iron stores. RBC counts may temporarily rise above normal before haemoglobin response. The red cell indices may remain abnormal for sometime after the normal haemoglobin level has been restored. The microcytic population is gradually replaced by a normocytic population.
4. Pica pagophagia and other nonspecific symptoms disappear within one week of therapy. With the onset of treatment the patients shows rapid subjective improvement with disappearance of fatiguability, lassitude, and impaired cognitive functions of the epithelial lesions those affecting tongue and nails are most responsive to treatment. After 1-2 weeks of therapy, small filiform papillae are seen on the tongue. By 3 months the tongue is usually normal and koilonychia usually disappears within 3 -6 months.

PREVENTION OF IDA

Appropriate nutritional strategies are an important factor in prevention of IDA. The basic approaches to the prevention of IDA include

1. Protection and promotion of breast feeding 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 %. Processes like germination (Sprouting of green gram) consumption of green leafy vegetables would be additional long-term methods for prevention of IDA.
3. Periodic de-worming with anti-helminthic drugs for hookworm infestation and schistosoma should be considered in endemic areas.
4. Supplementation with medicinal iron is considered necessary to reduce the extent of anaemia 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 with iron content of 1 mg per gram of salt is the most effective preparation.

Self care Procedures for iron deficiency anaemia

1. Eat more foods that are good source of iron
2. Concentrate on green leafy vegetable, red meat, beef liver, poultry, fish, wheat germs, oysters, dried fruit and fortified cereals.
3. Boost iron absorption. 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 to 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. Eat good sources of folic acid daily
11. These include vegetables like asparagus, sprouts, spinach and lettuce.
12. Black-eyed peas, cantaloupe orange juice, oatmeal, whole grain cereals, wheat germ, liver and other organ meats are excellent sources also
13. Eat fresh uncooked fruits and vegetables often. Don't parboil vegetables. Heat destroys folic acid.

Take a multivitamin supplement daily that has 100% of the RDA for folic acid deficiencycan lead to infertility and an increased risk of infection.

DRUG REVIEW

The Trail Drug is Tirudharatchatha Chooranam(TC)

INGREDIENTS

- Munthiri (Anacardium occidentale)-35 gms
- Pereechu (Phonex dactilifera) - 35gms
- Thippli (Piper longum) - 35gms.
- Athimathuram (Glycyrrhiza glabra) - 35gms
- Lavangapathri (Cinnamomum tamala) - 35gms
- Mutha kasu (Cyperus Rotundus) - 35gms
- Kothumalli (Coriandrum sativum) - 35gms
- Kirambu (Syzygium aromaticum) - 35gms
- Nerpori (Oryza sativa) - 35gms.
- Elam (Elettaria cardamomum)-35 gms
- Kodiveli (Plumpago indica)-35 gms
- Kookai neeru(Maranta arundiacae)-35 gms
- Milagu (Piper nigrum) – 35 gms
- Mutka velai (tephorosia purpurea)-35 gms
- Sugar – Equal amount of chooranam.

1. முந்திரி

தாவரவியல் பெயர் : Anacardium occidentale

குடும்பம் : Anacardiaceae.

வேறு பெயர் : கொட்டைமுந்திரிமா, கொல்லம்மா.

சுவை : இனிப்பு.

தன்மை : தட்பம்.

பிரிவு : இனிப்பு.

செய்கை :

- சிறுநீர்பெருக்கி,
- வெப்பமுண்டாக்கி.

குணம் : ஐம்புலன்களுக்கும் நன்மை பயக்கும்.

2. பேரிச்சம்

தாவரவியல் பெயர் : Oryza sativa

குடும்பம் : Arecaceae

வேறு பெயர் : பேரிச்சம், பேரிச்சை.

சுவை : இனிப்பு.

தன்மை : தட்பம்.

பிரிவு : இனிப்பு.

செய்கை :

- உடலுரமாக்கி,
- உடல்தேற்றி.

குணம் : குருதியை பெருக்கும், குருதிபோக்கை நிறுத்தும்,

3. ஏலம்

தாவரவியல் பெயர் : Elettaria cardamomum.

குடும்பம் : Zingiberaceae

வேறு பெயர் : இளஞ்சி, கோரங்கம், துடி.

சுவை : கார்ப்பு.

தன்மை : வெப்பம்.

பிரிவு : கார்ப்பு.

செய்கை :

- வெப்பமுண்டாக்கி,
- அகட்டுவய்வகற்றி,
- பசித்தீத்தூண்டி.

குணம் : இது, தொண்டை, தாள், கீழ்வாய் இவைகளில் உண்டாகும் நோய்களையும், இருமல், கழிச்சல், நீர்ச்சுருக்கு, சிலந்தி நஞ்சு இவற்றையும் போக்கும், அழலை ஆற்றும்.

4. கொடிவேலி

தாவரவியல் பெயர் : Plumpago indica.

குடும்பம் : Plumbaginaceae.

வேறு பெயர் : சித்திரமூலம், ஒலி, சித்திரமூலி, வன்னி.

சுவை : கார்ப்பு.

தன்மை : வெப்பம்.

பிரிவு : கார்ப்பு.

செய்கை :

- உரமாக்கி,
- பசித்தீத்தூண்டி.

குணம் : இதனால், கட்டி, புண், கழலை, வளிநோய், சோபை, உதிரக்கட்டு, பெரு வயிறு இவற்றை குணபடுத்தும்.

5. கூகைநீர்.

தாவரவியல் பெயர் : Maranta arundiacae.

குடும்பம் : Zingiberaceae

வேறு பெயர் : அரரூட்கிழங்கு, கூவாமாக்கிழங்கு, கூகைகிழங்கு.

சுவை : இனிப்பு.

தன்மை : தட்பம்.

பிரிவு : இனிப்பு.

செய்கை :

- உடலுரமாக்கி,
- உடல்தேற்றி,
- குளிர்சியுண்டக்கி.

குணம் : இதனால், இருமல், சுரம், நீர்வேட்கை நீங்கும், உடற்கு ஊட்டம் தரும்.

6. மிளகு.

தாவரவியல் பெயர் : Piper nigrum.

குடும்பம் : Piperaceae.

வேறு பெயர் : கலினை, கறி, காயம், திரங்கல், மிரியல், சருமபந்தம், வள்ளிசம், மாசம், குறுமிளகு, மலையாளி, கோளகம்.

சுவை : கைப்பு, கார்ப்பு..

தன்மை : வெட்பம்.

பிரிவு : கார்ப்பு.

செய்கை :

- காறலுண்டக்கி,
- அகட்டுவய்வகற்றி,
- முறைவேப்பகற்றி,
- தடிப்புண்டாக்கி.

குணம் : இதனால், குளிர்சுரம், பாண்டு, கோழை, கழிச்சல், குன்மம், வாயு, சுவையின்மை இவை போகும்.

It contain Chavicine, Piperine, Piperidine these alkalodies.

7. முட்காய் வேளை.

தாவரவியல் பெயர் : tephorosia purpurea.

குடும்பம் : Favaceae.

வேறு பெயர் : காய்வேளை.

சுவை : கார்ப்பு.

தன்மை : வெட்பம்.

பிரிவு : கார்ப்பு.

செய்கை :

- உடலுரமாக்கி,
- கொழையகற்றி,
- சிறுநீர்பெருக்கி,
- வீக்கமுருக்கி,
- மலப்புழுவகற்றி.

குணம் : இதனால், வாதாதிக்கமும், நாவறட்சியும், பல் நோயும், சொள்ளுவடியும் நோயும் போகும்.

8. திப்பிலி.

தாவரவியல் பெயர் : Piper longum.

குடும்பம் : Piperaceae.

வேறு பெயர் : காமன், குடாரி, கோலகம், கோலி, சரம், சாடி, மாகதி, வைதேகி, அம்பு, ஆதி மருந்து..

சுவை : இனிப்பு.

தன்மை : தட்பம்.

பிரிவு : இனிப்பு.

செய்கை :

- அகட்டுவய்வகற்றி,
- வெப்பமுண்டாக்கி.

குணம் : இது, ஐயப்பிணிகளை அகற்றி, உடற்கு வன்மையை அளித்திடும். ஈளை, இருமல், இரைப்பு, உப்பிசம் முதலிய பிணிகளை போக்கும்.
It contains iperine, alkaloides.

9. அதிமதுரம்.

தாவரவியல் பெயர் : Glycyrrhiza glabra.
குடும்பம் : Favaceae.
வேறு பெயர் : அதிங்கம், அட்டி மதூகம், குன்றிவேர்.
சுவை : இனிப்பு.
தன்மை : சீதம்.
பிரிவு : இனிப்பு.

செய்கை :

- வறட்சியகற்றி,
- உள்ளழலாற்றி,
- கோழையகற்றி,
- மலமிளக்கி,
- உரமாக்கி.

குணம் : இது, முப்பினியால் வரும் புண், நீர் வேட்கை, கண் நோய்கள்,சிறு நீர் எரிச்சல், காமாலை, ஐயத்தாலுண்டன கோழையை இளகச் செய்யும். தீக்குற்றத்தின் வன்மையைத் தாழச் செய்யும்.
It contain Glycyrrhizin, Glycyrrhizic acid, Glycyrrhetinic Acid.

10. இலவங்கப்பத்திரி.

தாவரவியல் பெயர் : Cinnamomum tamala.
குடும்பம் : Laraceae.
வேறு பெயர் : தாளிசபத்திரி, தமாலபத்திரி.
சுவை : கார்ப்பு.
தன்மை : வெப்பம்..
பிரிவு : கார்ப்பு.
செய்கை :

- அகட்டுவய்வகற்றி,
- வெப்பமுண்டாக்கி.
- பசித்தீத்தூண்டி,
- வியர்வைபெருக்கி.

குணம் : மேகசுரம், ஐயசுரம், வெட்டை, இரைப்பு, இருமல், நீர் வேட்கை, அழல், வாந்தி, தாது நட்டம் போக்கும்.
It contain Cinnamic Acid, Tannin.

11. முத்தக்காசு.

தாவரவியல் பெயர் : Cyperus rotundus.

குடும்பம் : Cyperaceae.

வேறு பெயர் : கோரை.

சுவை : .

தன்மை : வெப்பம்..

பிரிவு : கார்ப்பு.

செய்கை :

- உரமாக்கி,
- வெப்பமுண்டாக்கி.
- துவர்ப்பி,
- சிறுநீர்பெருக்கி,
- வியர்வைபெருக்கி.

குணம் : இதனால், நளிர்ச்சுரம், குருதியழல் நோய், சுரவகைகள், நீர்வேட்கை, முப்பிணி, பித்ததாகம் ஆகிய இவைகள் போகும்.

It contains Starch, Carbohydrate, Pinene, Cineole, Sesquiterpenges, Glycerol, Linolenicacid, Linolic acid, Oleic Acid.

12. கொத்துமல்லி.

தாவரவியல் பெயர் : Coriandrum sativum.

குடும்பம் : Apiaceae.

வேறு பெயர் : உருள் அரிசி, தனியா.

சுவை : கார்ப்பு.

தன்மை : வெப்பம்.

பிரிவு : கார்ப்பு.

செய்கை :

- அகட்டுவய்வகற்றி,
- வெப்பமுண்டாக்கி.
- பசித்தீத்தூண்டி,
- சிறுநீர்பெருக்கி.

குணம் : இதனால், சுவையின்மை, தீகுற்றத்தால் வரும் சுரம் முதலிய இவை போகும். இது வன்மையை உண்டாக்கும்.

It contain Coriandrol terbine.

13. கிராம்பு.

தாவரவியல் பெயர் : Syzygium aromaticum.

குடும்பம் :Myrtaceae.

வேறு பெயர் : அஞ்சுகம், உற்கடம், கருவாய்க் கிராம்பு, சோசம், திரளி, வராங்கம்..
சுவை : காரமும் விறுவிறுப்புமுள்ளது..
தன்மை : வெப்பம்..
பிரிவு : கார்ப்பு.
செய்கை :

- அகட்டுவய்வகற்றி,
- இசிவகற்றி.
- பசித்தீத்தூண்டி,

குணம் : இது கிராம்பு, மயக்கம், பேதி, வாந்தி, குருதிக்கழிச்சல், நாட்பட்ட கழிச்சல், எருவாய்க்கடுப்பு, செவிநோய், சிவந்தமச்சம், கருத்தமச்சம், கண்ணில் பூ, படைகள் ஆகியவற்றை நீக்கும்.

14. நெற்பொறி.

தாவரவியல் பெயர் : Oryza sativa.
குடும்பம் : Poaceae.
வேறு பெயர் : தோரை, வை, விரிகி, சாலி, வரி.
சுவை : கார்ப்பு.
தன்மை : வெப்பம்..
பிரிவு : கார்ப்பு.
செய்கை :

- உடலுரமாக்கி,
- உள்ளழலாற்றி,

குணம் : நெற்பொறியை கஞ்சி காய்ச்சி நோயாளிகளுக்கு கொடுக்கலாம். வாயிற்று நோய் வெள்ளை சுரம் முதலிய நோய்கள் நீங்கும்.

15. சர்க்கரை.

தாவரவியல் பெயர்: Saccharum Officicinarum
குடும்பம்: Poaceae
செய்கை :

- உள்ளழலாற்றி,
- அழுகலகற்றி.

குணம் : உணவு பொருளை கெடாமல் வைக்கவும், ஜலதோஷம், நீரபீனசநோய் இவைகளை போக்கும்.

4. MATERIALS AND METHODS

Pandu is one of the most common problem in Paediatric practice among young children in India. Hence it was proposed to study about the disease. A Protocol was prepared and submitted before IEC & IAEC meeting, a After obtaining approval from the commitee, the study Preclinical & clinical study on Pandu Noi **(**IDA) in children and the drug of choice was Tirudharatchatha Chooranam(TC) was carried out in National Institute of Siddha.

The ingredients for preparation of experimental formulation Tirudharatchatha Chooranam was purchased from a well reputed country shop and raw drugs were authenticated by Herbal botanist. The medicine was prepared in Gunapadam lab of National institute of Siddha after proper purification.The prepared medicine was also authenticated by the concerned Head Of The Dept for its completeness.

INGREDIENTS OF TC

- Munthiri (Anacardium occidentale)-35 gms
- Pereechu (Phonex dactilifera) - 35gms
- Thippli (Piper longum) - 35gms.
- Athimathuram (Glycyrrhiza glabra) - 35gms
- Lavangapathri (Cinnamomum tamala) - 35gms
- Mutha kasu (Cyperus Rotundus) - 35gms
- Kothumalli (Coriandrum sativum) - 35gms
- Kirambu (Syzygium aromaticum) - 35gms
- Nerpori (Oryza sativa) - 35gms.
- Elam (Elettaria cardamomum)-35 gms
- Kodiveli (Plumpago indica)-35 gms
- Kookai neeru(Maranta arundiacae)-35 gms
- Milagu (Piper nigrum) – 35 gms
- Mutka velai (tephorosia purpurea)-35 gms
- Sugar – Equal amount of chooranam.

4.1 PREPERATION:

Except sugar, Pereechu, all the drugs are made into a fine powder by using Iron mortar and pestle and then purified. It is then mixed with Pereechu and equal amount of sugar.

PURIFICATION:

All the raw drugs purchased are cleaned finely and the drugs which are to be purified by roasting, are roasted in allow heat until it becomes golden brown in colour. The drugs to be added in the preparation are grinded separately, filtered in a fine cloth, measured separately, then the fine powder (chooranam) is mixed with cows milk and made in to a solid form. Then it is kept in a clean cloth which is tied to the mouth of a mud vessel containing equal amount of cow's milk and water. Then it is finally covered over with a top vessel, sides are covered with a cloth, so that vapor does not escape over by boiling. After complete boiling of liquid, the solid mixture is taken and dried in sun light and grinded finally and equal amount of sugar is added to the chooranam.

