A STUDY ON

NEERIZHIVU

The dissertation Submitted by

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Under the Guidance of

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CERTIFICATE

This is to certify that this dissertation work on ***NEERIZHIVU*** has been carried out by **Dr.A.CHINNASAMY** during the year 2010-2013 in the Post Graduate Department of Maruthuvam, Government Siddha Medical College, Chennai- 600106 under my guidance and supervision in partial fulfillment of regulation laid by **The Tamilnadu Dr. M.G.R Medical University,Chennai** for the final **M.D(siddha) Branch I- MARUTHUVAM** examination to be held in **April 2013**.

This dissertation is a record of original work done and it has not been previously formed the basis for the award of any degree.

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INTRODUCTION

கற்றலுஞானக் கருத்தறிந்தோர்கள் கமலபதம்
பெற்றலுஞ்சித்திப் பேரின்பக் கல்வி பிறவாத முத்தி
வைத்தாலும் வெற்றி மருவியவர்பதம் வணங்கவும் யான்
சித்தாதி சித்தர் பதினெண்மர் தந்த திரவியமே.

மூலிகை மர்மம் - கண்ணுசாமி பிள்ளை ப - 1

Evergrace has its own natural system of healing and Tamil culture has very long history of unbroken tradition of healing system called siddha system of medicine.This well codified system of medicine is developed by eternal saints called “Siddhars’’ and hence it has the name as siddha system of medicine .

These spiritual scientist felt that the body is best tool for their spiritual attainment and they developed this system of medicine to preserve the same from disease, death and decay.Natural products like plants,minerals,metals and zoological products are used as medicine.They followed very methodical intricate procedures to prepare these medicine to convert them to bioavailable form. Many refractory and metabolic diseases are draft in siddha medicine since oldentime.

Neerizhivu (Diabetis mellitus) is probably the single most important metabolic disease and wildly recognized as one of the leading causes of death and disability.If affects everycell in the body and the body’s essential biochemical processes, it is major public health problem in developing countries like India.

In india alone, the prevalence of diabetes is enpected to increase from 31.7million in 2000to 79.4 million in 2030.In India, faces of paradox of families, in which the children are under weight.The prevalence of type 2 diabetes is 4-6 times higher in the urban areas as compared to rural areas.The prevelence of impaired Glucose tolerance (IGT) in the rural population is also high at 7-8%. This indicates presence of Genetic basis for Type2 diabetes in ethnic Indian population.

Risk factors for developing type 2 diabetes,peculiar to the Indian population are high familial aggregation,central obesity,insulin resistance and life style changes including non healthy food habits.Sedantory life style is one of the significant factors associated with diabetes in this population.The insidious onset of the disease and long duration of asymptomatic disease before the symptoms develop makes the prevalence of

complications quite high even at the time of diagnosis of Type2 diabetes like micro vascular disease and macrovascular diseases. Micro vascular complications like Retinopathy, Neropathy, Nephropathy and Macro vascular complications are Atherosclerosis which leads to myocardial infarction and stroke.Though many drugs are used in contemporary medicine.

The development of complications are also found to be high. The limitation of currently available oral antidiabetic agents inferms of efficacy and safely created the need for development of indigenous,natural inexpensive drugs are sought after for the prevention of disease complication and for better therapeutic effect.In siddha system of medicine many herbal drugs are indicated for Neerizhivu.disease

Serankottai (Semecarpaus anacardium) is one of the acclaimed kayakalpam drug mentioned in siddha classical literature extracts or crude drug extract of semicarpus seed for its antidiabetes effect.(semalty 2010, Jaya aseervatham 2010)

Usually in siddha system, Serankottai is used as Ghee, legium which are called as serankottai nei or mahawalladi legium.In siddha literature, yakobu vaidhya cinthamani 700 authored by Ramadevar clearly states medicated for various degenerative diseases including Neerizhivu. Many scientific studies done for these drugs for its anti-inflammatory, antioxidant coupled with immunomodulatory effects make this drug very essential as if was termed in siddha classics.The previous studies were also done with

About the Serankottai Thiravagam which is to be prepared by distillation method.

Thiravagam is a special type of acid or alkali distillate prepared with herbal or herbominerall salts.Thes preparations are generally indicated in chronic,degenerative diseases like Neerizhivu.This theeneer form makes this preparation unique,palatable,and easily dispousable one.No scientific study of Serankottai thiravagam is undertaken sofar.In the present study Serankottai thiravagam is prepared as per Yakobu vaidhya chinthamani 700 in our lab.This drug was studied for its preclinical, including Toxicological studies.Also this drug was studied for clinical efficacy on Neerizhivu noi.

AIM AND OBJECTIVE

AIM

In my experience that siddha is natural medicine and also to be considered as the people's medicine for ever.First I selected the disease Neerizhivu (Diabetes mellitus) one of the leading disease affected most of the people who are either in rich (or) poor among the developed and developing countries. So I had decided to select the best medicine having no side effect and economically low cost for using of all.When I had referred a book as "Yakobu vaidhya chinthamani 700" written by "Ramadevar", i selected the medicines which is "Serrankottai thiravagam" having the ability to control the type 2 Diabetes mellitus- Madhumegam as described in siddha literature.

OBJECTIVE

1. **To** make a detailed study on the course of the disease, etiology, pathogenesis, treatment and prognosis by making use of siddha concepts based on literature.
2. **T**o study Madhumegam in various literature in comparison with modern science.
3. To carry out a clinical trial with two compound formulations.
4. To understand the incidence of the disease with referece the age, sex, thainaigal, paruvakalam, socio economic conditions, diet and family history.
5. To explore the utilize,the diagnostic methods envagai thervu,mentioned by siddhars to know about the manifestation of the disease due to disproportionate mukkutram,udal thathukkal with specific reference to Naadi,Meerkuri,Neikuri.
6. To use siddha and modern parameters to confirm diagnosis severity and progress of the disease.
7. To evaluate the biochemical and Microbiological features of the drug.
8. To undertake pharmacological and toxicological study of the drug.

REVIEW OF LITERATURE

SIDDHA ASPECT

The siddha system is originated by LORD SHIVA''.Agasthiyar is the first disciple of shiva.nobody deny the fact that Agasthiyar was Guru and pioneer.propagate this siddha system of medicine.

In siddha system of medicine the NEER NOIKAL has been classified broadly into two categories.

1. Neerinai perukkum Noi (Adhi moothiram-Exceesive Urination)
2. Neer Arukkal Noi (Neer kattu-Neer Adangal-Retention of Urine)

The same has been described in siddha literature as follows:

நீரிருவினைக் குணத்தை நீயறிவிரித்துச் சொல்வோம்
நீரினை பெருக்கலொன்று நீரினை யருக்க லொன்று
நீரிழிவுடனே கொல்லும் நீர்க்கட்டு வினைகளென்று
நீணில முரைக்கும் மிந்த நீர்நிறைக் குணத்தைக் கேளாய்
நீரினைப் பெருக்க லென்னும் நீரிழிவிலக்கணங்கேள்
நீல வாரிதி போற்குக்கி நீட்டிக்கு முரை தள்ளாடும்
நீவி கூடாது கைகால் நீலமாவினை நேராடும்
நீஞ் சொணாவுரனின் மூச்சு நீசமாமுயக்கள் காட்டும்.

Neerizhivu has been described and classified under the Neerinai perukkal Noi'.

The Ayurvedic and Unani system of medicine have entertained similar views about this disease and postulated paralld views about this origin and management of this disease.

The Indian outstanding physicians Charaka as early as 300B.C was the first to give Information to the world about the sweet taste in urine Charaka samhita.

VERUPEYAR-SYNONYMS

Neerizhivu-Excess of urination (T.V.SAMBASIVAM PILLAI Dictionary)
Ennipu Neer - The urine is sweet in taste (Noi Naadal)
Vegu moothiram.
Madhu prameham.
Madhumegam.

Thithippu neer.

Migu neer.

DEFINITION (IYAL)

Neerizhivu is defined as large quantity and high frequency of urination.Derangement of the seven udal thathukkal and loss of weight.

Noi nadal Part - 1

Urine of the Neerizhivu patients attracts flies and ants in large numbers.when heated it gives a sweet aroma.

இனிப்பான இனிப்பல்ல ஈ வந்ததாடும்
ஒருதுளிவாய் விட்டார்கைப் பிணியாய் தோன்றும்''

குருநாடி நோய்நாடல் பாகம் – 135

''நீரினைப் பெருக்கலென்று நீரிழி விலக்கணங்கேள்
நீலவாரிதி போற் குக்கி நீட்டிக்கு முரை தள்ளாகும்
நீவி கூடாது கை, கால் நீலமா வினை நேராகும்
நீள் சொனாவுரனின் மூச்சு நீசமா முயங்கக்காட்டும்''

தேரையர் மகா கரிசல் ப–135

Abdomen distends like sea, slurring of speech peripheral neuritis, lassitude, dyspnoea are the symptoms of Neerizhivu.

''அண்மையாயடிக் கடிக்கு நீரிறங்கு
மடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையான தடிய தனிற்றான் பிடிக்கும்
மிக்கான சடம் வெளுத்து மேனிகன்றும்''

யூகி வைத்திய சிந்தாமணி ப–117

These lines quotes frequent micturition, more than the normal with large quantity resulting in detoriation of gradual dimnision of sevan udal thathukkal

As per Athma Rakshmirrtham body becomes weak, weight loss, dryness of skin and tongue, excessive thirst, tiredness, excess sleep indicate the presence of megaroham.Athma Rakshamirtham.

வெகு மூத்திரத்தின் குணம்

அன்னமே கேணி மாந்தர்
அருந்துநீர் மிகவும் பொங்கித்
தன்னுள்ளே சுவறா வண்ணம்
தங்கியே மிகுதியாகி
இந்நிறமில்லா நாளு
மிடைவிடா திறங்கு மாயின்
வின்னமும் செய்யும் வெய்ய
வெகு மூத்திரத்தின் வாறே

In spite of excessive intake of water for quenching the thirst, it is continuosly excreted like a spring in the well.

