A study on

VENPADAI

(DISSEPTION SUBJECCT)

For the partial fulfillment of the requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)

BRANCH IV – KUZHANTHAI MARUTHUVAM DEPARTMENT

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**Need For The Study**

Even though vitiligo is a pathological disorder, the community causes a major psychological stress to the victims.

There are some evidences from the individual trials, recorded short time benefit from steroids besides its side effects.

The methods currently available to treat vitiligo are largely unsatisfactory.

More cure the patients walk in to our national institute of siddha, with this vitiligo are children’s, particularly female, it seems the patients are particularly from kalpakkam to kuduvancherry. Hence it is a need to do a study to make on impact on
1. INTRODUCTION

The word siddha comes from the word siddhi which means an perfection or heavenly bliss. The siddhars were further the greatest scientists in ancient times. they were men of highly cultured intellectual and spiritual faculties combined with super nature powers.

Agathiya siddhar who is the chief of the siddhars school is said to have been a celebrated philosopher and physician who laboured amongst the tamils in southern india.

The entire Siddha or Tamil medicine system consists of three great subdivisions namely

a. Noyilla Neri (Preventive)

b. Noy Neeku Neri (Curative methods)

c. Uramakkumurai (Strengthening Methods)

Prevention can mostly save our body and soul but modernization results in diseased condition. Siddha system is playing major role in treating chronic diseases. The Siddha system is the holistic system of codified life style a health care perfected many thousands of years ago in the Tamil speaking peninsular India. The granite rocks of the southern peninsula belong to the category of Archaic Rocks and are estimated to be around 2.5 to 4 billion years old. This system is also called as Tamil Maruthuvam, Chinthamani Maruthuvam and Arivan Maruthuvam. Since the advent of Thirumoolar who wrote the classic Thirumanthiram this is popularly called as Siddha system and practiced by Siddhars – the enlightened seekers.

Siddha system clearly lays down the general principles of body constituents in the classic Bootha system. They hold that the universe is a macrocosm made up of the five primordial elements or Boothas, viz Nilam (Earth), Neer (Water), Thee (Fire), Vali (wind) and Veli (Space) and the human being is a microcosm made up of the same five elements.

The Siddha system is divisible in to three major divisions, which are totally interwoven.

They are

1. Kappu (Prevention and Protection)

2. Neekam (Remove of diseases and disability)

3. Nirappu (Restoration of people to their full potential of good health)

National Institute of Siddha, Chennai
A specific feature in Siddha care is the Kalpamurai also called as kaykalpa where the human being from the stage of foetus to around 80 years of age can be fortified in to rock like strength through serious of efforts with lifestyle, strengthening drugs and formulations.

Siddha system characterizes the basic guna of human beings

- Sathuva gunam – Saint like attitude
- Rasathuva gunam – King like attitude
- Thamova gunam – Lesser endowmends - lower level of attainment both materially and mentally

Any person may have varying propositions these guna’s in himself or herself.

Noi illa neri is the special approach of the siddha system where regular dietary habit, early rising, physical and mental disciplinaries are all emphasized.

Man is nothing but a world in miniature containing the fine elements and all the various principles which constitute the mineral the vegetable and the animal kingdoms

Venpudai is one of the most important skin disorder encountered in medical practice in india it depresses the patient mentally to a great deal, venpudai is also called ‘swetha kuttam’ which is one of the 18 varieties of skin disorders, under kuttam noted in yugi chithamani.

There are many misgivings about this diseases in the minds of the public.

- It is still a wrong notion of the people that it is contagious and it will lead to leprosy because of the name venkuttam and also believe that it is due to sin and karma.
- So the author decided to use form ‘venpudai’ instead of venkuttam for this clinical activity.
- The disease beard social stigma school children having venpudai are struggling to study.
- In the treatment of skin disease as “the siddha system of medicine has wonderful drugs’ the author choice of drugs for the clinical study is
  - Lagu seena chooranam- internal with hotwater.
  - Venpadai patru with –external cows urine
2. AIM AND OBJECTIVES

AIM:

Diseases of the skin are a common occurrence. They account for a great deal of misery, suffering, incapacity and economic loss. Venpadai affects the patients physically and mentally.

The clinical study of Venpadai was done in selected cases of both the gender and they were treated in the Inpatient ward in the Ayothidoss Pandithar Hospital of National Institute of Siddha, Tambaram Sanatorium, Chennai - 47.

OBJECTIVES:

• To have an idea of the incidence of this disease with gender, age, occupation, social status, diet and seasonal variations.

• Collection and detailed study of various literatures, dealing with definition, aetiology, classification, signs and symptoms, prognosis, treatment and diet for venpadai.

• To correlate the signs and symptoms of venpadai with that of modern science.

• To do bio-chemical analysis and toxicological study of the trial drug.

• To find out the efficacy of lagu seena chooranam and venpadai pattru(venkutta lapanam) (external) in venpadai patients.

• To find out the side effects or adverse reactions if any.
PROTOCOL

A PILOT OPEN CLINICAL TRIAL OF LAGU SEENA CHOORANAM AND VENPADAI PATTRU (VENKUTTA LABANAM) FOR THE TREATMENT OF VENPADAI (VITILIGO)

By

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1. INTRODUCTION

Vitiligo is an acquired idiopathic depigmentary condition, which though worldwide in distribution, is most common in India. It is a source of great social embarrassment to dark-skinned people. It affects all age groups with no predilection to either sex. Its incidence is markedly increased.

In Siddha system of medicine, the drugs which are commonly used in the treatment of Venpadai includes karpogi choornam and karpogi thylam.

In Agathiar pillai tamil text, and Siddha vaithiya patharthaguna vilakkam(kannusamiyam) text, there is a preparation for Venpadai, whose efficacy is not well known. So, I try to evaluate it in a pilot clinical trial in our OPD and IPD patients in National Institute of Siddha, Chennai.

2. AIM

To find out the efficacy of Lagu seena Chooranam (Internal) and venpadaipattru(Venkutta labanam) (External) in Venpadai patients.

1. To find out the side effects of the drugs, if any.
2. Biochemical Study
3. Toxicological Study.
3. POPULATION AND SAMPLE

The population consists of all patients with Venpadai (completely depigmented or hypopigmented patches without any structural change in the skin) satisfying the inclusion and exclusion criteria mentioned below. Sample consists of Venpadai patients attending the OPD of Ayothidoss Pandithar Hospital of the National Institute of Siddha, Chennai – 47.

4. SAMPLE SIZE

The trial size will be 50 patients.

5. INCLUSION CRITERIA

1. Age between 5 to 12 years.
2. Willing to give specimen of blood for the investigation when required.
3. Willing to be admitted in the Hospital for 48 days or willing to attend OPD once in 7 days for 48 days.

6. EXCLUSION CRITERIA

Patients of Jaundice, hypo–pigmented patches of leprosy and burns, connective tissue disorders, and heart ailments are not eligible for this trial.

7. WITHDRAWAL CRITERIA

1. Any drastic changes occurring in hematological/immunological findings during treatment period.
2. Development of any exacerbation in clinical features.
3. Occurrence of any serious illness

8. TRIAL DRUG, DOSAGE, DURATION

INTERNAL DRUG: *Lagu seena Chooranam* 1-2 gm with hotwater twice a day after food.

EXTERNAL DRUG : *Venpadai pattru*(Venkutta labanam)-10gm with cows urine applied twice a day

TRIAL PERIOD : 48 days
9. ASSESSMENT AND TESTS

   Clinical assessment
   Site, Size, Colour, Margin, Shape, Itching.

   Investigations
   Blood Test: RBC, Hb, Tc, Dc, ESR

SIDHHA ASPECTS

1. ENVAGAI THERVU
   Naa, Niram, Mozhi, Vizhi, Sparism, Naadi, Malam, Moothiram

2. NEERKURI
   Niram, Manam, Nurai, Enjal, Eadai.

3. NEIKURI

10. CONDUCT

   Venpadai patients satisfying the inclusion and exclusion criteria will be included in the trial. Informed consent will be obtained from the patients. A day before starting the trial treatment, cleaning of mukkutras by purgation will be carried out by Siddhathi ennai, early morning 1 ml with hot water.

   For O.P patients, the trail drugs will be issued for 7 days at a time. They will be asked to attend the clinic once in 7 days. Also, they will be instructed to bring back unconsumed trail drugs and return them during their next clinic visit. Tests will be carried out before treatment and at the end of the treatment. Photos will be taken before and after treatment.

11. FORM

Form I – Selection proforma
   It is used before admission of the patients to the trial

Form II – Assessment form
   It is used once in 7 days during treatment.

12. ANALYSIS

   Cure will be mainly assessed by comparing the before and after treatment photos.
   Quantitative parameters will be assessed by paired t-test.
4. REVIEW OF LITERATURE

SIDDHA ASPECTS

SYM: Venpadai (In yugi’s classification, it is under 18 types of kuttam as swethakuttam) Swethakuttam (Swetha – White)

WHEN TO SUSPECT/RECOGNISE (DIAGNOSTIC CRITERIA):

- Venpadai is a chronic skin disease characterized by various sizes of hypopigmentation of the skin.

- In siddha literatures if the lesions may be present in palms, palms and region (soles, anal region), genitalia are uncurable

- The colour of the patches look like burns is also uncurable.

According to Siddha System, the predisposing causes for this disease have been described as hereditary factor, stress and strain, malnutrition and venereal exposure and no other specific causes have been mentioned for venpadai.
According to Thirumoolar Karukkidai Vaithiya Nool

“எளிரேற்றி வரைய கிளைகளில் துள்ள்கிரமை
கிரேணி மீக்கி குட்டிய காரணத்தும்
பன்னிய முதுகு பவனருக்கு ஒரு
நிலை பயணங்கு, நிலைக்கின்ற தரீயனே”

Six types of Kuttam i.e. skin diseases are caused by kirandhi and megam, eight types are caused by insects in the soil, and four types are caused by worms.

Excessive heat and cold, laziness, excessive sleep in day time, unbridled sexual indulgence, robbery etc. These habits are supposed to be the factors which lower the immunity of the body (Iyarkkai vanmai) and make it vulnerable to the disease.

Excessive intake of food items which are hard to digest, imbalanced food, vomiting and frequent intake of food mixed with stone and hair.

Prolonged mental depression, intention to spoil others, raping, greed, use of indecent and disrespectful words against God and highly religious and noble people, neglecting or

phans and beggars, cursing the elders and so on have also been given as predisposing causes by Yughì Text.

According to Agasthiyar Kanma Kandam

“என் தெரு வெப்பந்தூத்து கறுப்புக்கை முதலே
என் தெரு பிற்புற விளக்கி மாந்தூத்து
நோயில் மிக்கெங்கிறோ ஐக்கிய மாந்தூத்து
நோயில் மிக்கெங்கிறோ ஐக்கிய வாசவு
நோயில் மிக்கெங்கிறோ ஐக்கிய வாசவு”
Kuttam may be hereditary.

Apart from all other etiological factors kuttam is also considered as ‘Karuma noi’.

Classification

According to Yugi Chinthamani

“நிதையால் பெறுவாலும் பெரியமுன்

பெரியப்பகுதியில் கொண்டு கொலையா

புகழ்வூடு பெரும்பாக வேறுபாக

பாலகர் தோரையில் அமைந்து

பாலகர் குளுக்கையோ குறுக்கையோ

சிங்கையூர் சிங்கையூர் அல்லது அல்ல

சுருக்க சோட்டல் சோட்டல் அல்லது

சுருக்கம் மற்றும் சோட்டல் அல்லது

சுருக்கம் மற்றும் சோட்டல் அல்லது

சுருக்கம் மற்றும் சோட்டல் அல்லது
SIGNS AND SYMPTOMS BASED ON *UYIR AND UDAL THATHUKKAL*

Etiological factor

Causative factor

Kanmam

Triggering factor

- Kirumihal (As per Gurunaadi Payil Mozhiyir threhathil kirumi thane…)
- Hormonal imbalance
- Malnutritious diet
- Chronic irritation of skin (i.e. cloth, chapel etc.,)
- Causative factors of kuttam
- Kiranthi noi

*Azhil vitiated*

Associate the *Vitiated iyam*

Derangement of *uyirthathukal* & Deteriorate the *udalthathukal*
Changes due to affected
- Melnokkukaal ($V^*$)
- Nadukaal ($V^*$)
- Vanna eri azhal

Decreased Saaram
- Dryness of the skin
- Thickness of the skin
- Numbness of skin
- Mild colour changes (hypo) lethargy, etc.

Changes due to affected
- Paravukaal ($V^*$)
- Vanna eri azhal ($A^*$)
- Olloli thee ($A^*$)

Decreased Senneer
- Neuritri
- Dryness of the skin
- Pallor of the skin

Changes due to affected
- Paravukaal ($V^*$)
- Vanna eri azhal ($A^*$)

Decreased Oon
- Burning sensation
- Pricking pain
- Impairment of

Changes due to affected
- Paravukaal ($V^*$)
- Vanna eri azhal ($A^*$)

Decreased Kozhuppu
- Increased sweating

Changes due to affected
- Keezhnokkukaal ($V^*$)
- Paravukaal ($V^*$)

Decreased Enbu
- Pain in joints
- Decolourisation of the hair
- Easy to brittle hair & nails.
1. Pundareeka kuttam
2. Virpodaka kuttam
3. Baama kuttam
4. Gaja saruma kuttam
5. Karna kuttam
6. Siguram
7. Krishna kuttam
8. Avudhumbaram
9. Mandala kuttam
10. Abarisa kuttam
11. Visarchika kuttam
12. Vibaathika kuttam
13. Kideeba kuttam
14. sarmathala kuttam
15. Thethru kuttam
16. Sithuma kuttam
17. Sathaaru kuttam
18. Swetha kuttam

According to Siddhar Aruvai Maruthuvam
Venpadai has been classified into 3 types on the basis of Mukkutram, they are,

1. Vaatha venpadi
2. Pittha venpadai
3. Kaba venpadai

According to Siddha Maruthuvam Sirappu :
Venpadai has been classified into 4 types :

1. Vaatha venpadai
2. Pittha venpadai
3. Kaba Venpadai
4. 4. Mega venpadai
According to Athma Rakshamirtha Vaidhya Sarasankiraham

Venpadai is classified into 4 types:

1. Venkuttam
2. Senkuttam
3. Karunkuttam
4. Peru viyathi

Clinical Features of Eighteen Kutta Rogam

According to Thanvanthiri Vaithyam

“மூகக்கண்ட இருமதையானையில் உள்ள இருமதையில்
செய்கின்ற அல்லது எச்சரித்து உள்ளதாலேயே”

Discolouration of skin into white

A. According to Vaithya Saarasangiraham - sole, hands, lips, scalp, fingers and wrist joint - all these organs are found with white coloured patches which are circumscribed along with thickened border and gradually spread which is known as “Venpadai”. Blood, muscle and adipose tissue are affected by disease.