முந்திரி (Anacardium occidentale)

பேரிச்சம் (Phonex dactilifera)

ஏலம் (Elettaria cardamomum)

கொடிவேலி (Plumpago indica)-

கூகைநீர் (Maranta arundiacae)

மிளகு (Piper nigrum)

முட்காய் வேளை (tephorosia purpurea)

திப்பிலி (Piper longum)

முத்தக்காசு (Cyperus Rotundus)

இலவங்கப்பத்திரி (Cinnamomum tamala)

கொத்துமல்லி (Coriandrum sativum)

கிராம்பு (Syzygium aromaticum)

நெற்பொறி (Oryza sativa)

சர்க்கரை (Saccharun Officinarum)

அதிமதுரம் (Glycyrrhiza glabra)

Tirudharatcha chooranam(TC)

Even though the drugs were pure herb safety of Tirudharatchatha Chooranam *by* Acute and Sub acute Toxicity studies were done in animal models according to WHO guidelines, 1993. For acute toxicity mice and for subacute toxicity study rat were used.

4.2 PRE CLINICAL STUDY

Method of Toxicity Study

4.2.1 Preparation of drug for dosing

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

4.2.2 Drugs and chemicals

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

4.2.3 Experimental animals

Colony inbred animals strains of wistar rats of either sex weighing 200 - 250 g were used for the pharmacological and toxicological studies. The animals were kept under standard conditions 12:12 (day/night cycles) at 22^0C room temperature, in polypropylene cages. The animals were fed on standard pelleted diet (Hindustan Lever Pvt Ltd., Bangalore) and tap water *ad libitum*. The animals were housed for one week in polypropylene cages prior to the experiments to acclimatize to laboratory conditions. The experimental protocol was approved by the Institutional Animal Ethical Committee (IAEC).

4.2.4 Acute oral toxicity study

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

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

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

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

4.2.5 Repeated oral toxicity study

Repeated oral toxicity studies can be used to get additional information regarding the toxicity profile of a chemical. Repeated oral toxicity studies are defined as those studies where the chemical is administered to the animal for a period covering approximately 10% of the expected life of the animal. Usually, the dose levels are lower than for acute studies and allow chemicals to accumulate in the body before lethality occurs, if the chemical possess this ability.

Experimental procedure

Group I : Control animals received 1%CMC, 10 mg/kg/p.o. for 14 days

Group II : Aqueous suspension of TC 180mg/kg/po for 14 days

Dose calculation: The adult dose (1500mg/day- of TC) is converted into rat dose by multiplying the human dose with a factor 0.018 (corresponding to body surface area)to get the dose for a rat weighing 200g. Multiply the dose for 200 grams rat x 5 to get the dose for kg body weight of the rat. 1000mg x0.018=180mg/200g body weight rat,multiply the rat dose for 200g x 5 to get rat dose for kg body weight.

Body weight, food intake and water intake was recorded at two intervals with simultaneous observation for toxic manifestation and mortality, if any. At the end of 21 days treatment all the animals were sacrificed by over dosage of ether anaesthesia. Blood was collected and used for haematological studies. Section of liver, kidney, and heart were dissected out and kept in 10% formalin for histopathological studies.

4.2.6 Biochemical studies

Estimation of glucose

Glucose was estimated using commercial Glucose estimation kit (Span Diagnostics) by the method of Barham *et al.,* (1972) and Tenscher. *et al.,* (1971).

Aspartate aminotransferase (AST)

Aspartate aminotransferase was estimated using commercial AST kit (Span Diagnostics) by the method of Reitman and Frankel (1957).

Alanine aminotransferase (ALT)

Alanine aminotransferase was estimated using commercial AST kit (Span Diagnostics) by the method of Reitman and Frankel (1957)

Alkaline phosphatase (ALP)

Alkaline phosphatase was assayed using commercial ALP kit (Span Diagnostics) by the method of King (1934).

Urea

Urea was assayed using the commercial kit (Span Diagnostics) by the method of Coulambe *et al*., (1965).

4.2.7 Haematological studies

Erythrocyte count

Erythocyte count was estimated by Hemocytometer method of Ghai (1995).

Total Leukocyte Count (WBC)

Total Leukocyte Count was estimated by Hemocytometer method of John (1972).

Haemoglobin

Haemoglobin was estimated by method of Ghai (1995).

4.2.8 Histopathological studies

Animals were sacrificed at the end of repeated oral toxicity and tissues were processed for histopathological studies

4.2.9 Haematinic study

One group(n=6)of animals fed with Tirudharatchatha Chooranam (TC) at the dose 180mg/kg/po for 14days. The hematological parameters were taken before and after the administration for 14 days and evaluated form its hematinic activity (table-4).

REFERENCES

- Barham D and Trinder, P.Analyst 1972:97:142.
- Coulambe G.G and Favrean L.A.Clin.Chem., (1965), 11, 624.
- Ghai C.L. A text book of practical physiology, Jaypee Brothers, India 1995; p.119-202.
- John MB. Laboratory Medicine Haematology. 4th Ed. C.V. Mosby co, St.Louis, 1972;p.1198-1209
- Kanai L Mukherjee. A text book of medical laboratory technology. A procedure manual for routine diagnostic tests. Tata McGraw Hill Publishing company ltd. 1999; **1**:p.242-276.
- King E.J and Armstrong A.R (1934), Can.Med.Ass.J., 31, 376.
- Moron M.S, Difieree J.W and Mannerwik K.B. Levels of glutathione, glutathione reductase and glutathione s- transferase activities in rat lung and liver. Biochem.Biophy Acta 1979;582:67-68.
- Reitman S and Frankel S (1957), Am.J.Clin.path., 28, 56
- Tenscher, A and Richterich, P. Schweiz Med. Wschr.1971 : 101:345 and 390.
- Yagi K. Simple fluorimetric assay for lipid peroxide in blood plasma. Biochem.Med.1976; 15:212-215

4.3 BIO CHEMICAL ANALYSIS

Method of Bio Chemical Analysis

1.1 Test Drugs

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

Tirudharatchatha chooranam (TC) – Book of Agasthiyar vaithiya rathinachurukkam.

1.2.Preliminary phytochemical screening

Procedure	Observation	inference
Test for Calcium : 2 ml of extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxide solution.	No white precipitate is formed	Absence of calcium
Test for Sulphate : 2 ml of the extract is added to 5 % barium chloride solution.	white precipitate is formed	Presence of Sulphate
Test for Chloride : The extract is treated with Silver nitrate solution	No white precipitate is formed	Absence of Chloride
Test for carbonate : The substance is treated with Conc. HCl.	No effervescence is formed	Absence of carbonate
Test for Starch : The extract is added with weak iodine solution	Blue colour is formed	Presence of starch
Test for Iron (Ferric) : The extract is treated with glacial acetic acid and potassium ferrocyanide	Blue colour is formed	Presence of Ferric iron
Test for Iron (Ferrous) : The extract is treated with Conc. HNO_3 and ammonium thiocynate	Brick red colour is formed	Presence of Ferrous iron
Test for phosphate : The extract is treated with ammonium molybdate and conc. HNO3	Yellow precipitate is formed	Presence of phosphate
Test for Tannic acid : The	Blue black precipitate is	Presence of Tannic acid

extract is treated with Ferric chloride	formed	
Test for Unsaturation : 1 ml of Potassium permanganate solution is added to the extract.	It is getting decolourised	Presence of unsaturated compound
Test for saponins: Dilute extract+ 1ml of distilled water shake well.	Froth formation	Presence of saponins
Test for sugars : Benedict method ; 5ml of Benedict solution heated gently then add 8 drops of diluted extract then heated in a boiling water bath.	colour changed	Indicates the Presence of sugar(1+)
Molisch test; Dilute extract+2 drops of Molisch+3ml conc.H2SO4.	No Reddish violet zones appeared	Absence of carbohydrate
Test for steroids : Liberman Burchard test ; Dilute extract +2 ml acetic anhydride+conc.H2SO4 .	No Formation of red colour	Absence of steroids
Test for amino acids: Dilute extract +2ml of Ninhydrin's soln	Formation of violet colour	Presence of amino acids
Test for proteins: Biuret method ; 1ml of dilute extract+1mlof5%$CuSO_4$+ 1%NaOH.	Formation of Violet colour	Presence of proteins
Test for Flavanoids : Dilute extract+ mg bits+2drops of conc.HCl and gently heated.	No formation of pink colour	Absence of Flavanoids
Test for phenol; Dilute extract+2drops of $FeCl_3$ soln.	No Deep green colour is formed	Absence of phenols

Test for Tannins ; dilute extract +2ml of 10%lead acetate add.	White precipitate formed	Presence of tannins
Test for alkaloids; Mayer's method;1ml of dilute extract + 1ml reagent. Dragendroff's method; 1ml of dilute extract+ 1ml of reagent.	Appearance of cream colour precipitate Appearance of orange colour precipitate	Presence of alkaloids Presence of alkaloids

After finishing the toxicity studies 40 cases were selected from the OPD & IPD of Kuzhandhai Maruthuvam Department, National Institute of Siddha. They were treated with the trial drug Tirudharatchatha Chooranam and observed for prognosis clinically.

CLINICAL STUDY

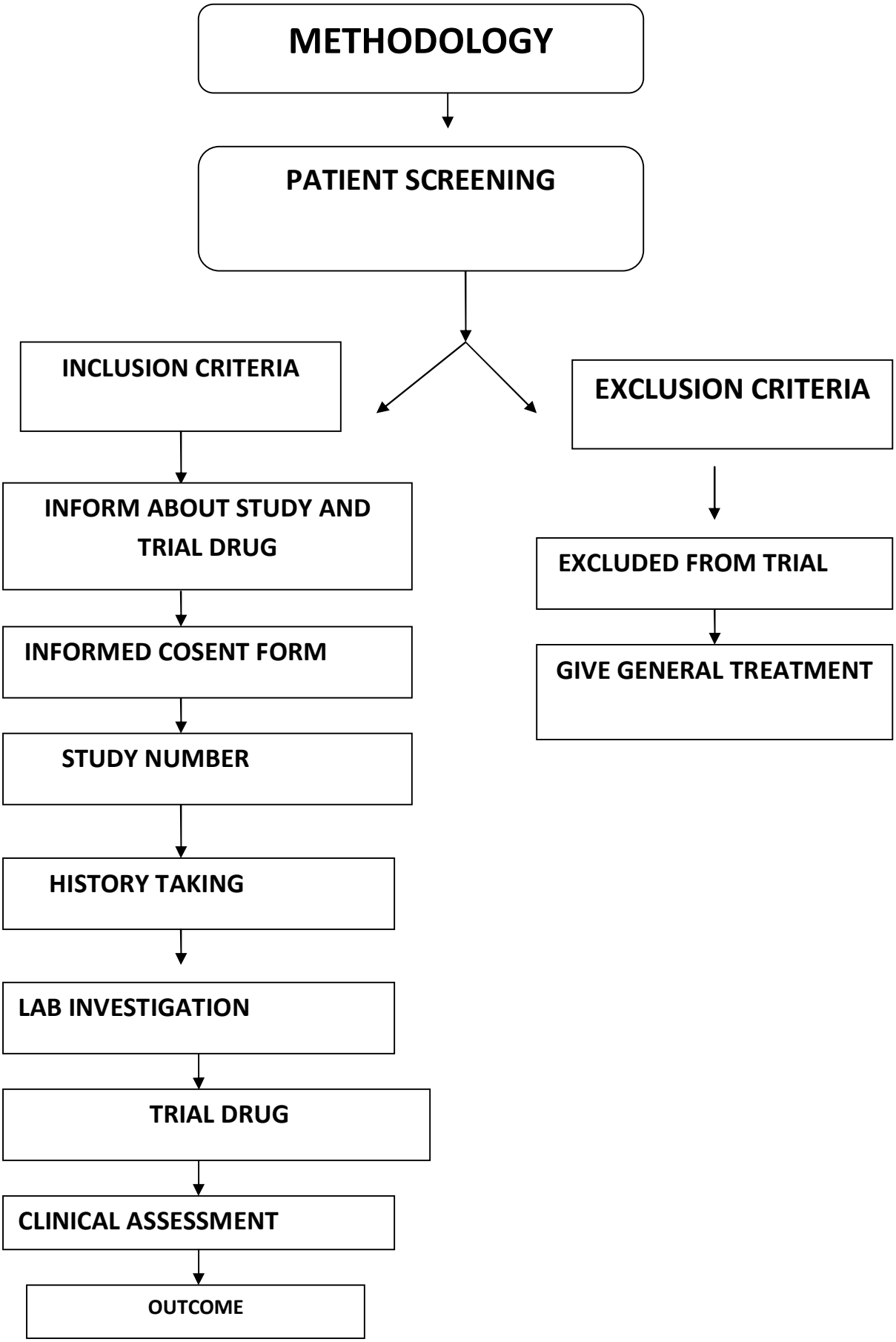
Study Design & Conduct Of Study

- Study type: An open clinical study.
- Study place: OPD & IPD Of Ayothidoss pandithar hospital, National Institute of Siddha , Tambaram sanatorium, Chennai-47.
- Treatment period: 40 Days
- Study Duration 12 Months
- Population and sample:

Population consists of pediatric patients attending OPD & IPD of Ayothidoss Pandithar Hospital, National Institute of Siddha, Chennai-47.

The sample consists of patients of 3 - 12 years of age group fulfilling all the inclusion criteria and passing the exclusion criteria mentioned below.

- Sample size: 40 patients.


METHODOLOGY
PATIENT SCREENING
INCLUSION CRITERIA
EXCLUSION CRITERIA
INFORM ABOUT STUDY AND TRIAL DRUG
EXCLUDED FROM TRIAL
INFORMED COSENT FORM
GIVE GENERAL TREATMENT
STUDY NUMBER
HISTORY TAKING
LAB INVESTIGATION
TRIAL DRUG
CLINICAL ASSESSMENT
OUTCOME

SUBJECT SELECTION

As and when patients reporting with symptoms of inclusion criteria will be subjected to screening test & documented using screening proforma**.**

INCLUSION CRITERIA

- Age: 3-12yrs of both sex.
- Hb level with < 11 gm/dl for aged 3-6 years and < 12 gm/dl for aged 7-12 years
- Patient willing to sign the informed consent form
- Patients who are willing to give biological samples for lab investigation.
- Smear showing hypochromic microcytic anemia.
- Patients with symptoms of pallor of skin, mucous membrane, conjunctiva, nail beds, Lassitude, Fatigue, Shortness of breath, Palpitation, Anorexia, Lack of interest, Frontal headache.

EXCLUSION CRITERIA

a) Based on Parental information and previous reports

- Congenital heart disease.
- Patients with chronic disease.
- Jaundice.
- Malaria and other haematological disorders
- Inherited defects.

b) Based on lab investigation:

- Smear not showing hypochromic microcytic.
- Patient not willing to give blood sample for investigation.

WITHDRAWAL CRITERIA

- Intolerance to the drug & development of adverse reactions such as nausea, vomiting, Abdominal discomfort during trial period.
- Poor patient compliance & defaulters.
- Patient turned unwilling to continue in the course of clinical trial.
- Increase in severity of symptoms.

TREATMENT

1. MEDICINE NAME **:** TIRUDHARATCHATHA CHOORANAM

2. DOSAGE

In Pediatrics: Age 3-5 yrs – 600mg.
Age 6-8 yrs- 750mg
Age 9-12 yrs-1gm

*Dose calculation for pediatric group is based on Height and Weight chart, ICMR, 1990; and the formula mentioned in the Essential of medical pharmacology by K.D. Tripathi from adult dosage of verukadi (1.25 gm – 1.5 gm).

