INTRODUCTION

The Siddha system of medicine contains rich Saiva Siddhantham, Philosophy, Alchemy and Materia Medica etc., Under Materia Medica the Siddha system deals with the combination of Herbs, Minerals, Metals, and Animal and Marine products.

According to Siddha medical science, the Universe originally consist of atoms which contribute to the five basic elements, viz., earth, water, fire, air and space which correspond to the organs of the human body and they constitute the base for all corporeal things in the world.

> "தலங்காட்டியிந்தச் சடமான வைம்பூதம் நிலங்காட்டி நீர்காட்டி நின்றிந் தீகாட்டி வலங்காட்டி வாயுவால் வளர்ந்தே யிருந்த குலங்காட்டி வானிற் குடியாயிருந்ததே இருந்திடுமிவ் வைந்தாலெடுத்த சடமிது" - திருமூலர் நாடி

Siddha system consists of 96 principles, 3 vital humors ie Vatham, Pitham, Kabham and 7 body constituents viz, Saram, Senneer, Oon, Kozhuppu, Enbu, Moolai and Sukkilam or Suronitham. Fewer than 7 body constituents Senneer is prioritized in the second order next to saram.

"சென்ம மாஞ்சரீரந்தொண் ணூற்றாறங் குலமாம் செய்கின்ற அவரவர்கள்கையா லேதான் தன்னமாம் ரசமிரத்த மாங்கிசமூ மேதை தசைமச்சை யொடுசுக் கிலந்தாதே ழாகி உன்னமாம் உருவென்னுஞ் சரீரமாகி உயர்சட்டை ரசஞ் சேர்ந்து ரோகமாகி வன்னவாம் வாதபித்த சேட்ப மாகி வருகின்ற சடத்தினது வன்மை கேளே."

- யூகி நாடி

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Siddhars developed methods and medication that are essential to strengthen the physical and mental status of the human being. Food is considered to be basic building material of human body, which gets processed into three humors viz, Vatha, Pitha and Kabha. The equilibrium of these three humors is considered as health and its disturbance or imbalance leads to disease or pathologic state.

According to Saint Yugi, the etiology of Pandu are frequent attacks of diarrhoea, excessive intake of salt and sour food, living in hot surroundings, excessive chewing of pawn and nuts, excessive intake of alcohol, day time sleep are correlated with the etiology of Anaemia in Modern science.

PithaPandu is characterised by yellowish colouration and pallor of the skin, diminished vision, thirst, and fainting, pungent taste like pepper, chest pain, dyspnoea, giddiness and bitter taste which may be correlated with iron deficiency anaemia in modern science.

Anaemia constitutes a serious health problem in many tropical countries because of the prevalence of malaria and other parasitic infections (Dacie and Lewis, 1994). In anaemia there is decreased level of circulating haemoglobin, less than 13 g/dl in male and 12 g/dl in females (Okochi et al., 2003). In the tropics, due to endemicity of malaria, between 10 to 20% of the population presents less than 10 g/dl of Hb (Diallo et al., 2008). Children are more vulnerable.

Iron Deficiency Anaemia (IDA) is the single most common disorder affecting mankind. It is estimated that more than 2 billion people suffer from IDA worldwide.

In India nearly, 90 percent of adolescent girls, women and children suffer from iron deficiency. Almost 20 percent of maternal deaths are because of iron deficiency anaemia.

Effective therapeutic Siddha formulations are traditionally employed to alleviate anaemia. The investigator selected **"KARISALANKANNI CHOORANAM"** which is indicated for pandu specifically in the sastric literature Sigicha Rathna Deepam (Part I : Ref: Page: 162). All the ingredients in this formula is used as pothu karpam and sirappu karpam for many ailments as rejuvenator. The major ingredient of Karisalankanni chooranam is Karisalai which has a potent haematinic action used for Pitha Paandu

The ingredients of this formulation are purely herbal drugs found to be efficacious and cost effective. The above said drug formulation, has not undergone any clinical trial, so far. Hence the Principal Investigator has selected the drug "**KARISALANKANNI CHOORANAM**" to find out the therapeutic efficacy in pittha paandu. As per the standard protocol designed by the investigator, safety studies and Clinical trial were conducted in Ayothidoss pandithar Hospital, National Institute of Siddha, Chennai. The study was explained in detailed.

AIM AND OBJECTIVES

The aim of this study is to introduce an effective Siddha medicine for pittha paandu noi and to document the formulation.

OBJECTIVES

1) Primary objective:

To evaluate the Siddha Therapeutic efficacy of the Poly Herbal formulation

"KARISALANKANNI CHOORNAM" (Internal) in " PITHA PANDU" (Iron Deficienccy Anaemia)

2) Secondary objective:

-To evaluate the safety profile (acute, long term toxicity studies) of the trial drug

-To study the effect of other co-factors such as age, sex & dietary influence

REVIEW OF LITERATURE SIDDHA ASPECTS

PANDU NOI

VERU PEYARKAL (SYNONYMS):

Veluppu noi, Venmai noi, Venpaandam.

IYAL (DEFINITION):

Pandu noi is a disease, characterized by changing of natural colour of the body, pallor of skin, nails, and conjunctiva.

இயற்கை நிறம் மாறி, உடல் வெளுத்து, கண், நகக்கண் இவைகளை நீக்கிப் பார்க்கில் குருதியின்றி வெளுத்திருக்கும் நோயாம்.

- நோய் நாடல், நோய் முதல் நாடல் திரட்டு.

The great Siddhar Agathiyar defined Pandu in the following verses;

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

- அகத்தியா் வைத்திய பிள்ளைத்தமிழ்.

"கழிவாகுந் தேகமப்பா காணத்தச வத்தாய் வற்றிவிடு மன்னவாசல் கேட்கில் பழிகாரா் முகத்தில் முழியாா் போலே பாண்டமெல்லாம் வெளுக்கடித்தது இரத்தம்"

-அகத்தியா் வைத்திய காவியம்.

"தேகத்தில் இரத்தம் வற்றித் தீங்கான விந்த நோய் காணுமப்பா." –அகத்தியா் குணவாகடம். In the text Uyir Kaakum Siddha Maruthuvam, it is quoted as follows;

உடல் பசிய நிறமாய் பளபளத்து, முகங்குளுப்பையிட்டு இருவிழியும் நீல நிறமாய், இடுப்புகுத்தோடு, நீரும் பொன்னிறமாய் இறங்குவதே பாண்டின் குணமாகும்."

- உயிர் காக்கும் சித்த மருத்துவம்

NOI VARUM VAZHI (ETIOLOGY)

According to Yugimuni the cause of Pandu are as follows;

"அறிந்துமே உற்பத்தி சொல்லக் கேளாய் அதிசார மலமிளகி யெந்நே ரந்தான் பிறிந்துமே புளியுப்பு பெருத்தலாலும் பெத்தமா மக்கினி யிருத்தலாலும் மிறிந்துதாம் பூலமிக அருந்த லாலும் மீறியே மதுக்களைத்தான் புசித்தலாலும் பறிந்துமே பகல் நித்திரையே செய்தலாலும் பாண்டு 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 pawn and nuts, excessive intake of alcohol, day time sleep are some of the factors causing Pandu noi.

According to Theraiyar vagadam;

"தாக முறும் பசியால் தாமுண்ணா ராமாகில் கோதருநீா் தாகத்தாற் கொள்ளாராய் - போத மிரத்தந் தனைக்குடிக்கும் மின்பவிடாய் போக்கும் பருத்த விழிகுருடாம் பாா்."

- தேரையா் வாகடம்

It is mentioned that negligence of food and water causes Pandu noi.

According to Thanvanthiri vaithyam;

"திருந்திடும் பாண்டு ரோகஞ் சோ்ந்திடுங் குணத்தைக் கேளாய் இருந்திடும் வாதபித்தச் சிலேற்பன மிவைதான் மாறும் புரிந்துதா னொன்றோடொன்று பொருந்துவதலு மண்ணோ டருந்துவதாலும் பாண்டு வணைந்திடு மென்னலாமே ஆகிய மூலந் தன்னிலைணைந்த வுட்டணத்திணாலுந் தோகையா் மேகத்தாலும் துயா்மிகு ரோகத்தாலும் தேக போஷணை யுள்ளோா்க்குத் தரித்திரஞ் சோ்தலாலும் வேகமாந் திரிதோஷங்கள் மிஞ்சிய பாண்டு வாமே." – தன்வந்திரி வைத்தியம்

Imbalance between the three thathus [Vatham, Pitham, and Kabam], perversion of appetite such as eating mud (PICA) and excessive heat accumulation due to altered Abana vayu, excessive sorrow, and psychosocial factors are some of the causes of Pandu noi.

NOI ENN (CLASSIFICATION):

According to Yugimuni Pandu noi is classified into 5 types

"கூறவே பாண்டுவிடப் பெயரைக் கேளாய் குறிப்பாக வைந்துவித மாகும் பாரு வாரவே வாதமாம் பாண்டு வோடு மார்க்கமாம் பித்தத்தின் பாண்டு வாகும் தேறவே சிலேட்டுமமாம் பாண்டு தானாம் திரிதோடப் பாண்டோடு விடப்பாண் டாகும் ஆறவே பாண்டுவிட வாண்மை யெல்லாம் ஆராய்ந்து சொல்லவே அறிந்து கொள்ளே." – யூகி வைத்திய சிந்தாமனி

Thanvanthiri classified Pandu into 7 types

"பயித்திய பாண்டு வாதபாண்டுவே சிலேத்தும பாண்டு வியத்திரி தோஷப்பாண்டு வெறும் பித்த சிலேத்தும பாண்டு பயித்திய வாதபாண்டு பகா் சன்னிவாத பாண்டு நயப்புறும் பாண்டு வேழின் குணத்தை நான் நவிலலுற்றேன்." – தன்வந்திரி வைத்தியம்

Classification of Pandu noi based on various Siddha books:

AGASTHIAR GUNAVAAGADAM

According to Agasthiyar gunavaagadam pandu noi is classified in to 5 types

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

VAITHYA SARA SANKRAHAM

According to vaithya sara sankraham pandu noi is classified in to 5 types

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

YUGI CHINTHAMANI-800

According to yugi chinthamani-800 pandu noi is classified in to 5 types

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

T.V. SAMBASIVAM PILLAI

According to T.V. sambasivam pillai pandu noi is classified in to 8 types

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

THANVANTHIRI VAITHIYAM

According to Thanvanthiri vaithiyam pandu noi is classified in to 7 types

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

ANUBOGA VAITHIYA DEVARAGASIYAM

According to Anuboga vaithiya devaragasiyam pandu noi is classified in to 6

types

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

PARARASA SEKARAM

According to Pararasa sekaram pandu noi is classified in to 5 types

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

MADAVA NITHANAM

According to Madava Nithanam pandu noi is classified in to 5 types

- 1. Vatha pandu
- 2. Pitha pandu
- 3. Kaba pandu
- 4. Sanni pandu
- 5. Mann pandu

JEEVARAKSHAMIRTHAM

According to Jeevarakshamirtham pandu noi is classified in to 5 types

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

ASHTANGA HRIDAYAM

According to Ashtanga hridayam pandu noi is classified in to 5 types

- 1. Vatha pandu
- 2. Pitha pandu
- 3. Kaba pandu
- 4. Sanni pandu
- 5. Mannul pandu

KURIKUNANGAL IN PANDU NOI (CLINICAL FEATURES) :

1. Murkurikunangal (Premonitory symptoms):

In Siddha Maruthuvam Pothu, Kuppusamy Mudaliar states;

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 நீா் சுருங்கினும் இதுவும் சலப்பொருள் செயற்கை யாயருந்தினும் சிறுத்த நீரிதுவும் பாண்டு நோய்ச் சம்பவத்தைத் தருமிதில்.

Oliguria occurs suddenly and sometimes it occurs even after intake of excessive of water are explained as premonitory symptom of Pandu noi.

2. POTHU KURIGUNANGAL (GENERAL SIGNS AND SYMPTOMS):

In Siddha Maruthuvam Pothu, Kuppusamy Mudaliar states;

Inability to walk, headache, palpitation, blurring of vision, giddiness, syncope, dyspnoea, anorexia, vomiting, paleness of the skin, nailbeds become swollen and pallor, fissured tongue, glossitis, hoarseness of voice are general signs and symptoms of Pandu noi. In females scanty menstruation, sometimes menorrhagia may occur. If it occurs in children and elderly, it may manifest because of worm infestation and blood disorders. If it occurs in pitha thegi, anorexia, indigestion, burning sensation, pallor of skin, glossitis, and dysphagia, vomitus with bile, bitter taste of the tongue and diarrhoea occurs. If the symptoms persist for longer duration it results in jaundice.

Agasthiyar Gunavaagadam states that;

"உண்டாகும் வேளை தன்னில் தேக நோ்மை

உறுதியாய்ச் சொல்லுகிறென் நன்றாய் பாரு குண்டான முகம் கண்கள் உதடு நாக்கு குறிப்பான வாய் வேகம் தேக முற்றும் வெண்டாக வேயுலா்ந்து வெண்மை யாகி விரல் கால்கள் முழுவதிலும் ரத்தம் வற்றி கண்டான கால்கள் தான் தணிந்து நிற்கும் கருவான நாடியது மெதுவாய் 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 are mentioned as the signs and symptoms of Pandu noi.

SYMPTOMS OF VARIOUS TYPES OF PANDU:

1. Vatha Pandu:

"கொள்ளவே வாதபாண்டு ரோகங் கேளாய் குடல்புரட்டி யடிவயிறு தான்வ லிக்கும் தள்ளவே தாகமொடு பசியு மில்லை தழலான சரசரப் பாகத் தாணும் நள்ளவே நரம்பெல்லாங் கறுப்பு மாகும் நடுக்கலொடு கண்சிவப்பு மலபந் தந்தான் விள்ளவே தலைவலிக்கு மேனி வீங்கும் வெளுப்பாகும் வாதத்தின் பாண்டுதானே. –யூகமுனி.

The symptoms of VathaPandu are lower abdominal pain, thirst, loss of appetite, dryness of the skin and 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 மஞ்ச ளித்து மகாவெளுப்பு உண்டாகி மந்தக் கண்ணாந் தாமென்ற தாகமொடு மூர்ச்சை யாகுந் தனிவாயில் மிளகுபோற் றானு றைக்கும் நேமென்ற நெஞ்சுமுள் தானு முண்டாய் நெருக்கியா மூச்சுமுட் டதுவே யாகுங் கோமென்ற கிறுகிறுத்து வாய்கைப் பாகுங் கிளா்பித்த பாண்டு வெனக் கூறலாமெ." – யூகிமுனி

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

3. Kaba Pandu :

"கூறியதோர் நரம்புதோல் மிகவெளுப்பு கிளர்நாவு உப்புறைக்கு மயிர்கூச் சாகும் வாறியதோர் வாந்தியாங் குரலுங் கம்மும் மெகத்தான தும்மலுடன் கோழை யாகும் ஈறியதோர் இருமலோடு மயக்க முண்டாம் இடுப்பசதி இந்திரிய நஷ்ட மாகும் சீறியதோர் சோபமொடு தாப மாகுஞ் சிலேட்டுமத்தின் பாண்டெனச் செப்பலாமே." – யூகிமுனி

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

4. Mukkutra Pandu:

"செப்பவே யருசியொடு சோப தாகஞ் செயலான சுவாசமொடு இளைப்பு மாகும் வெப்பவே மேகனத்தில் சிறுநீர்தான் வீழும் மிடுக்கான பலவீன மார்பி டித்தல் துப்பவே சூட்டோடு தியக்க மாகுந் தும்மலா யுடம்பெங்கு மூதிக் காணும் திப்பவே தேகமெங்கு மசதி யாகும் திரிதோடப் பாண்டென்னச் செப்பும் நூலே." – யூகிமுனி

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

5. Vida Pandu:

"நூலாக நலத்தோ டேயுடல்வெ ளுக்கும் நோய்நரம்பு சூடாகும் தாக மாகும் ஆலாக யருசியொடு சத்தி விக்கல் அதட்டியெ இருமலுட னதிசு வாசம் வாலாக வயிற்றிரைச்ச லதிசார தோடம் மாசுரமு முண்டாதல் மார்க் கனத்தல் வேலாக மேனியெங்கு மிகவே ஊதல் விடபாண்டு அசாத்தியமென்றே விளம்பலாமே." – யூகிமுனி

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

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):

It is based upon three main principles. They are

- 1. Porialarithal (Inspection)
- 2. Pulanalarithal (Palpation)
- 3. Vinathal (Interrogation)

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."

"மெய்க்குறி நிறந்தொனி விழிநாவிருமலம் கைக்குறி" – தேரையா்

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

There are three vital naadi viz, Vatham, Pitham, Kabham present in our body, as mentioned in the following poem,

"தெளிந்திவிட்ட நாடியது ஞானம் போலே சிறப்பான புரிமூன்று மொன்றாய்க் கூடி மொழிந்திட்ட வாதபித்த சிலேற்பன மென்று முன்கையில் பூண்டிருக்கு முறைமையாக." – சதக நாடி

Vatha, Pitha and Kabha naadi are in the ratio of 1:1/2:1/4 proportion in normal condition. This is stated as follows,

"வழங்கிய வாதம்மாத்திரை யொன்றாகில் தழங்கிய பித்தந் தன்னிலரை வாசி அழங்குங் கபந்தா னடங்கியே காலோடில் பிறங்கிய சீவா்க்குப் பிசகொன்று மில்லையே." – குணவாகடம்

By combination of the above said three naadi, six thontha naadi are formed. They are Vathapitham, Vathakabham, Pithavatham, Pithakabham, Kabhavatham and Kabhapitham. This is stated as follows,

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 ப**ாண்டு** சோகை ______

நாமேதான் சொன்னோமே பித்தக்கூறு நவின்றிட்டார் வாசமுனி நவின்றிட்டாரே." - அகத்தியர்

2. Kaba Naadi:

"தானமுள்ள சேத்துமந் தானிளகில் வெப்பு ------ஏன முறுங் காமாலை **பாண்டு** சோபை ஏழு சுரங்கள் பலதுக்கம் விட முண்டாமே." - சதகநாடி

3. Vatha Kaba Naadi:

"இருக்குமந்த வாதத்தில் சீதஞ் சேர்ந்தால் ------

ஒருக்கின்ற மலபந்தம் பொருமல் வீக்கம்

உள்வீச்சு சூலையொடு **பாண்டு** ரோகம்."

- சதக நாடி

4. Kaba Vatha Naadi:

"கண்டாயோ சிலேற்பனத்தில் வாத நாடி

விண்டாலே இளைப்பிருமல் சோகை **பாண்டு** விடபாகம் விடசூலை பக்கவாதம்."

- சதக நாடி

5. Kaba Pitha Naadi:

"இடமான சேத்துமத்தில் பித்த நாடி

------விடமான நெஞ்சடைப்பு சுவாசம் விக்கல் வெகுசுரமும் நாவறட்சி **பாண்டு** ரோகம்." – சதக நாடி

6. Pitha Vaatha Naadi:

"சிறப்பான பித்தத்தில் வாத நாடி சேரிலுந் **தாதுநட்ட முதிர பீடை** --------"

- சதக நாடி

2. Sparisam (Palpation):

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

In **PithaPandu**, hot sensation, dryness, roughness of the skin, sweating, swelling, hepatospleenomegaly are seen.

3. Naa (Tongue):

The colour, dry or wet, coating, texture, 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 **PithaPandu**, pallor of the tongue, dryness, ulceration, fissure, bitter or pungent taste of tongue, baldness 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 PithaPandu 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 and accumulation of secretion at the angle of eyes, visual disturbance and any specific diseases of the eyes should be noted.

In **PithaPandu** noi, pallor of conjuctiva and dull vision are noted.

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.

In PithaPandu noi, diarrhoea is noted.

8. Moothiram (Urine):

Neer Ilakkanam (Method of collection of urine):

"அறிந்துமாறிரதமும் அவிரோதமதாய் அஃகல் அலர்தல் அகாலவூன் தவிர்ந்தழற் குற்றளவருந்தி உறங்கி வைகறை ஆடிக்கலசத் தாவியே காது பெய் தொரு முகூர்த்தக் கலைக் குட்படு நீரின் நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே." - தேரையர் நீர்க்குறி நெய்க்குறி.

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 நீா்க்கரி எடை மணம் நுரை எஞ்சலன றைந்தியலுளவை யறைகுது முறையே." – தேரையா் நீா்க்குறி நெய்க்குறி.

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 நீா் சுருங்கினும் இதுவும் சலப்பொருள் செயற்கை யாயருந்தினும் சிறுத்த நீரிதுவும் பாண்டு நோய் சம்பவத்தைத் தருமிதில் தூண்டு றாய் பேதியும் சோா்வும் பிறக்குமே." – தேரையா் நீா்க்குறி நெய்க்குறி.

Neikkuri:

"நிறக்குறிக் குரைத்த நிருமான நீரிற் சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவிடுத் தென்றுறத் திறந்தொலி யேகா தமைத்ததி நின்றதிவலை போம் நெறிவிழியறிவும் சென்றது புகலுஞ் செய்தியை யுணரே." - தேரையர் நீர்க்குறி நெய்க்குறி.

The urine 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 நீண்டின்ஃதே வாதம்" "ஆழிபோற் பரவின் அஃதே பித்தம்" "முத்தொத்து நிற்கின் மொழிவ தென் கபமே" - தேரையர் நீர்க்குறி நெய்க்குறி. 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"

- தேரையா் நீா்க்குறி நெய்க்குறி.

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

ROLE OF MUKKUTRAM IN PANDU:

Vatham: Its mathirai alavu is 1 mathirai.

Location of vatham in the body: Vatham is located in the abanan, faeces, idakalai, spermatic cord, pelvic bone, skin, nerves, joints, hair and muscles.

Ten forms of Vatham:

1. Pranan (Uyirkaal): It resides in the heart and legs to nose and controls knowledge, mind and five objects of sense, useful for breathing.

In PithaPandu noi, dysnoea is present, if pranan is affected.

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 **PithaPandu** noi, diarrhoea, oliguria, ammenorohoea or oligomennorohoea are present, when abanan is affected.

3. Viyanan (Paravukaal): It resides mainly at the heart and responsible for movements of the body and sensation.

In **PithaPandu** noi, swelling of the body, pallor of eyes and lips, numbress are present due to the affection of viyanan.

4. Samanan (Nadukkaal): It is located in the stomach, helps for proper digestion and balances the above four vaayus in equillibirium.

In **PithaPandu** noi, anorexia and any of the above four vaayus affection are present when samanan is affected.

5. Uthanan (Melnokkukaal): It is located in the chest and responsible for vomiting, cough and sneezing reflexes.

In PithaPandu noi, excessive thirst is present due to the affection of uthanan.

6. Naagan: It resides in the eyes and is 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.

In PithaPandu noi, blurred vision is present when koorman is affected.

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

In **PithaPandu**, anorexia and dryness of mouth are present when kirukaran is affected.

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

In PithaPandu, fatigue and insomnia are present when devathathan is affected.

10. Dhananjeyan: It resides in the nose and escapes on the third day after death by bursting out of the cranium.

Pitham: Its mathirai alavu is 1/2 mathirai

General Characteristics of Pitham:

- ➢ Veppam Hot
- Koormai Sharpness
- Neippu Lubricative
- Nekizhchi Viscousity
- > Pitham conceives the properties of the substance to which it combines.

Location of Pitham in the Body

"தானான பித்தம் பிங்கலையைப் பற்றிச் சாய்வான பிராணவாயுவு தன்னைச்சேர்ந்து ஊனான நீர்ப்பையில் அணுகி மூலத் துதித்தெழுந்த அக்கினியை உறவு செய்து மானேகே ளிருதயத்தி லிருப்பு மாகி மயலாகி நினைவாகி மயக்க மாகி கானான சிரந்தனிலே இரக்க மாகிக் கொண்டுநின்ற பித்தநிலை கூறினோமே." – கண்ணுசாமியம்

- Pingalai
- Praanavayu
- Moolakkini
- Neerpai Urinary bladder
- Irudhayam Heart
- Thalai Head
- Kopuzh Naval
- Undhi Abdomen
- Iraippai Stomach
- Viyarvai Sweat
- Naavil oorukindra neer Salivary secreation
- Senneer Blood
- Saaram Chyle.
- ≻ Kan Eye.
- Thol Skin

Five forms of pitham:

1. Anala pitham: This gives appetite and helps for digestion.

In PithaPandu, loss of appetite is present when it is affected.

2. Ranjagam: It gives colour to the blood.

In PithaPandu, pallor of conjunctiva and skin are present when it is affected.

3. Saathagam: It controls the entire body functions responsible for the activities of the body

In **Pithapandu**, inability to do the works properly and sluggishness are present when it is affected.

4. Alosagam: This gives brightness to the eyes.

In PithaPandu, dull vision is present if alosagam is affected.

5. Prasagam: It gives complexion to the skin.

In Pithapandu, altered skin lusture is present when it is affected.

Kabam: Its mathirai alavu is ¹/₄ mathirai.

Location of Kabam in the body:

Kabam is located in Samanavayu, Sperm, Head, Tongue, Vulva, Fat tissue, Bone marrow, Blood, Nose, Chest, Nerve, Bone, Brain, Eyes and Joints.

Five forms of Kabam:

1. Avalambagam: It controls heart, lungs and supports other forms of kabam

In **PithaPandu**, dyspnoea is present when it is affected.

2. Kilethagam: It makes the food wet and helps for digestion.

In **PithaPandu**, indigestion is present when it is affected.

3. Pothagam: It is responsible for taste.

In **PithaPandu**, bitter or pungent taste is present when it is affected.

4. Tharpagam: It keeps the eyes cool.

In PithaPandu, burning sensation of eye is present when it is affected.

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

Paruvakaalam (Season):

The whole year is divided into six seasons, they are as follows;

1. Kaar kaalam	- Avani and Puratasi	- Aug 16 to Oct 15
2. Koothir kaalam	- Ayppasi 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

Seasonal influence of earth, Water bodies, Flora and Fauna will have its impact in human being's physiology inturn make them susceptible to certain specific diseases which are common in that season.