Discolouration of hairs, absence of normal skin texture comparing the adjoining normal skin area and appearance of burns is not curable.

B. According to Pararaasa Sekaram

1. Watery discharge
2. Gray Colour
3. Foul Smelling

According to Anubhava Vaithiya Deva Ragasiyam

“மூகக்கண்ட இருமதையானையில் உள்ள இருமதையில்
செய்கின்ற அல்லது எச்சரித்து உள்ளதாலேயே
செய்கின்ற குட்ட ரோகம் தேர்வு ஆகமலே
பாலம்பாசத் குறுநீர் அல்லது குல்லீர் தரும் தேர்வு.”
Three Types

1. Vaatha Venpadai
2. Piththa Venpadai
3. Kaba Venpadai - Indian Medicine P. 143

Clinical Features:

1. The Skin appears glittering and rough
2. There is an excessive perspiration or no perpiration
3. Discolouration
4. Heat and itching of the skin
5. Numbness in some parts of the body.

According to Sirappu Maruthuvam

1. Vaatha Venpadai
2. Piththa Venpadai
3. Kaba Venpadai
4. Mega Venpadai

1. Vaatha Venpadai
   It is characterised by the depigmented patches, which are dry, rough, reddish with some what pale-black in colour.

2. Pittha Venpadai
   It is characterised by the depigmented patches red in colour like lotus flower, spreading with burning sensation and loss of hairs on that area.
3. **Kaba Venpadai**

   It is characterised by the depigmented patches white in colour like leucus flower, spreads with rashes and itching.

4. **Mega Venpadai**

   It is due to the venereal diseased and it occurs after 4 or 6 months after the onset as disease, syphilis within four or six months of the attack. This venpadai develops initially along the nape and the adjoining spaces. Also gradually it affects the shoulder joints, back of trunk. Clinical features of this type are clearly defined by the author of “Siddha Maruthuvam Sirappu” as follows:

   Depigmented patches are small in number, pale in colour, with turmeric colour or dark colour margin marked with hyperpigmented signs. These lesions are circumscribed with 2 mm to 3 mm diameter or above. This correct picture of hypopigmented and hyper-pigmented skin seems to be more or less a multi eyed filter (sieve-like).

   Females are more prone to this Mega Venpadai, Therefore anti-syphilitic therapy is mandatory in the early period of the treatment.

**Character of Venpadai**

Skin colour will change to reddish black or reddish white or white colour with spreading nature. The imbalance of the three thathus produces certain lesions in skin known as kuttam.

Absence of perspiration and thickening of skin may produce the colour changes in skin.
In Thanvanthiri Vaithiyam

.curable - 11

1. Thethuru kuttam
2. Sadhaaru kuttam
3. Pundareega kuttam
4. Virpodaga kuttam
5. Sarma thala kuttam
6. Baama kuttam
7. Kaha nandhi
8. Venkuttam
9. Sithuma kuttam
10. Alasakuttam
11. Vibaathiga kuttam
Incurable - 7
1. Kabaala kuttam
2. Sarumamega kuttam
3. Avudhumbara kuttam
4. Kideeba kuttam
5. Visarchika kuttam
6. Aguvai kuttam
7. Mandala kuttam

In Yugi Chinthamani - 800

Curable - 10
1. Virpodaka kuttam
2. Baama kuttam
3. Gaja saruma kuttam
4. Krishna kuttam
5. Avuthumbara kuttam
6. Thethuru kuttam
7. Sithuma kuttam
8. Kideepa kuttam
9. Sathaaru kuttam
10. Sarmathala kuttam

Incurable - 11

1. Pundareeka kuttam
2. Karna kuttam
3. Sikura kuttam
4. Mandala kuttam
5. Abarisa kuttam
6. Visarchika kuttam
7. Swetha kuttam

சுருக்க சுருக்கங்கள் (Pathology)

According to Noi Nadal Noi Muthal Nadal Thirattu and Siddha Maruthuvam texts in Siddha System the manifestation of all diseases are the result of derangement of dhoshas that is vaatha piththa and kaba.

According to the theory of Siddha the human body is composed of 96 thathuvams (of constituent principles in nature including panchaboothams and thridhosam). The Siddha System of Medicine is based on the thridhosha theory. This includes the three humors vaatham, pitham and kabam. These three humours are primary and essential constititional factors of human body. These factors exist in $1 : \frac{1}{2} : \frac{1}{4}$ ratio.
respectively in the normal body. This hormonial existence is responsible for the proper functioning of the body. Any alteration in the above ratio can cause disease in the body like, vaatha disease, pitha disease and kaba disease.

Combination of five elements in muthathu:

- Vaatham = Vali + Vin
- Piththam = Thee
- Kaba = Mann + Neer

MUTHATHU IYAL OR UYIR THATHU IYAL:

The most important clinical approach of the Siddha physicians is to assess the status and function of the three uyir thatus. This is described as Naadi sothanai and Naadi nadai Arithal.

a) Vali – (Kattru + Veli)
b) Azhal – (Thee)
c) Iyyam – (Neer+Mann)

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1:½:¼) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

The three thathus are manifest at the wrist and are individually and collectively assessed. These three humour are divided in to various types and have their functions specifically.

VALI:

According to the physiological function vali is of ten types. They are

1. Uyir Kaal (pranan)

   This is the first of ten vital airs. According to Yugi muni, pranan starts from moolatharam and comes through the nostrils and causes the act of inspiration and expiration. The inspiration and expiration are not equal and their ratio is 8:12. The process helps in the digestion of ingested food.
2. **Kizhnokkumkaal (Abanan)**
Apanan, the downward air, starts from swathittanam and descends towards the pelvis and is responsible for excretion of urine and faeces. This is green in color. It contracts the anus. It helps to take the essence of the digested food to the different parts of the body which requires food. The god attributed is varadarajan.

3. **Paravukaal (Vyanan)**
Vyanan arises from the shoulders and go through all the 72,000 nerves and thus activate voluntary and involuntary movements of the body and thus make them to extend the contract. This appreciates the sense of touch, helps to take the essence of the food to the strategic points of the body and guards the body.

4. **Melnokkumkaal (Udhanan)**
Udhanan starts from the umbilical region (udarakkini) and takes the essence of food and stations it at appropriate places. It helps in digestion and assimilation of food.

5. **Nadukkal (Samanan)**
Samanan starts from the umbilical cord and spread out up to the lowerlimb. This is responsible for the balance of the other four vathas. It equalizes the six tastes, water, food etc and helps in assimilation.

6. **Naagan**
Naagan is responsible for higher intellectual functions, hearing, thinking etc. It causes closing and opening of the eye lids.

7. **Koorman**
Koorman starts from the mind and causes winking of the eyelids, yawning and closure of mouth. It gives strength and helps to visualize things and causes lacrimal secretion.
8. Kirukaran
Kirukaran lies in the tongue and causes nasal and salivary secretions. It induces hunger. Sneezing and cough are attributed to kirukaran. It is black in color.

9. Devadaththan
Laziness is attributed to devadaththan. Occular movements and human passions are attributed to this vatham. It stays either at the anus or at urinary orifice.

10. Dhananjeyan
Dhananjeyan functions from the nose and it is responsible for the bloating of the body after death and also for the foul smell.

AZHAL:
It is not just heat in our body. Functionally this human bodies warmth/heat of life is divided into five types. They are

1. Akkuanal (Analagam)
It lies between the stomach and the intestine and causes digestion and dries up moist ingested substances

2. Vanna eri (Ranjagam)
This fire lies in the stomach and gives red color to the chyme and produces blood. It improves blood.

3. Attralangi (Sadhagam)
This fire lies mainly in limbs. It gives energy for activities

4. Nokku Azhal(Alosagam)
It lies in the eyes and causes the faculty of vision. It helps to visualize things

5. Ollolithee (Prasagam)
It gives color and complexion and brightness to the skin
IYYAM:

It is of five types. They are

1. **Alli Iyyam (Avalambagam)**
   It lies in the lungs and helps in respiration. It causes firmness of the limbs. This is vital among all types of kapham for it controls the other four kapham and maintains equilibrium.

2. **Neerpi Iyyam (Kilethagam)**
   It lies in the stomach. It mixes the consumed food and water and promotes the digestive process.

3. **Suvaikanna Iyyam (Pothagam)**
   It lies in the tongue and helps to realize the taste of the consuming food.

4. **Niraivu Iyyam (Tharpagam)**
   Sustaining in the head, this gives refrigerant effect to cool the eyes and other sense organs.

5. **Ondri Iyyam (Sadhigam)**
   Sustaining in the joints this makes them more freely and easily.

Since venpadai patients are not having defined description of pathology, the pattern of disturbance in Vali, Azhal, Iyyam keeps on varying. The manifestation of these uyir thathus keep on changing according to the predominant symptom. But alteration in azhal thathu is seen mostly.

EZHU UDAL KOORUGAL:

**SEVEN PHYSICAL CONSTITUENTS**

The human body is made of seven basic, physical constituents. These constituents should be in harmony and function normally. Any variation in them will lead to their functional deviations.

The Natural characters of the seven physical constituents
1. **Saaram** (Chyle): This gives mental and physical perseverance

2. **Senner** (Blood): Imparts color to the body, nourishes the body and is responsible for the ability and intellect of an individual

3. **Oon** (Muscle): It gives shape to the body according to the physical activity and covers the bones.

4. **Kozhuppu** (Adipose tissue): It lubricates the joints and other parts of the body to function smoothly

5. **Enbu** (Bone): Supports the frame and responsible for the postures and movements of the body

6. **Moolai** (Bone marrow): It occupies the medulla of the bones and gives strength and softness to them

7. **Sukkilam/Suronitham** (Sperm and Ovum): It is responsible for reproduction

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**THE VARIATIONS OF THE PHYSICAL CONSTITUENTS**

1. **SAARAM**
   - Increased Saaram: Leads to diseases of increased kapham like indigestion etc.
   - Decreased Saaram: Leads to loss of weight, tiredness, lassitude, dryness of the skin and diminished activity of the sense organs.

2. **SENNEER**
   - Increased Senneer: Causes boils in different parts of the body throbbing pain, anorexia, mental disorder, splenomegaly, Colicky pain, increased blood pressure, reddish eye and Skin, jaundice, haematuria etc.
   - Decreased Senneer: Leads to anaemia, tiredness, neuritis and lassitude, Pallor of body.

3. **OON**
   - Increased Oon: Oon in excess causes cervical lymph adenitis, venereal ulcer, tumour in face, abdomen, thigh genitalia etc are the signs of increased Oo
   - Decreased Oon: Leads to impairment of sense organs, joints jaw, thigh and genitalia gets shortened
4. KOZHUPPU

Increased Kozhuppu: Identical to that of increased Oon associated with Dyspnœa and loss of acidity Decreased Kozhuppu: Leads to pain in the hip region and diseases of the spleen

5. ENBU

Excess Enbu: Growth in bones and teeth Decreased Enbu: Loosening of teeth and nails and Splitting and falling of hair

6. MOOLAI

Increased Moolai: Causes heaviness, swollen eyes, swollen phalanges, Oliguria and non healing ulcers Decreased Moolai: Causes osteoporosis and sunken eyes

7. SUKKILAM / SURONITHAM

Excess Sukkilam/Suronitham: Causes lust towards women and cause Urinary calculai Decreased Sukkilam/Suronitham: Causes failure in reproduction, pain in the genitalia.

ENVAGAI THERVU

Nowadays advanced diagnostic tools have been developed by modern bio-medical scientists. But Siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

1. NAA

Signs and symptoms in the tongue are considered here. Size, appearance, thickness, color(pigmented, magenta) fissured (longitudinal, transverse) coated, geographical patches, oral hairy leucoplastia, candida, apthus ulcers, sense of taste, saliva secretion

2. NIRAM

The color of skin is mainly considered here but also the change in other organs.

3. MOZHI

The change in the normal sound of voice mainly uratha olli (Valithel), thazhntha olli (Melithal), physiological and mental status can also be noted during conversation.

4. VIZHI

Color, warm, burning sensation, irritation, visual perception
5. PARISAM

Observations by touch, temperature, sensory impairment, masses, nodes, swelling, texture of the skin, pain, hardness, odematus, and dullness shall be noted.