3. ADJUVANT **:** Honey

4. DURATION **:** 40 days.

METHODOLOGY

STUDY ENROLLMENT:

- In the open clinical trial, patients informant reporting at the OPD with the clinical symptoms of Pallor of skin, mucous membrane, conjunctiva, & nail beds, Lassitude, Fatigue, Shortness of breath, Palpitation, Anorexia, Lack of interest, Frontal headache will be examined clinically for enrolling in the study based on the inclusion and exclusion criteria**.**
- The patients who are to be enrolled would be informed about the study, trial drug, possible outcomes and the objectives of the study in the language and terms understandable to the patients informant.
- After ascertaining the patient's informant willingness, informed consent would be obtained in writing from them in the consent form.
- All these patients will be given unique registration card in which patient's Registration number of the study, Address, Phone number and Doctors phone number etc. will be given, so as to report easily if any complications arise.
- Complete clinical history, complaints and duration, examination findings all would be recorded in the prescribed Proforma in the history and clinical assessment forms separately.
- Patients would be advised to take the trial drug and appropriate dietary advice would be given according to the patient's informant perfect understanding**.**

CONDUCT OF THE STUDY:

The trial drug "TIRUDHARATCHATHA CHOORANAM" is given continuously for 40 days. Deworming will be done with available OPD medicine for needy patients after examining the stools. For OP patients, they should visit the hospital once in 7 days. The trial drug will be provided for seven days. The patients are requested to return the unconsumed trial drug if any. At each clinical visit clinical assessment is done and prognosis is noted..For IP patients medicine will be issued daily by investigator for 3-4 weeks in IPD and followed OPD for who is not in a situation to stay in the hospital for a long time. Laboratory investigations are done 0 day & 40th day of the trial. After the end of the treatment also, the patient is advised to visit the OPD for another 2 months for follow-up. If any trial patient who fails to collect the trial drug on the prescribed day but wants to continue in the trial from the next day or two, he/ she will be allowed, but

defaulters of one week and more will not be allowed to continue and be withdrawn from the study with fresh case being inducted**.**

DATA MANAGEMENT:

- After enrolling the patient in the study, a separate file for each patient will be opened and all forms will be filed in the file. Study No. and Patient No. will be entered on the top of file for easy identification. Whenever study patient visits OPD during the study period, the respective patient file will be taken and necessary recordings will be made at the assessment form or other suitable form.
- The screening forms will be filed separately.
- The Data recordings in all forms will be monitored and scrutinized by HOD, Dept of Kuzhanthai Maruthuvam.

Data analysis will be done with the help of senior research officer (statistics) of NIS

OUTCOME

- Primary – Results and observation during the study inclusive of clinical improvement etc.
- Secondary – Efficacy of the trail drug and its side effects if any.

ADVERSE EFFECT/SERIOUS EFFECT MANAGEMENT:

If the trial patient develops any adverse reaction, he/she would be immediately withdrawn from the trial and proper management will be given in OPD of National institute of siddha.

ETHICAL ISSUES:

1. To prevent any infection, while collecting blood sample from the patient, only disposable syringes, disposable gloves, with proper sterilization of lab equipments will be used.
2. No other external or internal medicines will be used. There will be no infringement on the rights of patient.
3. 3.The data collected from the patient informant will be kept confidentially.The patient informant will be informed about the diagnosis, treatment and follow-up.

4. After the consent of the patient informant (through consent form) they will be enrolled in the study.
5. Informed consent will be obtained from the patient informant explaining in the understandable language to the patient.
6. Treatment would be provided free of cost.
7. In conditions of treatment failure, adverse reactions, patients will be given alternative treatment at the National Institute of Siddha with full care throughout the end.

ASSESSMENT FORMS

FORM I	- SCREENING & SELECTION PROFORMA
FORM II	- CONSENT FORM
FORM III	- HISTORY PROFORMA
FORM IV	- CLINICAL ASSESSMENT FORM
FORM V	- LABORATORY INVESTIGATION FORM
FORM VI	- PATIENT'S INFORMATION SHEET
FORM VII	- WITHDRAWAL FORM
FORM VIII	- ADVERSE REACTION FORM

5. RESULTS AND OBSERVATIONS

Pre Clinical Study Results

5.1 Acute oral toxicity study

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

5.2 Repeated oral toxicity for 14 days

Test drug TC at the dose of 180 mg/kg/po when administered orally 14days in rats did not show toxicity in renal and Hematological parameters (Table 1). However hematological studies showed an increased HB% and total RBC count. Liver and kidney function biomarkers level were normal in drug treated rats when compared with control. The drug administration for 14 days did not alter the physiological levels of glucose and cholesterol in serum (Table 2 and 3).

5.3 Histopathological study

TC at the dose of 180mg/kg/po daily administered for 14 days did not show evidence of pathological lesions in the tissues tested

5.4 Haematinic effect

TC at the dose of 180mg/kg/po given for 14 days exhibited significant improvement in the RBC,Hb%Differential count, and MCV when compared to control animals

The test drug did not exhibit mortality at the dose of 2000 mg/kg/p.o. According to OECD 423, drugs do not show mortality at 2000 mg/kg and above are "Unclassified" under the toxicity scale. Hence further studies with higher doses were not attempted.

In repeated oral toxicity study (180/kg/p.o) for 14 days animals treated with TC did exhibit significant changes in Hb%, and RBC without any change in blood sugar, cholesterol, body weight, food and water intake and behavioural parameters when compared to control animals. TC did not alter Liver and kidney marker enzyme status when compared to control animals. .

The reverse pharmacological studies of TC have got good correlation with clinical study report presented in this thesis. The present study also shows the safety profile of the

drug in repeated dosing for 14 days Oral drug treatment for 14 days did not exhibit any alteration in the biomarkers of kidney. And liver The drug treatment for 14 days significantly improved the HB% and its use in the treatment of anemia is justified through this study. Since the drug elemental iron the administration of the drug for 14days at the dose of 180mg/kg/po showed significant improvement in the Hb%,clearly shows its therapeutic value in the iron deficiency anemia.

Table 1

Preliminary acid, basic radicals and phytochemical screening

S.No.	Constituents	TC
1.	Calcium	-
2.	Iron (Ferric)	+
3.	Iron (Ferrous)	+
4.	Sulphate	+
5.	Chloride	-
6.	Carbonate	+
7.	Starch	+
8.	Phosphate	+
9.		
10.	Unsaturated	+
11.	Sugar	+
12.	Alkaloids	+
13.	Steroids	-
14.	Protein	+
15.	Tannins	+
16.	Phenols	-
17.	Flavanoids	-
18.	Saponins	+
19.	Amino acid	+
20.	Glycosides	-
21	Sterols	-

(+) – Present

(-) - Absent

Table-2

Effect of Siddha Formulations TC on Haematological parameters after 14 days repeated oral dosing

Groups	Hb (gm/100 ml)	RBC (millions/cu. mm)	WBC (cells/cu.mm)	Differential leucocyte count (%)		
				Lymphocytes	Monocytes	Granulocytes
Control	12.08±0.348	5.20±0.347	5583.33±334.94	78.00±3.89	5.50±1.04	16.66±3.07
TC	13.22±0.24 **	6.27±0.53 *	6043.±349.23 **	78.33±4.32 ns	6.00±2.28 ns	17.5±4.27 ns

Groups	PCV %	MCV	MCH
Control	29.89 ± 1.16	90.02 ± 0.47	32.83 ± 0.11
TC	37.0 ± 0.43**	95.54 ± 1.09**	37.43 ± 0.52 **

n=6; Values are expressed as mean ± S.D followed by Students Paired 'T' Test

ns – non significant when compared to control groups

***p<0.001 **p<0.01

Table-3

Effect of Siddha formulation TC on Biochemical markers of liver and kidney after 14days repeated oral dosing in rats

Groups	ALP (K.A.Units)	AST (IU/L) SGOT	ALT (IU/L) SGPT	Urea (mg/100ml)	BUN (mg/ 100ml)	Glucose Mg.dl	Cholestrol Mg/dl
Control	2.973 ±0.3929	79.89 ±1.906	25.48 ±2.93	16.38 ±2.12	7.52 ±0.84	85.06 ±5.34	57.64 ±4.54
Test drug TC	2.850 ±0.2074 ns	82.3 ±5.164 ns	24.75 ±0.88 ns	16.93 ±0.79 ns	7.92 ±0.37ns	89.23 ±10.21ns	64.07 ±2.73ns

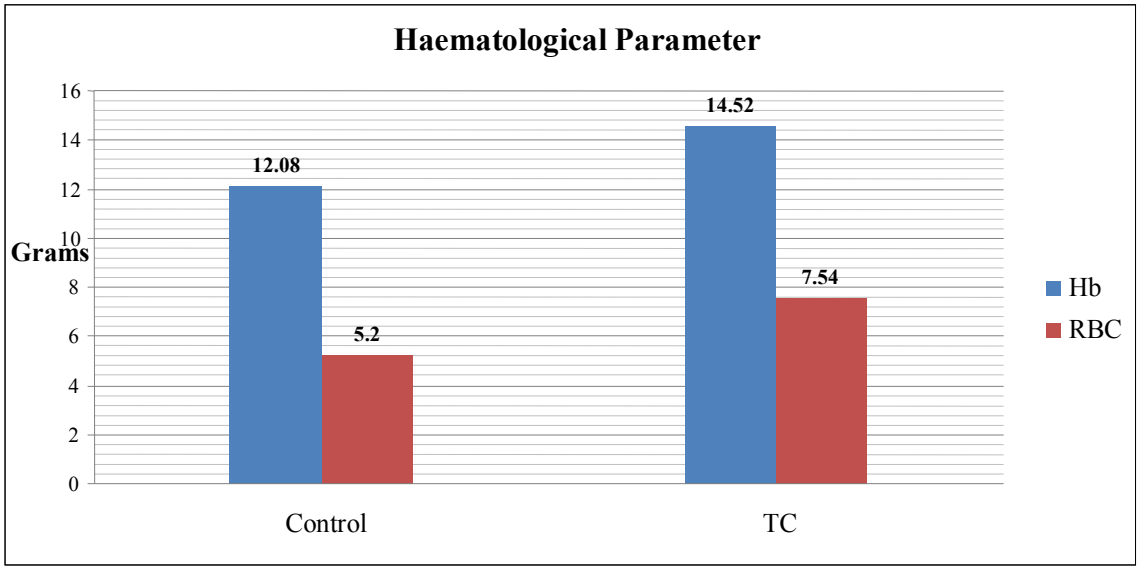
n=6; Values are expressed as mean ± S.D followed by Students Paired 'T' Test

ns – non significant when compared to control groups

Table-4

Effect of Siddha Formulations TC on Haematological parameters after 21 days treatment

Groups	Hb (gm/100 ml)	RBC (millions/cu. mm)	WBC (cells/cu.mm)	Differential leucocyte count (%)		
				Lymphocytes	Monocytes	Granulocytes
Control	12.08±0.348	5.20±0.347	5583.33±334.94	78.00±3.89	5.50±1.04	16.66±3.07
TC	14.52±0.74***	7.54±0.55***	6432.±152.23**	80.37±4.12ns	5.00±2.88ns	16.2±2.12ns



Observations

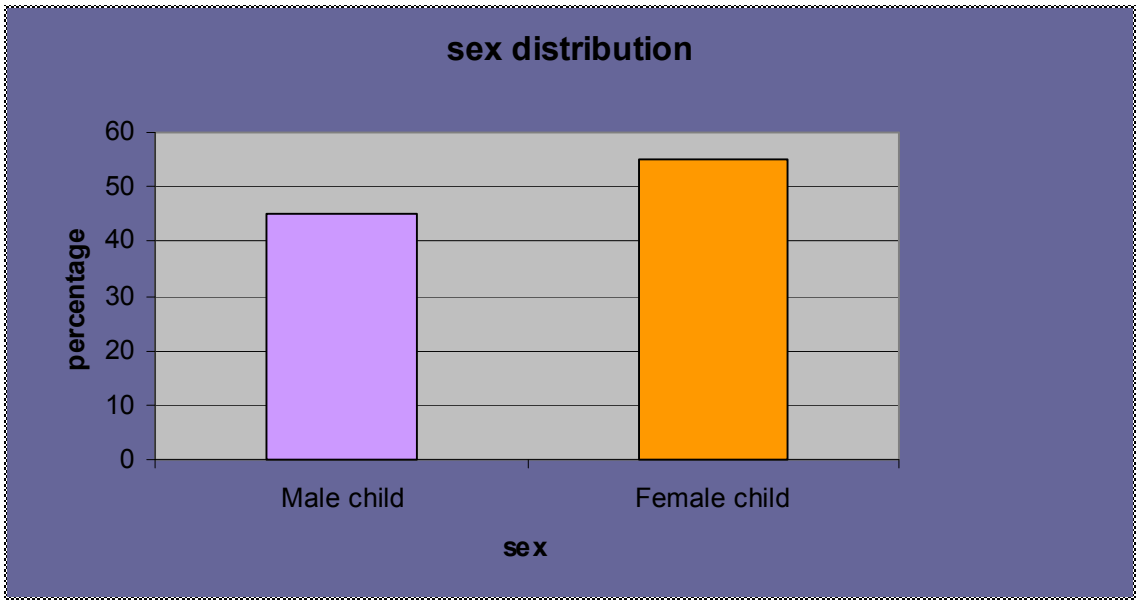
For this clinical study 40 cases were selected and treated in the In-patient department and Out-patient department of Ayothidoss Pandithar Hospital, National Institute of Siddha, Chennai-47. Results were observed with respect to the following criteria.

1. Sex distribution
2. Age distribution
3. Religion distribution
4. Family distribution
5. Socio-economic status
6. Dietary habits
7. Reference to Etiological factors
8. Seasonal Reference
9. Reference to Thinai
10. Reference to Ezhu udal kattugal
11. Reference to Ennvagai thervukakl
12. Neerkkuri and Neikkuri reference
13. Signs and symptoms of Pandu noi during admission and discharge
14. Statistical analysis
15. Results after treatment

The observation recorded are given below in tabular form

1. Sex distribution

S. No	Sex	No of cases	Percentage
1	Male child	18	45
2	Female child	22	55

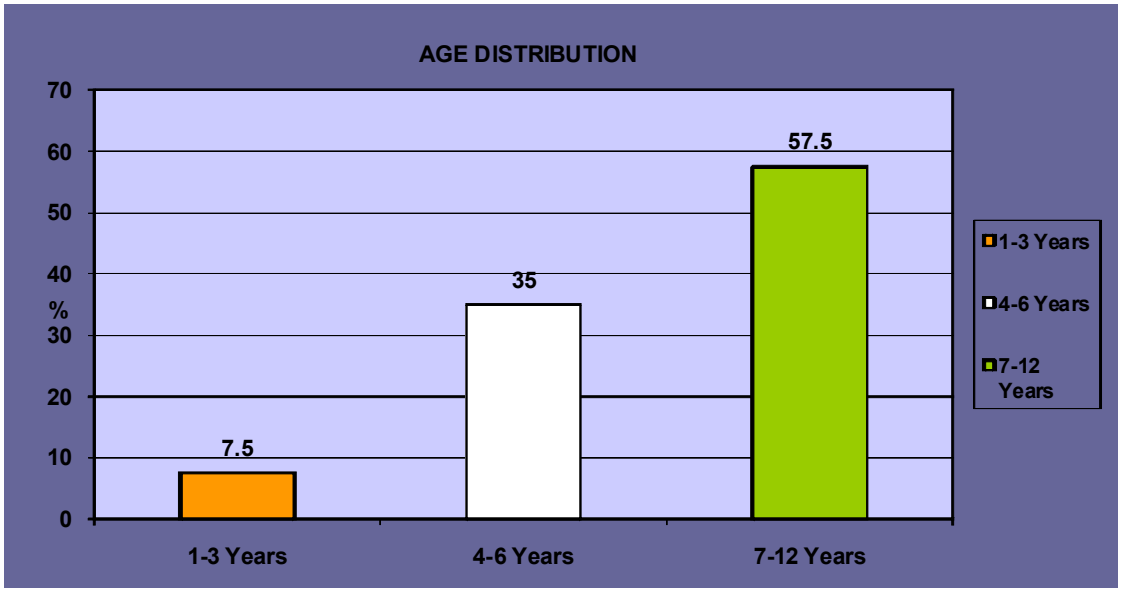


Among the 40 cases treated 18 patients were male children and 22 were female children. The percentage is more in the case of female children

.