Mukkutrm	Thannilai Valarchi	Vetrunilai Valarchi	Thannilai Adaithal
Vatham	Muthuvenil kaalam	Kaar kaalam	Koothir kaalam
D'41	77 1 1		
Pitnam	Kaar kaalam	Koothir kaalam	Munpani kaalam

Physiological alterations of Mukkutram:

Nilam:

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

People living in Kurinji, Mullai, Neithal and Palai may have in increased chance to acquire Pithapandu 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. Sent	eer - 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 1/	

7. Venneer - Responsible for the propagation of species.

In PithaPandu,

Fatigue, dyspnoea and tiredness are present, if Saaram is affected.Pallor of skin and conjunctiva are present, if Senneer is affected.Swelling of the body is present, if Oon is affected.Ammenorrhoea / oligomenorrhoea is present, if Suronitham is affected.Sluggishness in sexual life is present, if Sukkilam is affected.

PROGNOSIS OF PANDU:

Curable and Incurable Types:

According to Siddha Maruthuvam,

- > The possibilities of cure for Nanju Veluppu noi are rare.
- > All other types of Veluppunoi are curable.
- Eventhough, if any of the following symptoms or diseases like vomiting, diarrhoea, odema, thirst, diabetes, tuberculosis gets associated in the above said four Veluppu noi, then it is not curable easily.

According to Sarabendrar Vaidhya Muraigal:

The Pandu noi, which is chronic in nature, is not treatable. In acute stage, also odema with yellowish discolouration, if present is not curable. Constipation or greenish dysentery, if occurs also is not curable.

Extreme pallorness of teeth, nail and eyes and the vision to every object seems to be whitish is not curable.

Emaciation of the Pandu noi persons, with odema present in head, upperlimb and lower limb, swelling of the external genetalia, inguinal region, frequent fainting, diarrhoea and fever in pandu noi is not curable.

According to Agathiya Vaithiya Pillai Tamil;

"பாண்டுரோகிக்கு 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 தானிறங்கிந் செப்பும் கிராணியிற் பாண்டில்கிளா் நீா்சுருங்கிற் பிராணன் பிரியுமென பேசு."

"சொல்லு பித்ததிற் 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."

- கண்ணுசாமியம்.

According to Sadhaga Naadi;

"தானான பிரமேகம் வாத சூலை சாா்வான நீரிழிவு குன்மரோகம் மானான சயரோகங் சன்னிதோடம் யடுத்த விடங்காமாலை பாண்டு சோகை மானே கேள் கபரோக மந்திர வியாதி மஞ்சள் நோய் குலைநோவு பயித்தியரோகம் ஊனாகும் வருமிடத்தில் யதிசாரங்கள் உண்டாகிய சாத்தியமா முறுதி தானே." – சதக நாடி

NOI NEEKKAM (TREATMENT):

The speciality of Siddha treatment emphasise not only for complete healing 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 the disease the following principles must be noted.

"நோய் நாடி நோய்முதல் நாடியது தணிக்கும் வாய்நாடி வய்ப்பச் செயல்"

"உற்றா னளவும் பிணியளவும் காலமும் கற்றான் கருதி செயல்" – திருக்குறள்

So, it is essential to know about the disease and the Cause for the onset of disease, body constituent of the patient, severity and chronicity of the illness, the season and the time of onset of the disease must be observed.

Line of Treatment of Pandu:

The aim is to normalize the vitiated Mukkutram, Vayus and the affected Saram and Senneer thathu.

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

"சத்தியால் பித்தந் தாழும்" "பேதியால் வாதந் தாழும்" "அஞ்சனத்தால் கபந் தாழும்"

Usually for pitha diseases, emetics are 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 drows, the administration of emetic medicine is excluded from the line of treatment.

As per Siddha Maruthuvam, the line of treatment includes:

- 1. Mild laxatives can be administrated to neutralize the deranged thathus.
- 2. To improve haemoglobin content of blood, iron preparations are used.
- 3. Pathiyam ie, diet restrictions are advised to normalize the affected thathu.

TREATMENT:

Medicine Name: KARISALANKANNI CHOORANAM (Internal) Dosage: Thirikadi pramanam [1g] [24]

(Twice/day) before food.

Adjuvant: white sugar

Course: 1/2 Mandalam[24 days]

DIET:

"மாறுபா டில்லா உண்டி மறுத்துண்ணின் ஊறுபா டில்லையு யிா்க்கு"

- திருக்குறள்.

"மருந்தே உணவு, உணவே மருந்து"

- திருமூலா்.

Diet regimen for Pandu Noi :

Diet should be of

- 1. Stimulating appetite
- 2. Strengthening the body
- 3. Easily digestable should be taken.

For Pandu noi, the following food items are advised.

- Easily digestible foods like porridge, mutton soup, and bone soup must be given in acute stages of Pandu noi.
- Tender brinjal, tender country bean, pepper, garlic, anise seed, ginger, onion green Peas, bengal gram, vegetable soups are advised to consume in diet.
- After the normal appetite is restored properly, prepared meat of Kaadai [quail], Udumbu [Monitor] can also be given. They tone up the deblitated system and also help in rejuvenation.
- Tamarind, tea, coffee, betel chewing, tobacco chewing and alcohol are advised to avoid as they prevent absorption of the drug.

MODERN ASPECT ANAEMIA

Definition

Anaemia is strictly defined as a decrease in red blood cell (RBC) mass. The function of the RBC is to deliver oxygen from the lungs to the tissues and carbon dioxide from the tissues to the lungs. This is accomplished by using hemoglobin (Hb), a tetramer protein composed of heme and globin. Anaemia impairs the body's ability for gas exchange by decreasing the number of RBCs transporting oxygen and carbon dioxide.

WHO's criterion for Anaemia in adults is an Hb value of less than 12.5 g/dL. Children aged 6 months to 6 years are considered anemic at Hb levels less than 11 g/dL, and children aged 6-14 years are considered anemic when Hb levels are less than 12 g/dL. The disadvantage of such arbitrary criteria is that a few healthy individuals fall below the reference range, and some people with an underlying disorder fall within the reference range for Hb concentration.

Erythrocyte life cycle

Erythroid precursors develop in bone marrow at rates usually determined by the requirement for sufficient circulating Hb to oxygenate tissues adequately. Erythroid precursors differentiate sequentially from stem cells to progenitor cells to erythroblasts to normoblasts in a process requiring growth factors and cytokines. This process of differentiation requires several days. Normally, erythroid precursors are released into circulation as reticulocytes.

Reticulocytes are so called because of the reticular meshwork of rRNA they harbor. They remain in the circulation for approximately 1 day before they mature into erythrocytes, after the digestion of RNA by reticuloendothelial cells. The mature erythrocyte remains in circulation for about 120 days before being engulfed and destroyed by phagocytic cells of the reticuloendothelial system. Erythrocytes are highly deformable and increase their diameter from 7 μ m to 13 μ m when they traverse capillaries with a 3- μ m diameter. They possess a negative charge on their surface, which may serve to discourage phagocytosis. Because erythrocytes have no nucleus, they lack a Krebs cycle and rely on glycolysis via the Embden-Meyerhof and pentose pathways for energy. Many enzymes required by the aerobic and anaerobic glycolytic pathways decrease within the cell as it ages. In addition, the aging cell has a decrease in potassium concentration and an increase in sodium concentration. These factors contribute to the demise of the erythrocyte at the end of its 120-day lifespan.

Pathophysiology

The physiologic response to Anaemia varies according to acuity and the type of insult. Gradual onset may allow for compensatory mechanisms to take place. With Anaemia due to acute blood loss, a reduction in oxygen-carrying capacity occurs along with a decrease in intravascular volume, with resultant hypoxia and hypovolemia. Hypovolemia leads to hypotension, which is detected by stretch receptors in the carotid bulb, aortic arch, heart, and lungs. These receptors transmit impulses along afferent fibers of the vagus and glossopharyngeal nerves to the medulla oblongata, cerebral cortex, and pituitary gland.

In the medulla, sympathetic outflow is enhanced, while parasympathetic activity is diminished. Increased sympathetic outflow leads to norepinephrine release from sympathetic nerve endings and discharge of epinephrine and norepinephrine from the adrenal medulla. Sympathetic connection to the hypothalamic nuclei increases antidiuretic hormone (ADH) secretion from the pituitary gland. ADH increases free water reabsorption in the distal collecting tubules. In response to decreased renal perfusion, juxtaglomerular cells in the afferent arterioles release renin into the renal circulation, leading to increased angiotensin I, which is converted by angiotensin-converting enzyme (ACE) to angiotensin II.

Angiotensin II has a potent pressor effect on arteriolar smooth muscle. Angiotensin II also stimulates the zona glomerulosa of the adrenal cortex to produce aldosterone. Aldosterone increases sodium reabsorption from the proximal tubules of the kidney, thus increasing intravascular volume. The primary effect of the sympathetic nervous system is to maintain perfusion to the tissues by increasing systemic vascular resistance (SVR). The augmented venous tone increases the preload and, hence, the end-diastolic volume, which increases stroke volume. Therefore, stroke volume, heart rate, and SVR all are maximized by the sympathetic nervous system. Oxygen delivery is enhanced by the increased blood flow.

In states of hypovolemic hypoxia, the increased venous tone due to sympathetic discharge is thought to dominate the vasodilator effects of hypoxia. Counterregulatory hormones (eg, glucagon, epinephrine, cortisol) are thought to shift intracellular water to the intravascular space, perhaps because of the resultant hyperglycemia. This contribution to the intravascular volume has not been clearly elucidated.

Etiology of Anaemia

Basically, only 3 causes of Anaemia exist: blood loss, increased RBC destruction (hemolysis), and decreased production of RBCs. Each of these causes includes a number of etiologies that require specific and appropriate therapy. Genetic etiologies include the following:

- Hemoglobinopathies
- Thalassemias
- Enzyme abnormalities of the glycolytic pathways
- Defects of the RBC cytoskeleton
- Congenital dyserythropoietic Anaemia
- Rh null disease
- Hereditary xerocytosis
- Abetalipoproteinemia
- Fanconi Anaemia

Nutritional etiologies include the following:

- Iron deficiency
- Vitamin B-12 deficiency
- Folate deficiency
- Starvation and generalized malnutrition

Physical etiologies include the following:

- Trauma
- Burns
- Frostbite
- Prosthetic valves and surfaces

Chronic disease and malignant etiologies include the following:

- Renal disease
- Hepatic disease
- Chronic infections
- Neoplasia
- Collagen vascular diseases

Infectious etiologies include the following:

- Viral Hepatitis, infectious mononucleosis, cytomegalovirus
- Bacterial Clostridia, gram-negative sepsis
- Protozoal Malaria, leishmaniasis, toxoplasmosis

Thrombotic thrombocytopenic purpura (TTP) and hemolytic-uremic syndrome may be a cause of Anaemia. Hereditary spherocytosis either may present with a severe hemolytic Anaemia or may be asymptomatic with compensated hemolysis. Similarly, glucose-6-phosphate dehydrogenase (G-6-PD) deficiency may manifest as chronic hemolytic Anaemia or exist without Anaemia until the patient receives an oxidant medication. Immunologic etiologies for Anaemia may include antibody-mediated abnormalities. In the emergency department (ED), acute hemorrhage is by far the most common etiology for Anaemia.

Examine optic fundi carefully but not at the expense of the conjunctivae and the sclerae, which can show pallor, icterus, splinter hemorrhages, petechiae, comma signs in the conjunctival vessels, or telangiectasia that can be helpful in planning additional studies.
Iron Deficiency Anaemia

Definition

Iron deficiency is defined as a decreased total iron body content. Iron deficiency Anaemia occurs when iron deficiency is severe enough to diminish erythropoiesis and cause the development of Anaemia.

Iron deficiency is the most prevalent single deficiency state on a worldwide basis. It is important economically because it diminishes the capability of individuals who are affected to perform physical labor, and it diminishes both growth and learning in children.

Pathophysiology

Iron is vital for all living organisms because it is essential for multiple metabolic processes, including oxygen transport, DNA synthesis, and electron transport. Iron equilibrium in the body is regulated carefully to ensure that sufficient iron is absorbed in order to compensate for body losses of iron. Whereas body loss of iron quantitatively is as important as absorption in terms of maintaining iron equilibrium, it is a more passive process than absorption.

The total body iron in a 70-kg man is about 4 g. This is maintained by a balance between absorption and body losses. Although the body only absorbs 1 mg daily to maintain equilibrium, the internal requirement for iron is greater (20-25 mg). An erythrocyte has a lifespan of 120 days so that 0.8% of red blood cells are destroyed and replaced each day. A man with 5 L of blood volume has 2.5 g of iron incorporated into the hemoglobin, with a daily turnover of 20 mg for hemoglobin synthesis and degradation and another 5 mg for other requirements. Most of this iron passes through the plasma for reutilization. Iron in excess of these requirements is deposited in body stores as ferritin or hemosiderin.

In healthy people, the body concentration of iron (approximately 60 parts per million [ppm]) is regulated carefully by absorptive cells in the proximal small intestine, which alter iron absorption to match body losses of iron. Persistent errors in iron balance lead to either iron deficiency Anaemia or hemosiderosis. Both are disorders with potential adverse consequences.

Mucosal cells in the proximal small intestine mediate iron absorption. Intestinal cells are born in the crypts of Lieberkuhn and migrate to the tips of the villi. The cells are sloughed into the intestinal lumen at the end of their 2- to 3-day lifespan. Absorptive cells remain attuned to the body requirement for iron by incorporating proportionate quantities of body iron into the absorptive cells. This iron and recently absorbed iron decrease uptake of iron from the gut lumen by satiation of iron-binding proteins with iron, by stimulating an iron regulatory element, or both. The incorporation of iron into these cells in quantities proportional to body stores of iron also provides a limited method of increasing iron excretion in individuals replete in iron.

Either diminished absorbable dietary iron or excessive loss of body iron can cause iron deficiency. Diminished absorption usually is due to an insufficient intake of dietary iron in an absorbable form. Hemorrhage is the most common cause of excessive loss of body iron, but it can occur with hemoglobinuria from intravascular hemolysis. Malabsorption of iron is relatively uncommon in the absence of small bowel disease (sprue, celiac disease, regional enteritis) or previous GI surgery.

Iron uptake in the proximal small bowel occurs by 3 separate pathways. These are the heme pathway and 2 distinct pathways for ferric and ferrous iron.

Three pathways exist in enterocytes for uptake of food iron. In the United States and Europe, most absorbed iron is derived from heme. Heme is digested enzymatically free of globin and enters the enterocyte as a metalloporphyrin. Within the cell iron is released from heme by heme oxygenase to pass into the body as inorganic iron. Most dietary inorganic iron is ferric iron. This can enter the absorptive cell via the integrin-mobilferrin pathway (IMP).Some dietary iron is reduced in the gut lumen and enters the absorptive cell via the divalent metal transporter-1 (DMT-1/DCT-1/Nramp-2). The proteins of both pathways interact within the enterocyte with paraferritin, a large protein complex capable of ferrireduction. Excess iron is stored as ferritin to protect

the cell from oxidative damage. Iron leaves the cell to enter plasma facilitated by ferroportin and hephaestin, which associate with an apotransferrin receptor. The enterocyte is informed of body requirements for iron by transporting iron from plasma into the cell using a holotransferrin receptor.

Dietary iron contains both heme and nonheme iron. Both chemical forms are absorbed noncompetitively into duodenal and jejunal mucosal cells. Many of the factors that alter the absorption of nonheme iron have little effect upon the absorption of heme iron because of the differences in their chemical structures. Iron is released from heme within the intestinal absorptive cell by heme oxygenase and then transferred into the body as nonheme iron.

Heme enters the cell as an intact metalloporphyrin, presumably by a vesicular mechanism. It is degraded within the enterocyte by heme oxygenase with release of iron so that it traverses the basolateral cell membrane in competition with nonheme iron to bind transferrin in the plasma.

Ferric iron utilizes a different pathway to enter cells than ferrous iron. This was shown by competitive inhibition studies, the use of blocking antibodies against divalent metal transporter-1 (DMT-1) and beta3-integrin, and transfection experiments using DMT-1 DNA. This research indicated that ferric iron utilizes beta3-integrin and mobilferrin, while ferrous iron uses DMT-1 to enter cells.

Which pathway transports most nonheme iron in humans is not known. Most nonheme dietary iron is ferric iron. Iron absorption in mice and rats may involve more ferrous iron because they excrete moderate quantities of ascorbate in intestinal secretions. Humans, however, are a scorbutic species and are unable to synthesize ascorbate to reduce ferric iron.

Other proteins appear to be related to iron absorption. These are stimulators of iron transport (SFT), which are reported to increase the absorption of both ferric and ferrous iron, and hephaestin, which is postulated to be important in the transfer of iron from enterocytes into the plasma. The relationships and interactions among the newly described proteins are not known at this time and are being explored in a number of laboratories.

The iron concentration within enterocytes varies directly with the body's requirement for iron. Absorptive cells of iron-deficient humans and animals contain little stainable iron, whereas those of subjects who are replete in iron contain significantly higher amounts. Untreated phenotypic hemochromatosis creates little stainable iron in the enterocyte, similar to iron deficiency. Iron within the enterocyte may operate by up-regulation of a receptor, saturation of an iron-binding protein, or both.

In contrast to findings in iron deficiency, enhanced erythropoiesis, or hypoxia, endotoxin rapidly diminishes iron absorption without altering enterocyte iron concentration. This suggests that endotoxin and, perhaps, cytokines alter iron absorption by a different mechanism. This is the effect of hepcidin and the balance of hepcidin versus erythropoietin.

Most iron delivered to nonintestinal cells is bound to transferrin. Transferrin iron is delivered into nonintestinal cells via 2 pathways: the classical transferrin receptor pathway (high affinity, low capacity) and the pathway independent of the transferrin receptor (low affinity, high capacity). Otherwise, the nonsaturability of transferrin binding to cells cannot be explained.

In the classical transferrin pathway, the transferrin iron complex enters the cell within an endosome. Acidification of the endosome releases the iron from transferrin so that it can enter the cell. The apotransferrin is delivered by the endosome to the plasma for reutilization. The method by which the transferrin receptor–independent pathway delivers iron to the cell is not known.

Nonintestinal cells also possess the mobilferrin integrin and DMT-1 pathways. Their function in the absence of an iron-saturated transferrin is uncertain; however, their presence in nonintestinal cells suggests that they may participate in intracellular functions in addition to their capability to facilitate cellular uptake of iron.

Etiology

Dietary factors

Meat provides a source of heme iron, which is less affected by the dietary constituents that markedly diminish bioavailability than nonheme iron is. The prevalence of iron deficiency Anaemia is low in geographic areas where meat is an important constituent of the diet. In areas where meat is sparse, iron deficiency is commonplace.

Substances that diminish the absorption of ferrous and ferric iron include phytates, oxalates, phosphates, carbonates, and tannates These substances have little effect upon the absorption of heme iron. Similarly, ascorbic acid increases the absorption of ferric and ferrous iron and has little effect upon the absorption of heme iron.

Both non heme iron and heme iron have 6 coordinating bonds; however, 4 of the bonds in heme bind pyrroles, making them unavailable for chelation by other compounds. Therefore, ascorbic acid chelates nonheme iron to enhance absorption but has no effect upon heme iron. Many dietary components, such as phytates, phosphates, oxalates, and tannates, bind nonheme iron to decrease nonheme iron absorption. They do not affect heme. This explains why heme is so effectively absorbed with foods containing these chelators. Iron hemoglobin structure.

Purified heme is absorbed poorly because heme polymerizes into macromolecules. Globin degradation products diminish heme polymerization, making it more available for absorption. They also increase the absorption of nonheme iron because the peptides from degraded globin bind the iron to prevent both precipitation and polymerization; thus, absorption of the iron in spinach is increased when the spinach eaten with meat. Heme and nonheme iron uptake by intestinal absorptive cells is noncompetitive.

Hemorrhage

Bleeding for any reason produces iron depletion. If sufficient blood loss occurs, iron deficiency Anaemia ensues A single sudden loss of blood produces a posthemorrhagic Anaemia that is normocytic. The bone marrow is stimulated to increase production of hemoglobin, thereby depleting iron in body stores. Once they are depleted, hemoglobin synthesis is impaired and microcytic hypochromic erythrocytes are produced.

Maximal changes in the red blood cell (RBC) cellular indices occur in approximately 120 days, at a time when all normal erythrocytes produced prior to the hemorrhage are replaced by microcytes. Before this time, the peripheral smear shows a dimorphic population of erythrocytes, normocytic cells produced before bleeding, and microcytic cells produced after bleeding. This is reflected in the red blood cell distribution width (RDW); thus, the earliest evidence of the development of an irondeficient erythropoiesis is seen in the peripheral smear, in the form of increased RDW.

Malabsorption of iron

Prolonged achlorhydria may produce iron deficiency because acidic conditions are required to release ferric iron from food. Then, it can be chelated with mucins and other substances (eg, amino acids, sugars, amino acids, or amides) to keep it soluble and available for absorption in the more alkaline duodenum.

Starch and clay eating produce malabsorption of iron and iron deficiency Anaemia. Specific inquiry is required to elicit a history of either starch or clay eating because patients do not volunteer the information.

Extensive surgical removal of the proximal small bowel or chronic diseases (eg, untreated sprue or celiac syndrome) can diminish iron absorption. Rarely, patients with no history of malabsorption have iron deficiency Anaemia and fail to respond to oral iron therapy. Most merely are noncompliant with therapy. Before placing these patients on parenteral therapy, document iron malabsorption either by measuring absorption of radioiron or by obtaining a baseline fasting serum-iron concentration and repeating the test 30 minutes and 1 hour after administration of a freshly prepared oral solution of ferrous sulfate (50-60 mg of iron) under observation. The serum iron should increase by 50% over the fasting specimen.

Genetic abnormalities producing iron deficiency have been shown in rodents (sex-linked Anaemia [sla] mice, microcytic Anaemia [mk] mice, Belgrade rat). This phenomenon has not been clearly demonstrated in humans; if it exists, it is probably an uncommon cause of iron deficiency Anaemia

Epidemiology

International statistics

In countries where little meat is in the diet, iron deficiency Anaemia is 6-8 times more prevalent than in North America and Europe. This occurs despite consumption of a diet that contains an equivalent amount of total dietary iron; the reason is that heme iron is absorbed better from the diet than nonheme iron. In certain geographic areas, intestinal parasites, particularly hookworm, worsen the iron deficiency because of blood loss from the GI tract. Anaemia is more profound among children and premenopausal women in these environs.

Age-related demographics

Healthy newborn infants have a total body iron of 250 mg (80 ppm), which is obtained from maternal sources. This decreases to approximately 60 ppm in the first 6 months of life, while the baby consumes an iron-deficient milk diet. Infants consuming cow milk have a greater incidence of iron deficiency because bovine milk has a higher concentration of calcium, which competes with iron for absorption. Subsequently, growing children must obtain approximately 0.5 mg more iron daily than is lost in order to maintain a normal body concentration of 60 ppm.

During adult life, equilibrium between body loss and gain is maintained. Children are more likely to develop iron deficiency Anaemia. In certain geographic areas, hookworm adds to the problem. Children are more likely to walk in soil without shoes and develop heavy infestations.

During childbearing years, women have a high incidence of iron deficiency Anaemia because of iron losses sustained with pregnancies and menses.

Gastrointestinal neoplasms become increasingly more prevalent with each decade of life. They frequently present with GI bleeding that may remain occult for long intervals before it is detected. Usually, bleeding from neoplasms in other organs is not occult, prompting the patient to seek medical attention before developing severe iron depletion. Investigate the etiology of the iron deficiency Anaemia to evaluate for a neoplasm.

Sex-related demographics

An adult male absorbs and loses about 1 mg of iron from a diet containing 10-20 mg daily. During childbearing years, an adult female loses an average of 2 mg of iron daily and must absorb a similar quantity of iron in order to maintain equilibrium. Because the average woman eats less than the average man does, she must be more than twice as efficient in absorbing dietary iron in order to maintain equilibrium and avoid developing iron deficiency Anaemia.

Healthy males lose body iron in sloughed epithelium, in secretions from the skin and gut lining, and from small daily losses of blood from the GI tract (0.7 mL daily). Cumulatively, this amounts to 1 mg of iron. Males with severe siderosis from blood transfusions can lose a maximum of 4 mg daily via these routes without additional blood loss.

A woman loses about 500 mg of iron with each pregnancy. Menstrual losses are highly variable, ranging from 10 to 250 mL (4-100 mg of iron) per period. These iron losses in women double their need to absorb iron in comparison to males. A special effort should be made to identify and treat iron deficiency during pregnancy and early childhood because of the effects of severe iron deficiency upon learning capability, growth, and development.

Race-related demographics

Race probably has no significant effect upon the occurrence of iron deficiency Anaemia; however, because diet and socioeconomic factors play a role in the prevalence of iron deficiency, it more frequently is observed in people of various racial backgrounds living in poorer areas of the world.

Prognosis

Iron deficiency Anaemia is an easily treated disorder with an excellent outcome; however, it may be caused by an underlying condition with a poor prognosis, such as neoplasia. Similarly, the prognosis may be altered by a comorbid condition such as coronary artery disease. Promptly and adequately treat a patient with iron deficiency Anaemia who is symptomatic with such comorbid conditions.

Chronic iron deficiency Anaemia is seldom a direct cause of death; however, moderate or severe iron deficiency Anaemia can produce sufficient hypoxia to aggravate underlying pulmonary and cardiovascular disorders. Hypoxic deaths have been observed in patients who refuse blood transfusions for religious reasons. Obviously, with brisk hemorrhage, patients may die from hypoxia related to posthemorrhagic Anaemia.

Whereas a number of symptoms, such as ice chewing and leg cramps, occur with iron deficiency, the major debility of moderately severe iron deficiency is fatigue and muscular dysfunction that impairs muscular work performance. In children, the growth rate may be slowed, and a decreased capability to learn is reported. In young children, severe iron deficiency Anaemia is associated with a lower intelligence quotient (IQ), a diminished capability to learn, and a suboptimal growth rate.

Patient Education

Physician education is needed to ensure a greater awareness of iron deficiency and the testing needed to establish the diagnosis properly. Physician education also is needed to investigate the etiology of the iron deficiency.