6. MALAM

The stools are examined for quantity, hardening (malakattu) loose motion (beethi) color, smell

7. MOOTHIRAM

Neer Kuri: Urine is to be observed for the following characters
- Niram (color)
- Alavu (quantity)
- Edai (specific gravity)
- Manam (smell)
- Nurai (froth)
- Enjal (deposit)

NEI KURI

A unique traditional method for diagnosis with urine is Neikkuri. Urine is freshly collected in a clean glass vessel and a drop of Gingili oil is dropped in to it. The gingelley oil should be prepared by wooden press. Machine pressed oil is not an effective tool. Mode of spreading is noted, usually
- Vali noi - Aravu (Snake like spread)
- Azhal noi - AAzhi (Ring like spread)
- Iyyam -Muthu (Pearl like spread)

venpadai starts with disturbed Azhal and eventually involves all the three uyir thathus – thus resulting in various patterns of oil spread in the urine surface. An unique pattern, is seen mostly like star fish with branches and sieve (plate with pores), irregular border, speed of spread is also to be noted. It has been observed that the initial star shaped spread reduces as the patients responds to treatment.
MANAGEMENT OF VENPADAI

REJUVENATION:

Kalpa marunthu:

Pothukalpam (General):

- Ponnangaani karpam (Alternanthera sessilis) (Used as recipes along with Milagu (Piper nigrum), Kariuppu (Sodium chloride)).

Sirappu:

- Kittikizhangu (Acalypha fruticosa) used as daily dishes like curry, vatral etc.,
- Lemon used as pickle or juices (to be continued for six months).
- Ayapirungaraja karpam
- Ayajambeera karpam

Kalpa Yogam:

- Sarvangaasana is most useful for this complained Shirasana is also useful, where as the other asanam have been included for general health and fitness.
- Pranayamam

The common benefits of yogam

Padmaasanam

Uses

The constant practice of this asana brings about good digestive power, cheerfulness and balancing of mukkutram.

Sarvangaasanam

Uses

It prevents narai, thirai and moopu. (i.e.) prevents ageing. By stimulating the thyroid gland it gives strength to all the organs of the body. It cures venpadai.
UNAVU (DIET):
To be add:
  • Bitter tasted foods

Greens:
  • *Sirukeerai* (Amarantus tricolor) – “Kanpuhaichal......Pongumpitham......Sirukeerai thanai kol.”
  • *Pannai keerai* (Celosia argentea) - Pannai yilankeerai yathu...Karappan sirangu pun maatrum.....
  • Paruppu keerai (Postulaca oleracea) – “Pillai parupilaiku pithamarum.....”
  • Keerai thandu (Amaranthus gangeticus) – Senkeerai thandathu than theeratha pithathai thengaamal oati vidum...
  • Agathi keerai (Sesbania grandiflora) – Pithmathu saanthinga hum agathiyilai thunuubavaiku...

To be avoid:
  • Sour, spicy, salty foods
  • Curd, oils, alcohol, sugar, non – vegetarian diet
  • Ulundhu (Vigna mango)
  • Mustard
  • Brinjal.

Other advices:
  • Oleation: Oil bath should be taken twice a week is advisable.
  • Be add bitter tasted herbs like Azadhirachta indica, Acacia cateche etc.,
  • *Savuri pazha thylam*
  • Palasu uppu (salt of Butea monosperma) – For external application
  • De-oleaginous substances:- The powdered form of Iluppai pinnakku (Bassia longifolia).
  • Eechaam paai (a type of mattress prepared from the leaves of Phoenix sylvestris)

Food stuffs that bring the vaatha, piththa and kaba dhoshas to the normal physiological level have to be consumed.
MODERN ASPECTS

The Skin

Dermatology is the study of skin diseases. Disease of the skin, are a common occurrence, account for a great deal of misery, suffering in capacity and economic loss. A very few skin diseases are contagious.

Hippocrates, Father of Medicine described many skin diseases and divided them into two groups according to their exogenous or endogenous causes. He attributed the origin of disease to abnormal mixing of black and yellow bile, blood and phlegm. The theory of adnormally mixed humors played a major role in dermatology for along time.

Dermatology is a branch of medicine dealing with the skin. Its roots reach back to antiquity. The obviously manifested skin diseases have drawn the attention of men since time immemorial.

Skin Anatomy

The human skin is the outer covering of the body and is continuous with the mucous membranes in the region of the mouth, nose, urogenital organs and the anus. In an adult the skin surface measures 1.5 - 2 m² while the thickness of the skin varies from fractions of a millimeter to 4 mm. The thickness of the epidermis varies from 0.06 - 0.9 mm to 0.5 - 0.6 mm. The thickness of the subcutaneous fat varies considerably. Some areas are devoid of fat while in others (on the abdomen and gluteal regions), it is several centimeters thick. The mass of skin of an adult accounts for approximately 5 % while together with subcutaneous fat for about 10 to 17.7 % of the total body mass.
Normal Skin Structure

The colour of the skin may change because the amount of the pigment in it varies due to external and internal factors.

The skin surface is covered with hairs over a great area. The areas devoid of hairs are the lips, palms and soles, glans penis, inner surface of the prepuce and the inner surface of the labia majorum and minorum.

Skin Histology

The skin develops from two germinative zones. The ectoderm which is represented by the epidermis (the most superficial skin layer) and the mesoderm (the middle embryonal layer) represented by two layers namely the true skin, or dermis (the middle layer) and the subcutaneous fat or hypoderm the deepest skin layer.

The boundary between the epidermis and dermis forms a wavy line because of the presence of skin papilla (special out growth on the surface of the true skin) the spaces between which are filled with epithelial processes.
Vascular System of Skin

Vascular system of the skin is formed of several networks of blood vessels. Large arterial vessels stretch from the bascia through the subcutaneous fat and give off small branches to the fat lobules. On the boundary of the dermis and hypoderm, they divide into branches which stretch horizontally and anastomose with one another. A deep arterial plexus of skinforms, which gives rise to branches supplying the holes of the sweat glands, the hair follicles and the fat lobules. The epidermis is devoid of blood vessels. The most powerful network of blood vessels is located in the skin of the face, palms, soles, lips, genitals and in the skin around the anus.

Lymphatic system of skin

The lymphatic system of the skin forms superficial and deep networks. The superficial lymphatic network arises on the papillary layer as blind rounded dilated capillaries between which there are numerous anastamoses. The second network of lymph vessels is in the lower part of the dermis and already has valves. There is a network of wide loops forming lymphatic plexus and deeper parts are continous with lymph trunks.

Epidermis

The epidermis is stratified epithelium undergoing keratinization; it consists of the following layers:

i) Germinative layer of stratum basale

ii) Prickle - cell layer or stratum spinosum

iii) Granular layer or stratum granulosum

iv) Stratum lucidum

v) Horny layer or stratum corneum

There are many nerve endings in the epidermis but no blood vessels and the cells are supplied with nutrients by the lymph flowing in the intercellular slits.
Dermis

The dermis is located between the epidermis and the subcutaneous fat. Two layers are distinguished in it, the papillary or subepithelial layers and the reticular layer. The papillary layer is that part of the dermis which is found between the epidermis and the superficial network of blood vessels. The reticular layer merges with the subcutaneous fat and is not demarcated from it sharply.

The Deep Part of the Skin

The subcutaneous tissue or hypoderm consists of thick bundles of collagen and elastic fibres stretching from the regular dermal layer and forming a wide loop reticulum in which accumulations of large fat cells, lobules or fatty tissues are lodged. The fat cells are almost completely formed of a large drop of fat which displace the cell nucleus to the periphery and a very small amount of protoplasm.

Vitiligo

The name ‘vitiligo’ is derived from the Latin word skin eruption, victim meaning a blemish (spoil the beauty of) happens to be a synonym for it.

White skin is the literal meaning of leucoderma, derma being derived from the Greek words, leucas and dermis. Leucas means white and dermis means skin.

Celeus was the first Roman physician of the 2nd century to coin the word vitilligo, because the disease resembles the white patches of a spotted calf (vitelus).

Vitiligo is characterized by the presence of non-pigmented areas of irregular shape, which develop on the epidermis of skin and hair. In this condition there is absence of deficiency of melanin, a dark pigment of the skin produced by melancytes under the stimulation of the sun light and possibly, under the control of a melanin stimulating hormone of the hypophysis.

It is also regarded to develop through eczema scar of prick by injection needle, injury by burn or from other accidents, by friction of foot, wearing tight clothes. It has also been observed in persons who have suffered serious illness due to typhoid, jaundice, liver diseases, diabetes, worms, constipation and diarrhoea.
The non pigmented patches whitish or reddish are round or oval in shape with smooth surface and slowly grow into large, irregularly outlined areas. It may be the result of skin diseases or it may be a harmless condition of unknown cause.

**Definition**

Vitiligo is a disorder of the skin especially due to loss of pigment without any disturbances and textural alterations.

A condition due to failure of melanin formation in the skin produces sharply demarcated, milky white patches with hyperpigmented borders.

But Leucoderma is an acquired no inherited (incurred as a result of factors acting from or originating outside the organism). Condition with localized loss of pigmentation of the skin. Inheritance means an acquisition of characters or qualities by transmission from parent or offspring.

It is an extremely common depigmentary disorder of great medicosocial significance among the dark people, aetiology is uncertain association with variable penetrance; no age is except, both sexes. A symptomatic puncture linear, oval, circular or irregular, discrete or confluent depigmented and or hypopigmented macules on otherwise normal skin is confined to mucocutaneous functions dermatomal unilateral or bilateral, symmetrical or asymmetrical generalized or universal over laying hair retain pigment or turn white, no autonomic or sensory disturbances, sub burn or chronic solar damage in longstanding cases, unpredictable and capricious course, stationary, self healing or progressive.

It is quite clear that vitiligo is due to some derangement in the pigment metabolism resulting in appearance of white patches in the skin. It is hard to say whether the site of derangement is usually general or local, but the main affected part is the skin, which is the most exposed part of the body. It can be examined by naked eye and can furnish a lot of information about the person and the disease. In certain cases the changes are not clear. Hence the study of the skin structure and its physiology is essential for proper assessment.
Epidemiology

Vitiligo is an acquired idiopathic depigmentary condition which, though worldwide in distribution, is most common in India, Egypt and other tropical countries. It is a source of great social embarrassment of dark-skinned people. It affects all age groups with no predilection to either sex.

Gross Anatomical Changes in Vitiligo

Vitiligo represents an acquired patchy loss of pigments of the skin. There are no gross changes seen except irregularly demarcated, depigmented patches of varying size, usually surrounded by hyperpigmented skin. These are seen distributed symmetrically or asymmetrically at various parts of the body.

Histopathologic changes in Vitiligo

Marked histological changes do not occur in cases of vitiligo. All the layers of the epidermis and dermis appear normal except a few changes which can be seen after special stains.

In the affected area the basal cells and the keratinizing cells of the other layers of epidermis do not contain melanin pigment granules in them. The contrast can be seen at a junction of the normal and vitiligenous areas of the skin, especially by silver staining or DOPA reaction. The pigment cells, the melanocytes are not seen in the affected area but they are present in the adjacent normal skin. At the border of the patches of vitiligo the melanocytes often appear large and posses long dendritic process filled with melanin granules. Electron microscopic studies confirm the absence of melanocytes in areas of long standing vitiligo.

There are collections of mononuclear cells at dermo epidermal junction at the border between vitisliginous and normal skin. These cells are predominately small lymphocytes. In the long standing cases where the skin has become thick and scaly, varying amount of the keratosis is seen.
Possible cellular and humoral immune mechanisms of vitiligo:

Melanin

Melanin - Derived from the Greek word Melas, meaning black.

Melanin is endogenous nonhaemoglobin derived brown or black pigment formed when the enzyme tyrosinase catalyses the oxidation of tyrosin to dihydroxy phenylalanine (DOPA) in melanocytes.

Distribution

It is widely distributed in the body but peculiarly enough it is limited only to those structures which have got an ectodermal origin, for eg: skin, hair, choroid coat of retina and substantia nigra of the brain. It is formed from tyrosine by oxidation,metabolism and polymerization.
**Pigmentation of the Skin**

The colour of the skin may be brown or even black according to the amount of pigment present.

Even in white races most parts of the skin contain brown pigment granules in the deepest layers of the germinative zone of the epidermis. In dark races they are more abundant and extend throughout the whole zone.

**Functions**

The function of melanin in the choroids is namely to convert the eye ball into a perfect dark chamber. Since nervous tissue is derived from ectoderm, the melanin in the substantia nigra may represent the vestigial remnants of the melanin in the substantia nigra may represent the vestigial remnants of the melanin forming properties. Melanin is the great protector of the skin against the actinic rays of the sun.

**Melanin Formation**

Melanin, wherever it is found, is formed in the local cells by the enzyme tyrosinase (or) melanase. The mother substance, upon which the enzyme acts, is a tyrosine derivative (DOPA) believed to be formed in the adrenals. Melanin synthesis from the oxidation of phenylalanine or tyrosine are as follow:

1. Tyrosine $\rightarrow$ DOPA $\rightarrow$ DOPA $\rightarrow$ Quinone
2. DOPA - Quinone $\rightarrow$ 2 - Carboxy 2,3 - dihydro - 5,6 - dihydroxyindole
   $\rightarrow$ 2 - Carboxy - 2,3 - dihydro - indole - 5,6 - Quinone $\rightarrow$ 5,6Dihydroxyindole.
3. 5.6 Dihydroxyindole $\rightarrow$ Indole - 5,6 Quinone $\rightarrow$ Melanin

Melanin formation in both human and amphibian skin is augmented by the hormone known as intermedian or melanocyte - stimulating hormone (MSH) secreted by the pars intermedial of the pituitary gland. Adrenocartico tropic hormone (ACTH) secreted by Anterior Pituitary has melanocyte - stimulating activity similar to MSH although to a much lower degree. In addison’s disease ACTH is secreted in a large amount and there is brownish black pigmentation of the exposed parts of the skin eg. hands, feet and mucous membrane.

Melatonin extract from bovine pineal gland, causes concentration of meanin near the nuclei of melanocytes in frog and as a result of this the skin becomes pale. Its role in the human is not
known. MSH causes the serum copper to rise and this is accompanied by in the melanin formation. Diminished formation of melanin is seen in albinism and leucoderms. In melanotic sarcoma, melanin may be found in the urine.