Epidemiology

A detailed study on the basis of geographical variation is also considered to be one of the important causes for Neerizhivu or Madhumegam.

KURINCHI

Mountain and their adjoining areas. In India, most of the pancreatic diabetes cases have been reported from the midland and the hilly tracts of the southern districts of Kerala and Tamil Nadu.

MULLAI

Forest and their adjoining areas.

MARUTHAM

Fertile agriculture land and their adjoining areas near river bed are always healthy.

NEITHAL

Sea and their adjoining areas.

PALAI

Desert and their adjoining areas because of the proverty,they suffer from ill health. Recent research indicates that the poor were also prone to diabetes. Research was being conducted to analyse whether rapid changes in their lifestyle or the stress of proverty triggered diabetes.

NOI VARUM VAZHI-AETIOLOGY

"மேகமெனு நீரழிவு வரும் விதத்தை
விளம்புகிறேன் முன்செய்த கர்மந்தன்னால்
தாகமுடன் மதுபதார்த்தங்கள் நன்றாய்த்
தான்புசித்த லாலுஞ்சிற்றினத்தின் மங்கை
போகமதி கரித்தலா லுட்டினந்தான்
போதவே மிஞ்சுதலால் தயிர்மோர் நெய்பால்
ஏகமாய்ப் புசித்தலாற் கொழுத்த வூனை
யென்று முண்ண லுவர்நீரைக் குடித்தலாலே
ஆசையுடன் சிறுவழுதலங்காய் தன்னை
யதிகமா யுண்பதால் காலந்தப்பில்
போசனங்கள் செய்தலால் நடையலைச்சல்
போதவே யிருத்தலிரா கண்விழித்தல்
தேசமெங்கு திரிதலா மிவைகளாலே
சிரந்தனிற்சூ டதிகங் கொண்டுடனே ரத்தம்
சோஷிதே யதிகமாய் **மேகந்தோன்றித்**
தொல்லை செய்யும் நீரழிவும் இருபதாமே.

சரபேந்திர மேக நிவாரண போதினி

என்னும் நீரிழிவு நோய் மருத்துவம். பக்கம் – 1

Sexual Indulgence:

"**கன்னி மயக்கத்தால் கண்டிடு மேகமே**

திருமூலர் சித்த மருத்துவம் ப– 485

"நிறை பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறை போற்றும் கருப்பத்தில் வளர்ந்தது மேகமே"

திருமூலர் சித்த மருத்துவம் ப - 485

"கிரந்திப் புண்ணிரண மேகக்
கீசக னென்னுந் துன் மார்க்கன்
அருந்ததி யென்னும் பாஞ்சாலி யன்னையைக்கண்ணுற்றானே"

மருத்துவ பாரதம் – சித்த மருத்துவம் ப–485

According to Thirumoolar and Therayar, excessive indulgence in sex causes megaroham.

ஸ்திரி போகம் செய்ததினால் வேவுகொண்டு
சிரசு மட்டும் வெந்துருகிக் கனலே மீறிக்
குறியுடனே மேகந்தான் கொடுமை செய்து
குறைந்து வரும் தாதுவெல்லாம் குன்றிப்போகும்

குருநாடி நோய் நாடல் ப - 250

Diet Habits

கோதையர் கலவி போதை கொழுத்தமீ னிறைச்சி போதை
பாதுவாய் நெய்யும் பாலும் பரிவுட னுண்பீ ராகில்
சோதபாண் டுருவ மிக்க சுக்கில **பிரமேகந்தான்**
ஒது நீரிழிவு சேர வண்டென வறிந்து கொள்ள

(அகத்தியர் 1200) சித்த மருத்துவம் ப– 470

''உற்பவிக்கும் பால் நெய்யா லிறைச்சி கள்ளால்
உரிசையாய் மீன்றன்னால் வருவிருத்த
மற்பவிக்கும் பதார்த்தத் தால் மதுர வஸ்தால்
மந்தங்கள் தனிற் பொசித்தல் வேகாப் பண்டம்
குற்பவிக்குங் குளிந்த வன்ன மங்கை கோஷ்டி
குறித்த நித் திரைதவிர்த லக்கினி மந்தம்''

யூகி வைத்திய சிந்தாமணி – ப– 146

Excessive intake of food rich in carbohydrate and fat, red meat, sweet food, raw food and sleeplessness give raise to Neerizhivu quotes Agathiyar and Yogi Munivar.

Psychosomatic Cause:

''இயம்பவே ஆறுகுளம் பின்னஞ் செய்தல்
ஏற்றமாய் பிராமணஸ்திரீ சங்கம் பண்ணல்
பயம்பவே பாலர்களுக் கொளித்துத் தின்னல்
பழவை சலம் போற வழிதனைத் தடுத்தல்
அயம்பவே சூலயத்திற் சலம்விட்ட டோர்க்கும்
ஆதியாம் வேதத்தை த்தூஷிற் தோர்க்கும்
துயம்பவே சூரியனை வணங் காதார்க்கும்
சுருக்கா மேகம் வந்துற்ப விக்குந்தானே''

யூகி வைத்திய சிந்தாமணி – 144

According to Yogi Vaidya Chinthamani, Megaroham may occur due to not giving proper respect to Guru, Father, Mother, Vedas and suriyan god.

தாளா மேகஞ் சாறும் வகை தன்னைக் கேளிர் மலக்கட்டாற்
கேளாச் சத்தால் காணாக்கண் கேட்டுக் கண்டும் போற் பேசிக்
கோழக் கருத்திற் றாய் தந்தை குருவைப் பழிக்குந் தோஷத்தால்
வுாழாக் கன்னிக் கற்பழித்து வரம்பு தவறில் வருமென்றே

தன்வந்திரி வைத்தியம ப – 75.

Obesity:

"தற்பிவிக்குஞ் **சரீரந்தான் மிகப்ப ருக்கல்**
சல்சலந்தான் பயன்படுதல் தரிக்கும் நோயே"

யூகி வைத்திய சிந்தாமணி ப– 146

Obesity is one of the main causes of Neerizhivu or Madhu megam.

Deeds:

"தானே பூருவ விதியினால் சாரும் பிணிக ளெல்லாம்
மானோர் விழியாள் வேட்கையினால் வருந்தும் பின்னும் பசியால்
தானே பொறுத்து உண்கையினால் தாகந்தன்னால் மிகச்சோர்ந்து
தானே கமலம் புண்ணாகி செய்யும் பிரமேகச் செயல்தானே."

தேரையர் மருத்துவபாரதம் ப –25

ஆமப்பா மனிதர் செய்த கருமத்தாலே
ஆரகரா மேக மென்ற ராசாவாலே
காமப்பா லதினால் பசியப்பா நாலும்
கைக்கடங்க நோய்கள் வரும் கர்மத்தாலே

அகத்தியர்கன்ம காண்டம ப–88

From the above poems, the diseases also occur as a result of bad deeds committed in previous or these births. So yogi, Therayar, Agathiyar, Thirumoolar andothers says that theNeerizhivu (madhumegam) comes by karmavinaigal and bad deed also.

Hereditary:

"முறைகேட்கில் ஒன்பது முயற்சியால் வந்தது
துறை கேட்கிற கருப்பத்திற் றுவங்கிய மேகங்கள்
நிறை பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறை போற்றுங் கருப்பத்தில் வளர்ந்தது மேகமே"

– திருமூலர் ப - 110.

"சுக்கிலத்தில் சுரோணி தங்கள் கலங்குமென்று
பூங்கிடும் வியாதி மூன்றும்"

(தன்வந்திரி நாடி) நோய் நாடல் ப

"தானே புருவ விதியதனாற் சாரும்
பிணிக ளல்லாமல"

தேரையர் வாகடம் ப - 116

Thirumoolar and Dhanvantri have noted in their literatures that Hereditary is one among the causes of the disease Neerizhivu.present day researchers have found out that genetic factors play an important role in Neerizhivu.

Excess Stimulation of Moolatharam:

சரியான **மேகத்தா லபான வாயு**
தான் புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெரிதான மேகத்தா லத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தச வாய்வால் மந்தங் கொண்டு
பெருந்தீனி மலபந்தம் உதான வாயு
விரிவாகித் தேகமெல்லாம் விட ரூ ராலே
மெய்யழிந்த தேகமென்ற திருபதாச்சே''

நோய்நாடல் பாகம் 1 ப– 420

Among the six Atharams the Moolatharam is situated in between recturn and genitals, just end of sacral plexus.

In the Neerizhivu disease, impaired Abana vayu (excretory junction) inactivate the moola agini during that time excess intake of food causes inactivation of dhasavayu

which create excessive appetite (Polyphagia) and constipation. Udanan is also affected. These changes in turn cause the derangement of seven udal thathukal.

மேக நோய் உண்டாவதற்கு ஏது:

"மாதர் மயக்க மிகுதியனான் மதியி லச்ச மிகுதலினால்
போதை தருங்கள் ளருந்துதலாற் புலான் மீனிறைச்சி நெய்பாலால்
சீதவுணவாற் பதனழிந்த திணை விரும்பித்தின் பதனால்
ஓது முணவின் மேலுணவாலுடம்பு பருத்தலினால்

அருந்து முணவி லினிப்பதிக மாக வுறலா ல்ராக்காலம்
பொருந்து முறக்கந் தவிர்தலினாற் பசிப்புக் குறைவான் மிகுதியினால்
திருந்து மடவார் போகமற விடலான் மிகலாற் றேகமிக
வருந்த வெயிலின் மிகுநடையால வந்துதோன்றும் பலமேகம்."

பரராச சேகரம ப – 3.