Public health officials in geographic regions where iron deficiency is prevalent need to be aware of the significance of iron deficiency, its effect upon work performance, and the importance of providing iron during pregnancy and childhood. The addition of iron to basic foodstuffs is employed in these areas to diminish the problem.

Iron Deficiency Anaemia Clinical Examinations

History

Whereas iron deficiency Anaemia is a laboratory diagnosis, a carefully obtained history can facilitate its recognition. The history can be useful in establishing the etiology of the Anaemia and, perhaps, in estimating its duration.

Iron deficiency in the absence of Anaemia is asymptomatic. One half of patients with moderate iron deficiency Anaemia develop pagophagia. Usually, they crave ice to suck or chew. Occasionally, patients are seen who prefer cold celery or other cold vegetables in lieu of ice. Leg cramps, which occur on climbing stairs, also are common in patients deficient in iron.

Often, patients can provide a distinct point in time when these symptoms first occurred, providing an estimate of the duration of the iron deficiency.

Fatigue and diminished capability to perform hard labor are attributed to the lack of circulating hemoglobin; however, they occur out of proportion to the degree of Anaemia and probably are due to a depletion of proteins that require iron as a part of their structure.

Increasing evidence suggests that deficiency or dysfunction of nonhemoglobin proteins has deleterious effects. These include muscle dysfunction, pagophagia, dysphagia with esophageal webbing, poor scholastic performance, altered resistance to infection, and altered behavior.

Dietary history

A dietary history is important. Vegetarians are more likely to develop iron deficiency, unless their diet is supplemented with iron. National programs of dietary iron supplementation are initiated in many portions of the world where meat is sparse in the diet and iron deficiency Anaemia is prevalent. Unfortunately, affluent nations also supplement iron in foodstuffs and vitamins without recognizing the potential contribution of iron to free radical formation and the prevalence of genetic iron overloading disorders.

Elderly patients, because of poor economic circumstances, may try to survive on a "tea and toast" diet because they do not wish to seek aid. They may also be hesitant to share this dietary information. This group is far more likely to develop protein-calorie mal nutrition before they develop iron deficiency Anaemia.

A fundamental concept is that after age 1 year, dietary deficiency alone is not sufficient to cause clinically significant iron deficiency and a source of blood loss should always be sought as part of the management of a patient with iron deficiency Anaemia. Infants and toddlers are the primary risk groups for dietary iron deficiency Anaemia. Neonates who double their birth weight are a special risk group. Pica is not a cause of iron deficiency Anaemia; pica is a symptom of iron deficiency Anaemia. It is the link between iron deficiency Anaemia and lead poisoning, which is why iron deficiency Anaemia should always be sought when a child is diagnosed with lead poisoning. Hippocrates recognized clay eating; however, modern physicians often do not recognize it unless the patient and family are specifically queried. Both substances decrease the absorption of dietary iron. Clay eating occurs worldwide in all races, though it is more common in Asia Minor. Starch eating is a habit in females of African heritage, and it often is started in pregnancy as a treatment for morning sickness.

History of hemorrhage

Two thirds of body iron is present in circulating red blood cells as hemoglobin. Each gram of hemoglobin contains 3.47 mg of iron; thus, each mL of blood lost from the body (hemoglobin 15 g/dL) results in a loss of 0.5 mg of iron.

Bleeding is the most common cause of iron deficiency, either from parasitic infection (hookworm) or other causes of blood loss. Patients report a history of bleeding from most orifices (hematuria, hematemesis, hemoptysis) before they develop chronic iron deficiency Anaemia; however, gastrointestinal bleeding may go unrecognized, and excessive menstrual losses may be overlooked.

Patients often do not understand the significance of a melanotic stool. Unless menstrual flow changes, patients do not seek medical attention. If they do, they report that their menses are normal in response to inquiry for self-evaluation. Because of the marked differences among women with regard to menstrual blood loss (10-250 mL per menses), query the patient about a specific history of clots, cramps, and the use of multiple tampons and pads.

Physical Examination

Anaemia produces nonspecific pallor of the mucous membranes. A number of abnormalities of epithelial tissues are described in association with iron deficiency Anaemia. These include esophageal webbing, koilonychia, glossitis, angular stomatitis, and gastric atrophy.



The exact relationship of these epithelial abnormalities to iron deficiency is unclear and may involve other factors. For example, in publications from the United Kingdom, esophageal webbing and atrophic changes of the tongue and the corner of the mouth are reported in as many as 15% of patients with iron deficiency; however, they are much less common in the United States and other portions of the world.

Spleenomegaly may occur with severe, persistent, untreated iron deficiency Anaemia.

Complications of Anaemia

Iron deficiency Anaemia diminishes work performance by forcing muscles to depend on anaerobic metabolism to a greater extent than they do in healthy individuals. This change is believed to be attributable to deficiency in iron-containing respiratory enzymes rather than to Anaemia.

Severe Anaemia due to any cause may produce hypoxemia and enhance the occurrence of coronary insufficiency and myocardial ischemia. Likewise, it can worsen the pulmonary status of patients with chronic pulmonary disease.

Defects in structure and function of epithelial tissues may be observed in iron deficiency. Fingernails may become brittle or longitudinally ridged, with the development of koilonychia (spoon-shaped nails). The tongue may show atrophy of the lingual papillae and develop a glossy appearance. Angular stomatitis may occur with fissures at the corners of the mouth.

Dysphagia may occur with solid foods, with webbing of the mucosa at the junction of the hypopharynx and the esophagus (Plummer-Vinson syndrome); this has been associated with squamous cell carcinoma of the cricoid area. Atrophic gastritis occurs in iron deficiency with progressive loss of acid secretion, pepsin, and intrinsic factor and development of an antibody to gastric parietal cells. Small intestinal villi become blunted.

Cold intolerance develops in one fifth of patients with chronic iron deficiency Anaemia and is manifested by vasomotor disturbances, neurologic pain, or numbness and tingling.

Rarely, severe iron deficiency Anaemia is associated with papilledema, increased intracranial pressure, and the clinical picture of pseudotumor cerebri. These manifestations are corrected with iron therapy.

Impaired immune function is reported in subjects who are iron deficient, and there are reports that these patients are prone to infection; however, because of the presence of other factors, the current evidence is insufficient to establish that this impairment is directly due to iron deficiency.

Children deficient in iron may exhibit behavioral disturbances. Neurologic development is impaired in infants and scholastic performance is reduced in children of school age. The intelligence quotients (IQs) of schoolchildren deficient in iron are reported to be significantly lower than those of their nonanemic peers. Behavioral disturbances may manifest as an attention deficit disorder. Growth is impaired in infants with iron deficiency. The neurologic damage to an iron-deficient fetus results in permanent neurologic injury and typically does not resolve on its own. Iron repletion stabilizes the patient so that his or her status does not further decline.

Differential Diagnosis

- Sideroblastic Anaemias
- Spherocytosis, Hereditary
- Thalassemia, Alpha
- Thalassemia, Beta

Prevention

Certain populations are at sufficiently high risk for iron deficiency to warrant consideration for prophylactic iron therapy. These include pregnant women, women with menorrhagia, consumers of a strict vegetarian diet, infants, adolescent females, and regular blood donors.

Pregnant women have been given supplemental iron since World War II, often in the form of all-purpose capsules containing vitamins, calcium, and iron. If the patient is anemic (hemoglobin < 11 g/dL), administer the iron at a different time of day than calcium because calcium inhibits iron absorption.

The practice of routinely administering iron to pregnant females in affluent societies has been challenged; however, it is recommended to provide prophylactic iron therapy during the last half of pregnancy, except in settings where careful followup for Anaemia and methods for measurement of serum iron and ferritin are readily available.

Iron supplementation of the diet of infants is advocated. Premature infants require more iron supplementation than term infants. Infants weaned early and fed bovine milk require more iron because the higher concentration of calcium in cow milk inhibits absorption of iron. Usually, infants receive iron from fortified cereal. Additional iron is present in commercial milk formulas.

Iron supplementation in populations living on a largely vegetarian diet is advisable because of the lower bioavailability of inorganic iron than heme iron. The addition of iron to basic foodstuffs in affluent nations where meat is an important part of the diet is of questionable value and may be harmful. The gene for familial hemochromatosis (HFe gene) is prevalent (8% of the US white population). Excess body iron is postulated to be important in the etiology of coronary artery disease, strokes, certain carcinomas, and neurodegenerative disorders because iron is important in free radical formation.

Long-Term Monitoring

Monitor patients with iron deficiency Anaemia on an outpatient basis to ensure that there is an adequate response to iron therapy and that iron therapy is continued until after correction of the Anaemia to replenish body iron stores. Followup also may be important to treat any underlying cause of the iron deficiency.

Response to iron therapy can be documented by an increase in reticulocytes 5-10 days after the initiation of iron therapy. The hemoglobin concentration increases by about 1 g/dL weekly until normal values are restored. These responses are blunted in the presence of sustained blood loss or coexistent factors that impair hemoglobin synthesis.

PRINCIPLES AND PROPERTIES OF TRIAL DRUG

1.Karisaalai

Botanical name

Eclipta prostrate, Roxb.

Family

Compositae.

English name.

Trailing eclipta.

Organoleptic characters

Taste: Kaipu

Potency : Vepam

Division: Karpu

Parts used.

Leaves, flowers.

Action

Cholagogue, emetic, tonic, aphrodisiac, hepatic tonic.

Pothu Gunam

குரற்கம்மற் காமாலை குட்டமொடு சோபை யுறற்பாண்டு பன்னோ யொழிய சொன்ன மெய்யாந் தகரையொத்த யீளி ண்ணூ நற்புலத்துக் கையாந் தகரையொத்தக் கால்.

குணபாடம் மூலிகை வகுப்பு.

Uses

Jaundice, pruritis, scabies, enlargement of spleen, liver.

2.Dhania

Botanical name

Coriandrum sativum, Linn.

Family

Umbelliferae

English name.

Coriander.

Organoleptic characters

Taste: Karpu Potency : Seetha Vepam Division: Karpu **Parts used.**

Fruit, leaves.

Pothugunam

கொத்துமல்லி வெப்பம் குளிர்காய்ச்சல் பித்தமந்தஞ் சாத்திவிக்கல் தாகமொடு தாதுநட்டம் - கத்தியெழும் வாத விகார்மடர் வன்கர்த்த பிவிரணம் பூதலத்தில் லாதகற்றும் போற்று.

குணபாடம் மூலிகை வகுப்பு.

Chemical Constituents

Coriandrol, dpinene, lpinene, mucilage, tannin, malic acid

Action

Aromatic, stimulant,tonic, carminative,anti-diabetic

Uses

Diabetes, flatulence, colic sore-throat, vertigo.

3. Mookkirattai

Botanical name

Boerhaavia diffusa, Linn.

Family

Nyctagineae.

English name.

Spreading hog-weed.

Organoleptic characters

Taste: Kaipu

Potency : Vepam

Division: Karpu

Parts used.

Herb, root. Punarnavine,

Chemical Constituents

potassium nitrate, fat, ash.

Action

Stomachic, laxative, diuretic, diaphoretic, expectorant.

Pothugunam

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சீத மகற்றுந் தினவடக்குங் கந்திதரும்
வாத வினையை மடிக்குங்காண் பேதி
கொடுக்குமதை உண்டாக்காற் கோமளமே! பித்தம்
அடுக்குமே மூக்குரட்டை யாய்.
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குணபாடம் மூலிகை வகுப்பு.

Uses

Dropsy, ascities, asthma, heart disease, stone in the kidney, colic.

4. Nellikai

Botanical name

Emblica officinalis.

Family

Euphorbiaceae.

English name

Emblicmyrobalan, Indian gooseberry.

Organoleptic Charecters

Taste	- Pulippu, Thuvarppu, Inippu
Potency	- Thatpam
Pirivu	- Inippu

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Parts used.

Dried fruit, nut or seed, leaves, rootbark, flowers

Action

Refrigerant, diuretic, laxative, astringent.

Pothugunam

பித்தமன லையம் பீநசம்வாய் நீா் வாந்தி மத்தமலக் காடும் மயக்கமுமில் - ஒத்தவுரு வில்லிக்கா யம்மருங்கா மென்னாட்கா லந்தோ்ந்தே நெல்லிகா யம்மருந் துணீ.

குணபாடம் மூலிகை வகுப்பு.

Uses

Jaundice, inflammation of lungs, hiccup, dyspepsia, nausea, vomiting, phthisis.

5.Elam

Botanical name

Elettaria cardamomum, Maton.

Family

Scitaminaceae.

English name

Cardamom.

Organoleptic characters

Taste: Karpu

Potency : Vepam

Division: Karpu

Parts used.

Dried ripe seeds, oil from fruits.

Chemical Constituents

Fixed oil, essential oil, volatile oil, cincole, free terpineol.

Action

Aromatic, stimulant, carminative, stomachic, diuretic.

Pothugunam

விக்கல் பெருவாந்தி வெய்யவழல் நீர்ப்பேதி மிக்கவெழும் பித்தம் மிகுமயக்கம் சிக்கலுற்ற மந்தம் வயிற்றுவலி மாதே விரைந்தோடும் அந்தமுறு மாஞ்சிக் கறி.

குணபாடம் மூலிகை வகுப்பு.

Uses

Stomach complaints, diarrhea, atonic dyspepsia, vomiting.

<u>6. Karunjeragam</u>

Botanical name

Nigella sativa, Linn.

Family

Rananculaceae

English name

Small fennel or Black cumin.

Parts used

Dried fruits and seeds.

Organoleptic Characters

Taste: KaippuPotency: Veppampirivu: Kaarppu

Chemical constituents

Volatile oil, mucilage, metarbin, melanthin, Arabic acid.

Action

Aromatic, diuretic, stomachic, stimulant, carminative, anthelmintic.

Pothu gunam

"கருஞ்சீ ரகத்தான் கரப்பனொடு பண்ணும் வருஞ்சிராய்ப் பீநசமு மாற்றும் – அருந்தினால் காய்ச்சல் தலைவலியுங் கண்வலியும் போமுலகில் வாய்ச்ச மருந்தெனவே வை." – குணபாடம் மூலிகை வகுப்பு.

Uses

Obstinate hiccup, fever, diarrhea, skin diseases, dropsy, puerperal diseases, sour belching.

7. Thippili

Botanical name

Piper longum, Linn.

Family

Piperaceae.

English name

Dried catkins, long-pepper.

Parts used

Immature berries, stems, roots.

Organileptic characters

Taste: Pungent Potency: Hot Division: Sweet

Chemical constituents

Resin, volatile oil, fatty oil, inorganic matter, piperine

Action

Stimulant, carminative, expectorant, diuretic.

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Pothu gunam
தாகபித்தஞ் சோகந் தணியாச் சுரமிருமல்
மேகங் குரற்கம்மல் மெய்க்கடுப்பும்-ஏகுங்கான்
திப்பிலிமூ லங்கண்டத் திப்பிலிய தாம்நறுக்குத்
திப்பிலியென் றேயொருக்காற் செப்பு.
- அகத்தியா் குணவாகடம்.
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Uses

Cough, cold, asthma, hoarseness, hiccup, colic, flatulence.

8.Milagu

Botanical name

Piper nigrum, Linn

Family

Piperaceae

English name

Black pepper, decorticated pepper; common pepper

Organoleptic characters

Taste: Kaipu,Karpu

Potency : Vepam

Division: Karpu

Part used

Dried unripe fruit

Chemical constituents

Piperine, piperidine, chavicin.

Action

Carminative, antiperiodic, resolvent, anti-pyretic.

Pothu gunam

"சீதசுரம் பாண்டு சிலேத்மங் கிராணிகுன்மம் வாதம் அருசிபித்தம் மாமூலம்-ஓடுசன்னி யாசமபஸ் மாரம் அடன்மேகம் காசமிவை நாசங் கறிமிளகினால்.

குணபாடம் மூலிகை வகுப்பு.

Uses

Dyspepsia, flatulence, colic, worms, ascitis, asthma, gonorrhoea, piles.

9. Thalisa pathiri

Botanical name

Taxus buccata, Linn.

Family

Coniferae.

English name

Himalayan yew.

Organoleptic characters

Taste: Karpu

Potency : Vepam

Division: Karpu

Part used

Leaves. Volatile oil

Chemical constituents

Volatile oil tannic and gallic acids, toxin, a resin.

Action

Carminative, expectorant, stomachic, tonic, anti lithic, anti spasmodic.

Pothugunam

நாசி களப்பிணிகள் நாட்பட்ட–காசஞ்சு வாசம் அருசி வனமங்கால் வீசிவரு மேகமந்தம் அத்திசுரம் விடேகுந் தாளிச்சத்தால் ஆகுஞ் சுகப்பிரச வம்.

குணபாடம் மூலிகை வகுப்பு.

Uses

Breast cancer, asthma, haemoptysis, epilepsy, calculus complaints, hysteria, spasmodic affections.

10. Thaandrikai

Botanical name

Terminalia bellerica, Roxb.

Family

Combretaceae.

English name

Belericmyrobalans.

Part used

Fruits.

Pothu gunam

"ஆணிப்பொன் மேனிக் கழகும் ஒளியுமிகும் கோணிக்கொள் வாதபித்தக்கொள்கைபோம் – தானிக்காய் கொண்டவர்க்கு மேகமறும் கூறா அனற்றணியும் கண்டவர்க்கு வாதம்போம் காண்".

குணபாடம் மூலிகை வகுப்பு.

Organoleptic characters:

Taste : Bitter Potency : Hot Pirivu : Sweet

Chemical constituents

Gallo-tannic acid, coloring matter, resin, oil.

Action

Astringent, tonic, laxative, expectorant.

Uses

Cough, hoarseness, sore-throat, dropsy, dysentery, diarrhea, fractures, asthma.

11.Kadukkai

Botanical name

Terminalia chebula, Retz.

Family

Combretaceae.

English name

Myrobalan, chebulic myrobalan.

Part used

Dried fruits, galls.

Organoleptic characters:

- **Taste :** Astringent, sweet, sour, acrid, bitter.
- Potency : Hot
- **Pirivu** : Sweet

Chemical constituents

Tannin, tannic acid, gallic acid, mucilage, chebulinic acid.

Action

Astringent, purgative, alterative, stomachic, laxative, tonic.

Pothu gunam

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"தாடை கழுத்தக்கி தாலு குறியிவிடப்
பீடை சிலிபதமுற் பேதிமுடம் - ஆடையெட்டாத்
தூலமிடி புண்வாத சோணிகா மாலையிரண்
டாலமிடி போம்வரிக்கா யால்".
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குணபாடம் மூலிகை வகுப்பு.

Uses

Worms, fever, cough, asthma, urinary disease, piles, hiccup, vomiting, swellings.

<u>12.Inji</u>

Botanical name

Zingiber officinale, Rosc.

Family

Zingiberaceae.

English name

Ginger.

Part used

Rhizome.

Organoleptic characters

Taste: Kaarpu Potency: Veppam Division: Kaarpu

Chemical constituents

Phellandrene, gingerol, gingerin.

Action

Carminative, aromatic, stimulant, increases

prostaglandins, adjuvant to tonic remedies.

Pothu gunam

சூலைமந்தம் நெஞ்செரிப்பு தோடமே பம்மழலை மூலம் இரைப்பிருமல் மூக்குநீர் - வாலகப தோடமதி சாரந் தொடர்வாத குன்மநீர்த் தோடம்ஆ மம்போக்குஞ் சுக்கு. .

குணபாடம் மூலிகை வகுப்பு.

Uses

Cough, cold, indigestion, dysentery, peptic ulcer, flatulence.

13.Seerakam

Botanical name

Cuminum cyminum, Linn

Family

Umbelliferae.

English name

Cumin seed, caraway seed.

Organoleptic characters

Taste: Karpu, Inipu

Potency : Thatpam

Division: Inipu

Part used

Fruit or seed,

Chemical constituents

essential oil. Thymine, carvone, cuminol or cumic aldehyde, cymene or cymol,

terpene.

Action

Carminative, aromatic, stomachic, stimulant, astringent.

Pothugunam

பித்தமெனு மந்திரியைப் பின்னப் படுத்தியவன் சத்துருவை யுந்துறந்து சாதித்து–மத்தனெனும் ரசனையு யீவென்று நண்பைப் பலப்படுத்தி போசனகு டாரிசெயும் போர்.

குணபாடம் மூலிகை வகுப்பு.

Uses

Hoarseness of voice, dyspepsia, chronic diarrhea, bilious nausea in pregnant women.

14. Athimathuram

Botanical name

Glycyrrhiza glabra, Linn

Family

Liquorice

Organoleptic characters

Taste: Inipu

Potency : Thatpam

Division: Inipu

Part used

Root and Rhizomes

Chemical constituents

Triterpenoids , saponin , glycyrrhizin , glycyrrhizinic acid , glabrin A&B, glycyrrhetol , glabrolide, isoglabrolide, isoflavones, coumarins, triterpene sterols etc..

Actions

Tonic, demulcent, expectorant, diuretic, mild laxative, anti-arthritic, antiinflammatory, anti-biotic, anti-viral, anti-ulcer, memory stimulant (being MAO inhibitor), anti-tussive, aphrodisiac, anti-mytotic, estrogenic, anti-oxidant, anti-caries agent, anti-neoplastic, anti cholinergic, anti-diuretic, hypolipidemic activity, etc.

Pothugunam

அதிமதுரம் பேரீந்துக் காணகுணங் கேளீர் கொதிமருவாப் பித்தங் குருகுமே–துதிமருவாய் நீரதுவு முண்டாம் நிலையாத தாபம்போம் பாரறியச் சொன்னோம் பகிர்ந்து.

குணபாடம் மூலிகை வகுப்பு.

Uses

Anaemia, menorrhagia-metrorrhagia, hoarseness of voice, cardiac tonic,

haematemisis. intrinsic haemorrhage.

15.Maramanjal

Botanical name

Coscinium fenestratum (Gaertn.) Colebr.

Family

Menispermaceae

English name

Tree turmeric

Part used

Stem.

Pothu gunam

அழன்றகண மூலம் அருசி யுடனே உழன்ற கணச்சுரமும் ஓடுஞ் - சுழன்றுள்ளே வீறுசுர முந்தணியும் வீசுமர மஞ்சளுக்குத் தேறு மொழியனமே செப்பு.

குணபாடம் மூலிகை வகுப்பு.

Organoleptic Charecters

Taste	- Kaippu
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Potency - Veppam

Pirivu - Kaarppu

Chemical constituents

Phenols, Alkaloids, Terpenoids, flavanoids

Uses

Diabetes mellitus, Fever, Dysentery, Piles, Ulcers











KARISALAI

DHANIA

MOOKIRATTAI

NELLIKAI

ELAM

65







KARUNJEERAKAM

THIPPILI

MILAGU



THALISAPATHIRI









SUKKU



SEERAGAM





ATHIMATHURAM

MARAMANJAL

METHODS OF THE STUDY

PREPARATION OF "KARISALANKANNI CHOORANAM"

Required raw drugs:

- 1. Karisali Eclipta alba,linn
- 2. Kadukai Terminalia chebula, Retz.
- 3. Nellivatral– Phyllanthus emblica, Linn
- 4. Thaandrikai Terminalia bellarica.(Gaertn) Rox
- 5. Mookkirattai Boerhaavia diffusa ,linn.
- 6. Sukku Zingiber officinalis, Rosc.
- 7. Milagu Piper nigrum, Linn.
- 8. Thippili Piper longum, Linn.
- 9. Karunjeragam Nigella sativa ,Linn
- 10. Seeragam Cuminum cyminum, Linn
- 11. Athimathuram Glycrrhiza glabra, Linn
- 12. Thalisa pathiri Abies spectabilis (d.don) mirr
- 13. Mara manjal Coscinium fenestratum, (Gaertn)Colebr.
- 14. Dhania- Coriandrum sativum ,linn
- 15. Elam: Elettaria cardamomum ,maton

Proportion of Drugs: Karisalai- 4 Thola (48 g) Other drugs-1 Thola (12 g) (each) [24]

Source of raw drugs

The above said raw drugs are purchased from a well reputed country shop .The raw drugs will be authenticated by the Head of the department of Medicinal Botany ,at NIS. The raw drugs are purified and the medicine is prepared in Gunapadam laborotary of NIS.The prepared medicine is again authenticated by the Head of the department of Gunapadam.

Purification of the raw drugs:

1.Karisali - Eclipta alba,linn

Clean the leaves with pure cloth and remove the rotten leaves([7]

2.Kadukai – Terminalia chebulla.Retz.

Soak the kadukai in rice water(kazhuneer), remove the yellowish tint of the water and seed and dry it.[8]

3.Nellikai – Phyllanthus emblica,Linn

Boil it with milk, remove the seed and dry it.[9]

4. Thaandrikai – Terminalia bellarica. (Gaertn) Roxb

Soak it in Pandanus odoratissimus(Thaludhalai),Linn.f juice for three hours (1-Samam) remove the seed and dry it. [10]

5.Sukku – Zingiber officinalis, Rosc

Double the proportion of lime stone[calcium carbonate] solution is poured and boiled for three hours, then wash it, dry and remove the peel. [11]

6.Milagu – *Piper nigrum,Linn.* Soak it in sour butter milk for three hours. [12]

7. Thippili – Piper longum, Linn.

Soak it in plumbago zeylanica,Linn(Kodiveli) leaf juice for twenty four minutes (1 Nazhigai) and dry it sun.[13]

8.Mara manjal – Coscinium fenestratum,(gaertn)Colebr.

Remove the peel cut it into pieces and dry it in sunlight. [14]

9.Karunjeragam – Nigella sativa ,Linn

Dry it in sunlight & fry it like as golden yellow colour [15]

10.Seeragam – Cuminum cyminum, Linn

Dry it in sunlight & fry it like as golden yellow colour[16]

11.Athimathuram – Glycrrhiza glabra,Linn

Wash with clean water and Remove the peel cut it into pieces[17]

12.Mookkirattai – Boerhaavia diffusa ,linn

Clean the leaves with pure cloth and remove the rotten leaves[18]

13.Thalisa pathiri – Abies spectabilis (d.don) mirr

Clean the leaves with pure cloth and remove the rotten leaves[19]

14.Dhania- coriandrum sativum ,linn

Boil Kothumali seed with Hotwater & dry it in sunlight[20]

15.Elam: elettaria cardamomum ,maton

Remove the peel & take the seeds[21]

METHOD OF PREPARATION

The above mentioned drugs are purified properly as said above and they are dried in shade & made into fine powder



KARISALANKANNI CHOORANAM

DRUG STORAGE: The drug thus prepared is stored in a clean and dry glass bottles.