**Aetiology - Vitiligo**

Melanocytes in areas of depigmented skin are destroyed and the cause is unknown. Anti-melanocytic anti-bodies directed against intra cellular components of melnaocytes have been shown. The presence of organ specific auto immune disease occurs in about 10 % of patients. Such conditions are more common in their families than in a normal population. A neurogenic defect has been postulated for the rare dermatomal pattern of vitiligo which affects principally the limbs.

**Endocrines - Association with thrototoxicosis and diabetes.**

**Trophoneurosis and autonomic imbalance - emotional stress and strain.**

Infections and toxic products, Enteric fever ill health, focal sepsis.

**Drugs and chemicals - like quinines, guano furacin, amylphenol, chlorthiazide, broad spectrum antibiotics and chloroquin.**

Auto-immune thyroid disease is one of a group of organ specific auto immune diseases that include pernicious anaemia, Addison’s disease and hypo para - thyroidism.

**Hereditary Factors**

Hereditary is one of the factors supposed to be related with venpadai to some extent. Familial incidence has been reported in 7.5 to 21 % in India and 33 to 40 % in western countries.

It is every day knowledge and observation that emotional factors affect the skin as shown by the blushing of embracement, the pallor of fear and the pallor or redness of Change, depending on the subject and his emotional state. Experiments have demonstrated that emotional change can affect the following, which has direct relevance in the aetiology of certain skin disorders.

- Control of vascularity of the skin
- Control of sebaceous gland secreation.
- Influencing the degree of oxidation.
- Influencing the tendency of pruritus.
There is due to the causative factor of this disease, vanpadai from the following basic facts, it is generally considered to be a trophoneurosis. Psychological factors are known to be responsible for the precipitation and aggravation of the disease.

Pathology

Chemically melanin pigment is a group of chromo proteins with coloured prosthetic groups, which is derived from the precursor tyrosine in the following way Tyrosine Tyrinase Dihydroxy phenylanin (DOPA) Melanogenase Melanin (Dopa oxidase).

Melanin + Protein = Melano protein

The possible pathogenetic mechanism. a schematic picture of the causal and temporal sequence leading to the melanocyte functional impairment during vitiligo.

In the skin, the pigment is produced by the melanocytes of their precursors melanoblasts. The melanoblasts are supposed to be derived from the cells of neuro ectodermal origin during the embryonic life. After birth, these cells migrate to their definitive position. The melanocytes appear as clear cells within the basal cell layer of the epidermis and show dendritic processes after special staining. These processes come in contact with similar process of other melanocytes and epithelial cells through which the melanin pigments are donated to the basal
cells of epidermis. The dermis of normal skin also shows macrophages containing malanin pigments known as melanophores, which are incapable to produce the melanin pigments.

**Causes of Hypopigmentation**

Generalised depigmentation is found mostly in albinos. In this case, the characteristic dendritic melanocytes are present in the skin, but they are unable to produce melanin pigment due to defective tyrosinase activity. In albinism, the skin looks milky white, the hairs are pale looking and the iris is transparent. The generalized pallor is also noticed in panhypopituitariarism, male eunuchoidism and phenyl ketonuria.

Localised depigmentation is often noticed in the skin of patterned leucoderma. The white patches on the skin may be quite extensive and the condition is inherited as an autosomal dominant character.

Sometimes sharply defined focal depigmented areas are found on skin of persons suffering from vitiligo. In the affected areas, melanocytes are absent and there is no trace of melanin. The condition is an acquired one and shows some familial tendency.

Vitiligo in patients in whom the disease spreads very fast or those having halo-navi or malignant melanoma is believed to be based on auto-immune mechanisms, where auto antibodies or sensitized lymphocytes are supposed to act on the melanocytes. Trauma on the skin including that produced by scratching can lead to depigmentation of the skin even when it does not lead to ulceration. Leucoderma is also commonly seen on the flanks of ladies wearing tight petticoat strings where the prolonged pressure is presumed to lead to depigmentation. Sometimes vitiligo can be caused by the action of monobenzyle either on hydroquinone present in the slippers, gloves (or) other articles made of rubber or used as a depigmenting agent in the form of an irritant for pigmentary disorders. Recently vitiligo has also been observed to occur from plastic slippers as well as plastic ‘hindis’.

However most people with vitiligo have no other autoimmune disease. Vitiligo may also be hereditary, that is, it can run in families. Children whose parents have the disorder are more likely to develop vitiligo. However, most children will not get vitiligo even if a parent has it, and most people with vitiligo do not have a family history of the disorder.
Clinical features:

1) In this condition patches of skin lose their pigment and become perfectly white, though no other changes take place in them and particularly there is no scaling.

2) Vitiligo may occur in either sex and at any time.

3) The white patches may appear on any part of the skin but commonest on the face, neck, hands and wrist, lower abdomen and thighs and may be precipitated by trauma to the skin.

4) They may be of any size or shape and are usually though not always, roughly symmetrical.

5) They slowly increase in size until large areas of the skin are completely discoloured.

6) The remaining small patches of normal skin may then be mistaken for pigmented areas. The mistake may be avoided by remembering that the vitiligo areas have convex margins and the normal areas therefore have concave ones.

7) When vitiligo occurs on a hairy area such as eyebrows or pubis the hair on the white patch may also become white.

8) The depigmented areas are sometimes surrounded by an excess of pigmentation in the immediately adjoining skin but this appearance is often illusory and the result of visual contrast.

9) Vitiligo is most noticeable in the summer when the normal skin is tanned by the sun.

10) Vitiligo sometimes disappears spontaneously after months or years but more usually the condition spreads slowly and may eventually involve nearly whole of the skin.

11) It is characterized by completely depigmented macules and patches of varying sizes and shapes.

12) There are no other changes except depigmentation.

13) Early lesions may be pale white and ill defined. At this stage, Wood’s lamp helps to confirm the diagnosis. Patches enlarge slowly and may affect the whole body.
14) Any part of the body can be affected but the sites of predilection are the face, dorsa of fingers and hands, wrist and the legs.

15) Involvement of mucous membrane especially the lip is not uncommon; it can precede cutaneous involvement by years.

**Clinical Criteria for Classification of Vitiligo**

**Stages of Clinical Features**

**Vitiligo**

- **Active (VI)**
  - (i) New lesions developing
  - (ii) Lesions increasing in size
  - (iii) Border ill-defined

- **Quiescent (V2)**
  - (i) No new lesions developing
  - (ii) Lesions stationary in size
  - (iii) Border hyper pigmented and well-defined.

- **Improving (V3)**
  - (i) Lesions decreasing in size
  - (ii) No new lesions developing
  - (iii) Border defined and signs of spontaneous regimentation

**Zosteriform:** Unilateral distribution of lesions, preferably along the course of nerves.

Besides typing the stage of disease, it is useful to decide the variety (acral, Vulgaris, Zosteriform). Severity (Localized or extensive) and acuity (insidious or galloping) of vitiligo.

**Diagnosis:**

- The distribution, the age of onset and the hyperpigmented border will suggest the diagnosis.

- It is usually apparent; in doubtful and early case, Wood’s lamp is of great help in diagnosis.

- Usually in macular leprosy, seborrhoeides, pityriasis versicolor and nevoid condition, its assistance is called for.
• In piebaldism the lesions are present at birth, are usually confined to the head and trunk and rarely show a hyperpigmented border.

• Careful examination of the texture of the unpigmented skin should exclude lichen sclerosus and scleroderma.

• Post-inflammatory leucoderma, which is frequent in the darker races, shows an irregular mottling of hyperpigmented and hypopigmented blotches.

• Hypomelanosis of the affected skin is commonly seen in pityriasis alba, producing slightly scaly areas with rather ill defined edges of children’s faces.

• Hypopigmented, slightly scaly macules are seen in pityriasis versicolor.

• Vitiliginous areas are milky white while other lacks this milky white colouration.

• Stationary patches are well-defined and have hyperpigmented borders.

• Absence of scaling, crusting and itching help to eliminate seborrhoeids and pityriasis versicolor.

• These areas often fluorescence a golder yellow when examined under a Wood’s lamp. The hypomelanotic macules in leprosy are anaesthetic.

• Examination of the skin in long wave UVR helps distinguish whether there is total depigmentation (as in Vitiligo) or not. It may also detect areas of depigmentation not easily seen in ordinary daylight, as well as detecting a lemon-yellow fluorescence seen in some cases of pityriasis versicolor.

**Prognosis**

It has improved considerably in recent years because of better understanding of etiological factors and advances made in therapy.

Following conditions are said to be of poor prognosis.

1) Poor nutritional state or digestion, use of broad-spectrum antibiotics over long period. Emotional stress and nervous debility.

2) Presence of vitiligo on resistant sites like the hands and the feet, front of wrists, the elbow, the waist, the eyelids and lips.

3) Depigmented hair in vitiliginous areas.
## Causes of Localised Hypopigmentation

<table>
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<th>Description</th>
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<td>Vitiligo</td>
<td>Destruction of melanocytes; common; acquired, multiple sharply defined nonpigmented patches any where.</td>
</tr>
<tr>
<td>Pityriasis versicolor</td>
<td>Superficial fungus infection leading to disturbance in pigment production, common multiple pale scaling patches on trunk.</td>
</tr>
<tr>
<td>Pityrisis alba</td>
<td>Mild patchy eczema of the face in children causing a disturbance in pigment production.</td>
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<tr>
<td>Leprosy</td>
<td>One or several paler macules on trunk or limbs that are hypoaesthetic.</td>
</tr>
<tr>
<td>White macules of affecting tuberous sclerosis</td>
<td>Uncommon development of anomaly of CNS, connective tissue and skin; several “maple leaf” shaped hypopigmented macules.</td>
</tr>
<tr>
<td>Postinflammatory hypopigmentation</td>
<td>After inflammatory skin disease (after eczema or trauma to the skin; irregular in shape and in depth of pallor).</td>
</tr>
<tr>
<td>Naevous anaemicus</td>
<td>Rare developmental solitary white patch usually on trunk; thought to have vascular basis.</td>
</tr>
<tr>
<td>Chemical toxicity</td>
<td>May look very much like vitiligo; seen in workers in rubber industry exposed to parateriary benzyltoluence.</td>
</tr>
</tbody>
</table>
## Differential Diagnosis of the important Depigmentary Disorders

<table>
<thead>
<tr>
<th>Distinguish Features</th>
<th>Albinism</th>
<th>Naevus Depigmentosus</th>
<th>Vitiligo</th>
<th>Leprosy</th>
<th>Pityriasis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>Congential present at birth</td>
<td>Congential-present at birth</td>
<td>Acquired</td>
<td>Any age</td>
<td>Any age</td>
</tr>
<tr>
<td><strong>Distribution</strong></td>
<td>Complete (or) partial</td>
<td>Unilateral</td>
<td>Any area</td>
<td>Any area</td>
<td>Trunk, Neck, and Face</td>
</tr>
<tr>
<td><strong>Course</strong></td>
<td>Stationary</td>
<td>Does not increase in size or changing shape</td>
<td>Progressive</td>
<td>Progressive</td>
<td>Progressive; worse in mansoon and summer</td>
</tr>
<tr>
<td><strong>Hyperpigmentary border</strong></td>
<td>Nil</td>
<td>Nil</td>
<td>Present</td>
<td>Inflammatory</td>
<td>Nil</td>
</tr>
<tr>
<td><strong>Heredofamilial</strong></td>
<td>Hereditary</td>
<td>Not hereditary</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td><strong>Other features</strong></td>
<td>Hair and eyes may be effected</td>
<td>Nil</td>
<td>Nil</td>
<td>Anesthesia thickened nerves, nasal, bleeding slit smear and biopsy</td>
<td>Furfuraceous like dandruff, scaling in head macules and large patches.</td>
</tr>
</tbody>
</table>
5. RESULTS AND OBSERVATIONS

Results were observed with respect to the following criteria

1. Age reference
2. Sex reference
3. Religion reference
4. Socio Economic status of the reference
5. Etiology reference
6. Family history reference
7. Paruva kaalam
8. Diet reference
9. Thinai
10. Site of lesion
11. Derangement in the types of Vatham
12. Derangement in the types of Pitham
13. Derangement in the types of Kabam
14. Ezhu Udar Kattugal reference
15. Ennvagai Thervugal reference
16. Distribution of nadi among the patient with venpadai
17. Neikuri reference
18. Results after treatment reference

The observations recorded with the above said criteria are given in the tabular form.
1. Age Reference

<table>
<thead>
<tr>
<th>Age (in Years)</th>
<th>No. of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-8 Years</td>
<td>23</td>
<td>46%</td>
</tr>
<tr>
<td>9-10 Years</td>
<td>11</td>
<td>22%</td>
</tr>
<tr>
<td>11-12 Years</td>
<td>16</td>
<td>32%</td>
</tr>
</tbody>
</table>

Inference

About 46% of them were aged 5-8 years and 32% cases 11-12 years, 22% cases 9-10 years.
2. Sex Reference

<table>
<thead>
<tr>
<th>Sex</th>
<th>No. of Cases (Out of 50)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male Children</td>
<td>21</td>
<td>42%</td>
</tr>
<tr>
<td>Female Children</td>
<td>29</td>
<td>58%</td>
</tr>
</tbody>
</table>

Inference
Out of 29 patients 58% were Female Children and 21 patients 42% were Male children.
3. Religion Reference

<table>
<thead>
<tr>
<th>Religion</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hindu</td>
<td>41</td>
<td>82</td>
</tr>
<tr>
<td>Christian</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Muslim</td>
<td>7</td>
<td>14</td>
</tr>
</tbody>
</table>

Inference
Most of the patients religion Hindu (82%) and Muslims 14% Christians 4%.
4. Socio – Economic Status