2. Age distribution

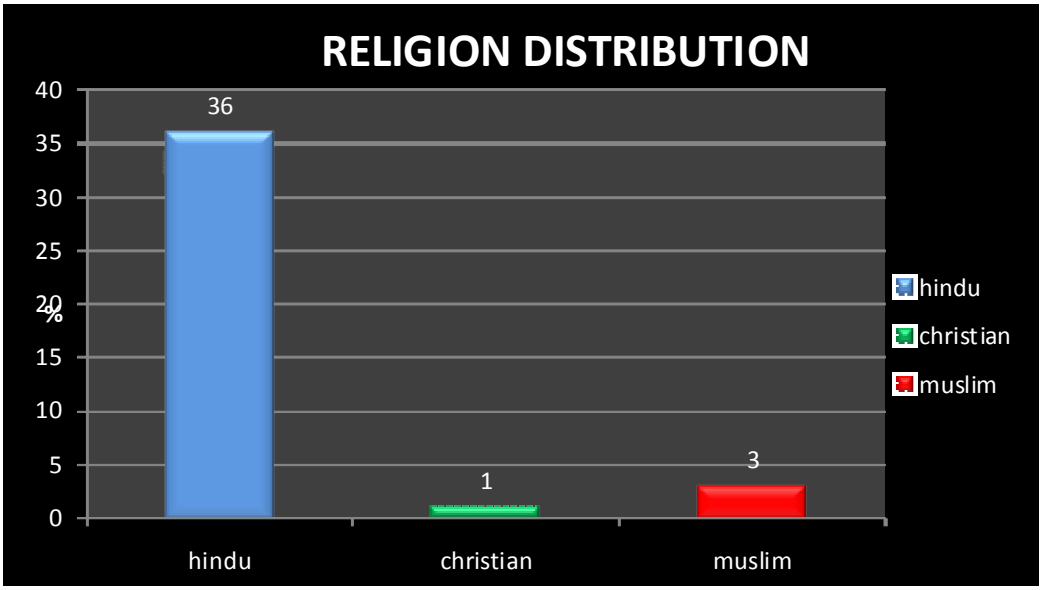
S. No	Age	No of cases	Percentage
1	1 – 6 months (Kaappu paruvam)	-	-
2	7 – 12 months (Senkeerai paruvam)	-	-
3	1 – 3 years (Thalattu, Sappani, Mutha and Varugai paruvams)	3	7.5
4	4 – 6 years (Pillai paruvam)	14	35
5	7 – 12 years (Siruparuvam-male child. Paethai and Pethumbai – female child)	23	57.5



Among the 40 cases treated 14 cases (35%) belonged to 4- 6 years and 23 cases (57.5%) belonged to 6- 12 years. The percentage is more in the age group of 6 – 12 years.

3. Religion distribution

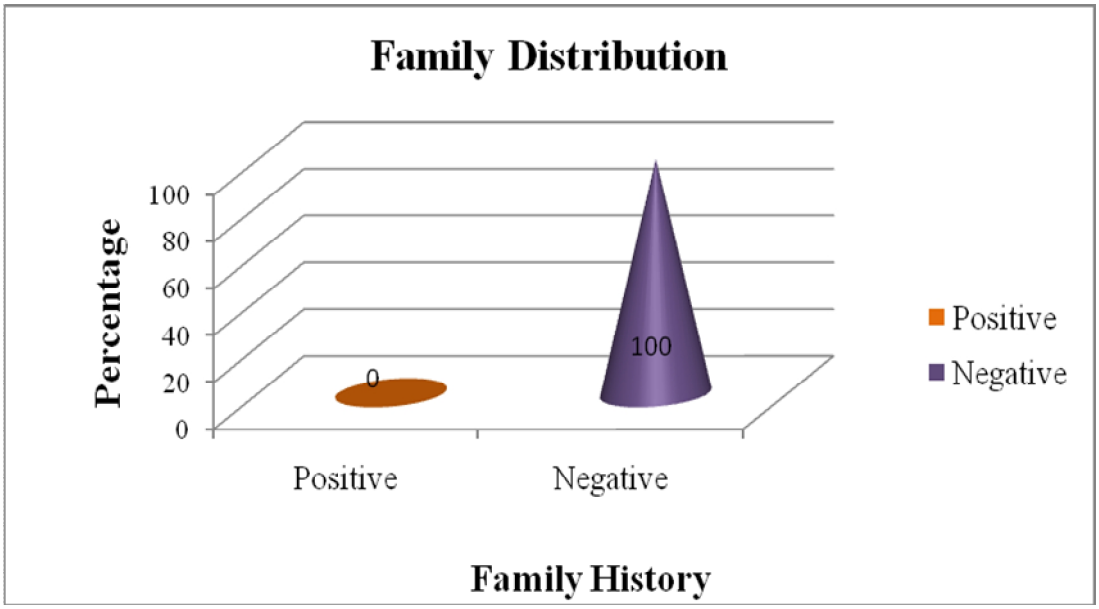
S. No	Religion	No of cases	Percentage
1	Hindu	34	85
2	Christian	2	5
3	Muslim	4	10



Out of 40 cases, 34 cases (85%) belonged to Hindu, 2cases (5%) from Christian religion and 4 cases (10%) belonged to Muslim.

4. Family Distribution

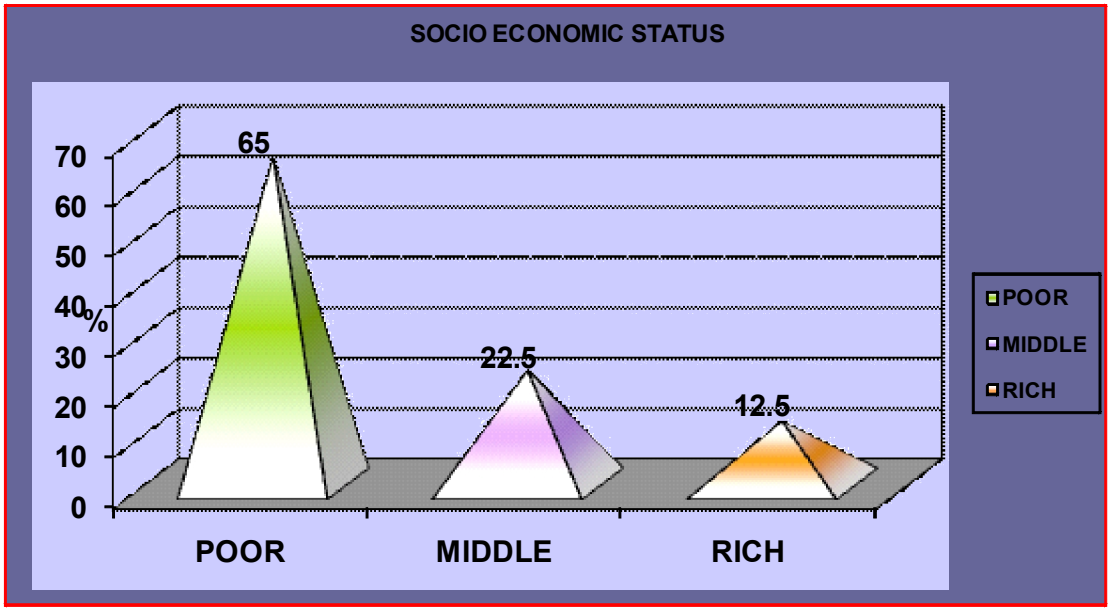
S. No	Family History	No of cases	Percentage
1	Positive	-	-
2	Negative	40	100



All the 40 cases had negative family history

5. Socio-economic status

S. No	Economic Status	No of cases	Percentage
1	Poor	26	65
2	Middle class	9	22.5
3	Rich	5	12.5

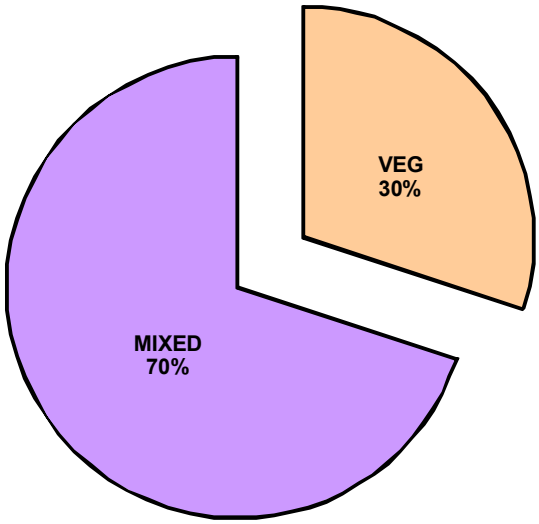


Out of 40 cases, 26 cases belonged to poor socio-economic status.

6. Dietary habits

S. No	Diet	No of cases	Percentage
1	Vegetarian	12	30
2	Mixed	28	70

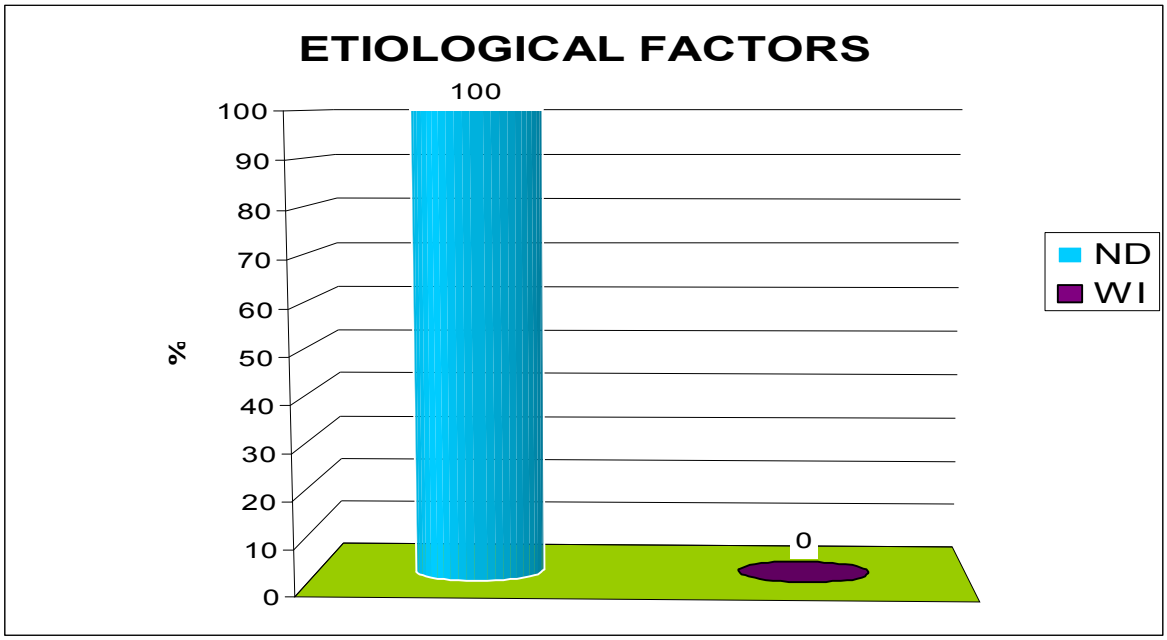
DIETARY HABITS



70 % of cases belonged to mixed diet and 30% belonged to vegetarian diet habits.

7. Etiological factors

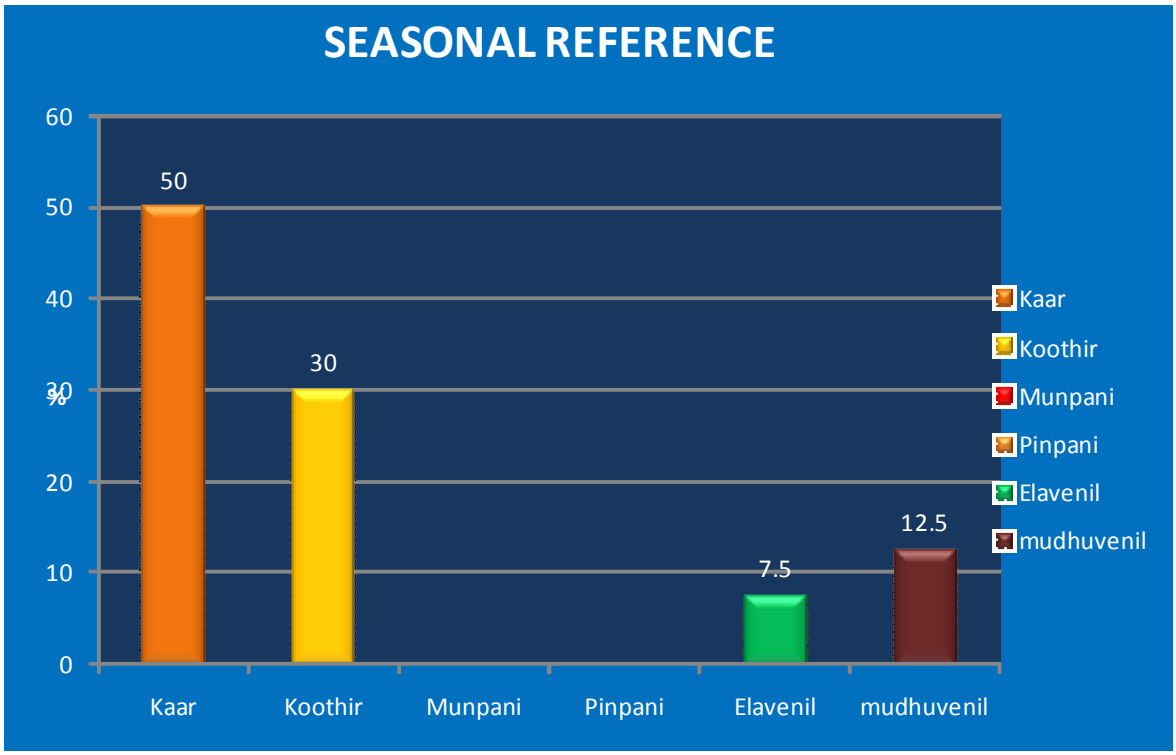
S. No	Etiological factors	No of cases	Percentage
1	Nutritional deficiency	45	100
2	Worm infestation	-	-



Out of 45 cases treated, the etiological factor was found to be nutritional deficiency in 100% cases.

8. Seasonal Referance

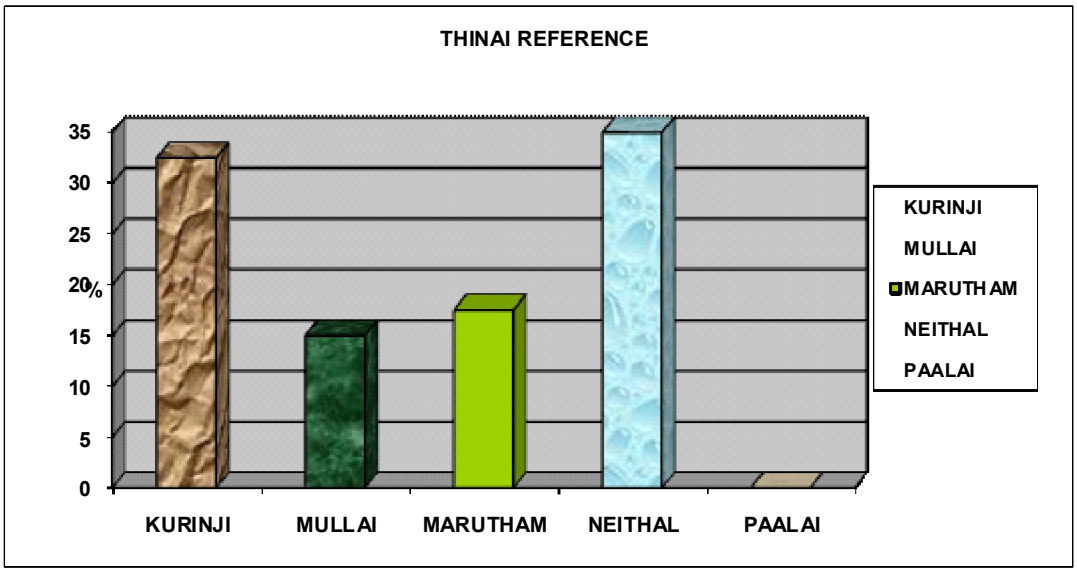
S. No	Paruva kaalam	No of cases	Percentage
1	Kaar(Avani, Purattasi)	20	50
2	Koothir(Iypasi, Karthigai)	12	30
3	Munpani(Margali, Thai)	-	-
4	Pinpani(Masi, Pankuni)	-	-
5	Elavenil(Chithirai, Vaikasi)	3	7.5
6	Mudhuvenil(Aani, Aadi)	5	12.5



Among the 40 cases, 20 cases (50%), Belonged to Kaarkalam, 12 Cases (30%) Belonged to Koothir Kalam, 3 Cases (7.5%), Belonged to Elavenil Kalam, 5 Cases (12.5%), Belonged to Mudhuvenil Kalam. The percentage is more in the kaar and Koothir kalam.