PROTOCOL

1.0 TITLE :

A study on the **PITHA PANDU** (**Iron Deficiency Anaemia**) and the drug of choice is KARISALANKANNI CHOORANAM"

2.0 REG NO:32101202 /DD/ MM/2011 DATE OF SUBMISSION:14-12-2011

3.0 NAME OF THE INSTITUTION

National Institute of Siddha Tambarm Sanatorium, Chennai-47 Telephone No : 044-22411611 Fax : 044-22381314 E.Mail : nischennaisiddha@yahoo.co.in Website : www.nischennai.org

4.0 NAME AND DESIGNATION:

PG STUDENT:

DR. M.GOBI KRISHNAN MD II YR[2011-2012] DEPARTMENT OF MARUTHUVAM

5.0 BACKGROUND:

Anaemia is one of the worldwide health problem. It has significant prevalence in developing countries, like India because of low dietary intake of iron, chronic blood loss due to hookworm infestation Malaria. The World Health Organization estimates that 30% of the world's population is anaemic of which 50% is attributed to iron deficiency anaemia [1].[2]

Every age group is vulnerable to iron deficiency anaemia, and women are more likely to suffer than men because of the loss of blood every month, due to mensuration.

To overcome iron deficiency anaemia in India, The measures to be taken both in Preventive& curative aspects.

A well defined system developed by siddhars, known as Siddha system of medicine, in which the diseases are classified into 4448 types on the basis of Mukkutram. In Yoogi Vaithiya Chinthamani text Yoogi Munivar classified PANDU into 5types and PITHA PANDU^[3] is one among them.

It include the symptoms like *pallor of the skin and mucous membrane*, *fatigue,lassitude,chest discomfort,breathlessness,pica,giddiness,dizziness,angular stomatitis,glossitis,pungent or bitter taste* [3][4][5] well explained in siddha literature may be correlated with Iron Deficiency Anaemia,in Modern science.

I have Selected Karisalankani Chooranam, Because the main ingredients of this drug are available through out the year& It is affordable for the patient. The major ingredients have the potency to cure Iron deficiency Anaemia as per our text. Eventhough currently available oral iron preparations can correct iron deficiency, it has gastrointestinal side effects like nausea, constipation or diarrhea and it takes long duration to iron get replenshied.

In Sigicha Rathna Deepam **"KARISALANKANNI CHOORANAM"** is indicated for pandu specifically (Ref:Page: 162)

The ingredients of this formulation are found to be efficacious & cost effective.

In Gunapadam-Mooligai Vaguppu Karisalankanni has the indication for anaemia [6]

The above said drug formulation, has not undergone any clinical trial, so far. So, it is proposed to carry a Clinical Trial to find out its efficacy in Pitha Pandu.

6.0 OBJECTIVES

2) Primary objective:

To evaluate the Siddha Therapeutic efficacy of the Poly Herbal formulation

"KARISALANKANNI CHOORNAM" (Internal) in " PITHA PANDU" (Iron Deficienccy Anaemia)

2)Secondary objective:

-To evaluate the safety profile(acute,long term toxicity studies) of the trial drug -To study the effect of other co-factors such as age, sex & dietary influence

7.0 STUDY DESIGN AND CONDUCT OF THE STUDY

7.1 Study type: An Open Clinical Trial

7.2**Study place**:

Ayothidasar Pandithar Hospital, Dept of Maruthuvam National Institute of Siddha, Tambaram Sanatorium, Chennai-47.

7.3 **Study period**:12 Months

7.4 Sample size:40 patients.
7.5Treatment:

Medicine Name: KARISALANKANNI CHOORANAM (Internal)

Ref: Sigicha Ratna Deepam- pg 162

Dosage: Thirikadi pramanam [1g] [24] (Twice/day) before food.

Adjuvant: white sugar

Course: 1/2 Mandalam[24 days]

DISPENSING: The Chooranam is dispensed in Sachets.

QUANTITY OF MEDICINE:

A packet of 24 Sachets for 12 days, each sachet consist of 1 gm

The pt have to visit twice for 24 days

8.0 SUBJECT SELECTION

As and when patients reporting at OPD of Ayothidasar Pandithar Hospital,NIS with symptoms of inclusion criteria will be subjected to screening test & documented using screening proforma.

9.0 SELECTION CRITERIA

INCLUSION CRITERIA

- Age 18-55 of both sexes.
- Clinical symtoms of Pallor, Breathlessness, Palpitation, Anorexia, Giddiness, Numbness, Glossitis, lassitude, Fatigue, koilonychias etc.,
- Hb level less than normal range ie.,7-13mg/dl for Men,7-10mg/dl for Women
- Patient blood smear shows microcytic hypochromic RBC

EXCLUSION CRITERIA

- infection
- Bleeding disorder(Bleeding piles,Menorrhagia)
- Pregnancy and lactation
- Presence of any associated severe systemic illness (e.g.CA,RA)
- Endocrine disorder(Thyroid abnormality, Diabetes mellitus)
- H/o Steroid exposure for prolonged period.
- Cardiac disease
- Renal disease
- Peptic Ulcer
- Inherited defects(sickle cell Anaemia, Thalassemia)
- Malabsorption syndrome

WITHDRAWAL CRITERIA

- Intolerance to the drug, and development of adverse reactions during the drug trial
- Severe abdominal pain
- Nausea
- Any other acute illness
- Poor patient compliance and defaulters

11.0 ASSESSMENTS AND INVESTIGATIONS:

A)Clinical assessment

Siddha assessment

B)Routine investigations

C)Special investigations

A)CLINICAL ASSESSMENT:

- Pallor
- Oedema of the Body
- Breathlessness
- Palpitation
- Tachycardia
- Anorexia
- Giddiness
- Numbness
- Tingling sensation
- Lack of concentration
- Amenorrhoea.
- Angular stomatitis
- Glossitis
- Cheliosis
- Koilonychia
- Hair fall
- Lassitude
- Fatigue
- Pica
- Faint
- Chest Discomfort
- Bitter / Pungent taste[22] [23]

SIDDHA ASSESSMENT

1.Thinai :

- Kurinchi (hill areas)
- Mullai (forest)
- Marutham (fertile land)
- Neidhal (coastal area)
- Palai (desert)

2. Paruva Kalam (season)

- Karkaalam
- Koothir kaalm
- Munpanikaalm
- Pinpani kaalam
- Ilavenil kaalam
- Muthuvenil kaalam

3. Poripulankal:

- Mei (Skin etc)
- Vaai (Tongue etc)
- Kan (Eye etc)
- Mooku (Nose etc)
- Sevi (Ear etc)

4.Kanmedriyam and Gnanenthiriyam:

- Vaai (Buccal cavity)
- Kaal (Lower limbs)
- Kai (Upper limbs)
- Eruvaai (Anorectal region)
- Karuvaai (Uro-genital re

5. Ezhuudalkattugal:

- Saram
- Senneer
- Uoon
- Kozhuppu
- Enbu
- Moolai
- Sukkilam /suronitham

6.Ennvagaithervu (Eight types of Examination):

- Naadi
- Sparisam
- Naa
- Niram
- Mozhi
- Vizhi
- Malam
- Moothiram
 - -Neerkuri

-Neikuri

SIDDHA PARAMETERS

Malam

Moothiram

B)ROUTINE INVESTIGATIONS Modern Parameters:

✓ Liver function test-

SGOT: 6-18IU/L

SGPT: 3-26IU/L

Serum alkaline phosphotase: 3-12mg/dl

✓ **Renal function test**-Blood Urea: 16-50mg/dl

Creatinine: 0.6-1.2mg/dl

Uric acid: Men:3-9mg/dl,Women:2.5-

7.5mg/dl

Blood sugar level- Fasting (80-120 mg/dl)

Postbrandial < 130 mg/dl Random: <140mg/dl

- ✓ **Bleeding time:** 2-6/min
- ✓ **Clotting time**: 3-8/min
- ✓ Urine:Albumin- Nil Sugar-Nil Deposits-Nil Bile salts-Nil Bile pigments-Nil Urobilinogen-Nil
- ✓ Motion:Ova-Nil
 Cyst-Nil
 Occult blood-Nil

C)SPECIAL INVESTIGATIONS: 1) Complete Blood count: (Routine Test):

* Hb-Men:14-18gms/dl,Women:11-15gms/dl
*Total RBC-Men:4.5-6.5million/cu.mm for both sex.
*Total WBC:4000-11,000cubic mm.
*Differential count:
Polymorphs:40-75 %
Lymphocytes:20-35%
Monocytes:2-10%
Esinophils:1-6%

Basophils:0-1%

*ESR –Men: 0-10 (mm/hr),Women 0-20 (mm/hr)

(Special Test)

- Reticulocyte count:1-2%
- PCV-Men:45-55%,Women:35-45%.
- MCV:76-96 fl or cubic mm.
- MCH:27-33 pg.
- MCHC:31-35%
- * CI-1%
- Platelet count-1,50000-5,00000 lakhs/cubic mm
- Morphology of RBC-Normocytic normochromic



12.1 STUDY ENROLLMENT

- In this Clinical Trial , patients reporting at the OPD with the clinical symptoms of pallor, anorexia, giddiness, odema, palpitation, numbress, fatigue etc., will be examined clinically for enrolling in the study based on the inclusion and exclusion criteria.
- The patients who are to be enrolled will be informed (Form IV) about the study, trial drug, possible outcomes and the objectives of the study in the language and terms understandable to them.
- After ascertaining the patient's willingness, informed consent would be obtained in writing from them in the consent form(Form IV-A).
- 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. Screening Form- I will be filled up; Form I-A,

Form -II and Form -III will be used for recording the patient's history, clinical

Examination of symptoms and signs and laboratory investigations respectively.

 Patients would be advised to take the trial drug and appropriate dietary advice (Form IV-D) would be given according to the patient's perfect understanding.

13.0CONDUCT OF THE STUDY:

As per siddha literature, before starting the treatment for PITHA PANDU, purgation is given with the soup of sesban leaves mixed with palm jaggery ¹/₂-1 aazhaku(84-168 ml) Od at early morning, in empty stomach for one day.[24]

The trial drug ": KARISALANKANNI CHOORANAM" - 1 gm is given continuously for 24 days. For OP patients ,they should visit the hospital once in 12 days. At each clinical visit clinical assessment is done and prognosis is noted. For IP patients the drug is provided daily and prognosis is noted

Laborotary investigations are done at 0th day& 24th day of the trial.For IP patients, who is not in a situation to stay in the hospital for a long time is advised to attend the OPD for further continuation of the treatment.

During the course of the treatment, patient is advised not to take tamarind,tea,coffee,tobacco,betel leaf and advised to take the diet as given in Form IV- D.

After the end of the treatment also, the patient is advised to visit the OPD for another 2months for follow-up. If any of the 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.

14.0 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's 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 will be monitored for completion by HOD-(Dept of Maruthuvam) ,and adverse reactions by the pharmacovigilance department of NIS .

All collected data will be entered into computer using the MS access software . All forms will be further scrutinized in presence of Investigator by Sr.Research Officer (Statistics) for logical errors and incompleteness of data to avoid any bias. No modification in the results is permitted for unbiased reports. Data entry will be 100% gross checked manually

STATISTICAL ANALYSIS:

All collected data will be entered into the computer and manually crosschecked the correctness of the data entry. The clinical symptoms and the Hb level will be analysed by comparing the two point of data(before and after treatment) paired test and chi-square test will be employed to study the efficacy of treatment. Further, the effect of age and sex will also be analysed.

15.00UT COME OF TREATMENT

Primary Outcome:

Primary Outcome is mainly assessed by comparing the pre and post treatmental **Hemoglobin level**, of the trial patient.

Secondary Outcome:

Secondary outcome is assessed by comparing the following parameters ,before and after the treatment.

- 1) Reduction of Clinical symptoms
- 2) Changes in Complete blood count

16.0ADVERSE EFFECT/SERIOUS EFFECT MANAGEMENT:If the trial patient develops any adverse reaction,he/she would be immediately withdrawn from the trial & referred to the Pharmacovigilence ,Dept of NIS.

17.0 ETHICAL ISSUES

1.Informed consent will be obtained from the patient explaining in the understandable language to the patient.

2.After the consent of the patient (through consent form) they will be enrolled in the study

3. Treatment would be provided free of cost.

4.No other external or internal medicines will be used. There will be no infringement on the rights of patient.

5.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.

6.The data collected from the patient will be kept confidentially. The patient will be Informed about the diagnosis, treatment and follow-up.7.The patients who are excluded (as per exclusion criteria) are given proper treatment at national institute of siddha

8.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.

18.0 ASSESSMENT FORMS

Screening and Selection Proforma
History Proforma on enrollment
Clinical Assessment on enrollment
Clinical Assessment during and after the trial
Laboratory investigations on enrollment during and after the trial.
Information sheet
Consent form
Withdrawal form
Drug Compliance form
Dietary Advice form.
Adverse Reaction form

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை- 47 அய**ோ**த்திதாசர் பண்டிதர் மருத்துவமனை

பித்த பாண்டு நேேπய்க்கான சித்த மருந்துகளின் (கரிசலாங்கண்ணி சூரணம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான ஒப்புதல் படிவம்

ஒப்புதல் படிவம்

நான் மேற்கூறிய தகவல் படிவத்தை படித்து அல்லது படிக்க கேட்டுக் க**ொ**ண்டேன். இது த∩ொடர்பான விளக்கங்களையும் கேட்டு தெரிந்துக∩ொண்டேன்.எந்த வித வற்புரத்தலின்றி என் ச∩ொந்த விருப்பத்தின் பேரில் என்னை இந்த ஆராய்ச்சிக்கு உட்படுத்த என் முழுமனதேோடும் சுயனநினைவோடும் சம்மதம் தெரிவிக்கின்றேன். எனக்கு விருப்ப்பம் இல்லாத பட்ச்சத்தில் இந்த ஆராய்ச்சியில் இருந்து என்னை எப்போது வேண்டுமானாலும் விடுவித்து க**ொ**ள்ளும் உரிமையை பெற்றுள்ளேன் என்பதையும் அறிவேன்

தேதி :		கைய ொ ப்பம்
இடம் :		பெயர் :
தேதி கைய ொ ப்பம் இடம் :		சாட்சிக்காரர் பெயர்
உறவுமுறை	:	

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OBSERVATION AND RESULTS

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

- 1. Age distribution
- 2. Sex distribution
- 3. Religion distribution
- 4. Socio-ecnomic distribution
- 5. Occupational distribution
- 6. Educational distribution
- 7. Dietary distribution
- 8. Reference to Thegi
- 9. Reference to Thinai
- 10. Reference to Season
- 11. Reference to Iymporigal
- 12. Reference to Iympulangal
- 13. Reference to Kosangal
- 14. Reference to Mukutram
- 15. Reference to Ezhu udalkattugal
- 16. Reference to Ennvagai thervugal
- 17. Reference to Neikkuri
- 18. Reference to Signs and Symptoms
- 19. Reference to OP / IP Investigation Results
- 20. Results after treatment

i) Primary outcome -	Hb before and after treatment.
ii) Secondary outcome	- Results from Complete Blood Count
	- Results from Iron supply studies
	- Results from clinical signs and symptoms

21. Statistical Analysis

The observation recorded are given below in tabular form

S. No	Age	No of cases	Percentage%
1	13-20	0	0%
2	20-40	25	62.5%
3	40-55	15	37.5%





Observation and Inference: Among the 40 cases treated 25(62.5%) cases belonged to 20-40 years and 15(37.5%) cases belonged to 40-55 years. The percentage is more in the age group of 20-40 years.

2.	SEX	DISTRIBUTIO)N
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S. No	Sex	No of Cases	Percentage%
1	Female	38	95%
2	Male	2	5%



Observation and Inference: 38 (95%) of female cases and 2(5%) cases of male cases presented with anaemia were included in this study.

3. RELIGION DISTRIBUTION

S. No	Religion	No of cases	Percentage%
1	Hindu	38	95%
2	Christian	0	0%
3	Muslim	2	5%



Observation and Inference: Out of 40 cases, 38 (95%) cases belonged to Hindu and

2(5%) case belonged to Muslim religion.

S.No	Socio-ecnomic status	Number of cases	Percentage %
1	Upper middle class	8	20%
2	Middle class	12	30%
3	Poor	20	50%

4. SOCIO-ECNOMIC DISTRIBUTION



Observation and Inference: Among the 40 cases, 8(20%) cases belonged to Upper middle class economic status, 12(30%) cases belonged to middle class people and 20(50%) belonged to poor economic status. The percentage is more in poor economic group.

S. No	Occupational status	No of cases	Percentage%
1	Students	0	0 %
2	working	16	40%
3	House wives	24	60%

5. OCCUPATIONAL DISTRIBUTION



Observation and Inference: Of the 40 cases, 16(40%) were working women and 24(60%) were house wives. The percentage is more in house wives.

S. No	Educational Status	No of cases	Percentage %
1	Literate – Degree holder	16	40%
2	Literate – Upto 12 th std	20	50%
3	Illiterate	4	10%

7. EDUCATIONAL DISTRIBUTION



Observation and Inference: Out of 40 cases, 16(40%) cases were degree holders, 20 (50%) were studied upto 12^{th} standard and 4(10%) were illiterates.

8.	DIETARY	DISTRIBUTION
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S. No	Diet	No of cases	Percentage%
1	Vegetarian	12	30%
2	Non-vegetarian	28	70%



Observation and Inference: Among 40 cases, 70 % of cases belonged to non-

vegetarian dietary habit and 30% belonged to vegetarian dietery habit.

8.REFERENCE TO THEGI [BODY CONSTITUTION]

S. No	Thegi	No of cases	Percentage%
1	Vatham	4	10%
2	Pitham	28	70%
3	Kabam	0	0%
4	Thontham	8	20%



Observation and Inference: Of the 40 cases, 4(10%) cases were in Vadha body constitution 28(70%) cases were in pitha body constitution, 8(20%) cases were in thontham body constitution and no cases were in kabam body constitution. The percentage was more in pitham type of body constitution.

S. No	Thinai	No of cases	Percentage%
1	Kurinji (Hill area)	0	0%
2	Mullai (Forest area)	0	0%
3	Marutham (Fertile area)	12	30%
4	Neithal (Coastal area)	28	70%
5	Paalai (Desert area)	0	0%

9. REFERENCE TO THINAI



Observation and Inference: Among the 40 cases, 12(30 %) belonged to Marutham nilam, 28(70 %) belonged to Neithal nilam.

S. No	Paruva kaalam	No of cases	Percentage%
1	Kaar(Aug 16-Oct15)	4	10%
2	Koothir(Oct 16-Dec 15)	0	0%
3	Munpani(Dec 16-Feb 15)	0	0%
4	Pinpani(Feb 16-Apr 15)	0	0%
5	Elavenil(Apr 16-June 16)	0	0%
6	Mudhuvenil(June16-Aug 15)	36	90%

10.REFERENCE TO SEASON



Observation and Inference: Among the 40 cases, 4 (10%) cases were treated during kaar and 36(90%) in Muthuvenil kalam.

S.No	Iymporigal	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Mei [skin]	32[80%]	32[80%]
2	Vaai [Buccalcavity]	12[30%]	12[30 %]
3	Kan [Eyes]	36[90%]	32[80%]
4	Mooku[Nose]	0[0%]	0[0%]
5	Sevi [Ear]	0[0%]	0[0%]

11. **REFERENCE TO IYMPORIGAL**



- Of the 40 cases, Mei [skin] was affected noted as pallor, numbress, dryness, in 32 (80%) cases and all the patients were improved after the treatment.
- Of the 40 cases, vaai [buccal cavity] was affected noted as glossitis, angular stomatitis, bitter or pungent taste, dryness, pallor, fissured and coated tongue in 12 (30%) cases and all the patients were improved after the treatment.
- Of the 40 cases, Kan [eye] was affected noted as pallor, blurred vision in 36(90%) cases and 32(80%) were improved after the treatment.

S.No	Iympulangal	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Kai [upper limb]	32[80%]	32[80%]
2	Kaal [lower limb]	28[70%]	28[70%]
3	Vaai[Buccalcavity]	12 [30%]	12[30%]
4	Eruvai[Anus]	8 [20%]	4[10%]
5	Karuvai[Genital organ]	4 [10%]	4 [4%]

12.REFERENCE TO IYMPULANGAL



- Of the 40 cases, Kai [upper limb] was affected noted as numbness, pain in 32 (80%) cases, Kaal [lower limb] was affected noted as numbness, pain, paedal edema in 28 (70%),Vaai [buccal cavity] was affected noted as glossitis, angular stomatitis, bitter or pungent taste, dryness, pallor, fissured and coated tongue in 12 (30%) cases, Karuvai was affected (ammenorrhoea), in 4(10%) cases and all the patients were improved after the treatment.
- Of the 40 cases, Eruvai organ was affected (Constipation) in 8(20%) and 4(10%) were improved after the treatment.

S.No	Kosam	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Annamaya kosam	32 [80%]	32 [80%]
2	Pranamaya kosam	32 [80%]	28 [70%]
3	Manomaya kosam	32 [80%]	24 [60%]
4	Vinganamayakosam	16 [40%]	16 [40%]
5	Anandamaya kosam	4 [10%]	4 [10%]

13.REFERENCE TO KOSANGAL



- Of the 40 cases, Annamayakosam was affected, noted as loss of appetite in 32(80%) cases, Manomayakosam was affected, noted as palpitation in 32 (80%) cases, Vinganamayakosam was affected, noted as pain, numbness and tingling sensation in 16 (40%) cases and Anandamayakosam was affected, noted as oligomenorrhoea in 4(10%) cases all the patients were improved after the treatment.
- Of the 40 cases, Pranamayakosam was affected, noted as breathlessness in 32(80%) cases and 28(70%) of the patients were improved after the treatment.

14. REFERENCE TO MUKKUTRAM

A.VATHAM:

S.No	Vatham	No of cases	No of cases
		affected before treatment	Improved after
		[Percentage%]	treatment[Percentage%]
1	Pranan	36 [90%]	32 [80%]
2	Abanan	12 [30%]	8 [20%]
3	Viyanan	32 [80%]	32 [80%]
4	Uthanan	28 [70%]	28 [70%]
5	Samanan	38[90%]	38 [90%]
6	Naagan	0[0%]	0[0%]
7	Koormam	8 [20%]	8 [20%]
8	Kirukaran	36 [90%]	36 [90%]
9	Devathathan	40 [100%]	40 [100%]
10	Dhananjeyan	Not Applicable	Not Applicable



Observation and Inference:

Of the 40 cases, Pranan was affected, noted as breathlessness in 36(90%) cases, Abanan was affected, noted as flattulence, constipation, ammenorrhoea and oligomenorrhoea in 12(30%) cases and Uthanan was affected, noted as breathlessness in 28(70%) cases before the treatment and 32(80%), 8(20%), 28(70%) cases were improved from the affection of Pranan, Abanan and Uthanan respectively, after the treatment. Of the 40 cases, Viyanan was affected noted as pain, numbress, and tingling sensation in 32(80%) cases, Samanan was affected noted as loss of appetite, pain, numbress and breathlessness in 38(90%) cases, Koorman was affected noted as blurred vision in 8(20%) cases, Kirukaran was affected noted as loss of appetite, dryness of mouth in 36(90%) cases , Devadhathan was affected noted as fatigue in 40(100%) of the cases and all of them were improved after the treatment.

B.PITHAM:

S. No	Pitham	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Analam	38 [90%]	38 [90%]
2	Ranjagam	40 [100%]	37 [92.5%]
3	Prasagam	40 [100%]	37 [92.5%]
4	Alosagam	2 [5%]	2 [5%]
5	Sathagam	37 [92.5%]	37 [92.5%]



- Of the 40 cases, Analam was affected noted as loss of appetite in38(90%) cases, Alosagam was affected noted as dull vision in 2(5%) cases, Sathagam was affected noted as fatigue in 37(92.5%) cases and all the cases were improved after the treatment.
- Of the 40 cases, Ranjagam and Prasagam were affected in all 40(100%) cases noted as pallor, of which 37(92.5%) cases were improved after the treatment.

C.KABHAM:

S.No	Kabam	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Avalambagam	32[80%]	30[75.5%]
2	Kilethagam	8 [20%]	8 [20%]
3	Pothagam	12[30%]	12[30%]
4	Tharpagam	0 [0%]	0 [0%]
5	Santhigam	0 [0%]	0 [0%]



- Of the 40 cases, Avalambagam was affected noted as breathlessness in 32(80%) cases before the treatment and 30(75.5%) cases were improved after the treatment.
- Of the 40 cases, Kilethagam was affected noted as indigestion in 8(20%) cases,
 Pothagam was affected in 12(30%) cases noted as feeling of pungent or bitter taste of the tongue of which all of them were improved after the treatment.

S.No	Udal kattugal	No of cases affected before treatment [Percentage%]	No of cases improved after treatment [Percentage%]
1	Saaram	40 [100%]	40 [100%]
2	Senneer	40 [100%]	37 [92.5%]
3	Oon	12 [30%]	12 [30%]
4	Kozhuppu	0 [0%]	0 [0%]
5	Enbu	0 [0%]	0 [0%]
6	Moolai	0 [0%]	0 [0%]
7	Suronitham	8[20%]	4 [10%]

14.REFERENCE TO EZHU UDALKATTUGAL



- Regarding seven Udal kattugal, Saaram [noted as fatigue] and Senneer [noted as pallor, reduction of Hemoglobin level] were affected in all 40 patients (100%) before the treatment and 40(100%) patients of saram affection and 37(92.5%) patients of senneer affection were improved after the treatment.
- Oon was affected noted as paedal edema in 12(30%) cases, before the treatment and all of them were improved after the treatment.
- Suronitham was affected in 8 (20%) patients noted as oligomennorhoea before the treatment and 4 (10%) cases were improved after the treatment.