MURKURIGUNAM (PREMONITORY SYMPTOMS):

தாகமே யதிக மாகித் தளர்ந்து நா உலர்ந்தமிக்க
சோகமாய்க் கிறுகிறுத்துத் தொடர்ந்துகை காலும் சோர்ந்து
ஏகமாய்ப் பகலு மல்லும் விடாது நீரிறங்கும் அன்றி
மோகமாய் மழை பனிக்கு முதிர்ந்திறங் கிடுஞ்சலந்தானே

. **வைத்திய விளக்கம் ப – 49**

Premonitory symptoms of Neerizhivu are poly uria, poly phagia, poly dipsia. Neerizhivu exhibits the following premonitory symptoms from its initial stage of development itself. The patient experiences voracious hunger, thirst, perspiration, exhaustion and giddiness. The excessive intake of water to quench his thirst is excreted as excessive quantity of urine (poly uria). In spite of abnormal consumption of food, stamina continues to decrease.

Noi Nadal Part I P .NO. 256

மதுமேகம் பொது குறிகுணம் :

கூறான **மேகமது இருபதுக்கும்**
குணந்தன்னைச் சிவன்சொல்ல தேவி கேட்க
தாறான தாகமொடு சோகமேகந்
தரியாமல் நீரிழித விருமல் மூச்சு
ஆறான அருசிசத்தி சித்தப் ரம்மை
அடிகடிக்கடிக்குத் தண்ணீர்தான் அங்கே கேட்கல்
ஈறான இடுப்புக்குள் கடுப்பு காணல்
எலும்புழற்ற லழற்றல டெரிவு டாமே
எரிவோடு சரீரமெல்லா மறைபட்டாற்போல்
எலும்பு நோதல் நித்திரையில் லாமைா

வரிவோடு மாய்விமெத் தவும்பறித்தல்
மனதுசஞ் சலப்படுதல் காற்று வேண்டல்
மொரிவோடு மேல் மூச்சு மிகவுண்டாதல்
விக்கலொடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளுருண்டாதல்
தேகமெத்த வாலோபப் படுதல் காணே''

யூகி வைத்திய சிந்தாமணி ப– 155

Common Symptoms:

Thirst	Polyuria
Poly dipsia	Cough
Anorexia	Dyspnoea
Delirium	Pain in the hip and burning sensation
Sleeplessness	loss of weight
Hiccough	Flatulance
Anaemia	Giddiness

NOI VAGAIKAL- (CLASSIFICATION)

Megarogam is classified into twenty varieties to quote from ***Agasthiar***

"உட்டிண ரோகத்தாலும் உறும்பெரும் பசியினாலுங்
கட்டவிழ் கோதை மாதர் கலவிமட்டிலா மையலாலு
முட்டறா நாலுமாறு மும் மூன்று மொன்று மொன்று
திட்டமாய் வருவதென்று திருமுனி யருளிச் செய்தார்"

அகத்தியர் சித்த மருத்துவம் – ப–471.

Yugi Munivar classifies the same as

வசனித்த **மேகமது யிரண்டு பத்து**
வாதத்திற் பிறந்தசலம் நாலேயாகும்
பிசனித்த பித்தத்திலு ற்றபவித்த
பேராசை லந்தானு மாறு மாகும்
தேசனிந்த சேட்டுமத்திலுற்ப வித்த
சீரான சலந்தானும் பத்தேயாகும்.

யூகி வைத்திய சிந்தாமணி ப–145.

According to ***Theraiyar***

"கழியும் வாதம் நான்காலும் காயும் பித்த மாறாலும்
சுழியும் சேத்துமம் பத்தாலும் சொல்லும் நாலஞ்சாய் தோன்றும்"

தேரையர் வாகடம் ப–42

NOI VAGAIGAL

நூல்கள்	நோய் எண்	வளி	அழல்	ஐயம்
அகத்தியர் 1200	20	4	6	10
யூகி வைத்திய சிந்தாமணி	20	4	6	10
தேரையர் வாகடம்	20	4	6	10
தன்வந்திரி வைத்தியம்	20	4	6	10
சரபேந்திர நீரிழிவு ரோக சிகிச்சை	20	4	6	10
யூகி முனி வைத்தியகாவியம்	20	4	6	10

The above books describe twenty different kinds of megam (urinary disorders) on the basis of colour, consistency, taste, smell, weight etc.

Out of this twenty different kinds

Four varieties are caused by vali

Six varieties are caused by Azhal

Ten varieties are caused by Iyam

Neerizhivu comes under the classification of Azhal.

Classification of Megam:

According to ***Yugi Vaidhya Chinthamani***

வாதநீர் வகைகள்

"தரித்திட்ட வாதத்தின் சலந்தா னாலு
தனியான நாலுக்கும் பேரே தென்னில்
அரித்திட்ட ஆச்சியகெந்தி மேகத்தோடு
அதன்பிறகு சுற்றமா மேகமென்று
பிரித்திட்ட பிரமிய மேகமொன்று
பேரான மாங்கரவி மேகமென்று

யூகி வைத்திய சிந்தாமணி ப–146.

Vali – 4

1. Neimananeer (ஆச்சியகந்தி மேகம்)
2. Pasumana neer (சுத்த மேகம்)
3. Seezhmana neer (வாதபிரமிய மேகம்)
4. Sathaimana neer (மாங்கிசசிராவி மேகம்)

பித்த நீர் வகைகள்

முறையான பித்த சல மாறுமாகும்
முதிர்ந்த அப்பிய மென்றும் பிரமிய மென்றும்
துறையான சாம்பீர்ணமதும்ப மென்றும்
சாத்திகமே யாறுவிதந் தன்னோ டாறு"

யூகி வைத்திய சிந்தாமணி – ப– 148

Azhal – 6

1. Yanai kozhupu mana neer (அப்பிய மேகம்)
2. Katrazhai mana neer (பித்தபிரமிய மேகம்)
3. Chunna mana neer (சவ்விரண மேகம்)
4. Innipu megam (மதுமேகம்)
5. Palingu neer (சாந்திர மேகம்)
6. Muyal kurithi neer (ஆர்க்க மேகம்)

ஐயநீர் வகைகள்

"ஆறான சிலேட்பசலம் பத்து தன்னை
அரன் சொல்ல ஆத்தாள் தான் கேட்கும் போது
வாறான வசாமேகம் உத்சமேகம்
மச்சியாமே கத்தோபா கீத மேகம்
தூறான சுராரி சுக்ல முத்த மேகம்
சுற்றமாம்பி னானியொட வலண மேகம்
கேறான தெயுத்தயமா மேக மென்று
செப்பினார் சிலேட் பத்தின் செலுத்துத் தானே"

யூகி வைத்திய சிந்தாமணி ப– 151

Iyam – 10

1. Iaya Neer (வசா மேகம்)
2. Thuimai Neer (உத்தம மேகம்)
3. Moolai neer (மச்சை மேகம்)
4. Ilaneer (ஆதிக்க மேகம்)
5. Kal neer (சுரா மேகம்)
6. Thavala Neer (சுக்கில மேகம்)
7. Kazhu neer (உதக மேகம்)
8. Then neer (பிரச மேகம்)
9. Uppu neer (சார மேகம்)
10. Avichi Neer (தைத்திய மேகம்)

Yugi described four types under the Vatha premeham, six types under the pitha prameham and ten types of under Kaba prameham. ***"Diabetes mellitus" a clinical entity in a modern medicine is closely resembles one of the type of pitha prameham ie "Neerizhivu".***

பரராச சேகரம் நூலில் கூறப்பட்டுள்ள நீரிழிவின் வகைகள்:

சிலேற்பன மேகம்-10

சிக்காய் மேகம:

"மிக்க சேற்பனத் தெழுந்த மேகநீர் பத்துங் கேளாய்
சிக்காயங் கலங்க னீராய்ச் சிறுகெண்டைச்சின்ன போற்றோன்றி
ஒக்கவே துவர்பாய் முற்றி யுடல் கரைந் திறங்கும வானீர்
விக்கல் கொட்டாவி தும்மன் மிகுந்திடுங் குணங்களாமே"

பரராச சேகரம் ப – 20

Urine is turbid and astrigngent.

Emaciation sets in.

There will be increase in hiccough, sneezing and yawning possibility of respiratory disorders.

புலால் மேகம:

"கேட்டிடாய் புலான்மே கந்தான் கெம்புமீன் கழுவு நீர்போல்
முட்டிய வனலாய்ச் சுட்டு மூக்குநீரிறக்க மாகும்
கோட்டிகள் பலவு முண்டாங்குறியடி நோகும் புண்போல்
நாட்டினி லிதன் குணத்தை நவின்றனர் பெரியோர் தாமே"

பரராச சேகரம் ப – 21

Urine resembles fish washed water.

Body temperature increase with rhinitis.Burning micturation with pain in the genitalia.

இலவண மேகம்:

"விரும்பிய லவணமேக மிகுந்தநீ ருப்பு றைக்கும்
துரும்பென மேனி வற்றுந் தொண்டையுங் கன்றி நோவாம்
திரும்பநீர் காய்ச்சிப் பார்கிற சிவந்துவுப் பாகித் தோன்றும்
குரும்பையின் முலைநன் மாதே கோசமு மெரிவுண்டாமே"

பரராச சேகரம் ப – 22

Urine is salty with reddish tinge.

Weight loss setsin.

Soar throat occurs due to infection.

மதுமேகம்:

"மதுமேகஞ் செய்கு ணந்தான் வாயது கசந்து காட்டும்
இதம்வரும் பின்னெடுக்கும் யானைமும் மதமே போல
முதயநீ ரெடுக்கும் பாலே முற்றிய புளிப்பு நாடும்

அதிகமாத் தாகமுணடா மயர்ந்துடல் வலக்கு மென்னே”

பரராச சேகரம் ப – 23

Bitter taste linger.

Patient became restless.

Urine ferments like milk.

Morbid thrist.

Extreme tiredness.