<table>
<thead>
<tr>
<th>Socio – Economic Status</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower Income Group</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Middle Income Group</td>
<td>25</td>
<td>50</td>
</tr>
<tr>
<td>High Income Group</td>
<td>10</td>
<td>20</td>
</tr>
</tbody>
</table>

Inference

About 50% of the parents were middle income group and 15% lower, 20% high income group.
5. Etiology Reference

<table>
<thead>
<tr>
<th>Etiology</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereditary</td>
<td>6</td>
<td>12%</td>
</tr>
<tr>
<td>Aneamia</td>
<td>10</td>
<td>20%</td>
</tr>
<tr>
<td>Unknown</td>
<td>34</td>
<td>68%</td>
</tr>
</tbody>
</table>

Inference

About 68% of the cases were of unknown aetiology and 20% Aneamia cases, 12% hereditary cases.
6. Family History Refernce

<table>
<thead>
<tr>
<th>Family History</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Absent</td>
<td>46</td>
<td>92</td>
</tr>
</tbody>
</table>

Inference

About 92% cases were having no family history absent and in 8% of cases family history was present.
7. Paruvakaalam

<table>
<thead>
<tr>
<th>Paruvakaalam</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaarkaalam</td>
<td>42</td>
<td>84</td>
</tr>
<tr>
<td>Koothirkaalam</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>Munpani</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pinpani</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Elavenil</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mudhuvenil</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Inference

According to Paruvakkalam, highest incidence 84% were noted in Karkaalam and Koothirkaalam 16% cases were noted (Table 7)
Out of fifty cases, 42 were admitted in Kaarkaalam and 8 admitted in Koothirkaalam and none in the remaining kaalangal.
### 8. Diet Reference

<table>
<thead>
<tr>
<th>Food Habit</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetarian</td>
<td>13</td>
<td>26%</td>
</tr>
<tr>
<td>Mixed</td>
<td>37</td>
<td>74%</td>
</tr>
</tbody>
</table>

**Inference**

About 74% of cases were taken mixed diet and 26% of cases have Vegetarian.
9. Thinai

<table>
<thead>
<tr>
<th>Thinai</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kurunchi</td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>Mullai</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Marutham</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Neithal</td>
<td>24</td>
<td>48</td>
</tr>
<tr>
<td>Paalai</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Inference

According to Thinai the highest distribution 48% was noted in Neithal, In Kurinchi 24%, in Marutham 20% and Mullai 8% were observed (Table 9).
10. Site of Lesion

<table>
<thead>
<tr>
<th>Site of Lesion</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scalp</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Face</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Lips</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Chest</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Upper Limb</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Lower Limb</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Genital</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Multiple</td>
<td>21</td>
<td>42</td>
</tr>
</tbody>
</table>

Inference

About 42% of cases were having multiple site of lesion, Scalp region 14%, Face 10% Chest 8%, upper limb 8% Lower limb 10% lips 4%, Genital region 4%.
### 11. Derangement in the types of vatham

<table>
<thead>
<tr>
<th>Classification of Vatham</th>
<th>No. of Cases (out of 50)</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piranan</td>
<td>20</td>
<td>40</td>
</tr>
<tr>
<td>Abanan</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Viyanan</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Uthanan</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Samanan</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Nagan</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Koorman</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Kirukaram</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Revathatham</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dananjeyam</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Inference**

According to classification Vatham derangement of Viyanan was 100%, Samanan was 100% Piranan was 40%, Uthanan was deranged in 30% Nagan 8% Koorman 4%.
12. Derangement in Types of Pitham

<table>
<thead>
<tr>
<th>Types of Pitham</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analapitham</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ranjagam</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Saathagam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Prasagam</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Alosagam</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

Inference

According to Pitham Prasayam (Oollolither 100%) was affected, Ranjagam (Varma eri) was affected in 20% Alosagam (Nokanal) was affected in 10%.
13. Derangement Types of Kabam

<table>
<thead>
<tr>
<th>Types of Kabam</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avalambagam</td>
<td>20</td>
<td>40</td>
</tr>
<tr>
<td>Kilethagam</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Pothagam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tharpagam</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Santhigam</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Inference
According to Kabam Avalambagam was deranged in 40%, Kilathagam was affected in 6% Pothagam was affected in 16% Pothagam was affected in 16% of cases.
14. Ezhu Udal Kattugal reference

<table>
<thead>
<tr>
<th>Udal Kattugal</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saaram</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Senneer</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Oon</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Kozhuppu</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Enbu</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Moolai</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sukkilam/Suronitham</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Saaram was affected in 100% of cases Senneer was affected in 100% of cases Oon was affected in 100% cases.
### 15 Enn Vagai Thervugal Reference

<table>
<thead>
<tr>
<th>Enn Vagai Thervugal</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naa</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Niram</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Mozhi</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Vizhi</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Parisam</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Malam</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Moothiram</td>
<td>10</td>
<td>20</td>
</tr>
</tbody>
</table>

**Inference**

Niram was affected in 100% of cases and Malam was affected in 30%, Moothiram 20% Naadi 20%, Sparisam 12% Vizhi 10% and Mozhi 6% of cases.
16. Distribution of nadi among the patient with venpadai

<table>
<thead>
<tr>
<th>Nadi</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vali Azhal</td>
<td>10</td>
<td>20%</td>
</tr>
<tr>
<td>Vali Ayyam</td>
<td>3</td>
<td>6%</td>
</tr>
<tr>
<td>Azhal Vali</td>
<td>20</td>
<td>40%</td>
</tr>
<tr>
<td>Azhal Ayyam</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Iyyam Vali</td>
<td>14</td>
<td>28%</td>
</tr>
<tr>
<td>Iyyam Azhal</td>
<td>3</td>
<td>6%</td>
</tr>
</tbody>
</table>

**Distribution of Naadi**

- Vali Azhal: 20%
- Vali Ayyam: 6%
- Azhal Vali: 40%
- Azhal Ayyam: 0%
- Iyyam Vali: 28%
- Iyyam Azhal: 6%

Inference

Azhal vali was observed in 40%
Azhal ayyam was observed in 20%
Vali Azhal 20%, Ayam vali 28%, Ayam azhal 6%, Vli ayam 6%
### 17. Neikuri Reference

<table>
<thead>
<tr>
<th>Character of Urine</th>
<th>Neikuri Reference</th>
<th>No. of Cases out of 50</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spreads Like Snake</td>
<td>Vatha Neer</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Spreads Like Ring</td>
<td>Pitha Neer</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Spreads Like Pearl</td>
<td>Kaba Neer</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Spreads Like Snake and Ring</td>
<td>Thontha Neer</td>
<td>34</td>
<td>68</td>
</tr>
</tbody>
</table>

#### Inference
Thontha Neer affected in 68% of cases Pitha neer 14% vatha neer 10% and Kaba neer 8%.
18. Results

<table>
<thead>
<tr>
<th>Results</th>
<th>No. of Cases out of 50</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good Relief</td>
<td>26</td>
<td>52%</td>
</tr>
<tr>
<td>Moderate Relief</td>
<td>20</td>
<td>40%</td>
</tr>
<tr>
<td>Mild Relief</td>
<td>4</td>
<td>8%</td>
</tr>
</tbody>
</table>

Inference

About 52% of cases had good relief and 40% had moderate relief and 8% mild relief.
6. DISCUSSION

According to Siddha literature Venpadai is one of the eighteen types of kuttam. The clinical entity of venpadai is more or less similar to that of vitiligo in modern medicine. It is an acquired idiopathic depigmentary condition and is characterized by completely depigmented macules and patches of varying sizes and shapes. Beside loss of colour there is no other structural change.

Anatomy and physiology of skin and aetiology, clinical feature of the disease etc., are discussed. Author collected these largely from kuzhanthai Maruthuvam and Siddha methods of diagnosis was carried out. Before and after the course of treatment patients were subjected to laboratory investigations and photographs were taken.

Laboratory Investigation:
- Blood – TC, DC, ESR, Hb, Urea and Serum cholesterol
- Urine – Albumin, Sugar and deposit
- Neerkkuri and Neikkuri

Aetiology

The causative factors of the disease can be found out from the history given by the patients, their diet, habits, occupation, mental stress and other signs and symptoms. According to the Siddha and modern science the aetiology of the disease has been arrived at as follows:

- Nutritional deficiency of copper, protein, vitamins in diet
- Gastro-intestinal problems like amoebiasis, helminthiasis, chronic diarrhoea and dysentery.
- Hereditary and autoimmune disorders.
- Thyrotoxicosis and diabetes.
- Industrial chemicals and dyes.
- Infections and toxic products, enteric fever, ill health, focal sepsis.
- Chronic irritation
- Anaemia
- Unknown aetiology
Gender Distribution:

50 patients of both the gender were selected for the dissertation study. Among the 50 cases 21 (42%) were males and 29 (58%) were females. The gender distribution was more or less equal.

Age Distribution:

23 (46%) Patients were in the age group between 5-8
11 (22%) patients were in the age group between 9-10
16 (32%) patients were in the age group between 11-12

Dietary Habits:

37 (74%) patients were non vegetarians and only 13 (26%) were vegetarian

Seasonal variation:

42 (82%) cases were admitted to trial in kar kaalam and 8 (16%) koothir kaalam.

Thinai:

12 (24%) patients belonged to kurunchi thinai,
4 (8%) patients belonged to mullai thinai,
10 (20%) patients belonged to marutham thinai,
24 (48%) patients belonged to Neithal thinai,

In Siddha literature the Marutham is mentioned as disease free land among the five lands. Because of various environmental changes in the life style, venpadai occurs irrespective of the land, Maximum patients came from in and around Chennai which belongs to Neithal thinai.

Reference of Mukkutram

1. Vaatham
   piranan was affected in 20 (40%),
   Abaanan was affected in 15 (30%),
   Uthanan was affected in 15 (30%),
   viyanan was affected in 50 (100%),
   samanann was affected in 50 (100%),
nagan was affected in 4 (8%),
koorman was affected in 2 (4%)
koorman in 3 (7.5 %)

2. Pitham
Ranjagam was affected in 10(20%) patients,
praasakam was affected in 50(100 %) patients,
alosagam was affected in 3(6%) patients
Praasakam is responsible for the complexion of the skin. Due to defect in praasakam
skin colour is changed in Venpadai.

3. Kabam
Avalambagam was affected 40 % Kilethagam was affected 6%

**Udal Kattugal**
Saaram, Senneer and oon which are responsible for the colour of the skin were affected
in all the 50 (100 %) cases.

**Ennvagai thervugal**
Naa was affected in 10(20%) cases
Niram was affected in all the (100 %) cases because in venpadai, the colour of Skin
changed into white.
Mozhi was affected in 3(6%) cases.
Vizhi was affected in 5 (10%) cases.
Sparisam was affected in 6 (12%) cases
Malam was affected in 15 (30 %) cases.
Moothiram was affected 15 (30%) cases

**Management:**
In Siddha System before starting the treatment it is necessary to bring the three thadu
into equilibrium. To bring down the vitiated vaatham, Viressanam (purgation) with
Poovarasampattai ennai –5 ml with hotwater was given in the early morning.
Drugs

I. Lagu seenachoora choornam – 1gm (BD) with hotwater. (Internal)

II. Venpadai patru(venkutta lepanam) – with cows urine (External)

Patients were instructed to take the medicines regularly and apply the patru twice a day and to expose the affected parts to sunlight. Diet restrictions were strictly imposed and followed.

The main reasons for choosing the drugs mentioned above as trial drugs are:

Venpadai is the disease that affects not only the skin (body) but also the mind. These patients face a lot of problems not only in their own families but also in the society. It leads them to mental depression and stress. It aggravates the clinical condition. The treatment already available for venpadai is a prolonged one and does not fully cure the condition. Resorting to plastic surgery proves costly and it is not affordable to all the patients. Moreover the relapse of the disease at the site of surgery is common.

The trial drug Lagu seenachoora choornam is the best combination of herbs. Each content of the Lagu seenachoora choornam is specifically given for skin diseases, as described in pothu gunam.
7. SUMMARY

- Venpadai has been chosen for the dissertation work by the author.
- Various literatures dealing with Venpadai have been collected from Siddha and modern text books.
- Preclinical analyses in toxicological and biochemical aspects were conducted for the trial drug *Lagu seenachoora choornam*. 50 patients of both sex and in age group between 5 to 12 were selected for the study.
- 20 cases were treated in inpatient ward at least for 30 days and followed up in the outpatient department after discharge. 30 cases were treated in the outpatient department for 48 days.
- All the details about the study and the drugs were informed to the patients and consent forms obtained from them. Before starting the treatment, the blood samples of the selected patients were subjected to investigation and photographs of the lesions were taken.
- A day before starting the treatment purgation was given by administering sithathi ennai 1ml with hot water in the early morning to bring the thridosha to equilibrium.
- From the second day onwards *Lagu seenachoora choornam* 1 gram BD along with *Venpadai pattu (venkutta lepanam)* – with cows urine for external use were given to the patients.
- Diet restrictions were strictly followed during the treatment period. Every 8th day the patients were assessed for clinical improvement and adverse effects. On the 48th day the laboratory investigations and photographs were repeated. The improvement was assessed.
- During the course of treatment there were no adverse effects or unwanted drug reactions like itching, abdominal discomfort, and nausea.
8. CONCLUSION

- Venpadai may occur due to various causes and it leads to mental stress and strain. Hence it is one of the Psychosomatic disorders. When the trial drug Lagu seena chooranam (Internal) with Venpadai pattru (venkutta lepanam - External) were administered to the venpadai patients, it showed improvement in varying degrees in all the cases.

- No adverse effects were noticed during treatment period. The ingredients are lagu seena chooranam are of plants origin, easily available and harmless to children. The cost comparatively very low the medicine has many properties to control the signs and symptoms of venpadai.