S.No	Ennvagai thervugal	No of cases observed before treatment [Percentage%]	No of cases observed after treatment[Percentage%]
1	Pithavatham Vathapitham	38 [95%] 2 [5%]	24 [60%] 16 [40%]
		Affected cases before treatment [percentage%]	Improved cases,after treatment [percentage%]
2	Sparisam	32 [80%]	32 [80%]
3	Naa	36 [90%]	36 [90%]
4	Niram	36 [90%]	36 [90%]
5	Mozhi	0[0%]	0[0%]
6	Vizhi	36 [90%]	36 [90%]
7	Malam	8 [20%]	8 [20%]
8	Moothiram	0[0%]	0[0%]

15.REFERENCE TO ENVAGAI THERVUGAL



Observation and Inference:

Among the Ennvagai Thervugal, Naa [pallor, coated, glossitis, angular stomatitis, baldness, fissure, dryness, pungent or bitter taste, decreased salivation] Niram [pallor] and Vizhi [pallor] were affected in 36 cases (90%) and all the 36 [90%] cases were improved after the treatment.

- ➤ 32 [80%] cases were affected from Sparisam [noted as dryness, hot or cold sensation, excessive sweat] and 8[20%] cases were affected from Malam [noted as constipation] before the treatment and all of them were improved after the treatment.
- ➤ 38[95%] were observed in pithavatham and 2[5%] vathapitham naadi before the treatment and 24[60%] were observed in pithavatham and 16[40%] vathapitham naadi after the treatment.

S. N	Neikkuri	No of cases observed before treatment [percentage%]	No of cases observedAfter treatment [percentage%]
1	Ring form-Pithaneer	28 [70%]	20 [50%]
2.	Serpentineform-Vathaneer	12 [30%]	20 [50%]

16.REFERENCE TO NEIKKURI



Observation and Inference: Of the 40 patients, 28 [70 %] cases were observed in Pitha neer and remaining 12 (30 %) cases were observed in Vatha neer before the treatment and 20 [50 %] cases were observed in Pitha neer and 20 (50 %) cases were observed in Vatha neer after the treatment

		No of cases	No of cases
S.No	Signs and symptoms	Affected before	improved after
		treatment	treatment
		[percentage%]	[percentage%]
1	Pallor	40 [100%]	37 [92.5%]
2	Anorexia	32 [80%]	30 [75%]
3	Fatigue	24 [60%]	24 [60%]
4	Tachycardia	28 [70%]	28 [70%]
5	Palpitation	28 [70%]	28 [70%]
6	Giddiness	28 [70%]	28 [70%]
7	Breathlessness	32 [80%]	30 [75%]
8	Pungent taste of tongue	4 [10%]	4 [10%]
9	Bitter taste of tongue	8 [20%]	8 [20%]
10	Angular stomatitis	8 [20%]	8 [20%]
11	Glossitis	4 [10%]	2 [5%]
12	Lack of concentration	24 [60%]	18 [45%]
13	Hairfall	28 [70%]	24[60%]
14	Numbness	16[40%]	16 [40%]
15	Tingling sensation	12 [30%]	8 [20%]
16	Ammenorrhoea	0 [0%]	0 [0%]
17	Oligomenorrhoea	8 [20%]	4 [10%]
18	Odema	0 [0%]	0 [0%]
19	Koilonychia	0 [0%]	0 [0%]
20	Pica	0 [0%]	0 [0%]

17.REFERENCE TO SIGNS AND SYMPTOMS

- Out of 40 cases, Pallor was noted in all the 40 [100%] cases before the treatment. Of which, 37 [92.5%] cases in Pallor showed improvement after the treatment.
- Out of 40 cases, Anorexia was noted in 32 [80%] cases of which 30[75%] showed improvement after the treatment.
- Tachycardia, Palpitation, Giddiness was noted in 28 [70%] cases before treatement and it was relieved all the 28[70%] after treatement.
- Anorexia and Breathlessness were noted in 32 [80%] cases before treatement and it was relieved all the 30[75%] after treatement.
- Out of 40 cases, Lack of concentration was noted in 24[60%] cases before the treatment and 18 [45%] showed improvement after the treatment.

- Out of 40 cases, Hairfall was noted in 28 [70%] cases before the treatment and 24 [60%] showed improvement after the treatment.
- Out of 40 cases, Oligomenorrhoea was noted in 8 [20%] cases before the treatment. Of which, 4[10%] cases showed improvement after the treatment.

19. OP / IP INVESTIGATION RESULTS:

Patients had undergone for Investigations [Blood, Urine and Motion] on 0th day and 48th day for assessing the prognosis of the treatment and the safety of the patients. The reports are given below in the tabular column.
OP/II	P INVESTIGATI	ON RESULTS															
				Before Tr	eatment						After Tr	eatment					
S No	O.P /IP No	Name	Age/ Sex	Hb gms/dl	RBC millio n/ cu.m m	PCV %	MCV fl	MCH pg	MCHC %	PBS	Hb gms/dl	RBC million/ cu.mm	PCV %	MCV fl	MCH pg	MCHC %	PBS
1	<i>B77139</i>	Rani	45/F	10.5	3.9	31.3	79.5	25.6	32.1	H.M	10.9	4.2	33.7	78.6	25.4	32.6	H.M
2	C84193	Sarojini	37/F	9.3	4.6	34.7	73.8	24.1	32.7	H.M	11.4	4.7	35.5	74.4	23.9	32.1	H.M
3	B75370	Surya Gandhi	21/F	9.8	3.6	29.9	78.5	25.2	32.1	H.M	12.8	4.1	37.0	86.7	29.9	34.5	H.M
4	C74519	Renuga	44/F	9.7	4.2	32.9	77.2	25.1	32.5	H.M	10.1	3.9	30.0	75.9	25.9	33.7	H.M
5	C80290	Sumathi	22/F	9.3	4.4	36.9	82.4	28.1	34.1	H.M	13.0	4.6	38.3	83.4	27.8	33.9	H.M
6	C71808	Rani	47/F	10.3	4.2	28.4	75.0	24.5	32.7	H.M	10.8	4.3	32.4	75.3	25.1	33.1	H.M
7	B85745	Usha	44/F	10.0	4.4	33.7	76.1	24.6	32.3	H.M	9.5	4.3	33.2	77.2	24.4	32.2	H.M
8	C84645	Chandrakantha	39/F	7.0	4.2	26.1	61.8	16.6	26.8	H.M	7.4	4.4	27.6	62.6	16.8	26.8	H.M
9	C69738	Jamuna	45/F	9.8	4.6	38.0	81.4	27.8	34.2	H.M	12.8	4.6	37.0	80.0	27.7	34.6	N.N
10	C82941	Rani	30/F	8.3	3.7	29.3	87.7	29.4	33.5	H.M	13.8	4.6	40.9	87.8	29.6	33.7	N.N
11	C82329	Lakshmi	48/F	9.2	4.2	36.8	86.2	29.0	33.7	H.M	13.3	4.4	38.9	87.5	29.5	34.6	N.N
12	C65259	Malarvili	35/F	9.4	4.2	36.1	84.3	29.0	34.3	H.M	12.5	4.2	36.4	84.9	29.4	34.6	H.M
13	B85887	Malasivakumar	42/M	9.6	5.0	32.1	63.4	19.0	29.9	H.M	9.3	4.8	30.9	63.2	19.0	30.1	H.M
14	B75701	Saraswathi	38/F	9.8	5.1	34.3	66.1	20.8	33.0	H.M	11.3	5.4	35.7	66.0	20.9	31.7	H.M
15	C84838	Shanthi	38/F	9.8	4.7	40.0	83.5	27.8	33.3	H.M	12.7	4.4	37.1	82.8	28.3	34.2	N.N
16	C82323	Yasim Khan	40/M	11.1	4.3	38.9	88.8	32.2	36.2	H.M	13.9	4.3	38.0	90.0	32.3	36.6	H.M
17	C79216	Radika	23/F	9.8	4.0	30.1	74.9	24.4	32.6	H.M	10.0	4.2	31.0	72.9	23.5	32.3	H.M
18	C75910	eshwari	21/F	9.8	4.7	40.6	85.5	29.5	34.5	H.M	14.0	4.8	40.4	86.2	29.2	34.7	N.N
19	C85414	Priya	<i>31/F</i>	9.8	4.1	37.5	85.2	29.8	34.9	H.M	13.1	4.4	36.8	83.3	29.6	35.6	H.M
20	C85572	Indra	28/F	8.9	4.3	38.2	87.6	29.4	33.5	H.M	12.8	4.2	37.0	89.6	30.2	34.6	H.M

				Befor	e Treatme	ent					After Tr	eatment					
S No	O.P/I.p No	Name	Age/ Sex	Hb gms/ dl	RBC millio n/ cu.mm	PCV %	MCV fl	MCH Pg	MCHC %	PBS	Hb gms/dl	RBC million/ cu.mm	PCV %	MCV fl	MCH pg	MCHC %	PBS
21	C85518	Saratha	36/ F	9.8	3.1	36.6	81.2	27.5	31.3	H.M	12.4	4.4	35.8	80.6	27.9	34.6	N.N
22	C85417	Janagi	44/ F	8.9	3.8	32.9	84.8	28.9	34.0	H.M	11.0	3.7	31.8	85.0	29.0	34.6	H.M
23	C86521/4016	Sarojamma	54/F	8.7	3.5	23.4	63.1	18.1	28.6	H.M	6.8	3.6	23.8	63.7	18.6	29.3	H.M
24	C27061/3986	Valliyammal	45/F	8.9	3.8	29.3	82.5	26.5	32.1	H.M	9.3	4.1	30.7	73.6	32.3	30.3	H.M
25	C85468	Tamilselvi	40/ F	9.0	5.2	40.0	76.7	25.4	33.2	H.M	12.8	5.0	38.6	78.6	35.4	33.2	N.N
26	C86420/4015	Subarathinam	50/F	9.3	3.8	33.7	88.2	29.8	33.8	H.M	11.7	4.0	40.4	95.4	30.1	45.2	H.M
27	C79659/4013	Lalitha	23/F	9.8	4.0	31.8	71.9	25.7	35.0	H.M	11.3	4.3	34.4	78.4	25.7	32.8	H.M
28	C86320/4014	Murugalakshmi	27/F	8.7	4.3	24.6	56.4	15.4	27.2	H.M	7.1	4.5	26.1	57.1	15.7	29.2	H.M
29	C26014	Sanmugavalli	51/ F	9.6	4.4	36.3	81.2	26.8	33.1	H.M	12.3	4.5	36.6	80.4	27.0	33.6	H.M
30	C85864	Rahamath Nisha	30/F	7.4	3.2	22.4	68.9	22.8	33.0	H.M	7.0	3.7	20.9	76.0	25.8	34.0	H.M
31	C85412	Renuga	39/F	9.4	4.0	34.1	84.2	27.7	32.8	H.M	10.9	3.9	32.5	88.5	27.4	33.5	H.M
32	C80021/4024	Malar	36/F	9.4	3.9	35.3	89.4	30.4	34.0	H.M	11.8	3.8	34.6	89.2	30.4	34.1	H.M
33	C86719/4027	Lalitha	33/F	9.0	3.9	34.7	87.6	31.1	35.4	H.M	12.6	4.0	35.7	88. <i>3</i>	31.2	35.3	N.N
34	C80773/4028	Malliga	40/F	9.4	4.6	39.1	84.1	29.1	34.8	H.M	14.0	4.7	40.3	84.7	29.4	34.7	N.N
35	C80725/4943	Srinivasan	44/ M	12.6	4.9	35.1	76.0	24.0	34.2	H.M	12.9	4.9	38.5	76.5	26.0	33.9	H.M
36	C83591	VijiyaGeetha	36/ F	9.2	4.3	30.0	68.8	21.1	30.7	H.M	8.5	4.1	27.9	67.7	27.9	30.5	H.M
37	C87572	Monidevi	35/F	7.8	2.6	86.5	30.0	34.7	25.5	H.M	7.1	4.7	17.9	80.6	34.3	34.1	H.M
38	C90816	Manimozhi	48/F	9.1	5.0	31.2	62.3	20.3	29.2	H.M	12.8	4.1	37.0	86.7	29.9	34.5	H.M
39	AL8984	Shanthi	35/F	9.4	4.3	30.5	70.4	21.7	30.8	H.M	10.3	4.6	31.5	70.8	22.2	31.4	H.M
40	C87999/4132	Renugadevi	42/F	7.3	4.0	23.8	58.6	16.0	27.3	H.M	6.2	4.0	24.9	64.7	17.0	27.0	H.M

Hb-Hemoglobin, PCV-Packed cell volume, MCV-Mean corpuscular volume, MCH-Mean corpuscular hemoglobin, MCHC-Mean corpuscular hemoglobin concentration, PBS-Pheripheral bloodsmear, H.M-Hypochromicmicrocytic, S.H.M-Slightly hypochromic microcytic, N.N-Normochromic normocytic.

									BLC	DOD							
S.N	NAME			Before T	reatment					After T	reatment			Before Tr	eatment	After Tree	utment
•		TC Cells	DC %			ESR		TC Cells	DC %			ESR		BT/min	CT/min	BT/min	CT/min
		cu.mm	Р	L	Ε	½ hr	1 hr	си.тт	Р	L	Ε	½ hr	1 hr				
1	Rani	6300	50	46	4	8	16	6600	40	58	2	6	12	2.30	4	2.3	3.45
2	Sarojini	7500	58	39	3	10	20	10200	50	46	4	8	16	2	4.30	2.30	5
3	Surya Gandhi	5600	66	27	1	15	2	6500	47	44	1	14	12	2	6	2.30	5
4	Renuga	5400	56	112	2	10	22	5300	58	38	4	8	16	3	6.15	1.4	3
5	Sumathi	6100	66	30	4	20	40	7200	39	59	2	10	20	2.30	3.30	2	4.30
6	Rani	7200	60	38	3	4	8	6400	46	48	6	6	16	2.35	8	2	4
7	Usha	9000	60	35	5	10	20	6900	64	35	1	24	48	3	5.45	2.3	4.30
8	Chandrakantha	7500	73	23	4	8	16	6400	63	35	2	8	16	2	5.05	2	4.30
9	Jamuna	8500	43	44	1	6	20	5200	71	30	6	6	10	1.30	3.30	2.45	2.50
10	Rani	6800	45	40	4	8	16	6400	65	34	3	4	12	.30	3.40	2	4.30
11	Lakshmi	4500	50	45	5	8	16	7800	54	37	9	14	12	2	3	2	2
12	Malarvili	4900	51	18	2	12	44	5100	69	23	3	4	8	3	4	3	4
13	Malasivakumar	4800	55	40	5	10	20	8300	60	39	1	10	20	2	4.15	2	4
14	Saraswathi	7700	66	28	6	8	16	6600	62	34	3	8	16	3	6	2	5
15	Shanthi	7000	63	35	2	10	20	8000	60	36	4	10	20	3	4	5	4.10
16	Yasim Khan	5200	60	36	4	8	16	5500	74	25	1	2	4	2	7	2	4.30
17	Radika	6700	55	40	5	20	40	8000	62	30	8	20	40	3	6.45	2.3	4.30
18	Mageshwari	6500	65	29	6	8	16	7000	68	28	4	4	8	3	6.30	2	4
19	Priya	5500	65	33	2	6	12	6300	78	18	4	6	12	2	4.30	3.30	4.30
20	Indra	6700	72	26	2	10	20	6500	79	19	2	10	20	2	4	2	3
				11	1												

		BLOOD															
S.N	NAME			Before T	reatment					After Ti	reatment			Before Tr	eatment	After Trea	tment
•		TC Cells	DC %			ESR		TC Cells	DC %			ESR		BT/min	CT/min	BT/min	CT/min
		cu.mm	Р	L	Ε	1/2 hr	1 hr	cu.mm	Р	L	Ε	1/2 hr	1 hr	DI/min	C1/min	DI/man	C1/man
21	Saratha	9000	41	58	1	22	44	8200	58	39	3	10	20	2	3.30	2	4.30
22	Janagi	4900	54	43	3	2	4	3800	53	44	1	6	12	1.30	4	1.3	3.45
23	Sarojamma	6500	43	44	2	14	12	4200	52	28	7	10	12	2.40	4.50	2.50	6
24	Valliyammal	5900	45	43	5	8	24	4800	49	47	4	16	32	2	2.45	4.15	5.30
25	Tamilselvi	7600	65	33	2	18	36	6700	67	30	3	12	24	2	4.45	3	4
26	Subarathinam	5000	51	28	3	4	28	7800	33	64	3	5	20	1.45	3	2.20	5.30
27	Lalitha	6600	62	36	2	2	4	5900	58	39	1	10	16	2	4.30	2	5.15
28	Murugalakshmi	7800	66	32	2	14	28	5600	56	40	13	12	24	2	5	2.45	5.45
29	Sanmugavalli	4400	72	27	1	10	20	6700	82	17	1	10	20	1.30	5.30	3.3	5
30	Rahamath Nisha	6100	72	26	2	4	30	6300	68	28	13	4	28	3	5	2.30	6
31	Renuga	7500	66	37	5	14	28	5900	56	42	2	6	30	2.30	3.30	1.45	3.30
32	Malar	5200	60	38	13	10	20	7900	56	35	4	10	12	1.45	6.30	1.45	4
33	Lalitha	5900	72	83	2	6	12	6900	56	40	12	6	12	1	4	2.15	5.15
34	Malliga	6800	66	25	4	6	8	6000	65	32	4	20	20	2.30	6.30	2.30	5.40
35	Srinivasan	7700	58	46	1	12	16	5200	56	41	3	8	40	1.30	2.20	4	3.15
36	VijiyaGeetha	7300	75	23	2	10	20	7600	71	27	2	12	24	3	4	2	4
37	Monidevi	6500	58	37	1	12	24	5800	79	18	3	12	12	2.15	4.30	2	4.45
38	Manimozhi	7100	62	30	3	2	8	6100	55	29	7	30	24	1	4	2	4.30
39	Shanthi	4600	66	43	9	10	12	6200	63	52	1	14	60	1.40	6	2.30	4
40	Renugadevi	4700	66	32	2	6	12	4200	60	35	5	6	20	2	6.50	3	4.30
TC-T	otal Count ; DC - Differ	ntial Cour	nt; P - Po	lymorp	hs ; L -	Lymphoc	ytes; $E - E$	Eosinophi	ls; ESR	-Erythr	ocyte s	edimentat	ion rate	e, BT-Blee	eding time	e,CT-Clott	ing
time,N	G-Not given																

					OP	/IP II	NVES	TIGATIONS					
					1	LIVER	FUNC	TION TEST					
		Before Trea	tment [IU/L]	After Treatn	nent [IU]/L]	Before Treatm	ient [IU/L]		After Treatme	nt [IU/L]	
S.No	Name	T.B	D.B	I.B	T.B	D.B	I.B	SGOT	SGPT	AP	SGOT	SGPT	AP
1	Rani	0.6	0.3	0.3	0.7	0.4	0.3	42	24	189	23	23	206
2	Sarojini	0.5	0.3	0.3	0.6	0.3	0.2	17	17	165	20	18	139
3	Surya Gandhi	0.5	0.3	0.2	0.6	0.4	0.2	24	28	191	11	19	142
4	Renuga	0.5	0.3	0.2	0.6	0.3	0.3	39	24	164	33	37	109
5	Sumathi	0.6	0.3	0.3	0.5	0.3	0.2	27	26	113	34	38	115
6	Rani	0.7	0.4	0.3	0.5	0.3	0.2	21	16	183	19	21	161
7	Usha	0.5	0.3	0.2	0.5	0.3	0.2	19	23	130	24	21	232
8	Chandrakantha	0.5	0.3	0.2	0.7	0.5	0.2	16	20	190	25	29	163
9	Jamuna	0.6	0.3	0.3	0.7	0.5	0.4	42	45	304	58	67	359
10	Rani	0.6	0.4	0.2	0.4	0.2	0.2	23	19	221	32	30	119
11	Lakshmi	0.5	0.3	0.2	0.7	0.5	0.2	37	17	129	24	26	192
12	Malarvili	0.6	0.4	0.2	0.6	0.4	0.2	13	16	159	16	19	164
13	Malasivakumar	0.5	0.3	0.2	0.6	0.3	0.3	29	19	235	36	31	221
14	Saraswathi	0.6	0.4	0.2	0.6	0.3	0.3	17	12	139	16	20	147
15	Shanthi	0.7	0.4	0.3	0.5	0.4	0.1	29	24	194	30	36	202
16	Yasim Khan	0.5	0.3	0.2	0.6	0.3	0.3	29	19	235	36	31	221
17	Radika	0.5	0.3	0.2	0.5	0.3	0.2	18	18	206	26	23	188
18	Mageshwari	0.5	0.3	0.2	0.6	0.4	0.2	29	19	254	21	25	256
19	Priya	0.6	0.3	0.3	0.5	0.3	0.2	28	19	124	16	12	156
20	Indra	0.7	0.4	0.3	0.6	0.4	0.2	30	33	130	40	38	206

					OP	/IP II	NVES	TIGATIONS					
					1	LIVER	FUNC	TION TEST					
		Before Trea	tment []	[U/L]	After Treatn	ient [IL	[/L]	Before Treatm	ent [IU/L]	•	After Treatme	nt [IU/L]	
S.No	Name	T.B	D.B	I.B	T.B	D.B	I.B	SGOT	SGPT	AP	SGOT	SGPT	AP
21	Saratha	0.5	0.3	0.2	0.5	0.3	0.2	20	20	196	29	28	129
22	Janagi	0.9	0.5	0.4	0.9	0.5	0.4	19	11	142	27	22	194
23	Sarojamma	0.4	0.2	0.2	0.8	0.5	0.3	30	26	276	22	16	168
24	Valliyammal	0.5	0.3	0.2	0.5	0.3	0.2	24	18	181	25	20	194
25	Tamilselvi	0.5	0.3	0.2	0.5	0.3	0.2	24	26	182	13	17	139
26	Subarathinam	0.5	0.3	0.2	0.7	0.4	0.3	23	25	180	20	20	229
27	Lalitha	0.5	0.4	0.1	0.7	0.5	0.2	17	19	161	32	23	101
28	Murugalakshmi	0.5	0.2	0.3	0.8	0.5	0.3	18	22	137	34	39	166
29	Sanmugavalli	0.7	0.4	0.3	0.6	0.4	0.2	30	33	130	40	38	206
30	Rahamath Nisha	0.8	0.5	0.3	0.5	0.3	0.2	21	23	206	17	19	134
31	Renuga	0.7	0.4	0.3	0.6	0.3	0.3	21	23	186	14	22	194
32	Malar	0.8	0.5	0.3	0.5	0.3	0.2	16	17	138	14	20	199
33	Lalitha	0.8	0.5	0.3	0.7	0.5	0.2	17	15	159	26	20	163
34	Malliga	0.5	0.3	0.2	0.6	0.3	0.2	18	15	218	25	21	214
35	Srinivasan	0.5	0.3	0.3	0.4	0.2	0.2	15	21	139	21	12	142
36	VijiyaGeetha	0.8	0.5	0.3	0.4	0.2	0.2	25	29	175	16	21	223
37	Monidevi	0.5	0.3	0.2	0.6	0.4	0.2	31	34	295	16	13	189
38	Manimozhi	0.9	0.5	0.4	0.5	0.3	0.2	20	19	272	16	12	272
39	Shanthi	0.5	0.3	0.2	0.7	0.4	0.3	19	20	127	36	26	143
40	Renugadevi	0.7	0.5	0.2	0.6	0.3	0.3	28	27	184	17	21	166

T.B-Total bilirubin, D.B-Direct bilirubin, I.B-Indirect bilirubin, SGOT-Serum glutamic oxaloacetic transaminase, SGPT-Serum glutamic pyruvate transaminase, AP-Alkaline phosphatase.