ஆனந்த மேகம்:

“திட்டமா னந்த மேகஞ் சிறுகரும் பினிற்சா றெடுத்து
விட்டநீர் குளிர்ந்து காட்டும் வேதிணை மயக்க முண்டாம்
முட்டமே லுளைந்து ளைந்து முதுகந்தண் டெலும்பு நோவாம்
அட்டமே தாதி சூத்திர மாய்ந்திடு மென்று காணே”

பரராச சேகரம் ப – 24

Urine resembles sugarance juice and is cold

Patient is exhausted and complains of lumbar pain

மகா மேகம்:

“செப்பிய மகாமே கந்தான் சிக்காய் மிறங்கு நீரின்
துப்பெனத் தெரித்து வீழுஞ் சுருக்கெனக் குறுக்கிற் குத்தும்
குப்புறத் தள்ளிக் காட்டுங் குணம்வரும் வெகு நீருண்டாம்
இப்படிக் குணங்க ளாமென் றியம்பினார் முனிவர் தாமே.”

பரராச சேகரம் ப – 25

There is obstruction in the urinary flow.

Intense pain in the lumbar region.

There is excessive urination in Neerizhivu.

காச மேகம்:

“இயம்பிய காச மேகம் மேதமாய் வாயினிக்கும்
வியன்பெற முகங்க றுக்கும் வெண்ணுரை நீறே வீழும்
அயர்ந்து கண் டூக்கமுண்டா மடிரண்டுங் காந்தலாகும்
நயந்துசற் றிருமா லுண்டா நயந்தவர் நவின்றவாறே.”

பரராச சேகரம் ப – 26

Sweet taste lingers

Face turns black.

Urine becomes white forthy.

Patient experiences deep sleep.

Burning sensation in the sole of foot.

Presence of cough.

வெறிமேகம்:

"வெறிமேகம் பக்க மாகி மிகவெறி போலக் காட்டும்
அறியுமாட் டிறைச்சித் தண்ணீரா மென நீரிறங்கும்
இறுகியே மலம்வி டாதாமிதற்குமுற குடிநீர் தன்னில்
நெறியதா யப்பி ரேக நிசமிடத் தீருமென்றே."

பரராச சேகரம் ப – 27

Aggressive mental attitude.

Beef washed water like urine.

Presence of hard stools.

NOIKURI KUNANGAL – (CLINICAL FEATURES)

"தண்மையாய்ச் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கும் பீசமுங்கோ சமுங்க டுக்கும்
அண்மையா யடிக்கடிக்கு நீரி றங்கும்
அடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையா யடிதனிற் றான்பி டிக்கும
மிக்கான சடம்வெளுத்து மேனி கன்றும்
பண்மையாய்ப் பஞ்சவாண் டதனிற் கொல்லும்
பகர் கின்ற மதுமேகப் பாங்குதானே"

யூகிவைத்திய சிந்தாமணி ப – 127

Poly urea, Poly phagia, poly dipsia, perspiration, exhaustion, insomnia, giddiness and loss of weight even at normal consumption of food.

Common sign and symptoms of Pitha Prameham

"அறியவே பித்தசல மாறுக்குந்தான்
அங்கமதிற் செய்கின்ற குணத்தைக் கேளாய்
தறியவே சரீரம் வற்றி யெரிவுண்டாகும்
சடத்திலுந் நீரிலுந்தான் கவிச்சுண்டாகும்.
தெறியவே சீப்போலுங் கற்றாழை போலும்

சேல் போலுந் தேன் போலும் நாற்ற முண்டாம்
வெறியவே பீசத்திற் கோசத்திற் குதத்தில்
மிகுமீரல் நாபியிலும் வேக்கா டாமே
வேக்காடாய் விரண முண்டாய் வாய்தான் நாறும்
விக்கலொடு அருசியாயச் சுரமுண்டாகும்
தீக்காடாய்த் தேகந்தான் கிடைகொட்டாது
தியக்கமொடு மூர்ச்சையுண்டா மயக்க மாகும்
சாக்காடாய் நாவறந் தண்ணீர் தாகம்
சக்தியொடு சரீரயாகும்
தாக்கடா மலசஞ்சலந்தான் மிகவுண்டாகும்
சமகுணந்தான் பித்த சல மாறு மாச்சே

யூகி சிந்தாமணி ப– 181

As per the above poem, poly uria, poly phagia, Poly dipsia, fever, angular stomatitis, pruritis vulvae, balano posthtis, burning sensation all over the body, loss of weight, are common signs and symptoms of pitha prameham.

MUKKUTRAIYAL:

"வாதமாய் படைத்து,பித்த அனலாய் காத்து
சேத்ம சீதமாய் துடைத்து"

தேரையர் மருத்துவபாரதம் ப– 81

Vali:

Sites of vali:

BelowNaval, Urinarybladder, intestines, pelvis, umbilicalcord thigh, bone, skin, Nerve endings, joints, musculature and hair root.

Properties: Dryness, lightness, clearness, coolness, mobile formless.

Functions:

Stimulation, respiration, thinking sensory function, coordination of seven physical constituents, reflex action.

In Neerizhivu:

Piranan	:	Normal
Abanan	:	Constipation, Noctural polyuria, frequency of micturation.
Viyanan	:	Symmetrical sensory disturbances, Peripheral neuritis, pain all over the body. Burning sensation in the sole of foot and palm, skin infection and carbuncle.
Udanan	:	Exessive thirst.
Samanan	:	Poly Phagia
Nagan	:	Normal
Koorman	:	Diabetic retinopathy / Cataract
Kirukaran	:	polyphagia
Devathathan	:	Normal
Thananjeyan	:	-

Azhal:

Sites of Pitha:

Between the heart and the naval, sweat, lymph, blood, stomach, urinary bladder, heart, saliva, eye and skin.

Properties:

Dry, cold, hot, light, subtle, keen, soft, liquid, bitter.

Function:

Body temperature, digestion of food,colouring of the skin, vision,sweat.

In Neerizhivu:

Anala Pitham	-	Excess hunger
Ranjaga pitham	-	Pallor sometimes
Alosagapitham	-	Diminishing of vision
Saathaga pitham	-	Lassitude
Pirasaga pitham	-	Dry skin

Iyam:

Sites of Kapha:

Above the heart, stomach, fat, sperm, tongue, uvula, bone marrow, blood, nose, nerves, bones, large intestine, eyes, joints.

Properties:

Heavy, cold, mild, watery, sweet stable and slimmy.

Function:

Iyam gives streanth, builts the body, gives streanth for joints,gives shiney appearece to skin, moistens food,cools the eyes,gives a whitish colour to the conjuctiva skin,urine and faecal matter,softness,firmness the capacity to bear or endure,patience.

In Neerizhivu

Avalambagam	-	Normal
Tharpagam	-	Burning sensation in the eye
Santhigam	-	Joint pain
Kilethagam	-	Excessive appetite
Pothagam	-	Normal

Seven Udal Thathukkal (Physical constituents):

Annamaya kosa is constituted by seven thathus.They are the basic tissues of our body.

Normal functions:

Saram:

It is responsible for the growth and development. It keeps the individual in good spirit and it nourishes the blood.

Senneer:

Blood imparts colour to the body and nourishes the muscle responsible for the ability, intellect of the individual.

Oon:

It gives shape to the body according to the requirements for the physical activity, nourishes fat.

Kozhuppu:

It helps in lubricating the different organs and maintains only matter of the body.

Enbu

Supports the system and responsible for posture and movements of the body.

Moolai:

It fills the bony cavity, nourishes semen, imparts strength endurance and shining appearance.

Sukkilam / Suronitham:

It is responsible for reproduction. In healthy people, they function in a harmony, while in diseased people, they are deranged.

In Neerizhivu:

Saaram	:	Tiredness, General weakness
Senneer	:	Pallor
Oon	:	Lean and thin
Kozhuppu	:	Dry skin
Enbu	:	Later stage due to infection it affects the bone and sometimes leads to amputation.
Moolai	:	Affected in Chronic stage.
Sukkilam / Suronitham:		Impotence, Sexual urge is reduced.

So, in Madhumegam, Seven Udal Thathukkal are deranged.

MUKKUTRA VERUPADUGAL-(SIDDHA PATHOLOGY):

"பகர் பித்த விந்தையலாது மேகம் வராது" –தேரையர்.

The disease megaroham, due to external (or) internal causes affect balance in the ratio of vali, Azhal, Iyam. This imbalance affects the Keelnokkukal, which inturn affect the seven udal thathukkal. Saram gets affected and there is loss of appetite. Seeneer also get affected with the net result even if the patient eats more nourished food (polyphagia) there won't be any improvement in health.

An imbalance in pitham does imply an imbalance in other two kutrams too and causes derangement of dasa vayu and seven udal thathukkal which cause the disease and other complications.

"குறியுடனே மேகந்தான் கொடுமை செய்து
குறைந்து வந்து வருந்தாது வெல்லாங் குன்றிப் போகும்"

பதிணெண்சித்தர நாடி நூல்.

PINIARI MURAIMAI- (DIAGNOSIS):

Diagnostic methods in Siddha system are very unique and solely based on clinical acumen of the physician.

1. ***Poriyal Arithal*** (or) understanding by the fire organs of perception (Mei, Vai, Kann, Mooku, Sevi)

2. ***Pulanal Arithal*** (or) understanding by the sense objects (Uraithal, Suvaithal, Parthal, Mugarthal, and Kettal).

3. ***Vinadal*** (or) Interrogation

The physician using his organs of perception and senses examines the patient and diagnoses the disease. Apart from this, on effective history taking also helps one to diagnose property.

Tools used by Siddha Physicians:

(1) Kanndal (Perception)

(2) Karuthal (Inference)

(3) Oorai (The instruction of the inspired)

(தோ.கி.ஆ) ப - 46

The application of these three is very extensive in diagnosis and treatment.