- Clinical results were found to be having good relief in 52% of cases, moderate relief 40% of cases, mild relief 8% of cases.

- Because of the encouraging result clinically the study may be undertaken with same drug for prolonged period of time in the large number of cases and it may throw new lights for the treatment of venpadai (vitiligo).

- The trial drug ‘lagu seena chooranam’ denoted in Agathiyar vaithya pillaitamil and venpadai pattru (External) denoted in pathartha guna villakam text as a common effective drug for venpadai and through this trial the effectiveness of lagu seena chooranam (Internal) and venpadai pattru (External) was conferred and re-established by the author.
1. PREPARATION AND PROPERTIES OF TRIAL DRUG

Trail Medicine I (Internal)

Name of the Medicine: இலங்கை செய்தி

Reference: அக்கிரமம் லாகைமை பின்னணி குறிப்பிட்டு

பகுதி- 72

சேர்ந்து சுருக்கத்தி

குறிப்பிட்டு மாற்று பாலன் 7½ அறவை

சிரசமான தவர் பாலன் 7½ அறவை

சிரசமான பெறுமான் 7½ அறவை

சிரசமான குழந்தை 7½ அறவை

லகை குழந்தை 7½ அறவை

எல்லாவை குழந்தைவழி 7½ அறவை

எல்லாவை குழந்தைவழி 7½ அறவை

எல்லாவை குழந்தைவழி 7½ அறவை

எல்லாவை குழந்தைவழி 7½ அறவை

சேர்மான், சதுர்க்கற்ற வருமாறு, வாரத்தில் முழுவதும் குறிப்பிட்டு காரணமாக நம்பக்கொள்ள விளங்கும்.

முறை

¼ - ½ அறவை வருமாறு

சேர்ந்து சுருக்கத்தி

னிதியை பெறலாம்
Trial Medicine II (External)

Name of the Medicine: அரையாளாம்புருந்து (அரையாளாம்புருந்து)

Reference : Patharthaguana vilakam Page No.234

விளையாட்டுகள்

காலம் சுமார் 40 கி
அதிகம் 10 கி
பெற்றியே வீசவெளிய அனுப்ப

சொவ்வலகு

அரையாளாவுக் கருத்து பலகு
தவாவுக்காக சாரணித்து வாழு நிற்கலாமலே மற்றும் விளையாட்டே குழு - பகுதி, அவற்றின்
சுற்றுக்கோடு நீக்கும்போது தவிர்த்து ரோஜே அளிக்கவும் பார்வை விளையாட்டே குழு
கத்திக்காதிரி மற்றும் பற்றிய அனுப்ப குறிப்பிட்டு.

சொவ்வலகு

தவாவுக்காக சாரணித்து வாழு நிற்கலாமலே தவிர்த்து ரோஜே விளையாட்டே குழு
நோய்க். வெளியான அனுப்ப

அனுப்ப வீசவெளிய அனுப்ப

தவாவுக்காக சாரணித்து
2. REVIEW LITERATURE OF TRIAL DRUG

Bot Name : Smilax China
Family : Liliaceae

 Constituents:

- Root contains fat, sugar, glucoside, colouring matter, saponin (Sarasasapogenin) gum and starch. Including psoriasis, rheumatoid artaritis, gout, enteritis, urinary tract Infections, smilax china – skin ulcers.

The root is

- Alterative
- Anti scropulatic
- Carminative
- Depurative
- Diaphoretic
- Diunetic, Tonic
Bot Name : Azima tetracantha
Family : Salvadoraceae

Azima Tetra Cantha Linn:

High concentrations of N – methoxy – 3 – indolymethyl – glucosinolate, a common glucosinate were found in the roots and seeds of a Tetra contha.

The seeds of Azima tetracantha contained a complex mixture of 267 lavonoids predominantly as glycosides.

Alkaloids :

Azimene, Zalarpine, carpine,
Bot Name : Indigofera aspalathoides
Family : Fabaceae

Leaves, flowers and tender shoots – coding, demulcent
Employed in decotion in leprosy and cancerous affections.
Indigofera aspalathoides works wonder in developing sustainable immunity in human body.

- Anti inflammatony agent
- Anti tuberulous agent
- Anti bacterial
- In activator of succinate dehydrogenase.
Bot Name : Eclipta prostrata
Family : Asteraceae (Compositate)

Constituents :

- A large amount of resin Alkarloid ecliptine
- Tonic and deobstruent
- Use in neputic and spleen encouragement
- Skin uleers
- Source of black stain
- Root emitic and purgative
**Bot Name**: Enicostemma axillare

**Uses**
- Dyspepsia, Flatulence
- Colic, Helminthiasis
- Abdominal ulcers, Hernia
- Snelling, alycosaria
- Leprosy, skin disease, pruritis
- Intermittent fever
- Local applications in snake poisons.

**Phytochemicals**
- Ophelic acid, tanniens, gentiamine, erythodentawin, gentioorunine, apigenin
Bot Name: Clerodendrum inerme

Family: Verbenaceae

Description:
- Constituents:
  - Leaves contain a bitter principle
  - Resin
  - Gum
  - A brown colouring matter &
  - Ash containing a large amount of sodium chloride

This plant possesses therefore ecbolic, hypertensine, and laxative effects. The plant proved to be non-toxic since it does not produce ill effects with doses as loose as 8 g / kg body weight of the powdered plant.
**Bot Name**: Capparis Sepiaria  
**Family**: Capparideae

<table>
<thead>
<tr>
<th>Scientific Name</th>
<th>Tamil Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capparis Sepiaria</td>
<td>கப்பாரிஸ் சேபியாரியா</td>
</tr>
<tr>
<td>Capparideae</td>
<td>கப்பாரி஡ேயேயே</td>
</tr>
</tbody>
</table>

**Constituents**

Bark contains a neutral bitter principle resembling senegen  
Flower buds: Contain capric acid & a glycoside which yield on boiling with sulphuric acid isodulite and a colouring matter similar to quercertin.  

**Alkaloids**

A new spermidine alkaloid capparidisin isolated from root bark and its structure capprisinine.  
B capparisine carotene isolated from leaves.
Bot Name: Aristolochia Indica Linn
Family: Aristolochiaceae

Phytochemicals
- Arstolochin, tannin, isoaristolochic acid
- allantonin

Uses
- Root used in fever and leucodema
- Respiratory parlysis
Bot Name : Gymnema Sylvestre (ectz)
Family : Menispermeceae
Part used : Root

Uses

- Fever
- Anti helminthatic effect
- Cough
- Bronchial asthma
- Skin diseases

Phytochemicals
Gymenmic acid, leaves contain anthroguione compound.
Venpadaipatru (External)

Bot Name : Vernonia anthelmintica
Family : Asteraceae

Constituents :
- Seeds contains resins, and alkaloid known as vernomine, an oil and ash amounting to about 7 P.C. of the dry material free from manganese.
- Avenasterol, renosterol, essential oil, resins, myristic, palmitic, stearic, oleic, lonoleic, vernolic acid.
CHEMICAL ASPECT OF (ORPIMENT) THALAGAM

Orpiment Is A Rare Mineral.

Name : Orpiment

Synonyms : Arsenic trisulphide, Yellow arsenic sulfide, Natural arsenic sulphides, Arrhenicum, Operment, Orpiment, Yellow Arsenic

Name origin : The name is derived from the Latin “auripigmentium” (“aurum”-gold and pigmentum) relating to the minerals “goldish color”. Orpiment is also known as “King’s Yellow”, “Chinese Yellow” and “Yellow Orpiment”.

Colour : Lemon –yellow, Orange Orange-brownish, Deep orange-colored Orpiment crystal aggregate.
**Chemical Formula:** A

**Class:** Sulfides and sulfosalts

**Chemical:** Arsenic Sulfide

**Elements:** As, S

**Composition:**
- Arsenic: 60.90% (As)
- Sulfur: 39.10% (S)

**Common Minerals:** Hg, Ge, Sb

**Associated Minerals:** Usually forms with realgar. In fact, the two minerals are almost always together. Other minerals are stivnite, pyrite, sphalerite, Calcite, antimony ores, barite, and gypsum.

**Hardness:** 1.5-2.0

**Specific Gravity:** 3.4-3.5

**Density:** 3.49-3.56, Average = 3.52

**Melting point:** 300°C

**Boiling point:** 707°C

---

**Chemical description of cow urine as per modern concepts and cure of diseases accordingly:**
### Table - Chemical contents of cow urine and cure of diseases as per them

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Name of chemical</th>
<th>Effect of chemical on diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Nitrogen N2 ,NH2</td>
<td>Removes blood abnormalities and toxins, Natural stimulant of urinary track, activates kidneys and it is diuretic.</td>
</tr>
<tr>
<td>3.</td>
<td>Copper Cu</td>
<td>Controls built up of unwanted fats</td>
</tr>
<tr>
<td>4.</td>
<td>Iron Fe</td>
<td>Maintains balance and helps in production of red blood cells &amp; haemoglobin. Stabilises working power.</td>
</tr>
<tr>
<td>5.</td>
<td>Urea CO(NH2)2</td>
<td>Affects urine formation and removal. Germicidal.</td>
</tr>
<tr>
<td>7.</td>
<td>Calcium Ca</td>
<td>Blood purifier, bone strengthener, germicidal</td>
</tr>
<tr>
<td>8.</td>
<td>Salt NaCl</td>
<td>Decreases acidic contents of blood, germicidal</td>
</tr>
<tr>
<td>9.</td>
<td>Vitamins A,B,C,D,E</td>
<td>Vitamin B is active ingredient for energetic life and saves from nervousness and thirst, strengthens bones and reproductive ingredient for energetic life and saves from nervousness and thirst, strengthens bones and reproductive power.</td>
</tr>
<tr>
<td>10.</td>
<td>Enzymes</td>
<td>Make healthy digestive juices, increase immunity</td>
</tr>
</tbody>
</table>

The invention relates to an absolutely novel use of cow urine distillate as activity enhancer and availability facilitator for bioactive molecules including anti-infective and anti-cancer agents. The molecules which express any activity in form of either inhibiting or promoting a biological function have been referred in this invention as bioactive molecule e.g. antibiotics, drugs, nutraceuticals, cardiovascular, hepatoprotective, neuro-tonics etc. The present invention has direct implication in drastically reducing the dosage of antibiotics, drugs and anti-cancer agent while increasing the efficiency of absorption of bioactive molecules.
3. **ANTIMICROBIAL ACTIVITY OF VENPADAI PATTRU**

Venkutta Lapanam

The anti microbial activity of different extracts were studied by disc diffusion method against the following organisms.

<table>
<thead>
<tr>
<th>Media used</th>
<th>Nutrient Agar.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ingredients</td>
<td>gms/litre.</td>
</tr>
<tr>
<td>Peptone</td>
<td>10.00</td>
</tr>
<tr>
<td>Beet extract</td>
<td>10.00</td>
</tr>
<tr>
<td>Sodium chloride</td>
<td>5.00</td>
</tr>
<tr>
<td>Agar</td>
<td>12.00</td>
</tr>
</tbody>
</table>

**Disc diffusion method:**

A suspension of staphylococcus aureus was added to sterile nutrient agar at 45’c the mixture was transferred to sterile petridishes and allowed to sterile discs 5mm in diameter (made from what man filter paper previously sterilized in u-v- lamp dipped in solution of the different extracts standard on the surface of agar plates.

Leave the plates were incubated at 37’c for 24 hours and observed for antibacterial activity the diameters of the zones of in inhibition Was measured for the plates in which the zone of inhibition was observed.

The average area of zone of inhibition was calculated and compared with that of the standard.

A similar procedure was carried out for the study of antibacterial activity of this sampler against mentioned organisms.
### Antimicrobial Activity Of Venpadai Pattru

<table>
<thead>
<tr>
<th>S. No</th>
<th>Organisms gram +ve.</th>
<th>Std</th>
<th>50</th>
<th>100</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Staphylococcs aureus</td>
<td>32</td>
<td>16</td>
<td>21</td>
<td>26</td>
</tr>
<tr>
<td>2</td>
<td>Staphylococcus epidermidis</td>
<td>31</td>
<td>15</td>
<td>19</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>Bacillus cereus</td>
<td>33</td>
<td>18</td>
<td>21</td>
<td>28</td>
</tr>
<tr>
<td>4</td>
<td>Bacillus subtilis</td>
<td>32</td>
<td>17</td>
<td>22</td>
<td>21</td>
</tr>
<tr>
<td>5</td>
<td>Micrococcus luteus</td>
<td>34</td>
<td>20</td>
<td>24</td>
<td>28</td>
</tr>
<tr>
<td>6</td>
<td>Streptococcus mutans</td>
<td>32</td>
<td>21</td>
<td>25</td>
<td>28</td>
</tr>
</tbody>
</table>

(Gram –ve)

<table>
<thead>
<tr>
<th></th>
<th>Organisms</th>
<th>Std</th>
<th>50</th>
<th>100</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Escherichia coli</td>
<td>33</td>
<td>15</td>
<td>19</td>
<td>24</td>
</tr>
<tr>
<td>2</td>
<td>Klebsiella pneumonia</td>
<td>32</td>
<td>15</td>
<td>18</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>Pseudomonas aeruginosa</td>
<td>32</td>
<td>14</td>
<td>18</td>
<td>22</td>
</tr>
</tbody>
</table>

Bacterial:

Standard used:

Ciproflaxacin hcl 50 mcg/disc.
4. ANTIFUNGAL ACTIVITY OF VENPADAI PATTRU
(VENKUTTAL LAPANAM)

The anti fungal activity of different extracts were studied by disc diffusion method against the following organisms.

Media used:
Sabouraud dextrose agar medium gm/ litre.