								OP/IP IN	VESTI	GATIO	N RESL	JLTS							
		BLOOD	SUGA	R/ LIPII	D PRO	FILE [m	g/dl]							RENA	L FUNCTIO	N TEST [mg/	[dl]		
		Before Tr	reatmer	ıt				After Tre	atment					Before	e Treatment	_	After 2	Treatment	-
		RBS	Lipid	profile	[mg/di]	-	RBS	Lipi	d profil	e [mg/d	[]]							
S.No	Name	[mg/dl]	TC	HDL	LDL	VLDL	TGL	[mg/dl]	TC	HDL	LDL	VLDL	TGL	urea	creatinine	Uric acid	urea	creatinine	uric acid
1	Rani	107	183	44	116	23	247	94	204	38	133	33	373	21	0.6	3.4	19	0.6	3
2	Sarojini	75	170	36	145	29	100	68	162	39	104	19	96	19	0.6	4.2	17	0.6	3.9
3	Surya Gandhi	77	118	35	145	21	105	73	177	36	121	20	99	15	0.6	4.3	33	0.7	4
4	Renuga	85	265	54	56	13	67	92	122	48	58	17	178	26	0.7	4.5	21	0.6	3.4
5	Sumathi	87	122	44	66	12	60	118	133	42	75	16	165	16	0.6	6.7	15	0.6	4.3
6	Rani	75	213	42	115	17	84	154	168	29	113	25	127	15	0.6	4.8	26	0.6	2.6
7	Usha	95	152	36	94	22	111	114	162	39	97	25	121	25	0.7	5.4	24	0.6	3.7
8	Chandrakantha	78	276	47	202	27	138	71	232	39	149	44	219	19	0.7	4.3	18	0.6	5.4
9	Jamuna	102	201	36	129	21	105	97	168	33	127	18	41	19	0.6	4.1	23	0.7	3.7
10	Rani	74	199	41	125	33	169	76	173	32	114	27	133	18	0.6	4.5	15	0.6	2.6
11	Lakshmi	104	180	55	102	23	116	92	193	36	92	20	100	20	0.6	4.2	23	0.7	4.6
12	Malarvili	85	140	41	78	26	104	104	137	37	59	35	174	16	0.6	4.7	19	0.6	3.4
13	Malasivakumar	93	198	39	121	33	164	95	239	36	170	33	168	16	0.6	3.2	26	0.7	4.3
14	Saraswathi	94	157	38	90	29	146	79	194	36	126	32	157	16	0.6	4.7	18	0.6	3.1
15	Shanthi	98	187	42	92	53	50	145	181	37	136	40	198	26	0.7	3.9	20	0.6	6.7
16	Yasim Khan	105	200	55	129	16	81	108	283	43	207	38	190	19	0.6	3.4	17	0.6	3.9
17	Radika	85	218	66	104	48	240	83	219	40	104	75	82	15	0.6	4.2	15	0.6	4.2
18	Mageshwari	119	123	44	162	59	116	124	324	28	221	79	396	17	.7	3.7	23	0.6	4.8
19	Priya	92	176	32	99	15	265	71	156	30	110	16	80	15	0.80	3.9	16	0.7	4.6
20	Indra	104	190	54	88	37	189	93	227	38	148	30	84	20	0.6	2.7	17	0.6	3.7

								OP/IP IN	VESTIC	GATION	V RESU	LTS							
		BLOOD	SUGA	R/ LIPI	D PRO	FILE [n	ıg/dl]							RENA	L FUNCTIO	N TEST [mg	/dl]		
		Before T	reatme	nt				After Tre	atment					Befor	e Treatment		After	Treatment	
		RBS	Lipid	profile	[mg/d	1]		RBS	Lipi	d profil	e [mg/d	11]							
S.No	Name	[mg/dl]	TC	HDL	LDL	VLDL	TGL	[mg/dl]	TC	HDL	LDL	VLDL	TGL	urea	creatinine	Uric acid	urea	creatinine	uric acid
21	Saratha	118	224	39	164	25	128	91	265	34	192	39	193	29	0.8	3.9	15	0.6	5.7
22	Janagi	94	140	42	129	10	79	129	213	36	115	36	115	34	0.8	2.9	17	0.6	5.5
23	Sarojamma	79	173	39	100	33	168	72	139	46	67	26	128	22	0.7	3.7	19	0.6	3.3
24	Valliyammal	70	245	39	145	21	105	88	175	41	115	19	95	22	0.6	2.8	15	0.6	3.5
25	Tamilselvi	85	231	37	39	235	40	90	274	<i>3</i> 8	205	31	155	34	0.9	4.7	25	0.7	3.9
26	Subarathinam	77	190	36	121	33	164	62	129	30	87	12	58	20	0.6	3.6	16	0.6	3.6
27	Lalitha	73	165	49	96	20	100	93	236	31	179	20	98	16	0.6	4.7	16	0.6	4.3
28	Murugalakshmi	90	217	46	142	29	145	79	183	48	109	25	128	25	0.7	3	26	0.7	2.5
29	Sanmugavalli	157	163	45	96	22	112	82	182	30	101	25	126	23	0.6	2.9	25	0.6	4.7
30	Rahamath Nisha	118	251	37	154	60	304	92	255	38	168	49	244	15	0.6	4.5	23	0.6	2.5
31	Renuga	80	191	32	132	27	136	83	160	45	88	27	135	20	0.7	5.1	15	0.6	3.9
32	Malar	80	183	39	116	28	140	89	162	41	96	19	97	22	0.6	4.7	22	0.6	3.2
33	Lalitha	87	172	35	99	38	193	91	150	48	75	27	136	22	0.6	3.4	28	0.7	2.6
34	Malliga	96	155	42	97	16	80	90	186	33	139	14	68	15	0.6	3.8	40	0.9	2.7
35	Srinivasan	88	250	51	161	38	189	92	241	42	162	37	181	27	0.7	3.4	15	0.6	5
36	VijiyaGeetha	77	141	28	99	14	73	101	132	39	216	17	87	26	0.6	4.3	20	0.6	2.7
37	Monidevi	88	148	35	93	20	102	77	126	26	83	17	88	16	0.6	3.3	16	0.6	2.3
38	Manimozhi	70	94	30	48	23	112	76	181	45	113	23	113	17	0.6	2.7	37	0.6	3
39	Shanthi	93	225	39	156	30	151	93	240	49	171	21	101	21	0.6	4.4	19	0.6	4.1
40	Renugadevi	85	208	42	130	36	183	91	119	46	50	23	113	21	0.6	5.2	25	0.6	3.7

RBS-Random blood sugar, TC-Total cholesterol, HDL-High density cholesterol, LDL-Low density cholesterol, VLDL-Very low density chlosterol, TGL-Triglycerides.

	Μ	IOTION	N TEST										URINE '	rest After treatment					
		Befor	e treatm	ent	After	treatme	ent			Befo	re treat	ment				Afte	r treatm	nent	
S.no	Name	ova	cyst	OB	ova	cyst	OB	А	RBS	BS	BP	UB	DEP	А	RBS	BS	BP	UB	DEP
1	Rani	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	4-6P 8-10E	nil	nil	nil	neg	Ν	4-8P E-plenty
2	Sarojini	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-2P,E	nil	nil	nil	neg	Ν	2-4P,E
3	Surya Gandhi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-3P 3-4E	nil	nil	nil	neg	Ν	1-2P,E
4	Renuga	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	nil	nil	nil	nil	neg	Ν	2-4P,E
5	Sumathi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-2P,E	nil	nil	nil	neg	Ν	2-4P,E
6	Rani	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P 8-10E	nil	nil	nil	neg	Ν	2-4P 1-2E
7	Usha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	3-5P,E	nil	nil	nil	neg	Ν	nil
8	Chandrakantha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	2-4P 4-6E	nil	nil	nil	neg	N	2-4P 3-6E
9	Jamuna	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	P,E-plenty	nil	nil	nil	neg	Ν	P,E-plenty
10	Rani	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	Ν	2-4P,E
11	Lakshmi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	6-8P,E	nil	nil	nil	neg	Ν	3-5P,E
12	Malarvili	Р	nil	nil	Р	nil	nil	nil	nil	nil	neg	Ν	1-2P,E	nil	nil	nil	neg	Ν	2-4P,E
13	Malasivakumar	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-2P,E	nil	nil	nil	neg	Ν	2-4P,E
14	Saraswathi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-2P 1-3E	nil	nil	nil	neg	Ν	nil
15	Shanthi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	2-4P,E	nil	nil	nil	neg	N	8-10P E-plenty
16	Yasim Khan	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	Ν	2-4P,E
17	Radika	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-2P,E	nil	nil	nil	neg	Ν	P,E-plenty
18	Mageshwari	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-3P 2-4E	nil	nil	nil	neg	N	2-4P 1-3E
19	Priya	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	nil	nil	nil	nil	neg	Ν	2-4P 3-6E
20	Indra	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	2-4P	nil	nil	nil	neg	Ν	2-4P 3-6E

		MOTIO	ON TEST			URINE TEST													
		Before	treatment		After tr	eatment		Befo	re treatme	nt				After tr	eatment				
S.no	Name	ova	cyst	OB	ova	cyst	OB	А	RBS	BS	BP	UB	DEP	А	RBS	BS	BP	UB	DEP
21	Saratha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	N	P,E-plenty
22	Janagi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	nil	nil	nil	nil	neg	Ν	4-8P 8-10 E
23	Sarojamma	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	6-8P,E	nil	nil	nil	neg	Ν	nil
24	Valliyammal	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	Ν	2-3P,E
25	Tamilselvi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	Ν	2-4P,E
26	Subarathinam	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	8-10E Pplenty	nil	nil	nil	neg	Ν	1-2P,E
27	Lalitha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	3-6P,E	nil	nil	nil	neg	Ν	2-3P 1-2E
28	Murugalakshmi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	P,E-plenty	nil	nil	nil	neg	N	P,E-plenty
29	Sanmugavalli	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	2-4P,E	nil	nil	nil	neg	N	2-4P,E
30	RahamathNisha	Р	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	P,E-plenty	nil	nil	nil	neg	Ν	1-2P 0-1E
31	Renuga	Р	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	4-5P 2-3E	nil	nil	nil	neg	N	2-4P,E
32	Malar	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	N	2-4P,E	nil	nil	nil	neg	N	1-2P,E
33	Lalitha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P 3-6E	nil	nil	nil	neg	Ν	1-2P 2-4E
34	Malliga	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P 3-6E	nil	nil	nil	neg	Ν	1-2P,E
35	Srinivasan	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P,E	nil	nil	nil	neg	Ν	2-3P,E
36	VijiyaGeetha	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	4-5P,E	nil	nil	nil	neg	Ν	2-4P,E
37	Monidevi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	1-3P 1-2E	nil	nil	nil	neg	Ν	2-4P,E
38	Manimozhi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	4-8P 8-10E	nil	nil	nil	neg	Ν	2-4P,E
39	Shanthi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	2-4P 5-6E	nil	nil	nil	neg	Ν	2-4P 1-2E
40	Renugadevi	nil	nil	nil	nil	nil	nil	nil	nil	nil	neg	Ν	nil	nil	nil	nil	neg	Ν	2-3 P,E

OB-Occult blood, A-Albumin, RBS-Random blood sugar, BS-Bile salt, BP-Bile pigment, UB-Urobilinogen, DEP-Deposits, N-Normal, Neg-Negative, P, E-Pus cells, Epithelial cells, P-Present, NG-not given.

20. RESULTS AFTER TREATMENT:

Results were observed on the basis of two main criteria.

Primary Outcome:

Primary Outcome is mainly assessed by comparing the pre and post treatmental **Hemoglobin level**, of the trial patient.

Secondary Outcome:

Secondary outcome is assessed by comparing the following parameters, before and after the treatment.

- 1) Reduction of Clinical symptoms
- 2) Changes in Complete Blood Count

HEMOGLOBIN CHART BEFORE AND AFTER TREATMENT

S.N	OP/IP NO	HB BEFORE TMT	HB AFTER TMT
1	B77139	10.5g	10.9g
2	C84193	9.3g	11.4g
3	B75370	9.8g	12.8g
4	C74519	9.7g	10.1g
5	C80290	9.3g	13.0g
6	C71808	10.3g	10.8g
7	B85745	10.0g	9.5g
8	C84645	7.0g	7.4g
9	C69738	9.8g	12.8g
10	C82941	8.3g	13.8g
11	C82329	9.2g	13.3g
12	C65259	9.4g	12.5g
13	B85887	9.6g	9.3g
14	B75701	9.8g	11.3g
15	C84838	9.8g	12.7g
16	C82323	11.1g	13.9g
17	C79216	9.8g	10.0g
18	C75910	9.8g	14.0g
19	C85414	9.8g	13.1g
20	C85572	8.9g	12.8g
21	C85518	9.8g	12.4g
22	C85417	8.9g	11.0g
23	C86521/4016	8.7g	6.8g
24	C27061/3986	8.9g	9.3g
25	C85468	9.0g	12.8g
26	C86420/4015	9.3g	11.7g
27	C79659/4013	9.8g	11.3g
28	C86320/4014	8.7g	7.1g
29	C26014	9.6g	12.3g
30	C85864	7.4g	7.0g
31	C85412	9.4g	10.9g
32	C80021/4024	9.4g	11.8g
33	C86719/4027	9.0g	12.6g
34	C80773/4028	9.4g	14.0g
35	C80725/4943	12.6g	12.9g
36	C83591	9.2g	8.5g
37	C87572	7.8g	7.1g
38	C90816	9.1g	12.8g
39	AL8984	9.4g	10.3g
40	C87999/4132	7.3g	6.2g

i) PRIMARY OUTCOME:

Results	derived	from	the	Hemoglobin
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S. N	Hemoglobin	Improvement	No of cases [Percentage %]
1	1-5 gms increase from its base level	Good	22 [55%]
2	0.1-0.9 gms increase from its base level	Moderate	10 [25%]
3	0.1-2 gms decrease from its base level	Poor	8 [20%]



Among the 40 cases, 32 [80%] cases showed Good improvement in the hemoglobin level, of which 22(55%) cases showed Moderate improvement of 1 to 5 grams in the hemoglobin level and 10(25%) cases showed increase of 0.1-0.9 grams in the hemoglobin level.8 [20%] cases showed no improvement in the Hemoglobin level.

ii) SECONDARY OUTCOME:

1. Results from clinical improvement:

Good, moderate and mild improvements were assessed on the basis as follows,

Good improvement

- Reduction of pallor, hairfall.
- > Restoration of regular menstrual cycle.
- Relief of signs and symptoms such as fatigue, palpitation, tachycardia, pungent or bitter taste of the tongue, giddiness, breathlessness, numbness, tingling sensation.
- > Absence of angular stomatitis, glossitis.

Moderate improvement

- ▶ No reduction in pallor, hairfall.
- Reduction of signs and symptoms such as fatigue, palpitation, tachycardia, pungent or bitter taste of the tongue, giddiness, breathlessness, numbness, tingling sensation.
- > Mild improvement in angular stomatitis, glossitis

Mild improvement

- No reduction of pallor, hairfall.
- Presence of oligomenorrhoea
- Reduction of signs and symptoms such as fatigue, palpitation, tachycardia, pungent or bitter taste of the tongue, giddiness, breathlessness, numbness, tingling sensation and koilonychia.
- Presence of angular stomatitis, glossitis

S. N	Signs and symptoms	No of cases [Percentage%]
1	Good	83.75%
2	Moderate	10.85%
3	Mild	5.40%



Among the 40 cases, 83.75% cases showed good improvement, 10.85% cases showed

moderate improvement and 5.40% cases showed mild improvement.

2. Results from Complete Blood Count:

Results from RBC:

S. N	RBC	No of cases [Percentage%]
1	Increased from its base level	26 [65%]
2	Did not change from its base level	9 [22.5%]
3	Reduced from its base level	5 [12.5%]

Regarding RBC of the 40 cases, 26 cases (65%) increased from its base level, 9 cases (22.5%) did not show any change from its base level and 5 cases (12.5%) showed reduction from its base level.

Results from PCV:

S. No	PCV	No of cases [Percentage %]
1	Increased from its base level	22[55%]
2	Did not change from its base level	0[0%]
3	Reduced from its base level	18[45%]

Regarding PCV of the 40 cases, 22[55%]showed increase from its base level, and 18[45%]showed reduction from its base level.

Results from MCV:

S. N	MCV	No of cases [percentage%]
1	Increased from its base level	27 [67.5%]
2	Did not change from its base level	0[0%]
3	Reduced from its base level	13 [32.5%]

Regarding MCV of the 40 cases, 27 [67.5%]showed increase from its base level and 13 [32.5%]showed reduction from its base level.



Results from MCH:

S. N	МСН	No of cases [Percentage%]
1	Increased from its base level	28 [70%]
2	Did not change from its base level	2 [5%]
3	Reduced from its base level	10 [25%]

Regarding MCH of the 40 cases, 28 [70%] showed increase from its base level, 2 [5%] did not show any change in its base level and 10 [25%] showed reduction from its base level.

Results from MCHC:

S. N	МСНС	No of cases [percentage%]
1	Increased from its base level	26 [65%]
2	Did not change in its base level	2[5%]
3	Reduced from its base level	12 [30%]

Regarding MCHC of the 40 cases, 26 [65%] showed increase from its base level and 12 [30%] showed reduction from its base level. 2[5%] did not change in its base level.

Results from Morphology of RBC:

S. No	Morphology of RBC	No of cases [Percentage%] After Tmt.
1	Normocytic Normochromic	9 [22.5%]
2	Microcytic Hypochromic	31 [77.5%]

Regarding Morphology of RBC, Out of 40 cases, 9 [22.5%] cases became Normocytic Normochromic after the treatment and 31 [77.5%] cases remained in Microcytic Hypochromic after the treatment.



22.STASTICAL ANALYSIS:

Student Paired 't' test was used to test the significance of treatment using before and after treatment data on HB, RBC, PCV, MC,MCH, MCHC. The level of significance probablity 0.05 was used to test the treatment difference and the values were statistically significant.

Statistical data for Hemoglobin:

Variable	N	Mean	Std.dev	Std. Error Mean	p.value
Before treatment HB	40	9.34	.97	.1544	<0.001
After treatment HB	40	11.10	2.24	.3552	

The mean \pm standard deviation of HB at before and after treatment were 9.34 \pm .97 and 11.10 \pm 2.24 which is statistically significant (p <0.001).

Stastical data for RBC:

Variable	Ν	Mean	Std.dev	Std.ErrorMean	t.value
Before treatment RBC	40	4.175	.5429	.0858	< 0.35
After treatment RBC	40	4.335	.3833	.0606	

RBC before treatment is $4.175 \pm .5429$ and after treatment is $4.335 \pm .3833$ which is stastically significant (P< 0.35)

Stastical data for PCV:

Variable	Ν	Mean	Std.dev	Std.ErrorMean	t.value
Before treatment PCV	40	34.427	9.6885	1.5319	< 0.685
After treatment PCV	40	33.693	5.4620	.8636	

PCV before treatment is 34.427 ± 9.6885 and after treatment is 33.693 ± 5.4620 which is stastically significant (P< 0.685)

Stastical data for MCV:

Variable	Ν	Mean	Std.dev	Std.ErrorMean	t.value
Before treatment MCV	40	76.130	11.8449	1.8728	< 0.64
After treatment MCV	40	78.870	9.0539	1.4315	

MCV before treatment is 76.130 ± 11.8449 and after treatment is 78.870 ± 9.0539 which is stastically significant (P< 0.64)

Stastical data for MCH:

Variable	Ν	Mean	Std.dev	Std.Error Mean	t.value
Before treatment MCH	40	25.623	4.5625	.7214	< 0.000
After treatment MCH	40	32.373	2.4997	.3952	

MCH before treatment is 25.623 ± 4.5625 and after treatment is 32.373 ± 2.4997 which is stastically significant (P< 0.000)

Stastical data for MCHC:

Variable	Ν	Mean	Std.dev	Std.Error Mean	t.value
Beforetreatment MCHC	40	31.735	2.1601	.3415	-1.050
After treatment MCHC	40	32.0183	1.91430	.30268	

MCHC before treatment is 31.735 ± 2.1601 and after treatment is 32.0183 ± 1.91430 which is stastically significant (P< 0.05)

Paired Sample Test

Variable	Т	Df	Sig. (2- tailed)	Significancy
HB (BT) – HB (AT)	-5.890	39	.000	Highly Significant
RBC (BT)- RBC(AT_	-2.181	39	.035	Significant
PCV(BT) – PCV(AT)	.409	39	.685	Not Significant
MCV(BT) MCV(AT)	-1.90	39	.064	Not Significant
MCH(BT)- MCH(AT)	-11.824	39	.000	Highly Significant

According to the data obtained from Paired Sample Test the level of Haemoglobin increased after treatment is **highly significant.**

DISCUSSION

- In Siddha Science, PithaPandu is caused due to derangement of Pitham. The signs and symptoms of PithaPandu such as pallor, anorexia, dyspnoea, palpitation, blurred vision, pungent taste of tongue, pica etc., are related with Iron Deficiency Anaemia in Modern Science.
- PithaPandu [Iron Deficiency Anaemia] is one of the global diseases affecting 2 billion people all over the world and in India 1 in 77 people are affected.
- Hence the Principal Investigator focused to treat PithaPandu [IDA]. The aim of the study was to find the theurapeutic efficacy of "KARISALANKANNI CHOORANAM" which is indicated for pandu specifically in the ancient literature Sigicha Rathna Deepam (Ref:Page: 162).
- Before the initiation of the study, Institutional Ethical Committee approval [Reg No: 1248/ac/oa/CPCSEA/4-02/2011] and Institutional Animal Ethical Committee approval [NIS/IEC/2011/03/02] was obtained by submitting well defined protocol, at NIS.
- All the raw drugs were authenticated by Botanist in National Institute of Siddha.Certificate no:NIS/MB/43/2012
- The Siddha formulation KARISALANKANNI CHOORANAM was prepared by following the standard operating procedure in Gunapadam labarotary, NIS, under the supervision of the HOD and Lecturers of Department of Gunapadam.
- The prepared drug KARISALANKANNI CHOORANAM is then subjected for Preclinical, Biochemical and Clinical studies and the observation and results are given as follows,

PRECLINICAL STUDIES:

The animals for preclinical studies were purchased from Kings Institute, Guindy, Chennai and the study was conducted in Pharmacology Laboratory, NIS.

i) Acute toxicity study(WHO Guidelines):

- Single dosage of the drug 36mg/animal [10 X] was administered orally to Swiss albino mice and observed for the period of 14 days.
- No mortality and behavior changes were noted for the first 4 hours and for the next 24 hours and throughout the study period of 14 days.
- ▶ No weight reduction was noted before and after the acute study duration.
- ▶ Reflexes were found to be normal before and after the study
- Observations such as grooming, lacrimation, alertness, skin changes etc., were found to be normal before and after the study.
- In Necropsy, the organs of the animal such as Liver, Heart, Lungs, Pancreas, Spleen, Stomach, Intestine, Kidney, Urinary bladder and Uterus all appeared normal

ii) Sub-Acute toxicity study results:

- 1X dose [36mg/animal], 5X dose [180mg/animal] and 10X dose [360mg/animal] were administered orally once per day to Wistar albino rats for the period of 3 months.
- No weight loss, abnormal animal behaviours, abnormal metabolic functions [urination, lacrimation, defaecation etc.,] and mortality were noted.
- > Necropsy of the animal organs showed normal appearance and weight.
- > All Haematological and Biochemical parameters were within normal limits.
- The stastical report of the Haematological and Biochemical datas did not show any significant difference, between the control and test groups.
- In Histopathological studies, No abnormal findings were observed in the organs such as Heart, Liver, Lungs, Kidneys and Stomach in 1X, 5X and 10X compared with control group.

QUALITATIVE ANALYSIS OF KARISALANKANNI CHOORANAM:

The Qualitative study was done in Biochemistry Laboratory, NIS and the results are as follows,

- > Presence of Iron in **Ferrous** form which is a readily absorbable form.
- Presence of Magnesium, Sodium, calcium, alkaloids, tanic acid and starch which are essential to fulfill the therapeutic need.

CLINICAL STUDIES:

- > The clinical study was conducted in OPD and IPD of NIS.
- > The patients with the complaints of pallor, anorexia, glossitis, breathlessness, palpitation, numbress etc., were screened using screening proforma, for PithaPandu.
- Out of 60 patients screened, 40 patients who satisfied the inclusion and exclusion criteria were recruited for the trial.
- > Before the start of the trial, Informed Consent was obtained from the patients.
- > Out of 40 patients 30 were treated in OPD and 10 were treated in IPD.
- The treatment aim was to regulate the deranged Pitha dosha and to improve the Hemoglobin level.
- Specific diet restrictions such as tobacco, betel chewing, tea, coffee and alcohol were advised to avoid, during the trial period.
- Labarotary investigations were done on 0th and 24th day for the assessment of safety of the patients and efficacy of the drug.

- After completion of the trial, patients were followed up for the next 2 months in the OPD.
- After the completion of treatment with the trial drug in 40 cases, highly encouraging results were observed in the following Haematological, Stastical and Clinical parameters as follows,

RESULTS FROM HAEMATOLOGICAL PARAMETERS:

- 1. **Hemoglobin** : Out of 40 cases,
 - ▶ 32 [80%] cases showed improvement, of which 8 cases reached nomal level.

i) Increase of 1 to 5 grams of Hb - 22 (55%)

ii) Increase of 0.1- 2.0 grams of Hb – 10 (25%)

- Remaining 8 [20%] cases showed no improvement.
- Total RBC: Out of 40 cases, in 26 cases (65%)total RBC increased from its base level, 9 cases (22.5%) did not show any change from its base level and 5 cases (12.5%) showed reduction from its base level.
- 3. **MCV**: Out of 40 cases, 27(67.5%) cases showed increase from its base level and 13(32.5%) cases showed reduction from its base level.
- 4. **PCV**: Out of 40 cases, 22(55%) cases showed increase from its base level, and 18(45%) cases showed reduction from its base level.
- MCH: Of the 40 cases, 28(70%) cases showed increase from its base level, 2(5%) did not show any change and 10 (25%) cases showed reduction from its base level.
- 6. MCHC: Of the 40 cases, 26(65%) cases showed Increase from its base level and 2(5%) cases showed reduction from its base level.

- Morphology of RBC: Out of 40 cases, in 9(22.5%) cases showed Normocytic Normochromic status after the treatment and 31(77.5%) cases remained in Microcytic Hypochromic status after the treatment.
- 8. Liver function tests, Renal function tests and other blood parameters were found to be in normal limits, during the treatmental period and after the treatment.

STATISTICAL REPORT:

The Statistical report states that the Mean ± Standard deviation for,

- i) Hemoglobin before treatment is $9.34 \pm .97$ and after treatment is $11.10 \pm .3552$ which is statistically significant (P < 0.001).
- ii) RBC before treatment is $4.175 \pm .5429$ and after treatment is $4.335 \pm .3833$ which is stastically significant (P<0.35)
- iii) PCV before treatment is 34.427 ± 9.6885 and after treatment is 33.693 ± 5.4620 which is stastically significant (P< 0.685)
- iv) MCV before treatment is 76.130 ± 11.8449 and after treatment is 78.870 ± 9.0539 which is stastically significant (P< 0.64)
- v) MCH before treatment is 25.623 ± 4.5625 and after treatment is 32.373 ± 2.4997 which is stastically significant (P< 0.000)
- vi) MCHC before treatment is 31.735 ± 2.1601 and after treatment is 32.0183 ± 1.91430 which is stastically significant (P< 0.05)

RESULTS FROM CLINICAL PARAMETERS:

- Adverse reaction of the drug was not observed during the course of the study.
- In the clinical trial, Out of 40 cases 32(80%) cases showed good improvement,
 8 cases showed poor improvement and some of the important siddha parameters are stated below.

Age incidence: The age limit for the cases taken for study ranged from 13 to 55 years. Among the 40 cases treated, 0 cases (0%) belonged to 13-20years, 25 cases (62.5%) belonged to 20-40 years and 15 cases (37.5%) belonged to 40-55 years. The percentage is more in the age group of 20-40 years.

Sex incidence: Out of 40 patients screened for the trial, 2 cases were males and all of them were found to be under the exclusion criteria were not included and 38 cases were females of which 40 of the cases, who satisfied the inclusion and exclusion criteria were included in the trial. The inference obtained from the study showed, the vulnerability of the female population towards the disease Pithapandu [IDA].

Occupational incidence: Among the 40 cases, 0 (0%) of them were students, 16 (40%) of them were working and 24 (60%) of them were house wives. The percentage is more in house wives.