"Enn Vagai Thervu (Eight tools of Diagnosis)

தரணியுள்ள வியாதி தன்னை யட்டாங்கத்தால்
தானறிய வேண்டுவது யேதோ வென்னில்
திரணியதோர் நாடி கண்கள் சத்தத்தோடு
தேகத்தினது பரிசம் வருணம் நாக்கு
யிரண மலமூத்திரமா மிவை களெட்டும்
யிதம் படவேதான் பார்த்துக் குறிப்புங்கண்டு
பரணருளால் பெரியோர்கள் பாதம் போற்றிப்
பண்பு தவறாமல் பண்டிதஞ் செய்வீரே"

குணவாகட நாடி. ப - 112

Naa:

The tongue is the organ of taste and speech. Naa is one of the Gnanenthirium. Colour of the tongue, size, shape, anomalies, surface, mobility and local lesion should be noted. Coating deposition of the tongue, increased salivation, and dryness of the tongue. Pervested taste, tongue ties must be noted.character of speech is noted in mozhi. Mozhi is one of 5 kanmenthiriyam.

Neer Bhoothams is related tongue which makes it wet always.

In Neerizhivu the tongue remains dry and at times black.

In Neerizhivu, the tongue remains dry and at times black.

Niram:

Colour of the skin all over the body, a local region of affection, conjunctiva, tongue, nail bud, hair etc.

Colour changes in the excretion from Nava thuvaram is noted (i.e) we have to observe colour changes in malam, muthiram, vomited matter, sputum, saliva, semen, teeth and face.

Vatha Udal	-	Black and whitish colour
Pitha Udal	-	Yellowish (or) Reddish colour
Kapha udal	-	White or golden colour
Thontha udal	-	Mix of two udal colours

In Neerizhivu, the colour of skin is different from original complexion, discoloured.

Mozhi:

To quote from Agasthiar Vallathi

"வார்த்தையைப் பார்"

Observation of speech and voice.The different components of speech should be properly as curtained.

Voice is a sound produced by the speech organ and uttered by mouth .In 96 thathuvas vai (mouth)one of the Gnanethiriyams and Mozhi one of the kanmenthiriyams.it belongs to Akaya Bootham.

In uncontrolled Neerizhivu which leads to cerebrovascular disorder, speech disorder sets in.

Vizhi:

Colour, character, vision should be observed.Apart from any discharge chronic diseases can be identified.

In uncontrolled Neerizhivu cataract set in last. In longstanding cases, the Neerizhivu affects retina and causes diabetic retinopathy which is the major cause of blindness.

Sparisam:

Therayar quates "மெய்க்குறி"

Colour of the skin (Vali, Azhal, Iyya udal), Eruption, Hemorrhages, Ulcers, Boils, trophic changes, in the skin can be identified.

Any changes in the internal organs can be noted by palpation (or) percussion.

In Neerizhivu, increased tendency ***for fungal infection like moniliasis and vulvities.***

Necrobiosis lipoidica diabeticorum the usual site in pre labial surface of lower limb and non healing ulcers in foot.

In Neerizhivu the skin is ***dry and pale****.*

Malam:

To quote from therayar "இருமலம்" three times evacuation of bowel is must.

Quantity, colour, smell, froth should be observed.

In Neerizhivu, ***constipation sometimes yellowish loose stool are passed*****.**

Muthiram:

Quantity colour froth smell and specific gravity of urine should be noted.

நீரின் பொது குறிகுணம:

"வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவை யறைகுது முறையே"

Urine:

Colour*:*

In Neerizhivu clear and white.

Specific Gravity:

In Neerizhivu, urine is thick in consistency like honey.

Smell:

Honey like smell, In diabetic keto acidosis,the urine has fruit smell.

Froths:

In Neerizhivu the urine is frothy at the time of urination.

Deposits:

In Neerizhivu few epithelial cells are present in urine.

Normal quantity of adult urine is 750 – 2500 ml in 24 hours.

In Neerizhivu polyuriya is present.Both quantity and frequency of urine are increased. Disturbing polyuria at night (nocturia) and Glucosuria (the presence of sugar in urine) are present.

நீர் நிறக்குறி:

"அருந்து மாறிரதமும் அவிரோதமதாய்
அஃகல் அலர்தல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காது பெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே"

நோய் நாடல் 1 ப – 198

Collection of Sample Urine:

The patient must take well cooked food in the previous day. The intake must be proportionate to the degree of his appetite. Food intake should be taken, at appropriate time. We must have sound sleep on the previous night. The urine is collected on the down of the next day in a glass container and closed immediately to prevent contamination. This specimen must be examination with in one and half hours. This procedure should be followed strictly to get accurate observation of Neerkuri and Neikuri.

நெய்குறி:

"நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடு விடுத்
தென்னுறத் திறந்தவெளி யேகா தமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுந் செய்தியை யுணரே"

நோய் நாடல் 1 ப– 209

NEIKURI:

The diagnosis and prognosis of deranged Mukkutrams are studied on the bais of the behaviour of a drop of gingelly oil gently droped on the surface of the urine kept in a wide vessel in the sunlight.

"முத்தொத்து நிற்கின் மொழவதென் கபமே"

நோய் நாடல் 1 ப-209.

In Neerizhivu, the oil dropped in urine is like a pearl and if the oil spreads slowly, the prognosis of the disease is slow and good.

நாடி:

Pulse diagnosis is the confirmatory diagnosis. In Neerizhivu,

இருமியே பித்தமும் வாதமுங் கூடிடில்
மருவுசல மேகம் வாருதி போலாகும்
உருவம் வேறாரு முண்டவுடன் காந்திடும்
உருகவே வூனோடு உறிஞ்சி இனிக்குமே''

திருமூலர் சித்த மருத்துவம் ப– 487

The above poem says that excessive elimination of urine containing sugar are always primarily due to continued vitiation of Azhal and Vazhi.Functional factors in the body ,the vitiation of "Azhal vazhi" is indicated clinically by excessive hunger,thirst,emaciationandpassing of large quantity of urine.

"தூரணமுடன் நீர்ப்பாடு நேர்பாடானால்
சொல்லுகின்றேன் நாடியெல்லாம் தளர்ந்து காணும்"

பரிபூரண நாடி ப - 150

From the above lines,all the three Naadies are feeble and week in Neerizhivu patients.

மதுமேக குணம்:

''இனிக்கின்ற வாதத் திடைசேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்
கனிக்கின்ற மேனி கரைந்து வெளுப்பேறும்
தனிக்கும் மதுமேகந் தப்பாது ஐயனே''

திருமூலர் சித்த மருத்துவம் ப– 488

The coupling of the Vali and Iya naadi causes increase tension in azhal Naadi. This is called "kudila naadi" it causes Neerizhivu urine is like today patient is emerciated.

வாதத்துடன் சிலேத்துமம் கூடி மேக வியாதி
ஏற்பட்டால் கள்ளுபதனிப் போ நீர் வெளிப்படும்
மேலும்உடல் குலைந்து வெளுத்த நிறம்
கொள்ளும் இதற்கு மதுமேகம் என்று பெயர்

பதினென் சித்தர்கள் நாடி சாஸ்திரம்– பக்கம் 89.

When kabha merges with vadha, glucosuria, emaciation, anaemia, develops.

பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மே வந் தோன்றியே பொருந்திமெய்யில்

– திருமூலர் நாடி ப–110

When all the three nadis ,runs in low volume,diabetes develops.

"பற்பித்த மேக மென்றால பித்த மீளும்
பாலகனே காங்கை கொண்டு நீராம் பாரே"

From the above lines,it is clearly stated that vitiation of Azhal results in Neerizhivu.

"நீர்மேக மானவர்க்கு நாடி தானும்
நீர்மயமாய் நாடியெல்லாம பலமே கெட்டுக்
கார்மேகம் போலவந் தெரிமேல் புரண்டு
விழுப்புழுப போலவே புரண்டு காட்டும்"

பரிபூரண நாடி ப - 155

All the three Naadies are felt feeble in these suffering from "Meganoi". The chrater of the pulse is compared to that of wrigging movements of a worm that have fallen in to the fire.

உறுதியுள்ள பித்தமது தோன்றில்……..
……..பிரமேகங்கள்……
சதக நாடி.

The above stanzas are indicating vitiation of Azhal, resulting Neerizhivu.

நீரழிவு நோயில் காணும் அவத்தைகள்:

The following complication follow gradually. If the disease is not controlled (or) left untreated.

அவத்தை – 1

"காணவே முதவலத்தைச் சரீரந் தானும்
கனமாகப் பருத்திறுகு நீர்த்து வாரம்
வேணவே வெண்டாக்கி யகலம் பண்ணும்"

–சித்த மருத்துவம் ப– 483

Obesity sets in.

There is obstruction in urinary flow.

Urinary passage expands due to inflammation.

அவத்தை – 2

"மிக்க இரண்டு டாமவத்தை விளம்பக் கேளாய்
மூணவே மூத்திரப் தீடையுமாச் சுக்ல
முகமழுகித் தேஜசுதான் மிகவே குன்றும்"

சித்த மருத்துவம் ப – 483

Micturation is frequent.

Sexual desire gets.

There is also loss of complexion.

அவத்தை – 3

"நாணவே மூன்றாகு மவத்தைக் குந்தான்
நாவறளும் வாயுவது மீறுந்தானே"

சித்த மருத்துவம் ப – 483

Tongue generally becomes dry.

Abdomen is distended due to flatulence.

அவத்தை – 4

"தானான நாலவத்தை யங்க தாகம்
சன்னியது பாத முண்டாம்"

சித்த மருத்துவம் ப– 483

Severe thrist occurs.

Causes delirium

அவத்தை – 5

"ஐந்து வத்தைத்
தேறான நீர்பெருகந் தாது நஷ்டம்

சித்த மருத்துவம் ப– 483

Quantify of urine increased.

Loss of semen (impotence).