Mycological peptone 10.0
Dextrose 40.0
Agar 15.0

DISC DIFFUSION METHOD:

A suspension of A. Niger was added to sterile sabouraud dextrose agar at 45. the mixture was transferred to sterile petridishes and allowed to sterile petridishes and allowed to solidity, sterile disc 5mm in diameter (made from whatman filter paper previously sterilized in u-v- lamp) dipped in solution of the different concentrations, standards and a blank were placed on the surface of agar plates

Leave the plates standing for 4 hours at room pre-incubation diffusion o minimize the effects of variations in time between the application of the different solutions, then the plates were incubated at 37°C for antibacterial activity the diameter of the zones of inhibition was observed.

The average area of zone of inhibition was calculated and compared with that of the standards.

A similar procedure was carried out for the study of anti fungal of extract against aspergillus fumigates & candida albicans
# ANTIFUNGAL ACTIVITY OF VENPADAIPATTRU (VENKUTALAPANAM)

**Fungus**

<table>
<thead>
<tr>
<th></th>
<th>Fungus Type</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Aspergillus niger</td>
<td>29</td>
<td>16</td>
<td>18</td>
</tr>
<tr>
<td>2</td>
<td>Aspergillus fumigates</td>
<td>29</td>
<td>17</td>
<td>19</td>
</tr>
<tr>
<td>3</td>
<td>Candida albicans</td>
<td>33</td>
<td>18</td>
<td>22</td>
</tr>
</tbody>
</table>

**Fungus:**

**Standard used:**

- Ketoconazole → 50 mcg/disc

**Sample concentration**

- 1 gm → 100ml solvent water 50,100,150 ul/disc
5. BIO CHEMICAL ANALYSIS OF LAGU SEENA CHOORANAM

Physical properties:

Loss on drying:

Five grams of Lagu seena chooranam is heated in a hot oven at 40°C to constant weight. The percentage of loss of weight was calculated.

Determination of ash value:

Weigh accurately 2-3 grams of Lagu seena chooranam in tarred platinum or silica dish and incinerate at a temperature not exceeding 450°C until free from carbon, cool and weigh. Calculate the percentage of ash with reference to the air dried drug.

Acid Insoluble ash

Boil the ash for 5 minutes with 25 ml of 1:1 dilute HCL. Collected the insoluble matter in Gooch – crucible on an ash less filter paper, wash with hot water and ignite, cool in a dessicator and weigh. Calculate the percentage of acid insoluble ash with reference to the air dried drug.

Water soluble ash:

To the Gooch crucible containing the total ash, add 25 ml of water and boil for 5 minutes. Collect the insoluble matter in a sintered glass crucible or on ash less filter paper. Wash with hot water and ignite in a crucible for 15 minutes at a temperature not exceeding 450°C. Subtract the weight of the insoluble matter from the weight of the ash; the difference of weight represents the water soluble ash. Calculate the percentage of water soluble ash with reference to the air dried drug.
Alkalinity of water soluble ash:

Five grams of Lagu seena chooranam converted to ash, boiled with water, filtered. Filtrate was titrated against 0.1N of HCl using phenolphthalein as an indicator.

$$\text{Alkalinity of water soluble ash} = \frac{X \times \text{of acid}}{0.1 \times W}$$

$X = \text{Titre value.}$

$W = \text{Weight of the material taken.}$

Alkalinity is given as ml of 0.1N of HCL equated to 1 gm.

PH:

Five grams of Lagu seena chooranam is weighed accurately and placed in clear 100 ml beaker. Then 50 ml of distilled water is added to it and dissolved well. Wait for 30 minutes and then apply in to pH meter at standard buffer solution of 4.0, 7.0, and 9.2.

**QUANTITATIVE ANALYSIS**

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>PARAMETER</th>
<th>RESULTS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>01.</td>
<td>Loss of drying @ 105°C</td>
<td>6.72</td>
</tr>
<tr>
<td>02.</td>
<td>Ash Value</td>
<td>12.89</td>
</tr>
<tr>
<td>03.</td>
<td>Water soluble</td>
<td>9.80</td>
</tr>
<tr>
<td>04.</td>
<td>Alkalinity as CaCO3 in water soluble ash</td>
<td>0.05</td>
</tr>
<tr>
<td>05.</td>
<td>Acid insoluble ash</td>
<td>5.25</td>
</tr>
<tr>
<td>06.</td>
<td>PH at 10% aqueous solution</td>
<td>5.68</td>
</tr>
</tbody>
</table>
### QUALITATIVE ANALYSIS

<table>
<thead>
<tr>
<th>SL.NO</th>
<th>EXPERIMENT</th>
<th>OBSERVATION</th>
<th>INFERENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Appearance of the sample</td>
<td>Brown in colour</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Solubility:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>a. A little of the sample is</td>
<td>Completely soluble</td>
<td>Absence of Silicate</td>
</tr>
<tr>
<td></td>
<td>shaken well with distilled</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>water.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>b. A little of the sample is</td>
<td>Completely soluble</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Shaken well with con. Hcl</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Con. H₂SO₄.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Action of Heat:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>A small amount of the sample is</td>
<td>White fumes not</td>
<td>Presence of Nitrate.</td>
</tr>
<tr>
<td></td>
<td>taken in a dry test tube and</td>
<td>evolved</td>
<td></td>
</tr>
<tr>
<td></td>
<td>heated gartly at first and then</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Strong.</td>
<td>Brown fumes evolved</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Flame Test:</td>
<td>White flame is</td>
<td>Absence of Copper.</td>
</tr>
<tr>
<td></td>
<td>A small amount of the sample is</td>
<td>appeared</td>
<td></td>
</tr>
<tr>
<td></td>
<td>made into a paste with con. Hcl</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>in a watch glass and introduced</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>into non-luminous part of the</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bunsen flame.</td>
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<td>5</td>
<td>Ash Test:</td>
<td>No Yellow colour</td>
<td>Absence of Sodium.</td>
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<tr>
<td></td>
<td>A filter paper is soaked into a</td>
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<tr>
<td></td>
<td>mixture of sample and cobalt</td>
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<tr>
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<td>nitrate solution and introduced</td>
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<td>into the Bunsen flame and</td>
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</table>
**Preparation of the extract**

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

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<tbody>
<tr>
<td>TEST FOR ACID RADICALS</td>
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</tr>
<tr>
<td>1.</td>
<td><strong>Test For Sulphate:</strong>&lt;br&gt;a. 2 ml of the above prepared extract is taken in a test tube to this added 2ml of 4% ammonium oxalate solution</td>
<td>Cloudy appearance present</td>
<td><strong>Presence of Sulphate.</strong></td>
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<tr>
<td></td>
<td>b. 2 ml of the above prepared extract is added with 2 ml of dil- Hcl is added until the effervescence ceases off. Then 2ml of Barium chloride solution is added</td>
<td>A white precipitate insoluble in con. Hcl is obtained</td>
<td>Sulphate is confirmed.</td>
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<td>2.</td>
<td><strong>Test For Chloride:</strong></td>
<td>No Cloudy appearance present</td>
<td>Absence of Chloride.</td>
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<tr>
<td></td>
<td>2 ml of the above prepared extract is added with dil. HNO₃ till the effervescence ceases. Then 2 ml of silver nitrate solution is added.</td>
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<td>Procedure</td>
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<td>3.</td>
<td>Test For Phosphate:</td>
<td>2 ml of the extract is treated with 2 ml of ammonium molybdate solution and 2 ml of con. HNO₃</td>
<td>No cloudy yellow appearance</td>
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<td>4.</td>
<td>Test For Carbonate:</td>
<td>2 ml of the extract is treated with 2 ml magnesium sulphate solution</td>
<td>No cloudy appearance</td>
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<td>5.</td>
<td>Test For Nitrate:</td>
<td>1 gm of the substance is heated with copper turnings and concentrated H₂SO₄ and viewed the test tube vertically down.</td>
<td>Brown gas is evolved</td>
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<td>6.</td>
<td>Test For Sulphide:</td>
<td>1 gm of the substance is treated with 2 ml of con. Hcl.</td>
<td>No Rotten egg smelling gas evolved</td>
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<tr>
<td>7.</td>
<td>Test For Fluoride &amp; Oxalate</td>
<td>2 ml of the Extract Is Added With 2ml of Acetic Acid and 2 ml calcium Chloride solution and heated.</td>
<td>No Cloudy appearance.</td>
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<tr>
<td>8.</td>
<td>Test For Nitrite</td>
<td>3 drops of extract is placed on a filter paper, n that 2 drops of acetic Acid and 2 drops of benzidine solution is placed.</td>
<td>No characteristic changes</td>
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</table>
9. **Test For Borate:**
   2 pinches of the substance is made into paste by using sulphuric acid and alcohol (95%) and introduced into the blue flame.  
   | Bluish green colour flame not appeared.. | Absence of borate. |

### II. TEST FOR BASIC RADICALS

1. **Test For Lead:**
   2 ml of the extract is added with 2 ml of potassium iodide solution.  
   | No Yellow precipitate is obtained | Absence of Lead. |

2. **Test for Copper:**
   a. one pinch of substance is made into paste with con. Hcl in a watch glass and introduced into the non-luminous part of the flame.  
   b. 2 ml of extract is added with excess of ammonia solution.  
   | No Blue colour flame precipitate | Absence of Copper. |

3. **Test For Aluminium:**
   Take the 2 ml of the extract sodium hydroxide is added in drops to excess.  
   | No characteristic changes | Absence of Aluminium. |

4. **Test For Iron: (Ferrous)**
   To the 2 ml of extract 2 ml ammonium thiocyanate solution and 2 ml of con. HNO₃ is added  
<p>| Blood red colour Appearance | Presence of Iron. |</p>
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<th><strong>Test For Calcium:</strong></th>
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<tr>
<td></td>
<td>2ml of the extract is added with 2ml of 4% ammonium oxalate Solution.</td>
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<td>To 2ml of extract sodium hydroxide solution is added in drops to excess.</td>
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<th><strong>Test For Ammonium:</strong></th>
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<td>To 2ml of extract few ml of Nessler's reagent and excess of sodium hydroxide solution are added.</td>
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<th><strong>Test For Potassium:</strong></th>
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<tr>
<td></td>
<td>A pinch of substance is treated with 2ml of sodium nitrite solution and then treated with 2ml of cobalt nitrate in 30% glacial acetic acid.</td>
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</tbody>
</table>
### Test For Sodium:
2 pinches of the substance is made into paste by using HCL and introduced into the blue flame of Bunsen burner. 
No Yellow colour Flame appeared. 
Absence of Sodium.

### Test For Mercury:
2m1 of the extract is treated with 2ml of sodium hydroxide solution. 
No Yellow precipitate is obtained 
Absence of Mercury.

### Test For Arsenic:
2m1 of the extract is treated with 2m1 of sodium hydroxide solution. 
No brownish red Precipitate is obtained 
Absence of Arsenic.

### III. MISCELLANEOUS

### Test for Starch:
2ml of extract is treated with weak iodine solution. 
No blue colour developed 
Absence of Strarch.

### Test For Reducing Sugar:
5. ml of Benedict's qualitative solution is taken in a test tube and 
No colour Changes 
Absence of Reducing sugar.
allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boil it for 2 minutes. The colour changes are noted.

3. **Test For The Alkaloids:**
   a. 2ml of the extract is treated with 2ml of potassium iodide solution.
      No Red colour developed
      Absence of Alkaloid.
   b. 2ml of extract is treated with 2ml of picric acid.
      Trace Yellow colour developed
      Trace of Alkaloid present.
   c. 2ml of the extract is treated with 2ml of phosphotungstic acid.
      No white precipitate developed
      Absence of Alkaloid

4. **Test for Tannic Acid:**
   2ml of extract is treated with 2ml of ferric chloride solution.
   No black precipitate obtained
   Absence of Tannic acid.

5. **Test for Unsaturated Compound:**
   To the 2ml of extract 2ml of Potassium Permanganate solution is added.
   Potassium Permanganate is not decolourised
   Absence of Unsaturated Compound.
6. **Test For Amino Acid:**

2 drops of the extract is placed on a filter paper and dried well and 2 ml of biuret reagent is added.

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<th>Phenomena</th>
<th>Result</th>
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<tbody>
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<td>No Violet colour developed</td>
<td>Absence of Amino acids.</td>
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7. **Test For type of Compound:**

2ml of the extract is treated with 2 ml of ferric chloride solution.

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<th>Result</th>
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<tbody>
<tr>
<td>No Green colour developed</td>
<td>Absence of oxy quinole epinephrine and pyro catechol.</td>
</tr>
<tr>
<td>No Red colour developed</td>
<td>Anti pyrine, Aliphatic amino acids and Meconic acid are absent.</td>
</tr>
<tr>
<td>No Violet colour developed</td>
<td>Apomorphine, Salicylate and Resorcinol are absent.</td>
</tr>
<tr>
<td>No blue colour developed</td>
<td>Morphine, Phenol cresol and hydro quinone are absent</td>
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**RESULT:**

Iron, Calcium, Sulphate, Nitrate, Traces of Alkaloids, Compounds are present in **lagu seena chooranam.**
6. TOXICOLOGICAL EVALUATION

ACUTE ORAL TOXICITY STUDY OF LAGU SEENA CHOORANAM

Experimental Animals

Adult female wistar albino rats (150-230 gm) were obtained from the animal housing facility of King Institute, Guindy, Chennai. The animals were maintained in a well – ventilated room with 12:12 hour light/dark cycle in polypropylene cages. Standard pellet feed and tap water were provided ad libitum throughout experimentation period. Animals were acclimatized to laboratory conditions one week prior to initiation of experiments. The project has got the ethical committee clearance from IAEC (Institutional Animal Ethics Committee) of CPCSEA (Committee for the Purpose of Control and Supervision of Experiments on Animals).