Socio-ecnomic Incidence: Among the 40 cases, 8(20%) cases belonged to Upper middle class economic status, 12(30%) cases belonged to middle class people and 20(50%) belonged to poor economic status. The percentage is more in poor economic group. The inference obtained from the study showed, poor socio-economic status is a main predisposing factor, since the poor people usually consume low nutritional food.

Dietary Factor: Among the 40 cases, 12 (30%) cases were observed to have pure vegetarian diet and 28(70%) were taken non-vegtarian diet. The incidence is high in non-vegtarians.

Etiology: Generally Pandu noi is due to dietic factors, which cause vitiation of Pitham and Kabam Thaathus. History of patients reveal that irregular food habits, excessive intake of ash, soil and clay (pica), over intake of salt, sour and pungent tasted food items and malnutritious diet cause this disease. Before treatment 18 of the cases had PICA and all of them relieved after the treatment.

Thegi [Body constitution]: Of the 40 cases, 4(10%) cases were in Vatham body constitution, 28(70%) cases were in Pitham body constitution, 8(20%) cases were in thontham body constitution. The percentage was more in pitham type of body constitution.

Iymporigal [Sensory organs]: Of the 40 cases,

- Mei [Skin] was affected noted as pallor, numbress, dryness, in 32 (80%) cases and 32 (80%) cases improved after the treatment.
- Vaai [Buccal cavity] was affected noted as glossitis, angular stomatitis, bitter or pungent taste, dryness, pallor, fissured and coated tongue in 12 (30%) cases and all the patients were improved after the treatment.
- Kan [Eye] was affected noted as pallor, blurred vision in 36 (90%) cases and 32(80%) cases improved after the treatment.

Iympulangal [Motor organs]: Out of 40 cases,

- Kai [Upper limb] was affected noted as numbress, pain in 32 (80%) cases and all the patients were improved after the treatment.
- Kaal [Lower limb] was affected noted as numbress, pain, paedal edema in 28 (70%) and all the patients were improved after the treatment.
- Vaai [Buccal cavity] was affected noted as glossitis, angular stomatitis, bitter or pungent taste, dryness, pallor, fissured and coated tongue in 12 (30%) cases and all the patients were improved after the treatment.
- Eruvai [Anus] was affected noted as constipation in 4 (10%) cases and all the patients were improved after the treatment.
- Karuvai [Genital organ] was affected (amennorhea in 4 (10%) all the patients were improved after the treatment.

Kosam: Of the 40 cases,

- Annamayakosam was affected, noted as loss of appetite in 32 (80%) cases and all the patients were improved after the treatment.
- Manomayakosam was affected, noted as palpitation in 24 (60%) cases and all the patients were improved after the treatment.
- Vinganamayakosam was affected, noted as pain, numbress and tingling sensation in 16 (40%) cases and all the patients were improved after the treatment.
- Pranamayakosam was affected, noted as breathlessness in 32(80%) cases and 28(70%) of the patients were improved after the treatment.
- Anandamayakosam was affected, noted as ammenorrhoea, oligomenorrhoea and constipation in 4 (10%) cases and 4(10%) were improved after the treatment.

Mukkutram:

Vatham: Out of 40 cases,

- Pranan was affected, noted as breathlessness in 36(90%) cases and 32(80%) were improved after the treatment.
- Abanan was affected, noted as constipation, flatulence, ammenorrhoea and oligomenorrhoea in 12(30%) cases and 8(20%) were improved after the treatment.
- Uthanan was affected, noted as breathlessness in 28(70%) of the cases before the treatment and 28(70%) of the cases were improved after the treatment.
- Viyanan was affected noted as pain, numbress and tingling sensation in 32(80%) cases and all of them were improved after the treatment.

- Samanan was affected noted as loss of appetite, pain, numbress and breathlessness in 38(90%) cases and all of them were improved after the treatment.
- Koorman was affected, noted as blurred vision in 8(20%) cases and all of them were improved after the treatment.
- Kirukaran was affected noted as loss of appetite, dryness of mouth in 36(90%) cases and all of them were improved after the treatment.
- Devadhathan was affected, noted as fatigue in 40(100%) of the cases and all of them were improved after the treatment.

Pitham: Out of 40 cases,

- Ranjagam was affected in all the 40 [100%] cases, noted as pallor before the treatment and 37(92.5%) cases showed improvement after treatment.
- Prasagam was affected in all the 40 [100%] cases, noted as pallor and 37 (92.5%) cases showed improvement after treatment.
- Analam was affected in 38 (90%) of the cases noted as loss of appetite before the treatment and all of them showed improvement after the treatment.
- Alosagam was affected in 2(5%) of the cases noted as dull vision before the treatment and all of them were improved after the treatment.
- Sathagam was affected noted as fatigue in 37(92.5%) cases and all the cases were improved from the affection after the treatment.

Kabam: Out of 40 cases,

- Avalambagam was affected noted as breathlessness in 32(80%) cases before the treatment and 30(75.5%) cases were improved after the treatment.
- Kilethagam was affected noted as indigestion in 8(20%) cases and all of them were improved after the treatment.
- Pothagam was affected in 12(30%) cases noted as feeling of pungent or bitter taste of the tongue and all of them were improved after the treatment.

Udal Kattugal: Of the 40 cases,

- Saaram [noted as fatigue] was affected in all 40 patients (100%) before the treatment and 40(100%) patients were improved after the treatment.
- Senneer [noted as pallor, reduction of Hemoglobin level] was affected in all 40 patients (100%) before the treatment and 37(92.5%) patients were improved after the treatment.
- Oon was affected noted as paedal edema in 12(30%) cases, before the treatment and all of them were improved from the affection after the treatment.
- Suronitham was affected in 8(20%) patients noted as oligomenorrhoea before the treatment of which 4(10%) were improved after the treatment.

Envagai Thervugal: Out of 40 cases,

➤ Naa [noted as pallor, coated, glossitis, angular stomatitis, baldness, fissure, dryness, pungent or bitter taste, decreased salivation] was affected in 36 [90%] cases before treatment and 36(90%) cases were improved after the treatment.

Niram [noted as pallor] was affected in 36 [90%] cases before treatment and 36(90%) cases were improved from the affection after the treatment.

➤ Vizhi [noted as pallor] was affected in all the 36(90%) cases of which 36(90%) cases were improved from the affection after the treatment.

➤ Sparisam was affected [noted as dryness, hot or cold sensation, excessive sweat] in 32(80%) cases and all of them were improved after the treatment

➤ **Malam** was affected [noted as constipation] in 8(20%) before the treatment and all of them were improved after the treatment.

Naadi: According to this study, among 40 cases, Pithavaatham naadi was observed in 38(95%) cases, Vathapitham naadi was observed in 2 cases before the treatment. After the treatment 24(60%) cases in Pithavatham naadi and 16(40%) cases in Vathapitham naadi were observed.

Neikkuri: Of the 40 patients, 28 [70 %] cases were observed in Pitha neer and remaining 12 (30 %) cases were observed in Vatha neer before the treatment and 20 [50 %] cases were observed in Pitha neer and 20 (50 %) cases were observed in Vatha neer and Pitha neer after the treatment

SUMMARY

- The clinical study was to evaluate the Therapeutic efficacy of the siddha formulation "KARISALANKANNI CHOORANAM" which is indicated for pandu specifically in the ancient literature Sigicha Rathna Deepam (Ref: Page: 162).
- Before the initiation of the study, Institutional Ethical Committee approval [Reg No: 1248/ac/oa/CPCSEA/4-02/2011] and Institutional Animal Ethical Committee approval [NIS/IEC/2011/03/02] was obtained by submitting well defined protocol, at NIS.
 - Institutional Ethical Committee and Institutional Animal Ethical Committee approval were obtained before the commencement of the trial by submitting the well defined protocol and proforma.
 - > The raw drugs were collected from the reputed raw drug market in Chennai.
 - > All the raw drugs were authenticated by Botanist in National Institute of Siddha.
 - The medicine was prepared by the Principal Investigator by following the standard operating procedure in Gunapadam laboratory, NIS.
 - Then the medicine was subjected to the preclinical studies, as per Who guideliness, in Pharmacology Lab, NIS and safety of the drug was ensured.
 - > The prepared medicine was subjected to clinical trial at OPD and IPD of NIS.
 - Among the 60 patients screened in the OPD of Department of Maruthuvam, 40 patients who satisfied the inclusion and exclusion criteria were selected and all were females. Since the males did not satisfy the inclusion criteria and exclusion criteria were excluded from the study.

Clinical diagnosis of PithaPandu was made by Siddha and Modern methodology.

- The trial drug ": KARISALANKANNI CHOORANAM" 1 gm is given continuously for 24 days.For OP patients ,they should visit the hospital once in 12 days.At each clinical visit clinical assessment is done and prognosis is noted.For IP patients the drug is provided daily and prognosis is noted.
- Laborotary investigations are done at 0th day& 24th day of the trial.For IP patients, who is not in a situation to stay in the hospital for a long time is advised to attend the OPD for further continuation of the treatment.
- > Out of 40 patients, 30 were treated in OPD and 10 were treated in IPD.
- Assessments and required Lab Investigations were carried out as per protocol and the concerned data was recorded in the proforma.
- Followups of the patient for next 2 months in the OPD after the trial period were also carried out, without the trial drug.
- The Statistical analysis showed the datas obtained from the Hematological parameters were statistically significant.

CONCLUSION

- To conclude Siddha way of approach is certainly the best treatment of Pitha pandu(Iron deficiency anaemia) in all aspects the trial drug Karisalankanni chooranam could avoid complications.
- The raw drugs are readily available and easily preparable with least cost and more safety.
- Toxicity study reveals safer study.
- Clinical study revealed that the trail drug possess good improvement in 83.75% cases moderate improvement in 10.85% cases and 5.40% cases showed mild improvement.
- The observed difference between the mean ± standard deviation of HB at before and after treatment were 9.34 ± .97 and 11.10 ± 2.24 which is statistically significant (p <0.001).

ANNEXURE –I TOXICOLOGICAL EVALUVATION OF KARISALANKANNI CHOORNAM

1. ACUTE TOXICITY STUDY OF KARISALANKANNI CHOORNAM:

Principle:

Acute toxicity is carried out in Swiss albino mice with a single exposure of 10 times of the recommended therapeutic dose of test compound .The study duration will be 14 days[WHO guidelines, 1993].

Animal species	:	Swiss albino mice
Age / Weight	:	6 weeks/ 20-25Gms.
Gender	:	Both male and female
Number of Animals	:	Mice: 20
Acclimatization Period	:	7 Days
Clinical dose	:	2000 mg/day

S. N	Group	No of mice
1	Vehicle control	10 (5 male, 5 female)
2	Toxic dose 10X therapeutic dose (72 mg/animal)	10 (5 male, 5 female)

Source of Test Animals: Test animals were obtained from the animal laboratory of the King institute, Chennai and stocked at National institute of siddha, Chennai.All the animals were kept under standard environmental condition $(27\pm 2 \text{ degree} \text{ celcius})$. The animals had free access to water and standard pellet diet (SaiDurga foods pvt.ltd, Bangalore). The principles of laboratory animal care were followed and the Institutional Animal Ethical Committee approved the use of animals and the study design. (1248/ac/09/CPCSEA/Dec/ 2012), [Reg No: NIS/IEC/2011/3/02].

Route of administration:Oral route was selected, because it is the normal route of clinical administrationt **substance and vehicle:** The KarisalankanniChoornam is black in colour with mild astringent taste and mild odour. The test substance is insoluble in water, in order to obtain and ensure the uniformity in drug distribution, the drug is dissolved by aqueous Tween 80 solution (10%).

Administration of doses: KarisalankanniChoornamwas suspended in aqueous Tween 80 solution (10%), with uniform mixing and it was administered to the groups in a single oral dose. The control groups were received equal volume of the vehicle. The animals were weighed before giving the drug. The dose level was calculated according to body weight and surface area. Since the clinical dose was 2000mg\day, it was converted to animal dose (36 mg/animal) and then administered. The principle of laboratory animal care was followed.

Observations: Observations were made and recorded systematically and continuously observed as per the guideline after substance administration. Animals were observed individually (visual observations included skin changes, alertness, grooming, aggressiveness, sensitivity to sound, touch and pain, restlessness, tremors, convulsion, righting reflex, gripping reflex, pinna reflex, corneal reflex, writhing reflex, papillary reflex, urination, salivation, lacrimation for first 4 hrs, then periodically during the first 24 hrs. Animals were observed for body weight and mortality for 14 days. If animals died during the period of study, the animals were sacrificed. At the end of the 14th day all animals were sacrificed and necroscopy was done.

Body Weight: Individual weight of animals was determined before the test substance was administered and daily for 14 days. Weight changes were calculated and recorded. At the end of the test surving animals were weighed and sacrificed.

Results: KarisalankanniChoornamat the dose 36mg/animal [10X] did not exhibit any mortalityin mice.No behavior changes were noted for the first 4 hours and for the next 24 hours and throughout the study period of 14 days. No weight reduction was noted before and after the acute study duration. Reflexes were found to be normal before and after the study. All other observations were found to be normal before and after the study. In Necropsy, the organs such as Liver, Heart, Lungs, Pancreas, Spleen, Stomach, Intestine, Kidney, Urinary bladder and Uterus all appeared normal.

2.SUB ACUTE TOXICITY STUDY OF KARISALANKANNI CHOORNAM:

)

S.No	Group	No of Rats
1	Vehicle control	10 (5male,5 female)
2	1XTherapeutic dose (36 mg/animal)	10 (5male,5 female)
3	5XTherapeutic dose (180 mg/animal)	10 (5male,5 female)
4	10XTherapeutic dose(360 mg/animal)	10(5male, 5 female)

Animal source: Test animals were obtained from the animal laboratory of the King institute, Chennai, and stocked at animal house in National Institute of Siddha, chennai. All the animals were kept under standard environmental condition (27 ± 2) degree celcius). The animals had free access to water and standard pellet diet (SaiDurga foods pvt.ltd, Bangalore). The principles of laboratory animal care were followed and Institutional Animal Ethical Committee approved the use of animals and the study design(1248/ac/09/CPCSEA/Dec/ 2012), [Reg No: NIS/IEC/2011/3/02].

Identification of animal:By cage number, animal number and individual marking on fur.

Housing & Environment:The animals were housed in polypropylene cages provided with bedding of husk under dark and light cycle each of 12 hours.

Administration period: The period of administration of the test substance to animals are depending on the expected period of clinical use. Since the clinical dose of the test drug is 48 days and as per WHO guidelines the administration period is reported to be 3 months.

Dose selection:The results of acute toxicity studies in Swiss albino mice indicated that KarisalankanniChoornamwas non toxic and no behavioral changes, mortality was observed. On the basis of these results, the doses were selected for the study as per WHO guidelines.

Preparation and administration of dose:KarisalankanniChoornam was suspended in aqueous Tween 80 solution (10%). It was administered to animalsat dose levels of 1X therapeutic dose (36 mg/animal), 5X therapeutic dose (180 mg/animal) and 10X therapeutic dose (360 mg/animal). The control animals were administered vehicle only. Administration was by oral (gavage) once a day for 90 days.

METHODOLODY:

Randomization, Numbering and Grouping of animal: The animals were randomly divided into four groups for dosing up to 90 days. Each group consist of 10 animals (5 per sex in each group) were allowed acclimatization period of 7 days to laboratory conditions prior to the initiation of treatment. Each animal was fur marked with picric acid. The females were nulliparous and non pregnant.

OBSERVATION: Experimental animals were kept under observation throughout the course of study for the following:

Body weight:Weight of each rat was recorded on day 1 and at weekly intervals throughout the course of study and at termination to calculate relative organ weights. From the data mean body weights and percent body gain were calculated.

Food and water consumption: The quantity of food consumed by groups consisting of 10 animals for different doses was recorded at weekly intervals. Food consumed per animal was calculated for control and the treated dose groups.

Clinical sings: All animals were observed daily for clinical sings. Time of onset intensity and duration of this symptom if any were recorded. Animal behaviours and metabolic functions [defaecation, urination, lacrimation etc.,] were noted.

Mortality:All animals were observed twice daily for mortality during entire course of study.

RESULTS:

10MG TREATED (Low dose)

Kidney: shows normal renal tissue with glomeruli and tubules.

Spleen: shows normal spleen with lymphoid aggregation.

Liver: shows almost normal hepatocytes and occasional binucleate cells.

Stomach: shows normal mucosal glands.

Ovary: shows ovarian stroma with follicles and corpus leuteum.

Lung: shows normal alveoli.

Testis: shows normal tubules with spermatogenesis.

Heart: shows normal cardiac muscle bundles.

Brain: shows normal brain with nerve fibers and astrocytes.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Bone: Shows normal osteocytes

Pancrea: shows normal acini with islets of β -cells
20MG TREATED (Mid dose)

Brain: shows brain with edema, microglial proliferation, shows brain with micro cystic change and astrocytic proliferation, shows brain with mononuclear infiltrate around vessel.

Kidney: shows renal tissue with focal tubular damage, interstitial inflammatory collection. Glomeruli shows epithelial proliferation.

Liver: shows hepatocytes with focal mild fatty change.

Spleen: shows congestion with lymphoid hyperplasia.

Stomach: shows near normal mucosal gland with mild exudates.

Lung: shows congested alveolar wall with mild thickening and mild emphysematous changes.

Pancreas: shows pancreas with acini and normal islets.

Testis: shows normal tubules with spermatogenesis.

Heart: shows congestion and mild inflammatory infiltration in between cardiac muscle bundles.

Ovary: shows ovarian stroma with follicles and corpus leuteum.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Bone: Shows normal osteocytes

40MG TREATED (High dose)

Stomach: shows stomach with superficial erosion and congestion.

Heart: shows hypertrophic cardiac muscle bundles.

Spleen: shows lymphoid hyperplasia.

Brain: shows brain with edema. Astrocytes show degenerative changes.shows brain with pyknotic irregular nucleus, shows brain with vesicular nuclei and micro cystic changes.

Liver: shows marked dilatation of sinusoids, degeneration of hepatocytes, necrosis.

Kidney: shows renal tissue with tubular epithelial damage.

Pancreas: shows atrophic islet cells.

Testis: Giant cells were formed in the lumen of the seminiferous tubules and the spermatogenic cells degenerated. **Lung:** shows congestion, narrowed alveolar space and thickened alveolar wall.

Ovary: shows ovarian follicles and corpus leuteum.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Bone: Shows normal osteocytes

HISTOPATHOLOGY PHOTOS







ANNEXURE – II

Biochemical Analysis of KarisalankanniChoornam

Test Drug details: The following formulation used in the study was processed by the methods prescribed in standard text books of Siddha Medicines.

Name of the drug:KarisalankanniChoornam.

QUALITATIVE ANALYSIS:

Preparation of the drug for Qualitative Analysis: 5 grams of thedrug accurately weighed and placed in 250 ml clean beaker. 50ml of distilled water is added and boiled for 10 minutes. Then it is cooled and filtered in a 100 ml volumetric flask and made up to 100 ml with distilled water.

ivicitiouology and Results.				
Procedure	Observation	Inference		
Solubility test:	Sparingly	Absence of Silicate		
Test for silicate: A little sample is	insoluble			
shaken well with distilled water and				
then with con.HCL/con.H2SO4				
Action of Heat:Test for Carbonate: A	No white fumes	Absence of		
Small amout of the sample is taken in	are formed	Carbonate		
a dry test tube and heated gently in the				
non-luminous part of the flame				
Flame test: Test for Copper: One	No Bluishgreen	Absence of Copper		
pinch of substance is made into paste	flames appeared			
with Con.Hydrochloric acid in a watch				
glass and introduced into the non-				
luminous part of the Bunsen flame.				
Ash Test:Test for sodium: A filter is	Yellow colour	Presence of		
soaked into a mixture of sample and	flame is	Sodium		
Cobalt Nitrate solution and introduced	developed			
into the Bunsen flame and ignited	_			
Test for Acid radicles:				
Test for Sulphate: 2 ml of the above	No cloudy	Absence of		
prepared extract is taken in a test	apperance is	Sulphate		
tube.To this add 2ml of 4%	formed			
Ammonium Oxalate solution.				
Test for Chloride: 2ml of the above	No Cloudy	Absence of		
prepared extract is treated with 2ml of	appearance is	Chloride		
dilute HCL, until the effervescence	formed			
ceases off.				
Test for phosphate : 2 ml extract is	No vellow	Absence of		

Methodology and Results:

treated with 2ml of Ammonium Molybdate and add 2 ml of Con. HNO2	appearance is formed	Phosphate
Test for carbonate : 2ml extract is treated with 2 ml of Magnesium	No Cloudy appearance is	Absence of Carbonate
Test for Fluoride and Oxalate: 2ml of extract is added with dil. Acetic acid and 2ml of Calcium Chloride and	No Cloudy appearance	Absence of Fluoride and Oxalate
heated Test for Lead: 2ml of the extract is	No Yellow	Absence of Lead
added with 2ml of Potassium Iodide solution.	precipitate is formed.	
Test for Aluminium: To the2ml of the extract, Sodium Hydroxide is added in drops to excess.Characteristic change is noted.	No reddish black colour is formed.	Absence of Aluminium
Test for Iron (Ferrous) : 2 ml extract is treated with 2ml of Ammonium Thiocynate solution and 2ml of Conc. HNO ₃ is added	Blood Red colour is formed	presence of Ferrous iron
Test for Iron (Ferric) : 2ml extract is treated with Glacial Acetic acid and Potassium Ferrocyanide	No Blue colour is formed	Absence of Ferric iron
Test for Zinc : To the 2ml of the extract Sodium Hydroxide solution is added in drops to excess.	White precipitate is not formed	Absence of Zinc
Test for Magnesium : To the 2ml of the extract Sodium Hydroxide solution is added in drops to excess.	White precipitate is not formed	Presence of Magnesium
Test for Calcium: 2 ml of extract is taken in a clean test tube. To this add 2 ml of 4% Ammonium Oxalate solution.	White precipitate or Cloudy appearance is formed	Prsence of Calcium
Test for Ammonium : To 2ml of extract 2ml of Nessler's reagent and excess of NaOH solution are added.	No formation of Brown colour	Absence of Ammonium
Test for Mercury : 2ml extract is treaed with 2ml of NaOH solution	Formation of Yellow precipitate	Absence of Mercury
Test for Arsenic: 2ml of the extract is treated with 2ml of Sodium Hydroxide solution.	Brownish Red precipitate is obtained	Absence of Arsenic

Miscellaneous:		
Test for Starch : 2ml extract is treaed	No Blue colour is	Presence of Starch
with weak Iodine solution	develoed	
Test for Reducing sugar: 5ml of	Brick Red colour	Absence of
Benedict's qualitative solution is taken	is developed	Reducing sugar
in a test tube and allowed to boil for 2		
minutes and add 8-10 drops of the		
extract and again boil it for 2 minutes.		
The colour change is noted.		
Test for Alkaloids: 2ml extract is	Presence of	presence of
treated with 2ml of Potassium Iodide	Reddish colour	Alkaloids
solution and 2ml Picric acid is added		
Test for Tannic acid : 2ml of the extract	No Black	Presence of Tannic
is treated with 2ml of Ferric Chloride	precipitate is	acid
solution.	formed	

ANNEXURE – III CERTIFICATES

•	NATIONAL INSTITUTE OF SIDDHA (An Autonomous Body under Department of AYUSH) Ministry Of Health & Family Welfare, Government of India	Tambaram Sanatorium, Chennai - 600 04 Tel : 044-22411611 Fax : 044-2238131 E mail : nischennaisiddha@yahoo.co.in Website : www.nischennai.org
	Name: Dr. M. GOBIKRISHNAN, Reg. NO	; 32 1 0/202
	No. NIS /IEC/2011/3/02 - 24/12	Y ON ' DI THO PANDU 'KARISALAN KONI CHODEANAM (INTERNAL) /2011
	DECSION	'
	Opinion of the Institutional Ethics Committee – Please	Check one
	Approval	
	Modifications required prior to appro	val (Please specify one space below)
	Disapproval	
		K. Traminch
	Date of review:	Dr.K. MANICKAVASAKAM)
	Signed: L. Jugo mh win (Please print name) Dr.V.SUBRAMANIAN
	(Please delee as appropriate Chairperson Sacratary)	
	Modifications needed	
	Modification given to candidate	
	B Construction	
	The research proponent is hereby informed that the require the following:	Institutional Ethics Committee will
	1. All adverse drug reactions (ADRs) that are	both serious and unexpected to be
	 reported promptly to the IEC within 7 working of The progress report to be submitted to the IEC a 	lays tleast annually
	3. Upon completion of the study, a final study sta	atus report needs to submitted to the

NO: 1248/ac/09/CPCSEA/4-02/2011 CERTIFICATE 20/12/2011 This is certify that the project title. Pre clinical & clinical Studyon pITHA PANDU (ANNEMIA) & The doug of choice is KARISDLANKANI CHOSENN has been approved by the IAEC. Prof. Dr. K. Manicka vasakam Dr. B. Jayachards an Dare Name of Chairman/Member Secretary IAEC: Name of CPCSEA nominee: Signature with date -4 20 K. Traminch 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, CHENNAI – 600047 CERTIFICATE OF BOTANICAL AUTHENTICITY

Certified that the following plant drugs used in the Siddha formulation Karisalankanni Chooranam (Internal) for the treatment of Pitha Pandu (Iron Deficiency Anaemia) taken up for Post Graduation Dissertation studies by Dr.M.Gobi Krishnan, M.D.(S), II year Department of Maruthuvam, 2011-12, are identified and authenticated through Visual inspection /Experience, Education & Training/ Organoleptic characters/ Morphology / Micromorphology / Taxonomical/ Microscopical methods. Eclipta alba Linn. (Asteraceae), Whole plant Terminalia chebula Retz. (Combretaceae), Fruit Terminalia belerica Roxb. (Combretaceae), Fruit Phyllanthus emblica Linn. (Euphorbiaceae), Fruit Boerhavia diffusa Linn. (Nyctaginaceae), Leaf and Root Plumbago zeylanica Linn. (Plumbaginaceae), Root Zingiber officinale Rosc. (Zingiberaceae), Rhizome Piper nigrum Linn. (Piperaceae), Fruit Piper longum Linn. (Piperaceae), Fruit Nigella sativa Linn. (Ranunculaceae), Seed Cuminum cyminum Linn. (Apiaceae), Fruit Glycyrrhiza glabra Linn. (Fabaceae), Root Taxus baccata Linn. (Taxaceae), Leaf Coscinium fenestratum Colebr. (Menispermaceae), Stem Coriandrum sativum Linn. (Apiaceae), Fruit Elettaria cardamomum Maton. (Zingiberaceae), Seed Certificate No: NIS/MB/43/2012