அவத்தை – 6

"நிலை யாறா மவத்தையுடற் கிடை கொள்ளாது
மூனான மூர்ச்சை வரும்"

Sleeplessness is present.

Difficulty in breathing is experienced.

அவத்தை – 7

"ஏழுவத்தை

மிக்கவரோ சிகஞ்சுவாசந் தேக சாட்யம்"

சித்த மருத்துவம் ப– 483

Tongue becomes tasteless.

Difficulty in breathing is experienced.

General weakness persists.

அவத்தை – 8

"ஏனான எட்டாவ தவத்தை தானே

எழுகிரந்தி பிளவையுந்தான் மிகவுண்டாமே"

சித்த மருத்துவம் ப – 483

Abscess is formed.

Presence of Carbuncle.

அவத்தை – 9

"உண்டாகு மொன்பதா மவத்தை கேளாய்

ஒழுங்கான ஆசாரங் கிருமி யுண்டாம்"

சித்த மருத்துவம் ப – 483

Irregularities in daily habits like bowel habits.

Bed soar may occur.

அவத்தை – 10

"பண்டான பத்தாந்த வைத்தைக் கேளாய்

பாரமாம் சயங்கண்டு பரத்துக்கேகும்"

சித்த மருத்துவம் ப – 483

Secondary infection like tuberculosis may sets indue to loss of immunity,

Other complication loading to death may occur.

These are the complication at 20 Neerizhivu.

Other complications: Meganeer Kattigal 10.

- Madaku Katti
- Ammaiodu Kattai
- Valai Kann Katti
- Athomuga Katti
- Phai surai katti

- Kadalai katti
- Kadugu Katti
- Vithirathi Katti
- Nilapoosani katti
- Magavithirathi katti

தீரும் தீராதவை (Prognosis)

செய்யவே வச்சரமாந் தண்ட மான
செயமான முதுகுதண்டைப் பற்றி நிற்கும்
பெய்யவே பெருநரம்பில் மேகந்தானும்
பிறக்குமென்றே தானறிந்து வாதந்தன்னால்
பிய்யவே பிறந்தகல மாறா லசாத்தியம்
பித்தத்திற் பிறந்தசல மாறும் யாப்யம்
பையவே சேட்டுமத்திற் பிறந்த பத்தும்
பரமனுரைத் தார் சாத்யம் பராபரிக்கே''

யூகி வைத்திய சிந்தாமணி ப– 156

''ஆனது பித்தந் தன்னி லாறஞ் சேற்பனம் பத்
தீனமாம் வாத நான்கா மிதில் வாதம் தீரா நீரே''

பரராச சேகரம் ப– 22

''வழியும் வாதம் நான்காமே மாறா தவிழ்தந் தன்னாலே
பொழியும் வாதம் நில்லாது போமே மருந்தைப் பொய்யெனவே''

தேரையர் வாகடம் ப–42

The four types of megam caused as a result of imbalance of vali are incurable.

The six types of megam arising with disparity of Azhal could be cured with great difficulty.

*But **ten types of megam arising due to Iyyam are curable**.*

எந்தெந்த ரோகங்களில் சிறுநீர் அதிகரித்தாலும் குறைந்தாலும் தீது
''வெப்பு பிணியதனில் வெம்மேகத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் – செப்பும்
கிராணியிற் பாண்டில் குளர்நீர் சுருங்கிற்
பிராணன் பிரியுமெனப் பேசு''

(கண்ணுசாமியம்) நோய் நாடல்1 ப – 153

Very excess of urination in Megaroham causes death.

"துதிப்பான மேகத்தில் நீரிழிவு மாகா

தோன்றிய நீரிழிவு தண்ணீர் வாதமுமாகா"

(சதக நாடி) நோய்நாடல் 1 ப – 153

If Megaroham is associated with excessive urination, it is difficult to cure. If megaroham coexist with vali it is incurable.

"மேகத்தில் நீரிழிவு மேவுமதில் வாத நோய்

வேக வயித்துள் வயிற்றுளைவு – சோக விக்கல்

பன்னு விக்கில் தன்னில் பகரிளைப்புப் பாங்க தனிற்

பின்னளை யாகாது பேசு"

நோய்நாடல் 1 ப – 153

In megaroham, if Valinoi, excurating pain in abdomen hiccough and tuberculosis coexist it is incurable.

"நீரிற் பிளவை நிரும்பிளவையில் தாகம்

ஒது மதில் தேகத்துறை மனலுந் – தேருமதில்

சோரு மயக்கமுஞ் சொல் மயக்கத்தில் வியர்வை

ஆரிற்றீதா மென்றறி"

நோய்நாடல் 1 – ப – 154

If in complications of Megaroham with carbuncle, Morbid thrist, excessive body heat, shock and sweat occurs the prognosis is bad.

"வேர்வைதனிற் கபமும் மேவுமதில் விக்கல் நோய்

கார்முகில் நேர் கூந்தலாய் கண்டு மேல் – சீர்கொள்

மருத்துவத்திற்றேர்ந்த மதியுடையாராவி

தரித்திரா தென்பர்சரி"

நோய்நாடல் 1 – ப – 154

If Iya megam is associated with sweat hiccough, the prognosis is bad.

Maruthuvam:

"வைத்தியச் செயல் வைத்தியமாமே"- திருமூலர் 8000

The treatment in Siddha Medicine is aimed at keeping the three kutrams (Vali, Azhal and Iyam) in equilibrium and maintenance of the seven udal thathukal.

"உற்றான ளவும் பிணியளவுங் காலமுங்

கற்றான் கருதிச் செயல்".

In siddha science, the treatment is not only for removal of the disease, but for the prevention and improving the body condition after the removal of the disease.

This is classified as

Kaappu -Prevention

Neekam -Treatment

Niraivu -Restoration

Kaappu:

Prevention is better than cure is a proverb. Siddha principles based mainly on prevention as mentioned in "Theraiyar pini Annuga vithi" by Theraiyar.

The aim of the treatment is to bring the affected thathus and Mukkutram to normal levels by eyamma, niyamma, diet and medicine.

The rules affecting healthy alliance have been elabourately described in the science of Astrology.The ideal marriage life should be on the basis of physical, emotional, intellectual and social compatabilities.

NEEKAM:

The mukkutrams are responsible for organisation, regularisation and integration of the body structures and their physio psychological functions which are kept in a state of equilibrium by word, throught, deed and food of the individual. The general actiological factors for constitutional discomfort is said to be incompatible diet, mentel and physical activities.

When treating the disease the following principles are noted.

"உற்றான ளவும் பிணியளவுங் காலமுங்

கற்றான் கருதிச் செயல்"·

For the disease neerizhivu, serrankottai thiravagam 10 drops twice daily with water before food.

Niraivu:

Physical, Psychological, social and economic rehabilitation of individual is known as Niraivu.

In Madhumegam, Azhal kutram and other two kutrams Vali and Iyam deranged and cause impairment of dasavayu which in turn affect the seven udal thathukkal.

Line of Treatment:

“பகர்பித்த விந்தையலாது மேகம் வராது”

நோய்நாடல்-1 ப.254

The aim of treatment in Neerizhivu is to normalize the vitiated mukkutram, vayus and theaffected seven udal kattugal.As this disease is caused by megam ,suitable effective medicinal preparations have to be administrated in the beginning itself to neutralize and eliminate the megam from the body tissue.

Siddhrs aimed at bringing the mukkutram in equilibrium as the basis of the treatment ofdisease. Initially the patient will be treated with herbs,if is not effective mineral preparations are used while treating the disease. Siddhars prescribed a minimum dosage initially and then increased the dose gradually.

“வேர்பாரு தழைப்பாரு மிஞ்சினக்கால்
மெல்லமெல்ல பற்ப செந்தூரம் பாரே.”

There are many preparations for Neerizhivu and its complications found in various siddha textbooks, like kudineer, choornam, maathirai, ilagam, parpam, chendhuram, thiravagam, etc.

Theran maruthuva Bharatham;

“கிரந்திப் புண்ணிரண மேகக்
கீச்சக னென்னுந் துன் மார்க்க
னருந்தி யென்னும பாஞ்சாலி
யன்னையைக் கண்ணுற்றானே”

Neerizhivu has been caused by indulgence of sex.It leads to the formation of meham which gradually all the seven udal kattugal and finally sets in genitourinary system, resulting in excessive secretion and elimination of urine with sweet taste.

In Theran Maruthuva Bharatham, meham is alluded to keesagam and Neerizhivu (madhumegam) is alluded to sainthavan. So the pancha pandavas who killed these keesagan and sainthavan.

In Neerizhivu seven udal thathukkals are affected. In Theran Maruthuva Bharatham some paadanam and metals are alluded to pancha pandavas for streanthening the seven udal thathukkal.

Saaram;

Abscess, boils, carbuncles, cellulitis, fungal bacterial infections are common when saaram is affected. Gandhagam (sulphur) is alluded to paanchali. To streangthen the saaram we used to give drug of Gandhagam.

Senneer;

Diabetic foot, gangrene and impotence are common due to vascular insufficiency. Ayam (iron) is alluded to Dharman. Ayam preparations are given to streangthen the senneer.

Oonn;

When the oonn is affected diabetic polyneuropathy will occur i.e weakness and wasting of the proximal muscles, loss of weight (Neuropathic cachexia) hyperasthesia, parasthesia with pulmonary tuberculosis are seen. To streangthen the oonn velli (silver) is given velli is alluded to Sagadevan.

Kozhuppu and Enbu

Impotence, pulmonary tuberculosis and emaciation supervene. Pon (Gold) preparations are given tostreangthen the kozhuppu and enbu.pon is alluded to abimanyu.

Moolai and sukkilam;

Diabetic polyneuropathy and impotence may occur.prepared medicine of kaareeyam (lead) and sembu is alluded to Arjunan.