Acute oral toxicity study (Ecobichnon, 1997)

The procedure was followed by using OECD guidelines (Organization of Economic Cooperation Development)-423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 animals of a single sex per step. Depending on the mortality and/or moribund status of the animals, on the average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. This procedure results in use of minimal number of animals while allowing for acceptable data based scientific conclusion. The method used defined doses (5, 50, 300, 2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which cause acute toxicity.

Preparation of Drug for administration to animals:

Lagu seena chooranam suspended in 1% sodium carboxyl methyl cellulose solution was used for the experimental studies.
Experimental Procedure:

Female wistar rats weighing about 150-200 g were used for the study. The starting dose level used was 2000 mg/kg p.o. Dose volume was administered 0.1 ml/ 10 gm body weight to the rat which were fasted over night with water ad libitum. Food was withheld for a further 3-4 hours after administration of Lagu seena chooranam and observed for signs of toxicity. Body weight of the rats before and after termination were noted and any changes in skin and fur, eyes and mucous membranes and also respiratory, circulatory, autonomic and central nervous system and somatomotor activity and behavior pattern were observed, and also sign of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma were noted. The onset of toxicity and signs of toxicity also noted.

RESULT

The Acute oral toxicity study was done according to the OECD guidelines 423(Acute toxic classic method ) A starting dose of 2000 mg /kg  b.w.p.o of Lagu seena chooranam was administered to 3 female rats observed for 14 days .There was no considerable change in body weight before and after treatment of the experiment and no signs of toxicity were observed .When the experiments were repeated again with the same dose level 2000 mg/kg b.w.p.o of Lagu seena chooranam observed for 14 days ,no changes were observed.so the LD₅₀ cut off value mg /kg body weight was observed as X-un classified

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7. STATISTICAL ANALYSIS

Paired t test – comparison of HB before and after treatment.

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<td>11.33 + 1.01</td>
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The HB before and After Treatment is statistically highly significant. Paired t test - Treatment was used to determine the significant different.
A PILOT OPEN CLINICAL TRIAL OF *LAGU SEENA CHOORANAM* AND *VENPANDAI PATTRU* (*Venkuttalapanam*) FOR THE TREATMENT OF *VENADAI* (VITILIGO)

FORM I-SELECTION PROFORMA

| 1. OP/IP No:_________________ | 2. BED No: ______ | 3. S.No: ________________ |
| 4. NAME: _____________________ | 5. AGE: ______ Yrs | 6. GENER: _______________ |

7. DATE OF ADMISSION TO THE TRIAL ________________________________
   Parent’s/ Guardian’s/ Name

8. PARENT’S OCCUPATION ________________________________

9. INFORMATION ADDRESS: ______________________________________
   INFORMATION RELATIONSHIP ________________________________
   COMPLAINS & DURATION: ________________________________
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………

11. HISTORY OF PRESENT ILLNESS:
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………

12. PAST HISTORY:
   ………………………………………………………………………………………
   ………………………………………………………………………………………
   ………………………………………………………………………………………

13. FAMILY HISTORY:
   ………………………………………………………………………………………
   ………………………………………………………………………………………

14. SOCIAL HISTORY:
   Low socio economic (1) □□□ Middle class (2) □□□ Higher class (3) □□□

IMMUNISATION HISTORY Yes(1) □□□ No(2) □□□
**HABITS:**

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**II) GENERAL EXAMINATION:**

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<td>18. Blood Pressure (mmHg)</td>
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**III) CLINICAL EXAMINATION OF SKIN:**

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| 29. ANATOMICAL LOCATION | .................
| 30. COLOUR - Normal | Hyperpigmented | Hypopigmented |
| 31. SIZE OF THE LESION (Length cm): | ..................|
| 32. SHAPE: | Irregular | Round | Dispersed |
| 33. PRURITUS: | Present | Absent |
| 34. SWELLING: | Present | Absent |
35. ERYTHEMA:  Present      Absent

36. DEPIGMENTATION OF HAIR

               Present      Absent

37. SENSATION

               Normal     Paraesthesia  Numbness
               Painful     Burning       Pricking

38. SCALING:  Present      Absent

39. CRUSTING: Present      Absent

40. OOZING:   Present      Absent

41. MACULES:  Present      Absent

42. PAPULES:  Present      Absent

43. VESICLES: Present      Absent

44. PUSTULES: Present      Absent

45. PALPATION:

               Normal      Smooth       Rough
               Warm        Cold

IV) EXAMINATION OF VITAL ORGANS:

46. CNS          Normal (1) Abnormal (2)

47. CVS                      ......................

48. RS                      ......................

49. ABDOMEN                ......................

SIDHA ASPECTS

50. NILAM:


51. KALAIYALBU:


### UDAL IYALBU:


### GUNAM:

1. Sathuvam  2. Rasatham  3. Thamasam

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### VI) KANMENTHIRIUM / KANMAVEDAYAM

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<td><strong>KABAM:</strong></td>
<td>Normal (1)</td>
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<tr>
<td>79. Avalambagam</td>
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<tr>
<td>80. Kilethagam</td>
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<td>81. Pothagam</td>
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<td>82. Tharpagam</td>
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<tr>
<td>83. Santhegam</td>
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<td><strong>UDAL THATHUKKAL:</strong></td>
<td>Normal (1)</td>
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<tr>
<td>84. Saaram</td>
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<tr>
<td>85. Senneer</td>
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<td>86. Oon</td>
<td></td>
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<td>87. Kozhuppu</td>
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<tr>
<td>88. Eanbu</td>
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<tr>
<td>89. Moolai</td>
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<td>90. Sukkilam/</td>
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### ENVAGAI THERVUGAL:

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<tbody>
<tr>
<td>91. Naadi</td>
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<tr>
<td>92. Naa</td>
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<td></td>
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<tr>
<td>93. Niram</td>
<td></td>
<td></td>
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<tr>
<td>94. Mozhi</td>
<td></td>
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<tr>
<td>95. Vizhi</td>
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<td>96. Sparisam</td>
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### Malam:

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<tr>
<td>97. Niram</td>
<td>Yes(1)</td>
<td>No(2)</td>
</tr>
<tr>
<td>98. Nurai</td>
<td></td>
<td></td>
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<tr>
<td>99. Kirumi</td>
<td></td>
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<td>100. Kalappu</td>
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<td>101. Erugal</td>
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<td>102. Elagal</td>
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### Mootheram:

### Neerkuri:

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<tr>
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<td>104. Manam</td>
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<td>105. Edai</td>
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<td>106. Nurai</td>
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<td>107. Enjal</td>
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### Neikuri:

- Vatha neer(1) ☐
- Pitha neer(2) ☐
- Kaba neer(3) ☐

### LAB INVESTIGATION:

#### BLOOD:

<p>| | |</p>
<table>
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<tr>
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<tr>
<td>108. TC (Cells/Cu mm)</td>
<td></td>
</tr>
<tr>
<td>DC (%)</td>
<td>113.N-</td>
</tr>
<tr>
<td>Test</td>
<td>Present</td>
</tr>
<tr>
<td>--------------</td>
<td>---------</td>
</tr>
<tr>
<td>ESR (mm)</td>
<td>118.1/2 hr</td>
</tr>
<tr>
<td>Hb (gm%)</td>
<td></td>
</tr>
<tr>
<td>RBC (Milliue / Cubic)</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td></td>
</tr>
<tr>
<td>Sugar</td>
<td></td>
</tr>
<tr>
<td>Pus cells</td>
<td></td>
</tr>
<tr>
<td>Epithelial cells</td>
<td></td>
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<tr>
<td>Red cells</td>
<td></td>
</tr>
<tr>
<td>Casts/Crystal</td>
<td></td>
</tr>
<tr>
<td>Ova</td>
<td></td>
</tr>
<tr>
<td>Cyst</td>
<td></td>
</tr>
<tr>
<td>Occult blood</td>
<td></td>
</tr>
<tr>
<td>Pus cells</td>
<td></td>
</tr>
<tr>
<td>Inclusion Criteria:</td>
<td>Yes(1)</td>
</tr>
<tr>
<td>Vitiligo patient.</td>
<td></td>
</tr>
<tr>
<td>Age between 5 to 12 years.</td>
<td></td>
</tr>
<tr>
<td>Willing to be admitted as In patient in our ward for 48 days or willing to attend OPD once in 7 days for 48 days.</td>
<td></td>
</tr>
<tr>
<td>Willing to give blood specimen and photograph before and after treatment.</td>
<td></td>
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<tr>
<td>Exclusion Criteria:</td>
<td>Yes(1)</td>
</tr>
<tr>
<td>Jaundice</td>
<td></td>
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<tr>
<td>Hypopigmended patches of leprosy and burns</td>
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</tbody>
</table>
126. Connective tissues disorders

127. Cerebral palsy

128. Cardiac disease

129. Admitted to trial:  
   Yes(1)  ☐  No(2) ☐

130. If yes, a) S.No: ☐  149. IP: ☐  OP: ☐

131. Drugs issued for O.P. Patients:

   1. No. of packs: ☐

   2. Weight of pattru ☐

132. Date: ☐

133. Station: ☐  Signature of Doctor
FORM II-ASSESSMENT PROFORMA

1. OP/IP No:_________________  2. BED No:_______  3. S.No:_______________

4. NAME:__________________________

5. DATE OF ADMISSION: ___________  ___________  ___________

6. DATE OF ASSESSMENT: ___________  ___________  ___________

7. DAY OF ASSESSMENT: ___________  ___________

8. ANATOMICAL LOCATION…………………………………..

9. COLOUR   Normal   □ □ Hyperpigmented □ □ Hypopigmented □ □

10. SIZE OF THE LESION (Length cm):…………………………

11. SHAPE:    Irregular □ □ Round □ □ Dispersed □ □

12. PRURITUS:    Present □ □ Absent □ □

13. SWELLING:    Present □ □ Absent □ □

14. ERYTHEMA:    Present □ □ Absent □ □

15. DEPIGMENTATION OF HAIR

    Present □ □ Absent □ □

16. SENSATION

    Normal □ □ Paraesthesia □ □ Numbness □ □

    Painful □ □ Burning □ □ Pricking □ □

17. SCALING:    Present □ □ Absent □ □
18. CRUSTING:  Present □  Absent □  
19. OOZING:  Present □  Absent □  
20. MACULES:  Present □  Absent □  
21. PAPULES:  Present □  Absent □  
22. VESICLES:  Present □  Absent □  
23. PUSTULES:  Present □  Absent □  
24. PALPATION:  
  Normal □  Smooth □  Rough □  
  Warm □  Cold □  
25. PIGMENTATION :  Present □  Absent □  

.................................................................

26. Naadi................................................................

LAB INVESTIGATION : (Only on Day 21 and Day 48)

BLOOD:  
27. TC (Cells/Cu mm)  
28. DC (%)  
34. ESR (mm)  
35. Hb (gm %)- □  

URINE:  
41. Albumin  Present (1) □  Absent (2) □  
42. Sugar  Present (1) □  Absent (2) □  
43. Pus cells  Present (1) □  Absent (2) □  
44. Epithelial cells  Present (1) □  Absent (2) □  
45. Red cells  Present (1) □  Absent (2) □  

**MOTION:**

<table>
<thead>
<tr>
<th></th>
<th>Present (1)</th>
<th>Absent (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>47. Ova</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>48. Cyst</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>49. Occult blood-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50. Pus cells</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

51. NEERKURI:  

52. NEIKURI:  Vatha neer(1) | Pitha neer(2) | Kaba neer(3)

53. RESULT:  Cured | Improved | No change

**FOR O.P. PATIENTS:**

54. Drugs issued:
   1. No. of pack: 
   2. Weight of pattu

55. Date:
56. Station:  Signature of Doctor
NATIONAL INSTITUTE OF SIDDHA, CHENNAI-47
AYODHIDOSS PANDITHAR HOSPITAL
DEPARTMENT OF KUZHANTHAI MARUTHUVAM
A OPEN CLINICAL TRIAL OF SIDDHA DRUGS LAGA SEENA CHOORANAM
AND VENPADAI PATTRU IN THE TREATMENT OF VENPADI (VITILIGO)
CONSENT FORM

Certificate by Investigator

I certify that I have disclosed all details about the study in the terms readily understood by the patient.

Date: ___________________  Signature: ________________

Name: ___________________

Consent of Informant

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

I am aware of my right to opt out of the trial to my Son/Daughter at any time during the course of the trial without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included my Son/Daughter as a subject in the clinical trial of Lagu seena Chooranam and Venpadai pattru in the treatment of Venpadai (Vitiligo).

Date: ________________  Signature

Name: ________________

Relationship: ________________

Date: ________________  Signature of witness:

Name: ________________

Relationship: ________________
BIBLIOGRAPHY

4. K. Uthamarayan, siddha maruthuvanga surukam, Indian Medicine, Chennai/2005
20. Dr. P.A. Mohammad Iqbal, Siddha maruthuvaththil Kann maruthuvam Based on the text Agasthiyar & Nagamuni Nayana Vidhi, March 2003 Thamarai Nooolagam, Chennai-26
## IN PATIENT DEPARTMENT

<table>
<thead>
<tr>
<th>S NO.</th>
<th>Name</th>
<th>Age/Sex</th>
<th>IP NO</th>
<th>DOA</th>
<th>DOD</th>
<th>RESULT</th>
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<td>1.</td>
<td>K. Subbulakshmi</td>
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<td>K. Manjubharathi</td>
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<td>3.</td>
<td>Akash</td>
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<td>4.</td>
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<tr>
<td>6.</td>
<td>Kavitha</td>
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The patient who are treated in IP Department after having slight improvement and discharged, then continued to OP Department.
<table>
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<tr>
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<th>NAME</th>
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<th>DOA</th>
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<td>Sudha</td>
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<td>18.</td>
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<td>20.</td>
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<td>23.</td>
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<td>24.</td>
<td>Arunsai</td>
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<td>25.</td>
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<td>26.</td>
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