9. **Thinai reference**

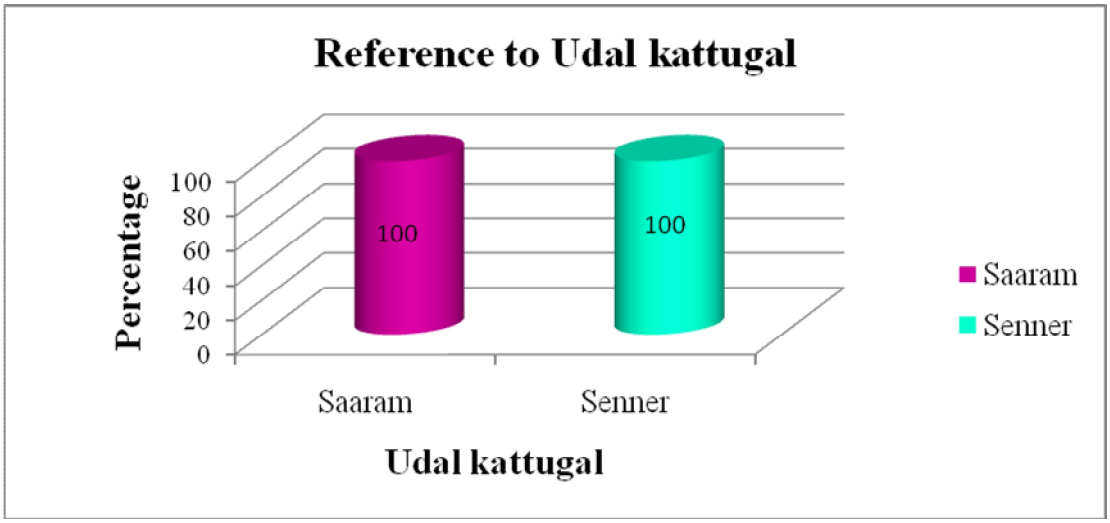
S. No	Thinai	No of cases	Percentage
1	Kurinji (Hill area)	13	32.5
2	Mullai (Forest area)	6	15
3	Marutham (Fertile area)	7	17.5
4	Neithal (Coastal area)	14	35
5	Paalai (Desert area)	-	-



Among the 40 cases, 32.5 % belonged to Kurinji nilam, 15 % belonged to Mullai nilam, 17.5 % belonged to Marutham nilam and 35 % belonged to Neithal nilam.

10. Reference to Udal kattugal

S. No	Udal kattugal	No of cases	Percentage
1	Saaram	40	100
2	Senner	40	100
3	Oon	-	-
4	Kozhuppu	-	-
5	Enbu	-	-
6	Moolai	-	-
7	Sukkilam/suronitham	Not applicable	Not applicable

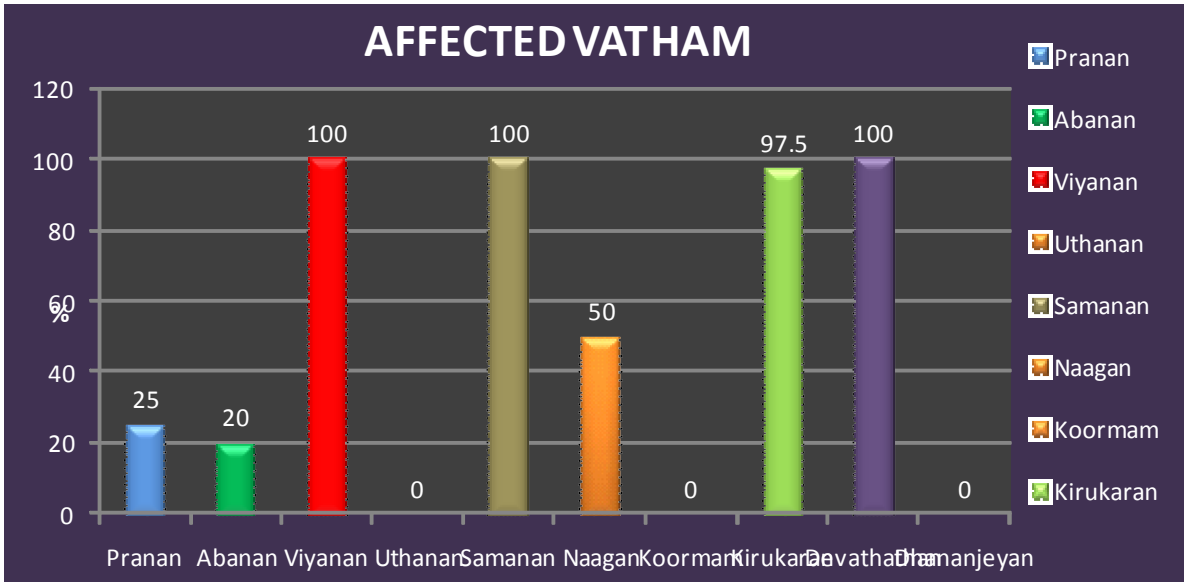


Regarding seven Udal kattugal, Saaram and Senner were affected in all 40 patients100%

11.Reference to Mukkutram

a. Affected Vatham

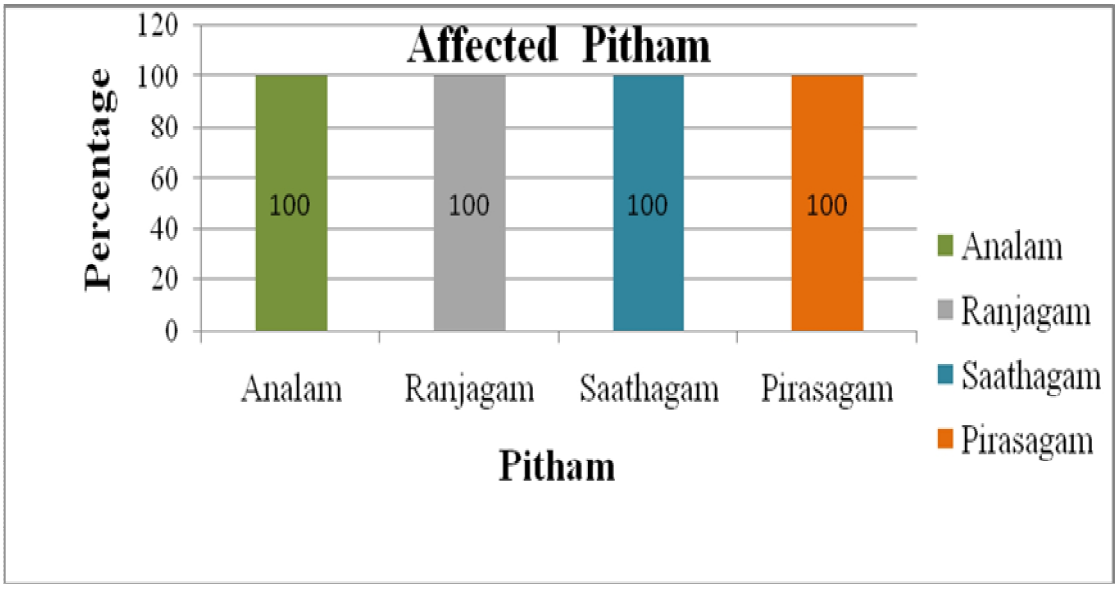
S. No	Vatham	No of cases	Percentage
1	Pranan	10	25
2	Abanan	8	20
3	Viyanan	40	100
4	Uthanan	-	-
5	Samanan	40	100
6	Naagan	20	50
7	Koormam	-	-
8	Kirukaran	39	97.5
9	Devathathan	40	100
10	Dhananjeyan	Not Applicable	Not Applicable



Among 10 types of Vatham Pranan, Abanan, Viyanan, Samanan, Naagan, Kirukaran and Devathathan were affected.

b. affected Pitham

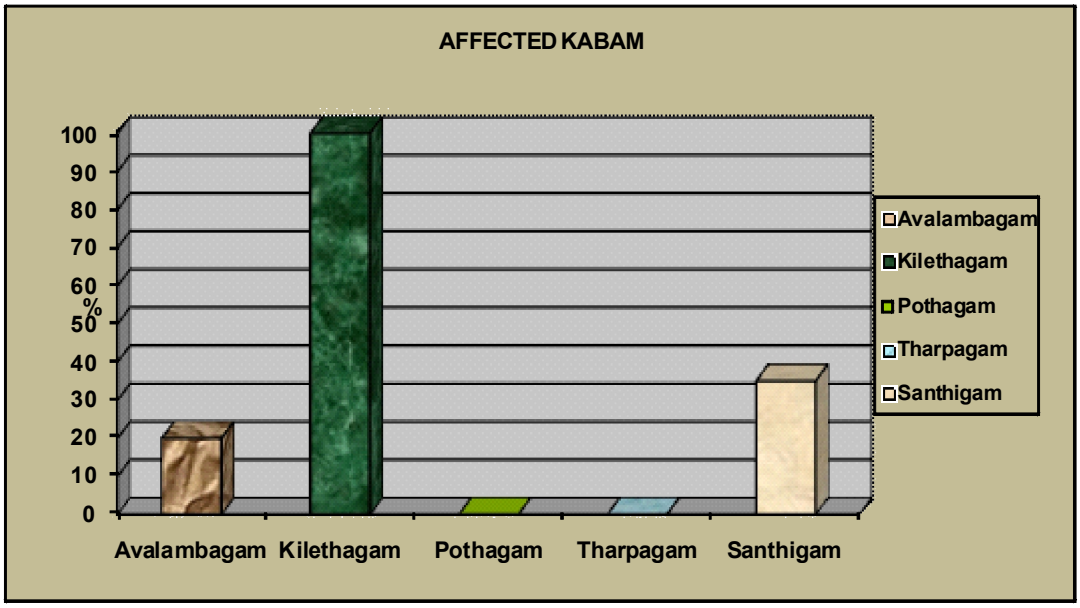
S. No	Pitham	No of cases	Percentage
1	Analam	40	100
2	Ranjagam	40	100
3	Saathagam	40	100
4	Alosagam	-	-
5	Pirasagam	40	100



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

c. Affected Kabam

S. No	Kabam	No of cases	Percentage
1	Avalambagam	8	20
2	Kilethagam	40	100
3	Pothagam	-	-
4	Tharpagam	-	-
5	Santhigam	14	35



Avalambagam was affected in 8 cases, Kilethagam was affected in all the cases. And Santhigam was affected in 14 cases.

12. Reference to Ennvagai thervukal

S. No	Ennvagai thervukal	No of cases	Percentage
1	Naadi: Vathapitham	21	52.5
	Pithavatham	12	30
	Pithakabam	7	17.5
2	Sparisam	40	100
3	Naa	40	100
4	Niram	40	100
5	Mozhi	-	-
6	Vizhi	40	100
7	Malam	8	20
8	Moothiram	13	32.5

Among the Ennvagai thervukal, Naa, Niram Vizhi and Sparisam were affected in all the cases (100%)

13.Reference to Neerkuri Neikuri

S. No	Type of test	No of cases	Percentage
1	Neerkuri	18	45
2	Neikuri		
	Ring(azhi) form	27	67.5
	Pearl (muthu) form	13	32.5

Out of 40 Cases 27 cases (67.5%) belonged to Ring(azhi) form, 13 cases (32.5%), belonged to Pearl (muthu) form

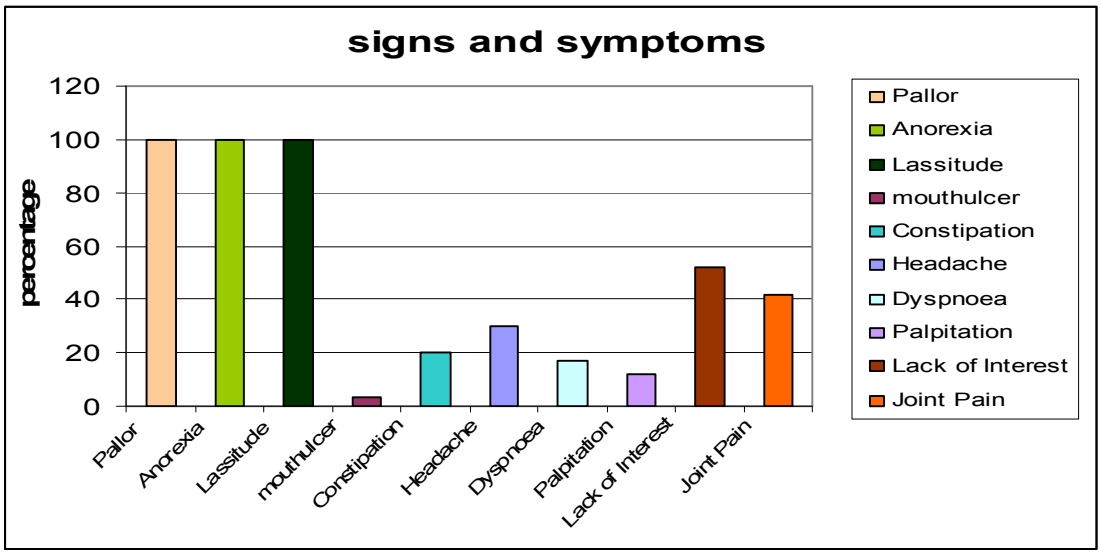
14. Seasonal Reference

S. No	Kaalam	No of cases	Percentage
1	Vatham	40	100
2	Pitham	-	-
3	Kabam	-	-

All the cases were in Vatha kaalam

15.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	40	10
2	Anorexia	40	-
3	Lassitude	40	5
4	Ulceration of mouth	1	-
5	Constipation	8	-
6	Headache	12	3
7	Dysnoea on exertion	7	2
8	Palpitation	5	2
9	Lack of Interest	21	5
10	Joint Pain	13	2



Regarding science and symptoms pailor, anorexia, lassitude were percentage in all 40 patient (100%)

16. Statistical analysis

Paried – t test is used to determine the significance of treatment before and after on Hb.

Variable	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf.	Interval]
hbbt	40	8.9834	.1501941	1.062027	8.681575	9.285225
hbat	40	11.523	.1349842	.9544819	11.25682	11.79926
diff	50	-2.5396	.0152099	.1075451	-2.575245	-2.514035

Ho: mean(hbbt - hbat) = mean(diff) = 0

Ha: mean(diff) < 0	Ha: mean(diff) ~= 0	Ha: mean(diff) > 0
t = -18.3361	t = -18.3361	t = -18.3361
P < t = 0.0000	P > |t| = 0.0000	P > t = 1.0000

There is significant difference between before and after treatment on Hb (P<0.0001)

17. Results after treatment

Results were observed on the basis of two main criteria. One on the basis of clinical improvement and the other on the results derived from the blood picture.

a.Results from clinical improvement

Good, moderate, mild improvements were assessed on the basis of relived signs and symptoms as follows

Good improvement

Anorexia-nil

. Lassitude-nil

Pallor of conjunctiva and nail beds-nil

Dyspnoea on exertion-nil

Lack of interest –improved

Moderate improvement

Anorexia-nil

Lassitude-nil

Pallor of conjunctiva and nail beds - improved

Dyspnoea on exertion - moderately improved

Lack of interest – slightly improved

Mild improvement

Anorexia-nil

Lassitude – on & off

Pallor of conjunctiva and nail beds - present

Dyspnoea on exertion - present

Lack of interest –present

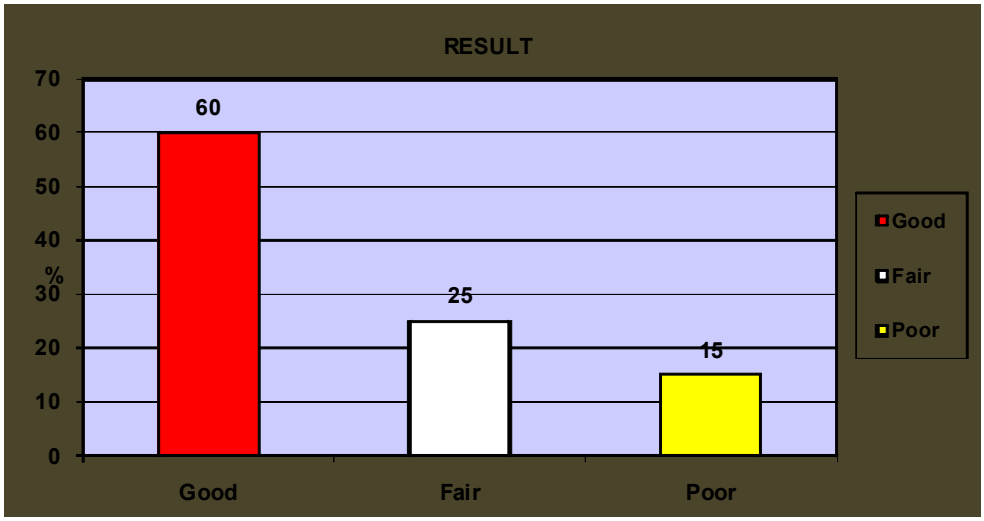
Among the 40 cases, 24 cases assessed as good improment,10 cases assessed as moderate improvement and 6 cases assessed as mild improvement.

b) Results derived from the blood picture

Among the 40 cases 24 cases showed hemoglobin 11.5 – 13 gms/dl, 10 cases showed hemoglobin 11 – 11.9 gms/dl and 6cases showed hemoglobin 10 – 11 gms/dl.