Nutritional Approach:

Lifestyle modifications are the cornerstone of management of diabetes mellitus and include the prescription of a healthy diet, regular exercise, the management of stress, and avoidance of tobacco

The approach consists of provisions of adequate calories for maintaining or attaining standard weight for age sex and height.

General consensus on proportion of food constituents are

Carbohydrate 60 – 65 % (complex form have more fibre)

Fat 20 – 25 % (saturated 7.8 % polyunsaturated 7-8% Mono unsaturated 7.7%) Proteins 10 – 15 %

Diet and nutrition has very important role in disease like Neerizhivu. Diseases are largely due to irregular dietary habits. The body requires no medicine no it new food is eaten only after the food that has already eaten before is fully digested and the food agrees with body.

Diet

The aims of dietary management are to achieve and maintain ideal body weight, euglycemia and desirable lipid profile, prevent and postpone complications related to diabetes and to provide optimal nutrition during pregnancy, lactation, growth, old age and associated conditions e.g. hypertension and catabolic illnesses.

The dietary recommendations should be individualized according to person's ethnicity, cultural and family background, and personal preferences and associated co-morbid conditions. It should be flexible in variety and preparation of food choices and timing of meals according to person's daily routine.

REVIEW OF MODERN LITERATURE

MODERN ASPECT

CONCEPTS OF DIABETES:

Diabetes is one of the ancient disease.The history of diabetes is stated in the Ebers papyrus(1500BC) polyuria and honey urine was noted as early as 400B.C by our Indian physician "susruta".He has described this disease as Madhumegam,the honey is the urine.

At present it is commonly known as "sugar in urine". In older days it was also known as melting of flesh.In Greek "diabetes"means "pass through" and in Latin "mellitus" means "honey" i.e presence of sugar in the urine.

This disease has been known to ancient Indian, chinesh, Arabic and Roman physicians.In 1674 Thomas wllis for the first time distinguish diabetes mellitus by the taste of urine. In 1688 Brunner removed the pancreas in dogs. In 1773 D0bson established that the urine sweet in diabetes mellitus because of the sugar in it.

In 1835 discovery of excess sugar in the blood in diabetes mellitus belongs to Ambrosiami. In 1855 Claude Bernand discovery of glucosuria in animal. In 1869 Langerhans found accumumalations of special cells in the pancrease, which lates were named "Islets of Langerhans" .Rassian scientist ULezko-stroganova was the first to emphasize the endocrine role of "Langerhans" is lets in 1881. Insulin affects carbohydrate metabolism was demonstrated by Mynkowsky and mehring in 1889-1892. In 1892 Mynkowsky first removed pancrease from a dog.

Canadian scientists Banting and Best in 1921 obtaining insulin from the pancrease of nnew born calf.The first insulin in the USSR was produced in 1922 under the supervision of Eigorn.

In 1936 Howsay demonstrated for the first time that besides Insulin.other hormones participate in the pathogenesis of diabetes mellitus.In 1942-1946 Loubatieres gave a explanation to many facts concerning the mechanism of action of sulphonyhreas in diabetes mellitus.In 1955 the English man Sanger established the molecular structure of insulin.

The synthesis of human crystalline insulin was accomplished by kastoyannis in the USA in 1963, and by zahn and fellow-workers in 1965.In 1972 a group of scientists leaded by Yudayev and sLvachkin in the USSR accomplished the laboratory synthesis of insulin that is identical with human insulin.

All the achievement in the past, the present diabetic can almost have a normal life span with usual activities by their diet.exexise, medicine and hygiene.The mortality was more due to diabetic coma, but now the longevity has increased by modern medicine.

Now a days the cause of death among diabetes with the onset at young age is due to diabetic nephrophathy and in adults, it is due to vascular disease.

DEFINITION:

Diabetes mellitus is a clinical syndrome, characterized by hyperglycemia due to absolute or relative deficiency of insulin.

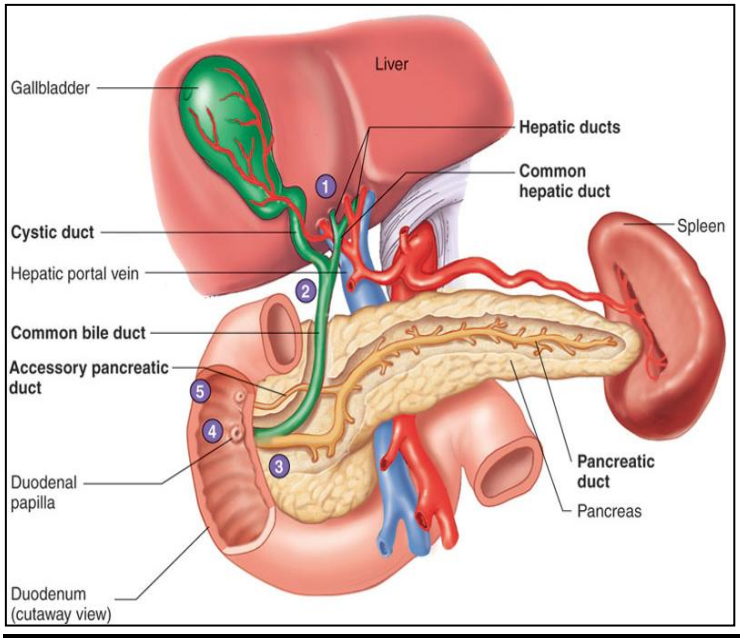
Lake of insulin affects the metabolism of carbohydrate protein, fat, and causes asignificant disturbance of water and electrolyte homeostasis.Death may result from acute metabolic decompensation while long-standing metabolic derangement is frequently associated with permanent and irreversible functional and structural changes in the cells of the body, with those of the vascular system being particularly susceptible.

These changes lead to the development of well defined clinical entities the so, called complication of diabetes.which characteristically affect the eye, the kidney,and the nervous system.

ANATOMY OF PANCREAS:

Definition:

The pancreas(pan=all,kreas=flesh)is a gland that is partly exocrine and partly endocrine.The exocrine part secretes the digestive pancreatic juice;and the endocrine part secretes hormones like insulin.it is soft ,lobulated and elongated organ.



LOCATION:

The pancreas lies more or less transversely across the posterior abdominal wall, at the level of first and second lumbar vertebrae.

SIZE AND SHAPE:

Pancrease is j-shaped or retort, set obliquely.the bowl of the retort represents its head, and the stem of the retort,its neck,body and tail. It is about 15-20cm long, 2.5-3.8cm broad and 1.2-1.8cm thick and weights about 90g.

The pancreas is divided (from right to left) into the head,the neck,the body and the tail.The hedd is enlarged and lies within the concavity of the duodenum.The tail reaches the hilum of the spleen.The entire organ lies posterior to stomach separated from it by the lesser sac.

Head of the pancreas:

Definition:

Head is the enlarged flattened right end of the pancreas,situated within the curve of the duodenum.

External features:

The head has three borders, superior, inferior, right lateral; twosurfaces, anterior and posterior; and one process, called the urcinate. This projects from the lower and left part of the head towards the left.

Relations:

The superior border is overlapped by the first part of the duedonum and is related to the superior pancreaticoduodenal artery.The inferior border is related to the third part of the duodenum and to the inferior pancreaticoduodenal artery.The right lateral border is related to the second part of the duodenum, the terminal part of the bile of the bile duct and the anastamosis between the two pancreaticoduodenal arteries.

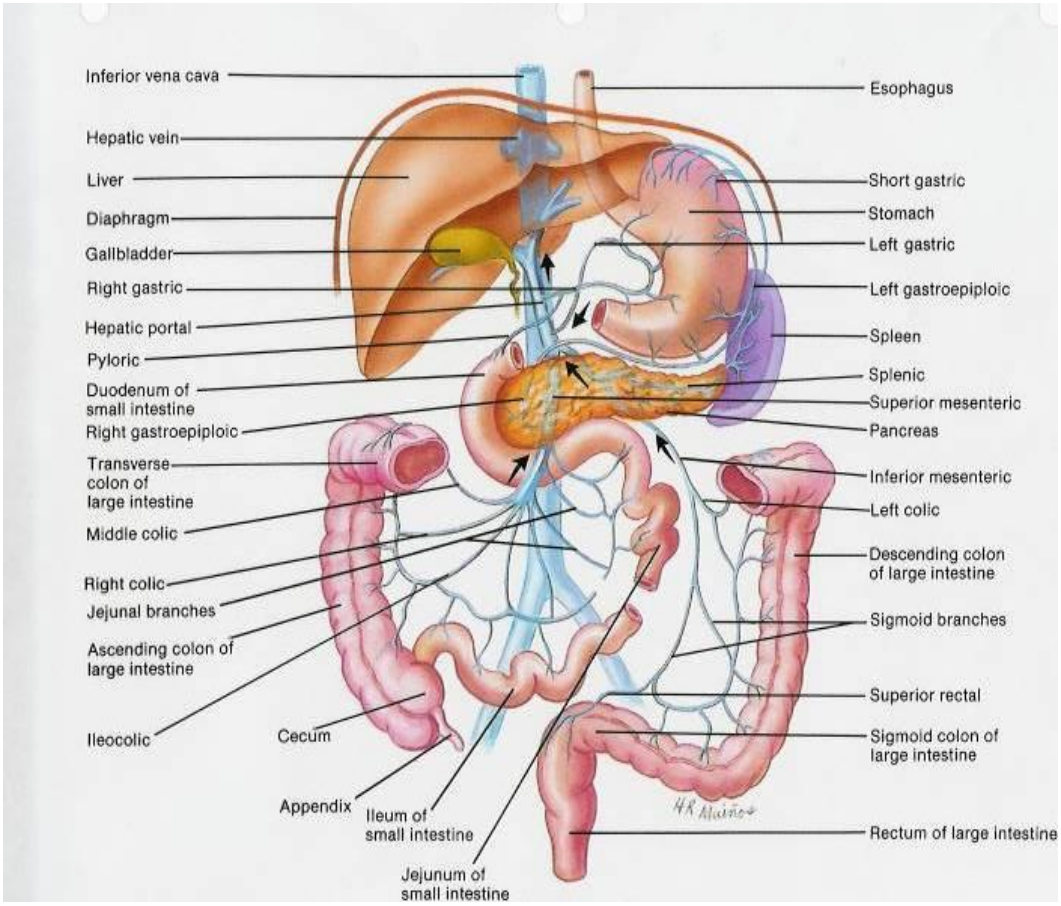
The anterior surface is related, from above downwards, to: 1) the gastroduodenal artery; 2) the transverse colon, and3) the jejunum which is separated from it by peritoneum.