Date: 24-8-12

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



ANNEXURE – IV PROFORMA

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF MARUTHUVAM AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THE SIDDHA DRUG "KARISALANKANNI CHOORANAM" IN"PITHA PANDU"(IRON DEFECIENCY ANAEMIA) <u>FORM I - SCREENING AND SELECTION PROFORMA</u>

1. O.P.No 2. I.P No	3	. S.No:	Reg No:321	01202	
4. Name: 5. A	ge (years)):	6. Gender: Femal	le/male	
7. Contact Nos:					
<u>8.INCLUSION CRITERIA:</u>Age 13-55 of both sexes					
Yes/No					
• Patient willing to undergo	blood inve	estigations			
Yes/No					
• Clinical symtoms of Pallor,	Breathles	sness, Palp	oitation, Anorexia,		
Yes/No					
Giddiness, Glossitis, la	assitude, F	Fatigue, koi	ilonychias etc.,		
• Hb less than normal range i	e.,men:7-1	13mg/dl,W	omen:7-10mg/dl		
Yes/No					
• Patient blood smear shows	microcyti	c hypochro	omic RBC		
Yes/No					
• Patient willingness for cons	ent to incl	ude in the	trial		
Yes/No					
9.EXCLUSION CRITERI	<u>A</u> :				
Pregnancy and lactation	Yes	No	Peptic Ulcer	Yes	No
Severe systemic illness(CA,RA)	Yes	No	Gasterectomy	Yes	No
Inheriteddefects(sicklecellAnaemi			Renal disease		

No

No

Diabetes

Yes

Yes

No

No

Yes

Yes

a, Thalassemia)

Steroid exposure for prolonged

period			mellitus		
Parasitic infection(Malaria,hook worm etc.,)	Yes	No	Hypertension	Yes	No
Hypothyroidism/Hyperthyroidism	Yes	No	Malabsoption syndrome	Yes	No
Cardiac disease	Yes	No	Chronic blood loss	Yes	No

10.BLOOD INVESTIGATION:Date:

Hemoglobin	Gms%
Red blood cells(RBC)	Millions/cu.mm
Morphology of RBC	

NO If Yes Serial NO:

11. ADMITED TO TRAIL YES

Date:

Station:

Signature of the Investigator:

Signature of the Lecturer:

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF MARUTHUVAM AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THE SIDDHA DRUG "KARISALANKANNI CHOORANAM" IN "PITHA PANDU" (IRON DEFECIENCY ANAEMIA)
FORM I-A HISTORY PROFORMA REG NO:
1. Serial No of the case:
2. Name: 3. Gender: Female/male
4. Age (years): DOB Date Month Vear
5.Address:
6.Occupation:
7. Educational Status: A) Illiterate B)Literate
8.Height: cms 9.Weight: kg
10. Complaints and Duration:
11.Habit of A) Smoking 1. Yes; duration years; number-
B)Tobacco chewing 1. Yes; duration years 2.No
C)Betel Nut chewing 1. Yes; duration years 2.No
D)Alcoholism 1. Yes; duration years; Quantity- ml 2.No
12.DrugHistory: Had the patient been treated before with allopathy drug?A)Yes 2)No
13.Dietary style: A.Pure vegetarian B.Non-vegetarian C. mixed diet
14.Mensural History:
Date: Station:
Signature of the Investigator:
Signature of the Lecturer: Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF MARUTHUVAM AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THESIDDHA DRUG "KARISALANKANNI CHOORANAM" IN "PITHA PANDU"(IRON DEFECIENCY ANAEMIA)

FORM II AND CLINICAL ASSESSMENT ON ENROLLMENT AND ON VISITS REG NO:

1. Serial No: _____

2. Name: _____

3. Date of assessments: $0^{\text{th}} \text{ day-}_/12^{\text{th}} \text{ day-}_/24^{\text{th}} \text{ day-}_/24^{t$

SIDDHA SYSTEM OF EXAMINATION

4.ENVAGAI THERVU:[EIGHT-FOLD EXAMINATION]

I.NAADI: [PULSE PERCEPTION]

	0 th Day	12 th Day	24 th Day		0 th Day	12 th Day	24 th Day
Vali				Iyya vali			
Azhal				Vali Iyyam			
Iyyam				Azhal Iyyam			
Vali Azhal				Iyya Azhal			
Azhal vali							

II.NAA:[TONGUE]

	0th Day	12th Day	24th Day
Colour	Dark/Yellow/Red/Pale	Dark/Yellow/Red/Pale	Dark/Yellow/Red/Pale
Taste	Sweet/Bitter/Sour/	Sweet/Bitter/Sour/	Sweet/Bitter/Sour/
	Pungent/None	Pungent/None	Pungent/None
Coating	Present/Absent	Present/Absent	Present/Absent
Fissure	Present/Absent	Present/Absent	Present/Absent
Saliva	Normal/Increased/Decreased	Normal/Increased/Decreased	Normal/Increased/Decreased
Dryness	Present/Absent	Present/Absent	Present/Absent
Glossitis	Present/Absent	Present/Absent	Present/Absent
Baldness	Present/Absent	Present/Absent	Present/Absent

III.NIRAM:[COMPLEXION]

0th Day	12th Day	24th Day
Dark/Yellow/ Pale/Other	Dark/Yellow/ Pale/Other	Dark/ Yellow/ Pale/Other

IV.MOZHI:[VOICE]

0th Day	12th Day	24th Day
Normal/High/Low pitched	Normal/High/Low pitched	Normal/High/Low pitched

v.vizhi:[EYES] (Lower palpabrel conjunctiva)

0th Day	12th Day	24th Day
Dark/Yellow/Red/ Pale	Dark/Yellow/Red/ Pale	Dark/Yellow/Red/ Pale

VI. MALAM; [BOWEL HABITS / STOOLS]

	0 th Day	12 th Day	24 th Day
Colour	Dark/ Yellow/Red	Dark/ Yellow/Red/Pale	Dark/ Yellow/Red/Pale
Consistency	Solid/Semisolid/Watery	Solid/Semisolid/Watery	Solid/Semisolid/Watery
stool bulk	Normal/Reduced	Normal/Reduced	Normal/Reduced
Constipation	Present/Absent	Present/Absent	Present/Absent
Diaarhoea	Present/Absent	Present/Absent	Present/Absent

VII.MOOTHIRAM:[URINE EXAMINATION]

Neerkuri	0 th Day	12 th Day	24 th Day
Niram[Colour]	White/Yellow/Straw coloured/Red/Crystal clear	White/Yellowish/Straw coloured/Red/Crystal clear	White/Yellowish/Straw coloured/Red/Crystal clear
Manam[Odour]	Present/ Absent	Present/ Absent	Present/ Absent
Nurai[Froth]	Nil/Reduced/Increased	Nil/Reduced/Increased	Nil/Reduced/Increased
Edai[Sp.gravity]	Normal/Increased/Reduced	Normal/Increased/Reduced	Normal/Increased/Reduced
Enjal[Deposits]	Present/ Absent	Present/ Absent	Present/ Absent
Volume	Normal/Increased/Reduced	Normal/Increased/Reduced	Normal/Increased/Reduced
NEIKURI			
Serpentine fashion			
Annular/Ringed fashion			
Pearlbeaded fashion			
Mixed fashion			
Other fashion			

VIII. SPARISAM: [PALPATORY PERCEPTION]

0th Day	12th Day	24th Day
Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat

5.THEGI:[**TYPE OF BODY CONSTITUTION**]

	0th day	12th day	24th day
Vatham predominant			
Pitham predominant			
Kabam predominant			
Thondha udal			

6.NILAM: [LAND WHERE PATIENT LIVED MOST]

	0 th day	12 th day	24 th day
Kurinji [Hilly terrain]			
Mullai [Forest ranges]			
Marutham [Plains]			
Neithal [Coastal belt]			
Paalai [Arid regions]			

7.KAALAM:[SEASON]

0 th day	12 th day	24 th day

8.GUNAM:[CHARACTER]

	0 th day	12 th day	24 th day
Sathuvam			
Rasatham			
Thamasam			

9.IYMPORIGAL: [SENSORY ORGANS]

	0th day	12th day	24th day
Mei [Skin]			
Vai[Buccal cavity]			
Kan [Eyes]			
Mooku [Nose]			
Sevi [ear]			

10.IYMPULANGAL:[MOTOR ORGANS]

	0th day	12th day	24th day	
Kai [upperlimb]				
Kal [lowerlimb]				
Vai [Buccal cavity]				
Eruvai [excretory organ]				
Karuvai[Reproduc-tive organ]				

11. KOSAM:[SHEATHS]

	0th day	12th day	24th day
Annamaya kosam			
Pranamayakosam			
Manonmayakosam			
Vingyanamaya kosam			
Anandhamaya kosam			

12.MUKKUTRAM: [AFFECTION OF THREE HUMORS]

A)VATHAM:

	0th day	12th day	24th day
Praanan			
Abaanan			
Samaanan			
Udhaanan			
Viyaanan			
Naahan			
Koorman			
Kirukaran			
Devathathan			
Dhananjeyan			

B) PITHAM:

	0th day	12th day	24th day
Analapitham			
Prasakam			
Ranjakam			
Aalosakam			
Saathakam			

C) KABAM:

	0th day	12th day	24th day
Avalambagam			
Kilethagam			
Pothagam			
Tharpagam			
Santhigam			

13.SEVEN DHATHUS: [SEVEN SOMATIC COMPONENTS]

	0th day	12th day	24th day
Saaram[chyme]			
Senneer[Blood]			
Oon[Muscle]			
Kozhuppu[Fat]			
Enbu[Bones]			
Moolai[Bonemarrow]			
Sukkilam/Suronitham [Genital discharges]			

14. SYSTEMIC EXAMINATION:

	0 th day	12 th day	24 th day
CardioVascularSystem			
Respiratory System			
Gastrointestinal\System			
CentralNervousSystem			
Urogenital System			
Endocrine System			

15.GENERAL EXAMINATION:

	0 th day	12 th day	24 th day
Height (cms)			
Weight (kg)			
Temperature(°F)			
Pulse rate (permin)			
Heart rate (per min)			
Respiratory rate(per min)			
Blood pressure(mm/Hg)			
Pallor			
Jaundice			
Cyanosis			
Lymphadenopathy			
Pedal edema			
Clubbing			
Jugular vein pulsation			
Peripheral pulse			

16. CLINICAL SYMPTOMS:

	0 th day	12 th day	24 th day
Pallor			
Anorexia			
Fatigue			
Tachycardia			
Palpitation			
Giddiness			
Breathlessness			
Pungent or bitter taste of tounge			
Angular stomatitis			
Glossitis			
Lack of concentration			
Hair fall			
Amenorrhoea			
Oligomennorhoea			
Anasarca			
Koilonychia			
Pica			

Date : Station: Signature of the Investigator:

Signature of the Lecturer:

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF MARUTHUVAM

AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THE SIDDHA DRUG

"KARISALANKANNI CHOORANAM " IN"PITHA PANDU"(IRON DEFECIENCY

ANAEMIA)

LABORATORY PARAMETERS-CHART 2.Reg No:

1. Serial No: _____ 2.R

.

3. Name: ______ 4.Age: _____ years

5.Gender: Male/Female

BLOOD INVESTIGATION		0 th DAY	25 th DAY	NORMAL	
		Date:	Date:	VALUES	
HB(gms%)				M:14-18 ;W:11-15	
T.RBC(milli/cu.mm)				M:4.5-6.5 ;W:3.5- 5.5	
		¹ / ₂ hr.		-	
ESR (mm)		1 hr.			M:0-10 ;W:0-20
MCV(fl or	cu.µ)				76-96
PCV (%)	• /				M:40-55 ;W:35-45
MCH (pg)					27-33
MCHC(gm/	dl)(%)				31-35
MORPHOL	OGY OF R	BC			Normocytic
	001011	20			Normochromic
T.WBC (cu.	mm)				4000-11,000
<u>`</u>		Polymorphs			40-75
		Lymphocutes			20-35
DIFFEREN	TIAL	Monocytes			2-10
COUNT (%)	Esonophils			1-6
		Basophils			0-1
Platelets(lak	/ cubic mm)			1,50000-500000
Blood	Fasting				70-110
glucose	РР				80-140
(mg/dl)	Random				80-120
	Serum cholesterol				150-250
Lipid	HDL				30-60
profile	LDL		_		Upto 130
(mg/dl)	VLDL				40
	TGL				Upto 160
RFT	Blood ure	a			16-50
(mg/dl)	Serum cr	eatinine	_		0.6-1.2
	Serum Ui	ric acid			M:3-9 ;W: 2.5-7.5
	Total bili	rubin			0.3-1
	Direct bil	irubin			0.1-0.3
	Indirect k	oilirubin			0.2-0.8
	Serum to	tal protein			6-8
Serum Al		bumin			3.5-5.5
LFT	Serum globulin				2-3.5
(mg/dl)	Fibrinogen(g/dl)				0.2-0.4
(Serum calcium Serum phosphorous				9-11
					<u> </u>
	SCPT (II				3_26
SGPT (IU Alkaline p		phosphatase (IU)			3-12

Urine investigation	Before TMT Date:	After TMT Date:
Nei kuri		
Albumin		
Fasting sugar		
PP sugar		
Random Sugar		
Deposits		
Bile salts		
Bile pigments		
Urobilinogen		
Motion test		
Ova		
Cyst		
Occult blood		

Date: Station:

Signature of the Investigator:

Signature of the Lecturer:

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை 47 அயோத்திதாசர் பண்டிதர் மருத்துவமனை பித்த பாண்டு நோய்க்கான நோய்க்கான சித்த மருந்தின் (கரிசலாங்கண்ணி சூரணம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

	FORM IV B தகவல படிவம
முதன்மை ஆராய்ச்சியாளர் பெயர்	: Dr.M. கோபிகிருஷ்ணன்
நிறுவனத்தின் பெயர்	: தேசிய சித்த மருத்துவ நிறுவனம்
	தாம்பரம் சானடடோரியம்
	சென்னை– 47

Dr. கோபி கிருஷ்ணன் ஆகிய நான் தேசிய சித்த மருத்துவமனையில் பட்ட மேற்படிப்பு பயின்று வருகிறேன். பித்த பாண்டு என்னும் நோயானது இரத்ததாதுவில் இரும்பு சத்து குறைவினால் ஏற்படும் ஒரு நோய்,இந்நோய் உடல் வெளுப்பு, உடல் சோர்வு, மயக்கம், மூச்சு வாங்கல், கை கால் ஓய்வு, கைப்பு சுவை, ஆகிய குறிகுணங்களை தோற்றுவிக்கும்,இந்நோய்க்கு தேசிய சித்த மருத்துவமனையில் பல சித்த மருந்துகள் பயன்படுத்தப்பட்டு வருகின்றது. சித்த மருத்துவ பட்ட மேற்படிப்பில், ஆய்வின் ஒரு பகுதியாக புதிய மருந்துகளை பயன்படுத்தும் நோக்கில் சர்வ நோய் இலிங்க செந்தூரம் இந்நோய்க்கு வழங்க பரிந்துரை செய்கிறோம். இந்த மருந்தின் செய்முறை, அளவு, அனுபானம் மற்றும் மருத்துவ பயன்கள் அனைத்தும் அங்கீகரிக்கப்பட்ட சித்த மருத்துவ நூலில் கூறப்பட்டுள்ளது. எந்தவித கட்டணமுமின்றி தாங்கள் இந்த மருந்தினை பெற்றுக்கொள்ளலாம். இந்த ஆய்வில் மருந்து உட்கொள்ளும் காலம் 24நாட்கள் ஆகும். வெளி நோயாளர்கள் 12 நாட்களுக்கு ஒரு முறை மருத்துவமனைக்கு வரவேண்டும். 24நாட்கள் மருந்து உட்கொள்ளும் காலம் முடிந்த பிறகு நோய்க்கான குறிகுணங்கள் மற்றும் ஆய்வக பரிசோதனைகள் இவற்றின் முடிவுகளின் அடிப்படையில் மருந்தின் பரிகரிப்புத்திறன் கண்டறியப்படும்.

இந்த ஆய்வு சம்பந்தமாக சில கேள்விகளை தங்களிடம் கேட்க இருக்கிறேன். தங்களிடமிருந்து பெறப்படும் கருத்துக்கள் மற்றும் குறிப்புகள் அனைத்தும் நம்பிக்கையாக பதிவு செய்யப்படும்.இந்த ஆய்வில் தங்களை உட்படுத்திக்கொள்வதின் மூலம் எந்த வகையிலும் பாதிப்புக்குள்ளாக மாட்டீர்கள் என உறகி அளிக்கிறேன்.எந்தவித வற்புறுத்தலுமின்றி, இந்த ஆய்வில் பங்கேற்கவும், இந்த ஆய்வு சம்பந்தமாக கேட்கப்படும் கேள்விகளுக்கு பதில் கூறவும் தங்களுக்கு முழு சுதந்திரம் அளிக்கப்படுகிறது. இந்த ஆய்வில் பங்கேற்பதற்கு எந்த சன்மானமும் வழங்கப்படமாட்டாது. ஆனால், ஆய்வு முழுவதும் எனது மேற்பார்வையிலும், தங்கள் உடல்நலன் குறித்த தனி கவனத்திலும் ஆய்வு மேற்கொள்ளப்படும். பித்த பாண்டு நோய்க்கானபுதிய மருந்தின் பரிகரிப்புத்திறனை சமூகத்திற்கு உணர்த்தும் வகையில் இந்த ஆய்வு மேற்கொள்ளப்படுகிறது, மாறுபட்ட குறிகுணங்கள் தொடர்ந்து இருக்கும் பட்சத்தில், முதன்மை ஆராய்ச்சியாளரான என்னிடம் தெரிவிக்கப்பட்டு, தேசிய சித்த மருத்துவமனையில் அதற்க்கான தீர்வு வழங்கப்படும். இந்த ஆய்வினைத் தொடர தங்களுக்கு விருப்பம் இல்லையெனில், எப்பொழுது வேண்டுமானலும் ஆய்வின் இடையில் விலகிக்கொள்ளவும், மருத்துவமனையில் வழங்கப்படும் இந்நோய்க்கான வழக்கமான மருந்துகளை பெற்றுக்கொள்ளவும் அறிவுறுத்தப்படுகிறீர்கள்.

இந்த ஆய்வில் சேகரிக்கப்படும் விபரங்கள் அனைத்தும் தங்களுக்கும் முதன்மை ஆராய்ச்சியாளரான எனக்கும் இடையில் இரகசியமாக வைக்கப்படும். கேள்வி பதில் வடிவத்தில் தங்களிடம் கேள்விகள் கேட்கப்படும். அனைத்துப் படிவங்களிலும் தங்களின் பெயர் தவிர்க்கப்பட்டு ஆய்வாளரால் தங்களுக்கென தனிக் குறியீடு வழங்கப்படும். அந்தக் குறியீடு ஆய்வாளருக்கு மட்டுமே தெரிந்ததாக இருக்கும். நீங்கள் இந்த ஆய்வில் பங்கேற்க விருப்பப்பட்டால், திட்ட வரைவு தகவல் படி தேர்வு செய்யப்படுவீர்கள்.

நீங்கள் இந்த ஆய்வில் பங்கேற்கும் முன், இந்த ஆய்வினைப் பற்றிய மேலும் விபரங்கள் பெற வேண்டுமென விருப்பப்பட்டால், இந்த ஆய்வின் முதன்மை ஆராய்ச்சியாளர் மற்றும் தேசிய சித்த மருத்துவமனை, பட்ட மேற்படிப்புத்துறை மாணவர்

Dr.M. கோபி கிருஷ்ணன் ஆகிய என்னை 9488085454 என்ற எண்ணில் தொடர்பு கொள்ளலாம். மேலும், நீங்கள் இந்த ஆய்வில், உங்களது பங்கேற்பு மற்றும் உரிமை பற்றி தெரிந்து கொள்ள தேசிய சித்த மருத்துவமனை, தலைவர்/செயற்க்குழு உறுப்பினர் அவர்களையும் 91-44-22411611 என்ற எண்ணில் தொடர்பு கொள்ளலாம்.

தேசிய சித்த மருத்துவ நிறுவனம் அயோத்திதாச பண்டிதர் மருத்துவமனை, சென்னை - 47. பட்ட மேற்படிப்பு மருத்துவத்துறை

பித்த பாண்டு நோய்க்கான சித்த மருந்தின் (கரிசலாங்கண்ணி சூரணம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான ஒப்புதல் படிவம்

FORM IV- C ஒப்புதல் படிவம்

நான் மேற்கூறிய தகவல் படிவத்தை படித்து அல்லது படிக்க கேட்டு கொண்டேன். - து தொடர்பான விளக்கங்களையும் கேட்டு தெரிந்து கொண்டேன். எந்த வித வற்புறுத்தலின்றி, என் சொந்த விருப்பத்தின் பேரில் என்னை - ந்த ஆராய்ச்சிக்கு உட்படுத்த என் முழுமனதோடும் சுயநினைவோடும் சம்மதம் தெரிவிக்கிறேன். எனக்கு விருப்பமில்லாத பட்சத்தில் இந்த ஆராய்ச்சியில் இருந்து என்னை எப்போதுவேண்டுமானாலும் விடுவித்து கொள்ளும் உரிமையை பெற்றுள்ளேன் என்பதையும் அறிவேன்.

தேதி:

கையொப்பம்:

இடம்:

பெயர் :

தேதி: இடம்: சாட்சிக்காரா் கையொப்பம்: பெயா் : உறவுமுறை :

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF POTHU MARUTHUVAM AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THE SIDDHA DRUG "SIDDHA MANDOORAM" IN"PITHA PANDU"(IRON DEFECIENCY ANAEMIA)

FORM IV -B (WITHDRAWAL FORM)

Name:

Age:

Serial No:

Reg.No: 32091202/2011-12

Date of trial commencement:

Date of withdrawal from the trial:

Reason(s) for withdrawal;

1. Long absence at reporting: Yes/No

2. Irregular treatment: Yes/No

- 3. Shift of locality: Yes/No
- 4. Complication/Adverse reactions if any:
- 5. Poor patient compliance:
- 6.presence of nausea Yes/No
- 7.Presence of abdominal pain:

Date :

Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

Yes/No

Yes/No

Yes/No

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47 AYOTHIDASAR PANDITHAR HOSPITAL DEPARTMENT OF POTHU MARUTHUVAM

AN OPEN CLINICAL TRIAL TO ASSESS THE THERAPEUTIC EFFICACY OF THE SIDDHA

DRUG

"KARISALANKANNI CHOORANAM" IN"PITHA PANDU"(IRON DEFECIENCY ANAEMIA)

FORM IV -C (DRUG COMPLIANCE FORM)

Name :______ Reg no:32101202 serial no____ Drug: Karisalankanni chooranam

 First Line of treatment:Sesban Leaves soup[84-168ml] with palm jaggery at early morning on empty stomach for one day.

 On 1st day-Date:
 ;Drugs issued:
 (Nos) / Drugs returned:
 (Nos)

On 12 th day-Dat	e: ;Drugs issued:	(Nos) / Drugs returned:	(Nos)
On 24 th day-Dat	e: ;Drugs issued:	(Nos) / Drugs returned:	(Nos

Day	Date/ தேதி	Morning/ காலை	Evening/ மாலை
Day 1			
Day2			
Day3			
Day4			
Day5			
Day6			
Day7			
Day8			
Day9			
Day10			
Day11			
Day12			
Day13			
Day14			
Day15			
Day16			
Day17			
Day18			
Day19			
Day20			
Day21			
Day22			
Day23			
Day 24			

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer:

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FORM IV D -DIETARY ADVICE FORM

சேர்க்க கூடிய உணவுகள்:

காய்கள்:கத்தரிபிஞ்சு,முருங்கைபிஞ்சு,வாழைபிஞ்சு,அவரைபிஞ்சு,சுண்டைகாய், நெல்லிகாய்,வாழைப்பூ,மிளகு,பூண்டு,வெங்காயம் ஆகியவை வாரத்திற்க்கு இரண்டு முறை சேர்க்க வேண்டும்.

கீரைகள்:பொன்னாகண்ணி,மணத்தக்காளி, முருங்கைகீரை, ஆகியவை வாரத்திற்க்கு இரண்டு முறை சேர்க்க வேண்டும்.

பழங்கள்:வாழை,பேரீச்சை,திராட்சை,கொய்யா,ஆரஞ்சு,எலுமிச்சை,நாவல், தக்காளி,

ஆகியவை வாரத்திற்க்கு இரண்டு முறை சேர்க்க வேண்டும்.

தானியங்கள்:பட்டானி,கொண்டைகடலை,எள், ஆகியவை வாரத்திற்க்கு இரண்டு முறை சேர்க்க வேண்டும்.

அசைவம்:வெள்ளாட்டுகறி,ஈரல், மீன்,ஆகியவை வாரத்திற்க்கு இரண்டு முறை சேர்க்க வேண்டும்.

சேர்க்க கூடாதவைகள்:

- புளி ,தேநீர், ஆகியவை சேர்க்க கூடாது.
- வெற்றிலை,பாக்கு போட கூடாது.
- புகையிலை போட கூடாது.
- மது அருந்துதல் கூடாது.

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"KARISALANKANNI CHOORANAM" IN"PITHA PANDU"(IRON DEFECIENCY ANAEMIA)

FORM –IV-E

ADVERSE REACTION FORM

Name:

Age:

Gender: Male/Female

Serial No:

Date of trial commencement:

Date of the adverse reaction occur; Time:

Description of Adverse reaction:

Date:

Station:

Signature of the Investigator:

Signature of the Lecturer:

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