Among the 40 cases studies the results were observed as follows

S. No	Results	No of cases	Percentage
1	Good	24	60
2	Fair	10	25
3	Poor	6	15



Among the 40 cases treated, 24 cases 70%) showed good result, 10 cases (25%) showed fair result and 6 cases (15%) showed poor result. The results were based on clinical improvement and results derived from the blood picture.

BLOOD INVESTIGATION BEFORE TREATMENT

S.No	OPD Number	Name of the Patient	Age/ Sex	HB-gm/dL	TC-Cells/µl	DC in %	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
1	**C94839**	R.Bagyalakshmi	12/FC	9.7	10000	P-50,L-41,E-6,M-3	32	71.6	24.6	33.4	4.5	3.8	Hypochromic Microcytic	Normal Morphology	NIL
2	**C95982**	R.Jeevika	09/FC	9	5500	P-31,L-55,E-12,M-2	34.4	80.8	26.8	34.2	3.8	2.3	Hypochromic Microcytic	Normal Morphology	NIL
3	**D006722**	P.Maheswaran	12/MC	9.1	7500	P-65,L-30E-4,M-1	35.1	72.8	24.1	33	3	3	Hypochromic Microcytic	Normal Morphology	NIL
4	**C61851**	T.Kishorekumar	05/MC	9.8	9300	P-68,L-23,E-18,M-1	35.6	77.4	27.8	36	2.7	3.6	Hypochromic Microcytic	Normal Morphology	NIL
5	**D003289**	Vishnupriya	12/FC	10.4	7200	P-45,L-44,E-5,M-1	38.5	81.1	29.1	35.8	3	3.7	Hypochromic Microcytic	Normal Morphology	NIL
6	**B64682**	Nareshkumar	06/MC	9.8	10600	P-40,L-54,E-6	25.5	60.6	18.6	26.6	2.5	3.36	Hypochromic Microcytic	Normal Morphology	NIL
7	**D004178**	S.Keerthika	08/FC	9	7500	P-50,L-44E-0.6	32.2	68.8	22.4	32.6	3.2	3.6	Hypochromic Microcytic	Normal Morphology	NIL
8	**D004179**	S.Mukesh	07/MC	9.5	8200	P-43,L-52,E-0.5	36.8	76.5	27.2	35.6	2.9	3.8	Hypochromic Microcytic	Normal Morphology	NIL
9	**C88766**	V.PratheepKanna	12/MC	9	7000	P-58,L-36,E-4,M-2	36	68.5	78.5	23.5	2.3	2.9	Hypochromic Microcytic	Normal Morphology	NIL
10	**D004763**	S.Arun	12/MC	8.9	9000	P-40,L-54,E-6	35.6	81.3	29.5	26.2	2.3	3.3	Hypochromic Microcytic	Normal Morphology	NIL
11	**C98887**	S.Iswaraya	04/FC	9.5	8500	P-49,L-46E-5,	30	68.9	20.9	34.1	4	3.6	Hypochromic Microcytic	Normal Morphology	NIL
12	**C96477**	V.Krithika	09/FC	8.3	10000	P-51,L-40,E-6M-3	23	61	24	23.2	4.5	3.1	Hypochromic Microcytic	Normal Morphology	NIL
13	**D00157**	G.Praveen	04/MC	9.3	11800	P-39,L-49E-11,M-1	22	65	25	22.3	4	3.6	Hypochromic Microcytic	Normal Morphology	NIL
14	**C87217**	E.Pavithra	03/MC	9.3	14900	P-405,L-55,E-5	35.4	77.8	27	34	2.1	3.5	Hypochromic Microcytic	Normal Morphology	NIL

BLOOD INVESTIGATION BEFORE TREATMENT															
S.No	OPD Number	Name of the Patient	Age/Sex	HB-gm/dL	TC-Cells/µl	DC in%	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
15	**C95889**	J.Gouthaman	05/MC	9..a6	10300	P-28,L-70,E-2	37.8	76.1	28.2	37	1.3	3.8	Hypochromic Microcytic	Normal Morphology	NIL
16	**B64681**	Lavanya	10/FC	10	6000	P-46,L-50,E-4	36.9	82.4	29.9	30.3	2.2	4.4	Hypochromic Microcytic	Normal Morphology	NIL
17	**C95982**	I.Ajeera	04/FC	9.5	10700	P-63,L-32,P-4,M-1	35.9	78.7	26.8	34	4.5	4.1	Hypochromic Microcytic	Normal Morphology	NIL
18	**C89216**	V.Vijayalakshmi	03/FC	10.8	15500	P-31,L-48,E-20,M-1	33.2	71.9	23.4	32.5	4.7	4	Hypochromic Microcytic	Normal Morphology	NIL
19	**C89215**	V.Rajeswari	06/FC	8.9	12700	P-60,L-45,E-5	32.5	68.3	19.3	28.3	6.9	4.2	Hypochromic Microcytic	Normal Morphology	NIL
20	**D003075**	N.Prasath	11/MC	7.9	9800	P-54,L-43,E-3	31.6	62.1	18.3	29.4	3.1	5	Hypochromic Microcytic	Normal Morphology	NIL
21	**D006422**	S.Deepikasri	04/FC	10	8000	P-63,L-37	30.6	62.1	17.3	25.2	1.8	3.5	Hypochromic Microcytic	Normal Morphology	NIL
22	**D003905**	J.Pooja	11/FC	9.8	9000	p-52,L-43,E-5	28.4	71.2	17.5	23.9	2	3.6	Hypochromic Microcytic	Normal Morphology	NIL
23	**C88373**	M.Mathivanan	12/MC	9.5	6200	P-60,L-35,E-5	25.9	68.9	27	34.3	2.9	4	Hypochromic Microcytic	Normal Morphology	NIL
24	**C98659**	E.Motheswaran	07/MC	9.5	9800	P-66,L-30,E-4	34.2	74.5	25.8	33	4.9	4.5	Hypochromic Microcytic	Normal Morphology	NIL
25	**D008631**	F.NaveenSingh	12/MC	8.5	6800	P-60,L-36,E-4	29	33	68	34.5	4.7	3.9	Hypochromic Microcytic	Normal Morphology	NIL
26	**C099668**	E.Dhanusree	10/FC	9.5	7700	P-41,L-52,E-6,M-1	34.3	15.4	26.8	35.6	3.2	4.5	Hypochromic Microcytic	Normal Morphology	NIL
27	**C48120**	S.AbdhulHalad	05/FC	10	8300	p-40,L-54,E-6	30.6	63.9	14.7	23.4	2.6	3.2	Hypochromic Microcytic	Normal Morphology	NIL
28	**C86040**	T.Kaviya	03/FC	9	13400	P-32,L-60,E-7,M-1	30	62	13.8	24	2.7	3.5	Hypochromic Microcytic	Normal Morphology	NIL

BLOOD INVESTIGATION BEFORE TREATMENT															
S. No	**OPD Number**	Name of the Patient	Age/ Sex	HB-gm/dL	TC-Cells/µl	DC in %	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
29	**C85039**	T.Balaji	07/MC	9.5	9200	P-53,L-37,E-I0	37.8	76.1	28.2	37	1.3	3.8	Hypochromic Microcytic	Normal Morphology	NIL
30	**D006423**	S.Datchanasri	04/FC	9.5	10800	P-40,L-40,E-20	30.6	62.1	17.3	25.2	1.8	3.5	Hypochromic Microcytic	Normal Morphology	NIL
31	**C97014**	R.E.SanjaKannan	12/MC	10.2	15400	P-32,L-47,E-20,M-1	33.3	71.8	21.4	32.6	4.5	4.5	Hypochromic Microcytic	Normal Morphology	NIL
32	**C66400**	C.Nivethana	10/FC	9.2	7800	P-50,L-42,E-8	36.9	74.4	27.5	35.5	2.4	4.7	Hypochromic Microcytic	Normal Morphology	NIL
33	**C099077**	A.AshokKumar	05/MC	9	11400	P-34,L-55,E-11	36.3	76.4	28	36.6	4.1	4.5	Hypochromic Microcytic	Normal Morphology	NIL
34	**C69026**	C.Abitha	05/FC	8.5	6800	P-60,L-36,E-4	29	33	68	34.5	4.7	3.9	Hypochromic Microcytic	Normal Morphology	NIL
35	**D002812**	M.AravindBala	07/MC	9.6	7400	P-38,L-49,E-13	35	81.4	28.1	34	2.9	4.1	Hypochromic Microcytic	Normal Morphology	NIL
36	**D005107**	M.Parthiban	06/MC	8.8	5800	P-40,L-56,E-4	37.4	76.3	27.6	35	2.4	3.5	Hypochromic Microcytic	Normal Morphology	NIL
37	**C91731**	K.Lakshmi	11/FC	9	11000	P-46,L-44,E-10	32.7	70.6	18.3	35.3	3.4	4.2	Hypochromic Microcytic	Normal Morphology	NIL
38	**IPD Number 1017**	A.Akshaya	09/FC	10.4	7600	P-39,L-58,E-3	34.3	15.4	26.8	35.6	3.2	4.5	Hypochromic Microcytic	Normal Morphology	NIL
39	**IPD Number 1027**	S.Shobana	04/FC	8.5	10900	P-18,L-77,E-5	29	33	68	34.5	4.7	3.9	Hypochromic Microcytic	Normal Morphology	NIL
40	**C54677**	Umapriyaa	10/FC	9	13400	P-32,L-60,E-7,M-1	30	62	13.8	24	2.7	3.5	Hypochromic Microcytic	Normal Morphology	NIL

BLOOD INVESTIGATION AFTER TREATMENT

S.No	OPD Number	Name of the Patient	Age/Sex	HB-gm/dL	TC-Cells/µl	DC in %	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
1	**C94839**	R.Bagyalakshmi	12/FC	12.7	10300	P-52,L-40,E-6,M-3	33	72	25.6	34.4	4.7	4.5	Normochromic Normo cytic	Normal Morphology	NIL
2	**C95982**	R.Jeevika	09/FC	9	5800	P-33,L-53,E-12,M-2	35.4	81	27.8	35.2	3.9	3.5	Hypochromic Microcytic	Normal Morphology	NIL
3	**D006722**	P.Maheswaran	12/MC	12	7700	P-66,L-31E-3,M-1	36.1	79	25.1	34.2	3.5	4.2	Hypochromic Microcytic	Normal Morphology	NIL
4	**C61851**	T.Kishorekumar	05/MC	11	9500	P-68,L-22,E-8,M-2	35.9	78	28.8	37.4	2.9	4	Normochromic Normo cytic	Normal Morphology	NIL
5	**D003289**	Vishnupriya	12/FC	9.8	7400	P-46,L-42,E-6,M-1	39	82	30.1	36.8	3.6	3.9	Hypochromic Microcytic	Normal Morphology	NIL
6	**B64682**	Nareshkumar	06/MC	11	10800	P-43,L-50,E-7	27.5	61	19.6	27.6	2.8	4	Hypochromic Microcytic	Normal Morphology	NIL
7	**D004178**	S.Keerthika	08/FC	9	7900	P-50,L-43E-,6,M-1	33.2	69	23.4	33.6	3.3	3.7	Normochromic Normo cytic	Normal Morphology	NIL
8	**D004179**	S.Mukesh	07/MC	9.5	8500	P-45,L-50,E-5	37.2	77	28.2	34.6	3	3.8	Hypochromic Microcytic	Normal Morphology	NIL
9	**C88766**	V.PratheepKanna	12/MC	11	7500	P-60,L-34,E-5,M-1	36.5	69	79.5	24.5	2.8	3.5	Hypochromic Microcytic	Normal Morphology	NIL
10	**D004763**	S.Arun	12/MC	12.5	9300	P-40,L-54,E-6	36	82	30.5	27.2	2.7	4.5	Normochromic Normo cytic	Normal Morphology	NIL
11	**C98887**	S.Iswaraya	04/FC	12.5	8700	P-50,L-47E-4	33	69	22.9	36.1	4.3	4.5	Normochromic Normo cytic	Normal Morphology	NIL
12	**C96477**	V.Krithika	09/FC	11.1	10500	P-50,L-41,E-7M-2	26	62	26	24.2	4.7	4	Hypochromic Microcytic	Normal Morphology	NIL
13	**D00157**	G.Praveen	04/MC	12.8	11500	P-40,L-50E-9,M-1	24	66	28	23.3	4.5	4.2	Normochromic Normo cytic	Normal Morphology	NIL
14	**C87217**	E.Pavithra	03/MC	11.8	15100	P-42,L-53,E-5	35	78	29	35	3.1	4	Normochromic Normo cytic	Normal Morphology	NIL

BLOOD INVESTIGATION AFTER TREATMENT															
S.No	OPD Number	Name of the Patient	Age/Sex	HB-gm/dL	TC-Cells/µl	DC in%	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
15	**C95889**	J.Gouthaman	05/MC	12.6	10500	P-30,L-68,E-2	38	77	28.6	37.9	1.8	4.3	Hypochromic Microcytic	Normal Morphology	NIL
16	**B64681**	Lavanya	10/FC	12.5	6300	P-48,L-48,E-4	37.5	82.4	30	30.6	2	4.5	Normochromic Normo cytic	Normal Morphology	NIL
17	**C95982**	I.Ajeera	04/FC	10.5	10900	P-64,L-31E-4,M-1	36	79	27.8	34.5	4.2	4	Hypochromic Microcytic	Normal Morphology	NIL
18	**C89216**	V.Vijayalakshmi	03/FC	9.5	15800	P-33,L-46,E-19,M-2	34	72	24	33	4.8	3.8	Hypochromic Microcytic	Normal Morphology	NIL
19	**C89215**	V.Rajeswari	06/FC	11.2	12900	P-61,L-44,E-5	33	69	20	28.9	7	4.3	Normochromic Normo cytic	Normal Morphology	NIL
20	**D003075**	N.Prasath	11/MC	9.3	10000	P-56,L-42,E-2	32	63	19	29.8	3.9	4.8	Hypochromic Microcytic	Normal Morphology	NIL
21	**D006422**	S.Deepikasri	04/FC	10.1	8500	P-64,L-36	31	62.5	18	26	1.7	3.8	Hypochromic Microcytic	Normal Morphology	NIL
22	**D003905**	J.Pooja	11/FC	12.4	9300	p-53,L-42,E-5	29	72.5	17.8	24.3	2.2	4.2	Normochromic Normo cytic	Normal Morphology	NIL
23	**C88373**	M.Mathivanan	12/MC	12.3	6300	P-61,L-34,E-5	26.5	69	27.5	34.9	2.8	4.2	Normochromic Normo cytic	Normal Morphology	NIL
24	**C98659**	E.Motheswaran	07/MC	11.9	9950	P-65,L-31,E-4	35	75	26	33.9	4.7	4.3	Normochromic Normo cytic	Normal Morphology	NIL
25	**D008631**	F.NaveenSingh	12/MC	12.7	6850	P-61,L-35,E-4	30	34	68.5	34.9	4.5	4.3	Normochromic Normo cytic	Normal Morphology	NIL
26	**C099668**	E.Dhanusree	10/FC	11.9	7750	P-40,L-53,E-6,M-1	35	16	27	35.7	3.4	4.3	Normochromic Normo cytic	Normal Morphology	NIL
27	**C48120**	S.AbdhulHalad	05/FC	11.9	8500	p-42,L-52,E-6	31.5	66	19	25.1	2.9	4.2	Normochromic Normo cytic	Normal Morphology	NIL
28	**C86040**	T.Kaviya	03/FC	11.5	13600	P-34,L-60,E-6,M-1	32	63	14.5	25.5	3	4.3	Normochromic Normo cytic	Normal Morphology	NIL

BLOOD INVESTIGATION AFTER TREATMENT															
S.No	OPD Number	Name of the Patient	Age/Sex	HB-gm/dL	TC-Cells/µl	DC in %	PCV-%	MCV-fL	MCH-pg	MCHC-gm/dL	PLC-Lakhs/µl	TRBC-C millions/µl	SemearStudy		Semear For MP
													RBC	WBC	
29	**C85039**	T.Balaji	07/MC	12.5	9300	P-56,L-34,E-I0	37.8	76.3	28.6	37.9	1.4	4.3	Normochromic Normo cytic	Normal Morphology	NIL
30	**D006423**	S.Datchanasri	04/FC	11.9	10900	P-42,L-39,E-19	31.6	62.5	17.6	25.6	1.9	4	HypochromicMicrocytic	Normal Morphology	NIL
31	**C97014**	R.E.SanjaKannan	12/MC	11.9	15500	P-34,L-45,E-19,M-2	33.5	72.3	21.8	32.8	4.6	4.6	Normochromic Normo cytic	Normal Morphology	NIL
32	**C66400**	C.Nivethana	10/FC	12.6	7950	P-48,L-43,E-9	37.9	74.6	27.8	35.9	2.5	4.8	Normochromic Normo cytic	Normal Morphology	NIL
33	**C099077**	A.AshokKumar	05/MC	11.6	11600	P-35,L-55,E-10	37.3	77.4	28.5	36.9	4.3	4.3	Normochromic Normo cytic	Normal Morphology	NIL
34	**C69026**	C.Abitha	05/FC	11.5	7700	P-62,L-34,E-4	29.5	33.9	68.8	34.9	4.8	4.2	Normochromic Normo cytic	Normal Morphology	NIL
35	**D002812**	M.AravindBala	07/MC	12.2	7700	P-37,L-51,E-12	35.3	81.6	28.6	33.8	3.2	4.5	Normochromic Normo cytic	Normal Morphology	NIL
36	**D005107**	M.Parthiban	06/MC	11.9	6000	P-40,L-55,E-5	37.8	76.8	27.8	34.9	2.6	4	Normochromic Normo cytic	Normal Morphology	NIL
37	**C91731**	K.Lakshmi	11/FC	13	11500	P-48,L-44,E-11	32.9	70.8	18.8	35.5	3.9	4.8	Normochromic Normo cytic	Normal Morphology	NIL
38	**IPD Number 1017**	A.Akshaya	09/FC	11.2	7900	P-40,L-57,E-3	34.6	15.6	26.9	35.7	3.6	4.6	HypochromicMicrocytic	Normal Morphology	NIL
39	**IPD Number 1027**	S.Shobana	04/FC	8.2	11000	P-28,L-67,E-5	29.5	33.8	68.6	34.9	4.9	3.6	HypochromicMicrocytic	Normal Morphology	NIL
40	**C54677**	Umapriyaa	10/FC	9	13700	P-36,L-57,E-5,M-2	29.8	62.8	13.9	24.5	3	3.5	HypochromicMicrocytic	Normal Morphology	NIL

DISCUSSION

- In preclinical study the result reveals, the dose level of 180mg/kg bw is highly safety.
- Haematological studies reveals increased level of parameters like **Hb%** and **Rbc** count.
- From the Clinical Study the following point were taken for discussion.

Sex

According to the clinical study, female children were affected more than male children. 45% of the cases were found to be male children and 55% were found to be female children. But in the Siddha literatures, there is no reference of incidence of pandu to a specific sex in children age.

Age

The age limit for the cases taken for study ranged from 3 to 12 years. Among the affected children 7.5% were found to be within the age limit of 1 - 3 Years. 35% were found to be within the age limit of 4 – 6 years and 57.5% were found to be within the age limit of 7 – 12 years.

Socio-economic status

In this pesent study 65% of cases were belonging to poor socio-economic status. Poor socio-economic status is a main predisposing factor, since the poor people usually consume low nutritional food.

Paruva Kaalam

Most of cases, subjected to this study existed predominantly in kaarkalam and less in koothir and venil kaalam. According to the siddha concept, Thannilai valarchi of pitham is during kaar kaalam.

Thinai

Among 40 cases that were affected, 13 cases are from Kurinji, 6 cases from Mullai, 7 cases from Marutham, 14 cases from Neithal Nilam. On doing this study most of the anemic children are came from kurinji nilam.

Etiology

Generally Pandu noi is mainly due to dietic factors, which cause vitiation of pitham and kabam. History of patients reveals that differential intake of food and food habits, worm infestation, excessive intake of ash, soil and clay (h/o pica) wandering in hot climate, over intake of salt, sour and pungent food items and malnutritious diet cause this disease.

In modern Paediatrics, the etiology is based upon malnutrition and worm infestation. In both systems, it is stated that any factor which causes vitiation of blood produce this disease.

Mukkutram

Among the three vital forces pitham is mainly affected. That too among the five types of pitham, ranjagam is affected which causes discolouration of mucous membrane. Also the other forms of pitham such as Analam, sathagam, prasagam are affected in 100% of the cases. The derangement of pitham is followed by derangement of kabam and vaatham.

Udal Kattugal

In this saaram and senner were affected in 100% of the cases

Ennvagi Theruvukal

In this study, the changes of Naadi, sparisam, Naa, Niram, Mozhi, Vizhi, Malam, Moothiram are noted.

Naadi

According to this study vathapitha naadi was found in 21 cases, pithavaatha naadi in 12 cases and pithakaaba naadi in 7 cases.

Sparisam

Palpable liver and spleen tenderness may be noted. Palpitation may be noted. The skin of the patient should also be noted.

Naa

In all of the cases, the tongue is pallor in colour

Niram

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

Mozhi

Mostly mozhi is not affected

Vizhi

In iron deficiency anaemia, pallor of conjunctiva was noted in almost all cases

Malam

The colour of the stool is pale yellow in colour. According to their study of patients were affected by hard stool, constipation.

Moothiram

In pandu noi, due to increased state of pitham the urine is yellow or dark yellow in colour.

Neikuri

The neikuri was ring shaped in 27 cases and pearl shaped in 13 cases.

The diagnosis was made on the basis of Envagai Thervukal and available modern investigation methods. In most of the cases, haemoglobin level, total red blood cells, PCV, MCV, MCH, and MCHC are reduced. After treatment there is a tremendous increase in the haemoglobin level, total red blood cells, PCV, MCV, MCH, and MCHC.

Pharmacological analysis

- Pharmacological analysis review that the drugs possess satisfactory hematinic action to treat pandu noi.
- Results of clinical study reveals the capability of the medicine to treat the pandu noi (anemia) Adverse reaction of the medicine was not observed during the coarse of the study.
- qualitative analysis and phytochemical screening showed that the trail medicine having iron in both ferric and ferrous forms and the other important minerals which are necessary to fulfill its therapeutic value.
- Results of pharmacological activity had good correlation with the clinical study report.
- At the end of the study the results reveals the exact preparation was selected for the disease pandu noi.

SUMMARY

- Who defines that “Health is the state of physical, mental social and spirituals wellbeing and not merely the absence of disease and infirmity”.

- Pandu noi is one of the major global disease in children community.prevelence is to be higher in children than adults and elder ones

- So the author focus to treat the pandu noi.with the foremost preparation of the medicine TIRUDHARATCHATHA CHOORANAM(TC)which is mentioned in Agasthiyar vaithiya rathina surukkam..

- The raw drugs were collected from the local market in Chennai.

- The medicine was prepared in NIS gunapadam lab.

- Then the medicine was subjected to the toxicity study.

- After done the preclinical study medicine was subjected to clinical study in NIS, OPD and IPD.

- Simultaneously the pharmacology activities, qualitative analysis of the medicine were evaluated.

CONCLUSION

- Based on the preclinical study the safety dose was 1500mg/kg bw, here we concluded that the therapeutic dose of **TIRUDHARATCHATHA CHOORANAM(TC)** which is mentioned in Agasthiyar vaithya rathina surukkam is a safe dose for clinical trial.

- From the clinical study findings I concluded that the foremost preparation of the medicine was selected is effective singnificantly in the management of Pandu noi.

- It could be one of the unavoidable medicine to treat pandu noi.

- In forthcoming days this study will be redouble by do the standardization of the medicine.

ANNEXURE

Profoma

NATIONAL INSTITUTE OF SIDDHA

AYOTHIDOSS PANDITHAR HOSPITAL

CHENNAI – 600047.

POST GRADUATE DEPARTMENT OF KUZHANDHAI MARUTHUVAM

An open clinical study on **PANDU** with

TIRUDHARATCHATHA CHOORANAM (TC).

CONSENT FORM

CERTIFICATE BY INVESTIGATOR

I certify that I have disclosed all the details about the study in the terms readily understood by the parent/guardian

Signature _____

Date _____

Name _____

CONSENT BY PARENT

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

I am aware of my right to opd my son/daughter out of the trail at any time during the course of the trail without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to include my son/daughter as a subject in the clinical trial of '**TIRUDHARATCHATHA CHOORANAM(TC)** for the treatment of '**PANDU'**

Date : Signature _____

Name _____

Date : Signature of witness _____

Name _____

NATIONAL INSTITUTE OF SIDDHA, CHENNAI-47

AYOTHIDOSS PANDITHAR HOSPITAL

DEPARTMENT OF KUZHANDHAI MARUTHUVAM

AN OPEN CLINICAL TRAIL OF SIDDHA DRUGS TIRUDHARATCHATHA CHOORANAMAND FOR THE TREATMENT OF PANDU IN CHILDREN

SELECTION PROFORMA

1. O.P /I.P No : Bed No: 2. S.NO ☐☐

3. Name : _____ 4. Age (Yr) : ☐☐

5. Gender : M ☐ F ☐

6. Father's Name :

7. Father's Occupation :

8. Mother's Name :

9. Monthly/Annual Income :

10. Religion :

11. Socio Economical Level :

12. Informant : Mother ☐ Father ☐ Guardian ☐

13. Postal Address : _____

_____

_____

Contact No. _____

14. Complaints and duration : _____

_____

_____

15. History of present illness : _____

16. Past History : _____

17. Immunization History : _____

18. Family History : 1. No ☐ 2. Yes ☐

19. Food Habits : 1. Veg ☐ 2. Non Veg ☐ 3. Mixed ☐

GENERAL EXAMINATION

20. Body weight (Kg) : ☐☐

21. Height (Cms) : ☐☐☐

22. Body temperature (F) : ☐☐☐

23. Heart rate/minute : ☐☐

24. Respiratory rate/minute : ☐☐

25. Pulse rate/minute : ☐☐

		1. YES	**2. NO**
26. Pallor	:	☐	☐
27. Jaundice	:	☐	☐
28. Clubbing	:	☐	☐
29. Cyanosis	:	☐	☐
30. Pedal Oedema	:	☐	☐
31. Lymphadenopathy	:	☐	☐
32. H/o Pica	:	☐	☐
33. Engorged veins	:	☐	☐

34. Abdominal Distension : ☐ ☐

35. Congenital Abnormalities : ☐ ☐

36. Jugular Venous Pulsation : ☐ ☐

EXAMINATION OF VITAL ORGANS

37. CVS Normal ☐ Abnormal ☐

38. RS Normal ☐ Abnormal ☐

39. Abdomen Normal ☐ Abnormal ☐

CLINICAL ASSESMENT : 1. YES 2. NO

40. Lassitude : ☐ ☐

41. Fatigue : ☐ ☐

42. Breathlessness on exertion : ☐ ☐

43. Palpitation : ☐ ☐

44. Pallor of skin and mucous membrane : ☐ ☐

45. Tachycardia : ☐ ☐

46. Anorexia : ☐ ☐

47. Irritability : ☐ ☐

48. Lack of interest : ☐ ☐

49. Koilonychia : ☐ ☐

50. Angular stomatitis : ☐ ☐

51. Dyspnoea on exertion : ☐ ☐

52. Recurrent infection : ☐ ☐

CLINICAL EXAMINATION

53. INSPECTION : _____

_____

54. PALPATION : _____

_____

55. PERCUSSION : _____

_____

56. AUSCULTATION : _____

_____

SIDDHA METHODS OF EXAMINATION

57. Nilam

1. Kurinji ☐ 2. Mullai ☐ 3.Marutham ☐

4. Neithel ☐ 5. Paalai ☐

58. Kaala Iyalbu

1. Kaarkalam ☐ 2. Koothirkaalam ☐

3 Munpanikaalam ☐ 4. Pinpanikaalam ☐

5 Illavenirkaalam ☐ 6. Muthuvenirkaalam ☐

59. Yaakai

1. Vatham ☐	2. Vatha pitham ☐	3. Vatha Kabam ☐
4. Pitham ☐	5. Pitha vatham ☐	6. Pitha Kabam ☐
7. Kabam ☐	8. Kaba vatham ☐	9. Kaba pitham ☐

60. Gunam

1. Sathuvam ☐	2. Rasatham ☐	3. Thamasam ☐

PORI PULANGAL

	1. Normal	**2. Affected**	
61. Mei	☐	☐	_____
62. Vaai	☐	☐	_____
63. Kan	☐	☐	_____
64. Mooku	☐	☐	_____
65. Sevi	☐	☐	_____

KANMENDHIRIUM / KANMAVIDAYAM

	1. Normal	2. Affected	
66. Kai	☐	☐	_____
67. Kaal	☐	☐	_____
68. Vaai	☐	☐	_____
69. Eruvai	☐	☐	_____
70. Karuvai	☐	☐	_____

UYIR THATHUKKAL

VATHAM :	**1. Normal**	**2. Affected**	
71. Pranam	☐	☐	_____
72. Abanam	☐	☐	_____
73. Viyanan	☐	☐	_____
74. Uthanan	☐	☐	_____
75. Samanan	☐	☐	_____
76. Nagan	☐	☐	_____
77. Koorman	☐	☐	_____
78. Kirukaran	☐	☐	_____

79. Devathathan ☐ ☐ _____

80. Dhananjeyan ☐ ☐ _____

PITHAM **1. Normal** **2. Affected**

81. Analam ☐ ☐ _____

82. Ranjagam ☐ ☐ _____

83. Saathagam ☐ ☐ _____

84. Alosagam ☐ ☐ _____

85. Prasagam ☐ ☐ _____

KABAM **1. Normal** **2. Affected**

86. Avalambagam ☐ ☐ _____

87. Kilethagam ☐ ☐ _____

88. Pothagam ☐ ☐ _____

89. Tharpagam ☐ ☐ _____

90. Santhigam ☐ ☐ _____

☐

UDAL THATHUKKAL	**1. Normal**	**2. Affected**	
91. Saaram	☐	☐	_____
92. Senneer	☐	☐	_____
93. Oon	☐	☐	_____
94. Kozhuppu	☐	☐	_____
95. Enbu	☐	☐	_____
96. Moolai	☐	☐	_____
97. Sukilam / Suronitham	☐	☐	_____

ENVAGAI THERVUGAL

	1. Normal	**2. Affected**	
98. Naa	☐	☐	_____
99. Niram	☐	☐	_____
100. Mozhi	☐	☐	_____
101. Vizhi	☐	☐	_____
102. Sparisam	☐	☐	_____

MALAM	**1. Normal**	**2. Affected**	
103. Niram	☐	☐	_____
104. Edai	☐	☐	_____
105. Nurai	☐	☐	_____
106. Elagal	☐	☐	_____
107. Erugal	☐	☐	_____

MOOTHIRAM

Neer kuri	**1. Normal**	**2. Affected**	
108. Niram	☐	☐	_____
109. Edai	☐	☐	_____
110. Manam	☐	☐	_____
111. Nurai	☐	☐	_____
112. Enjal	☐	☐	_____

113. Nei kuri

1. Vatham ☐ 2. Pitham ☐ 3. Kabam ☐ 4. Others ☐

114. Naadi

1. Vatham	☐	2.Vatha pitham	☐	3. Vatha kabam	☐
4. Pitham	☐	5. Pitha vatham	☐	6. Pitha kabam	☐
7. Kabam	☐	8. Kaba vatham	☐	9. Kaba pitham	☐

LAB INVESTIGATION

Blood :

115. Hb (gm %)

116. Total RBC

117. Smear study

118. PCV

119. MCV

120. MCHC

121. MCH

122. TC (cells / cumm) ☐☐☐☐

123.Platelet count

124.MP:

DC (%)

123. L ☐☐ 124. N ☐☐ 125. M ☐☐

126. E ☐☐ 127. B ☐☐

128. ESR (mm) ½ Hr ☐☐ 129. ESR (mm) 1 Hr ☐☐

URINE

130. Albumin - Nil ☐ + ☐ ++ ☐ +++ ☐

131. Sugar - Nil ☐ + ☐ ++ ☐ +++ ☐

DEPOSIT

	1. Absent	**2. Present**	
132. Pus cells	☐	☐	_____
133. Epithelial cells	☐	☐	_____
134. Red blood cells	☐	☐	_____
135. Casts / Crystal	☐	☐	_____
136.Bile Salts	☐	☐	_____
137.Bile Pigments	☐	☐	_____

MOTION

136. Ova ☐ ☐ _____

137. Cyst ☐ ☐ _____

138. Occult blood ☐ ☐ _____

INCLUSION CRITERIA

	1. Yes	**2. No**
139. Aged 3 year to 12 years	☐	☐
140. Hb level with < 11 gm/dl for aged 3-6 years and < 12 gm/dl for aged 7-12 years patients	☐	☐
141. Willing to give blood sample for the Investigation when required	☐	☐
142. Smear showing hypochromic microcytic Anaemia	☐	☐
143. pallor of skin, mucous membrane, conjunctiva, nail beds,	☐	☐

EXCLUSION CRITERIA

144. Jaundice	☐	☐
145. Malaria	☐	☐
146. Inherited defects	☐	☐

147. Congenital Heart Diseases ☐ ☐

148. Patient with chronic disease ☐ ☐

149. Smear not showing Hypochromic microcytic anaemia ☐ ☐

150. Patient not willing to give blood sample for investigation.

Admitted to trial 1. Yes ☐ 2. No ☐

151. If yes, S.No: ☐☐

152. Date : _____

153. Station : _____

Signature of Doctor

NATIONAL INSTITUTE OF SIDDHA, CHENNAI-47

AYOTHIDOSS PANDITHAR HOSPITAL

DEPARTMENT OF KUZHANDHAI MARUTHUVAM

AN OPEN CLINICAL TRAIL OF SIDDHA DRUGS TIRUDHARATCHATHA CHOORANAM FOR THE TREATMENT OF PANDU

ASSESSMENT PROFORMA

1. O.P/I.P No : Bed No: 2. S.NO ☐☐

3. Name : _____

4. Date of Admission : ☐☐☐☐☐☐

5. Date of this Assessment : ☐☐☐☐☐☐

6. Day of Assessment : ☐

CLINICAL ASSESMENT :	**1. YES**	**2. NO**
7. Lassitude :	☐	☐
8. Fatigue :	☐	☐
9. Breathlessness on exertion :	☐	☐

10. Palpitation : ☐ ☐

11. Pallor of skin and mucous membrane : ☐ ☐

12. Tachycardia : ☐ ☐

13. Anorexia : ☐ ☐

14. Irritability : ☐ ☐

15. Lack of interest : ☐ ☐

16. Koilonychia : ☐ ☐

17. Angular stomatitis : ☐ ☐

18. Dyspnoea on exertion : ☐ ☐

19. Recurrent infection : ☐ ☐

CLINICAL EXAMINATION

20. INSPECTION : _____

_____

21. PALPATION : _____

_____

22. PERCUSSION : _____

_____

23. AUSCULTATION : _____

_____

TEST IN SIDDHA ASPECTS

ENVAGAI THERVUGAL

	1. Normal	**2.Affected**	
24. Naa	☐	☐	_____
25. Niram	☐	☐	_____
26. Mozhi	☐	☐	_____
27. Vizhi	☐	☐	_____
28. Sparisam	☐	☐	_____

MALAM

	1. Normal	**2.Affected**	
29. Niram	☐	☐	_____
30. Edai	☐	☐	_____
31. Nurai	☐	☐	_____
32. Elagal	☐	☐	_____
33. Erugal	☐	☐	_____

MOOTHIRAM

Neer kuri	**1. Normal**	**2.Affected**	
34. Niram	☐	☐	_____
35. Edai	☐	☐	_____
36. Manam	☐	☐	_____
37. Nurai	☐	☐	_____
38. Enjal	☐	☐	_____

39. Nei kuri

1. Vatham ☐ 2. Pitham ☐ 3. Kabam ☐ 4. Others ☐

40. Naadi

1. Vatham	☐	2.Vatha pitham	☐	3. Vatha kabam	☐
4. Pitham	☐	5. Pitha vatham	☐	6. Pitha kabam	☐
7. Kabam	☐	8. Kaba vatham	☐	9. Kaba pitham	☐

LAB INVESTIGATION

Blood:

41. Hb (gm %)

42. Total RBC

43. Smear study

44. PCV

45. MCV

46. MCH

47. MCHC

48. TC (cells / cumm) ☐☐☐☐

49 Platelet Count

50 MP

DC (%)

51. L ☐☐ 50. N ☐☐ 51. M ☐☐

52. E ☐☐ 53. B ☐☐

53. ESR (mm) ½ Hr ☐☐ 55. ESR (mm) 1 Hr ☐☐

URINE

54. Albumin - Nil ☐ + ☐ ++ ☐ +++ ☐

55. Sugar - Nil ☐ + ☐ ++ ☐ +++ ☐

DEPOSIT

	1. Absent	**2. Present**	
56. Pus cells	☐	☐	_____
57. Epithelial cells	☐	☐	_____
58. Red blood cells	☐	☐	_____
59. Casts / Crystal	☐	☐	_____
60.Bile salts	☐	☐	_____
61.Bile Pigments	☐	☐	_____

MOTION

62. Ova ☐ ☐ _____

63. Cyst ☐ ☐ _____

64. Occult blood ☐ ☐ _____

65. RESULT

1. Good ☐ 2. Moderate ☐ 3.Mild ☐

66. Date _____

67. Station _____

Signature of Doctor

NATIONAL INSTITUTE OF SIDDHA

AYOTHIDOSS PANDITHAR HOSPITAL

CHENNAI – 600047.

POST GRADUATE DEPARTMENT OF KUZHANDHAI MARUTHUVAM

An open clinical study on **PANDU** with **TIRUDHARATCHATHA CHOORANAM(TC).**

WITHDRAWAL FORM

Name : OPD/IPD No:

Age : Reg.No :

Date of trial commencement :

Date of withdrawal from trial :

Reasons for withdrawal

1.Long absence at reportinging :

2.Irregular treatment :

3.Shift of locality :

4.complication/Adverse reactions if any :

5.exaribation of symptoms :

6.patient not willing to continue

Sign of lecturer sign of the principal

BIBLIOGRAPHY

1. Pon Gurusironmary, **Balavaganam (2nd edition)** Tamilnadu Publications Directorate, Chennai -1992

2. Kuppusamy Mudaliar K.N. **Siddha Maruthuvam** Tamilnadu Publications Directorate Chennai - 1987.

3. Ramachandran S.P **Theraiyar Vagadam** , Thamarai Noolagam, Chennai - 2000

4. Uttamarayan K.S **Thotrakirama Aarachiyum, Siddha Maruthuva Varalarum** Tamilnadu Publications Directorate Chennai - 1992.

5. Sambasivam Pillai T.V **Tamil-English Dictionary** Government of Tamilandu - 1994

6. Murugesa Mudaliyar K.S **Gunapadam Mooligai Vaguppu (Part-I)** Tamilnadu Siddha Medical Council - 2003.

7. Thiyagarajan R. **Gunapadam (Part II & Part III)** Indian Medicine and Homeopathy Department, Chennai - 2004.

8. Kannusamy Pillai C. **Sikitcha Rathna Deepam**, Pathna, Nayakkar & Sons, Chennai.

9. Uttamarayan K.S. **Siddha Maruthuvanga Churukkam (2nd Edition)** Siddha Science Department Council, Tamilnadu - 1983.

9. Duraivasan G. **Siddha Hygiene & Preventive Medicine** Indian Medicine & Homeopathy Chennai -1983.

10. Shanmugavelu M. **Siddha Maruthuvam Noinadal Noimuthal Nadu (Part I & Part II)**

11. Ramachandran S.P **Uyir Kakkum Siddha Maruthuvam,** Publishers by Thamarai Noolagam Chennai - 2000.
12. Abdulla Sahib, **Agathyar Vaidhya Pillai Tamil** , Thamarai Noolagam 1998.

13. Ramachandran S.P **Agathyar Paripooranam-400** Thamarai Noolagam.

14. Yugi Mamuniver, **Yugivaidhya Chinthamani** Indian Medical College Chennai - 1998.

15. Somasundaram S. **Medicinal Botany** Elangovan Pathippagam - 1997.

16. Ghai O.P **Essential Paediatries (6th Edition)** CBS-Publishers.

17. Partha Sarathy A. **I.A.P. Tex Book of Paediatries (2nd Edition)** 2002, Jaypee Publications.

18. Guvton C & Hall, **Text Book of Patholoby** XI Edition, Elesevier.

19. Harsh Mohan, **Text Book of Pathology (5th Edition)** Taypee Brothers, Medicinal Publishers Private Limited. New Delhi - 2005.

20. Rubbing and Cotran,**Pathologic basis of disease**, (7th Edition), 2005

21. Vishwanathan J **Achar's Text Book of Paediatries (3rd Edition)** Orient longman Limited, Chennai.

22. Nadkarni K.M, **Indian Metria Medicine,** Popular Prakashan Private Limited 1976.

23. Chopru R.N Nayar S.L. Chopra. I.C **Clossary of Indian Medicinial Plants**. National Institute of Science and Information Resources (1956).

24. Sathyanarayan V. **Biochemistry Books** and allied Private Limited Kolkatta 2004.

25. Jambulingam K. **Essential Medical Physiology (4th Edition)** Jaypee Brothers, New Delhi.

26. Krishnan Mary K.S **The Wealth of India (Volume 2 & 5).** A Dictionary of Indian Raw Materials and Industrial Products 2004.

27. Mahadevan Pandithar T.R **Roganirnaya Saram Negapatam** Scottish Brother Press 1898.

28. Subramania Pandithar, **Seevakshamirtham,** Baskar & Sons, Chennai.

29. Swamy C.P **Agathiyar Gunavagadam** 1973.

30. Venkatrayan, **Sarabendvar Vaidhya Muraiygal.** Garpini Balayoga Skikitchi Saraswathi Mahal 1949.

31. Pandit Duraisamy Iyangar, **Astanga Irudhaya (Volume-I)** Chennai - 1935.

32. Suraj Gupta, The **Short Text Book of Paediatries.**

33. Venkat Rayan, **Agathiyar 2000,** Saraswathi Mahal Publications 1963.

34. Christopher R.E Edwards - **Davidsons Principles & Practice** of medici ne (16th Edition -1991.

35. Kannusamy Pillai **Pathartha Guna Vilakkam,** Rathina Naya and Sons.

IAEC PROTOCOL no: 124&/ac/09/CPCSEA-25/2011

20/12/2011

CERTIFICATE

This is certify that the project title Pre clinical & clinical Study on Pandu (Iron deficiency anaemia) in children and the drug of choice is Tiruchoorathathya Choornam. has been approved by the IAEC.

Prof. Dr. K. Manickavasakam
Name of Chairman/Member Secretary IAEC:

Dr. B. Jayachandran Dare
Name of CPCSEA nominee:

Signature with date

K. Manickam

Chairman/Member Secretary of IAEC:

CPCSEA nominee:

(Kindly make sure that minutes of the meeting duly signed by all the participants are maintained by Office)

NATIONAL INSTITUTE OF SIDDHA

(An Autonomous Body under Department of AYUSH)
Ministry Of Health & Family Welfare, Government of India

Tambaram Sanatorium, Chennai - 600 047
Tel : 044-22411611 Fax : 044-22381314
E-mail : nischennaisiddha@yahoo.co.in
Website : www.nischennai.org

Name: P. BALA MURUGAN.... Reg. NO :- 32102701

Title: PRE CLINICAL AND CLINICAL STUDY ON PANDU (IRON DEFICIENCY ANEMIA) IN CHILDREN AND THE DRUG OF CHOICE IS TIRUDHARATCHAI CHOORANAM.

No. NIS/IEC/2011/3/25 - 24/12/2011

DECSION

Opinion of the Institutional Ethics Committee – Please Check one
✓ Approval _____ Modifications required prior to approval (Please specify one space below) _____ Disapproval Date of review: _____ K. Manickavasakam (Dr. K. MANICKAVASAKAM) Member Secretary Signed: _____ (Please print name) Dr.V.SUBRAMANIAN chair person (Please delee as appropriate, Chairperson, Secretary)
Modifications needed Modification given to candidate
The research proponent is hereby informed that the Institutional Ethics Committee will require the following: 1. All adverse drug reactions (ADRs) that are both serious and unexpected to be reported promptly to the IEC within 7 working days 2. The progress report to be submitted to the IEC atleast annually 3. Upon completion of the study, a final study status report needs to submitted to the IEC

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 600047

CERTIFICATE OF BOTANICAL AUTHENTICITY

Certified that the following plant drugs used in the formulation **"Tirudharatchatha chooranam"** (Internal) for **Pandu Noi** taken up for Post Graduation Dissertation by **Dr.P.Balamurugan**, M.D.(S), II year, Department of Kuzhandhai Maruthuvam, 2011-12, are identified and authenticated through Visual inspection / Experience, Education & Training / Organoleptic characters / Morphology / Taxonomical / Microscopical methods.

Anacardium occidentale (Anacardiaceae), Nut
Phoenix dactilifera Linn. (Arecaceae), Dried fruit
Piper longum Linn. (Piperaceae), Fruit
Piper nigrum Linn. (Piperaceae), Fruit
Glycyrrhiza glabra Linn. (Fabaceae), Root
Cinnamomum tamala Nees & Eberm. (Lauraceae), Leaf
Cyperus rotundus Linn. (Cyperaceae), Rhizome
Coriandrum sativum Linn. (Apiaceae), Fruit
Syzygium aromaticum (Linn.) Merr. & L.M. Perry (Myrtaceae), Flower bud
Tephrosia purpurea (Linn.) Pers. (Fabaceae), Root
Maranta aurundinacea Linn. (Marantaceae), Rhizome
Plumbago zeylanica Linn. (Plumbaginaceae), Root
Elettaria cardamomum Maton (Zingiberaceae), Fruit
Oryza sativa Linn. (Poaceae), Seed

MEDICINAL BOTANY CHENNAI 600 047

Certificate No: NIS/MB/78/2012

Date: 16-6-12

Authorized Signatory
Dr. D. ARAVIND, M.D.(s),M.Sc.,
Assistant Professor
Department of Medicinal Botany
National Institute of Siddha
Chennai - 600 047, INDIA

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to

~~Mr~~ / ~~Ms~~ / Dr .....P. BALAMURUGAN.....

for participating as a ~~Resource Person~~ / Delegate in the IX Workshop on **"Research Methodology & Biostatistics"** for AYUSH Post-Graduates & Researchers organized by the Department of Siddha The Tamil Nadu Dr. M.G.R. Medical University from 24th September 2012 to 28th September 2012.

Dr. N. KABILAN MD (Siddha)
READER, DEPT. OF SIDDHA

Dr. K. SIVASANGEETHA MD
REGISTRAR (FAC)

DR. MAYILVAHANAN NATARAJAN D.Sc.
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. (Orth. Onco.) F.R.C.S. (Eng)
7th VICE CHANCELLOR