The posterior surface is related to: 1)the inferior vena cava,2)the terminal parts of the renal veins,3)the right crus of the diaphragm and 4)the bile duct which runs downwards and to the right and often embedded in the substance of pancreas.

Uncinate process:

It is related anteriorly to the superior mesenteric vessels, and posteriorly to the aorta

RELATIONS OF PANCREAS



Neck of the pancreas:

This is the slightly constricted part of the pancreas between its head and body.it is directed forwards, upwards and to the left.it has two surfaces, anterior and posterior.

Relations:

The anterior surface is related toto:1) the peritoneum covering the posterior wall of the lesser sac,and 2)the pylorus.At its junction with the head there lie the gastroduodenal and superior pancreaticoduodenal arteries.

The posterior surface is related to the termination of the superior mesenteric vein and the beginning of the portal vein.

Body of the pancreas:

Defination:

The body of the pancreas is elongated.it extends from its neck to the tail.It passes towards the left with a slight upward and backward inclination.

External features:

It is triangular on cross-section,and has three borders(anterior,superior and inferior).A part of the body projects upwarts beyond the rest of the superior border,a little to the left of the neck. This projection is known as the tuber omentale.

Relations:

Three borders

The anterior border provides attachment to the root of the transverse mesocolon.The superior border is related to celiac trunk over the tuber omentale,the hepatic artery to the right,and the splenic artery to the left.The inferior border is related to to the superior mesenteric vessels at its end.

Three surfaces

The anterior surface is concave and directed forwards and upwards.It is covered by peritoneum. And is related to the lesser sac and to the stomach.

The posterior surface is devoid of peritoneum, and is related to

1) The aorta with the origin of the superior mesenteric artery,
2) The left crus of the diaphragm.
3) The suprarenal gland,
4) The left kidney,
5) The left renal vessels,and
6) The spleenic vein.

The inferior surfaces is covered by peritoneum,and is related to the duodenojejunal flexure,coils of jejunum and the left coliac flexure.

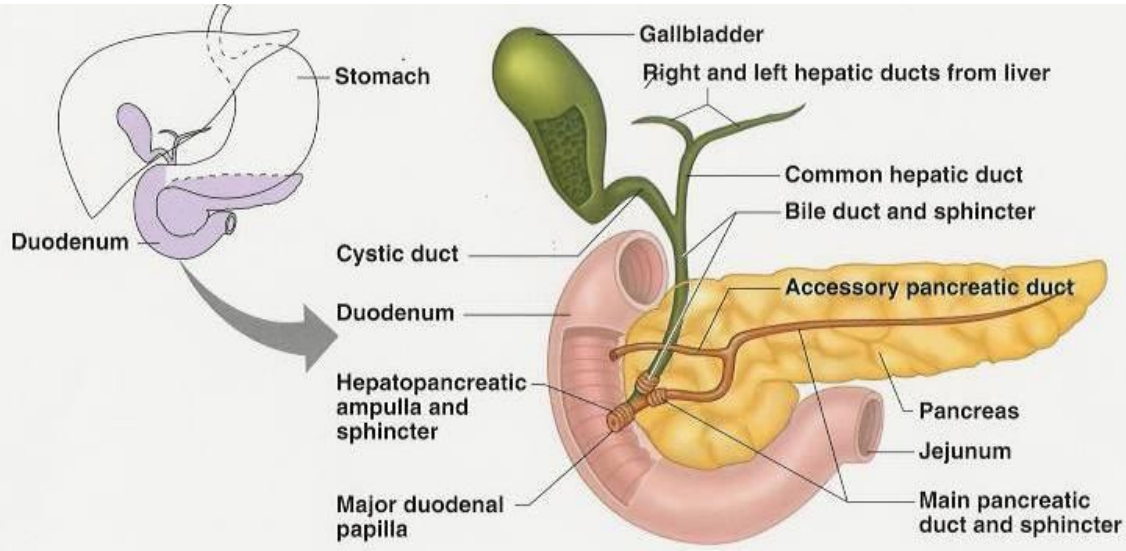
The tuber omentale projects upwards beyond the lesser curvature of the stomach, and is related to the lesser omentum across the lesser sac.it is the relationship to the omentum that gives process its name.

Tail of the pancreas:

This is the narrow left end of the pancreas. It lies in the linorenal ligament together with the splenic vessels.it comes into contact with the lower part of the gastric surface of the spleen.

DUCT OF THE PANCREAS:

PARTS OF PANCREAS



The exocrine pancreas is drained by two ducts, main and accessory.

1) The main pancreatic duct of wirsung lies near the posterior surface of the pancreas and is recognized easily by its white colour .I t begins at the tail;runs towards the right through the body;and bends at the neck to run downwards,backwards and to the right in the head.
2) Its lumen is about 3mm in diameter.
3) It receives numerous small tributaries which join it at right angles to its long axis forming what has been described as a 'herring bone pattern'.
4) Within the head of the pancreas the pancreatic duct is related to the bile duct which lies on its right side.The two ducts enter the wall of the second part of the duodenum and join to form the hepatiopancreatic ampulla of vater which opens by a narrow mouth on the summit of major duodenal papilla, 8to10 cm distal to the pylorus.
5) The accessory pancreatic duct of santorini begins in the lower part of the head,crosses the front of the main duct with which it communicates and opens into the duodenum aat the minor duodenal papilla.This papilla is situated 6to8 cm distal to the pylorus.The opening of the accessory duct lies cranial and ventral to the main duct.The two ducts remind the double origin of pancreas from the ventral and dorsal pancreatic buds.

Arterial supply:

The pancreas is supplied:

1) Mainly by pancreatic branches of the spleenic artery,
2) The superior pancreaticoduodenal artery and
3) The inferior pancreaticoduodenal artery

Like the duodenum the pancreas develops at the junction of the foregut and midgut, and is supplied by branches derived from both the celiac and superior mesenteric arteries.

Venous drainage:

Veins drain into spleenic, superior mesenteric and portal veins.

Lymphatic drainage:

Lymphatics follow the arteries and drain into the pancreaticosplenic, celiac and superior mesenteric groups of lymph nodes.

Nerve supply

1) Sympathetic
    a) coliac plexus
    b) Superior mesenteric plexus
2) Parasympathetic
    a) vagus nerve

Endocrine pancreas

The endocrine part of the gland is formed by the islets of Langerhan's. These islets are arranged into irregular plates. Tortuous blood capillaries supply them the cells are poly hedral in shape. The islemts are surrounded by awnar cells. There are about 2 million islets in the human pancreas. Each islet is about 200-300 micron in size.

Major cell Types:

1. Beta (β) cells: Comprise about 70% of islet cells and secrete insulin, the defective response (or) deficient synthesis of which causes diabetes mellities.
2. **Alpha (α) cells:** Comprise 20 % of islet cells and secrets glucagons which induces hyperglycemia.
3. **Delta (δ) Cells:** Comprise 5 to 10 % of islet cells and secrete somatostatin which suppresses both insulin and glucagons release.

4. **Pancreatic Poly peptide (P.P) cells (or) F Cells:** Comprise 1 to 2 % of islet cells sand secrete pancreatic polypeptide having some gastrointestinal effects.

A. Minor Cell Types:

1. D1 Cells elaborate vasoactive intestinal peptide (VIP), which induces glycogenolysis and hyperglycemia and causes secretory diarrhea by stimulation of gastro intestinal fluid secretion.

2. **Enterochromaff in cells:**

Synthesise serotonin, which in pancreatic tumours may induce carcinoid syndrome.

Physiology of the pancreas:

1. Functions of Exo crine pancreas:

The main functions of the exocrine pancreas are the alkaline secretion of digestive enzymes prominent among which are trypsin, chymotrypsin, Lactase, amylase, lipase and Phospholipase.

2. Functions of Endocrine Pancreas:

The endocrine portion of the gland the islets of Langerhans produce the hormones insulin and glucagons that play a key role in carbohydrate metabolism.

ISLET CELL STRUCTURE:

The islets of langerharns are ovoid, scattered throughout the pancrease, although they are more plentiful in the tail in the body and head.In humans, there are 1-2 million islets.

The cells in the islets can be divided into types on the basis of their staining properties and morphology.There are atleast four distinct cell types in humans.A,B,D and F cells.A,B and D cells are also called α,βand δ cells.

A cells 20% secretes Glucagon

B cells 75% secretes Insulin

D cells 5-10% secretes somatostain

F cell 1-2% secretes pancreatic polypeptide.

D cell 5-10% secretes vasoactive intestinal polypeptide.

Entro chromatin cells secrete serotonin.

The B cells is the predominant cell type that make up

INSULIN:

Insulin is secreted by Bcell or the beta cells in the islets of Langerhans of pancreas. It has molecular weight of 5808and contains 2 amino acid chains linked by disulfide bridges. Alpha chains of insulin contain 21 amino acids and beta chain contains 30 amino acids.

Insulin is a polypeptide containing two chains of amino acids linked by disulfide bridges Insulin is synthesized in the endoplasmic reticulam of the β cells. The half life of insulin in the circulation in humans is about 5 minutes.

EFFECTS OF INSULIN:

They are conveniently divided into rapid, intermediate and delayed actions.

Rapid Seconds)	Increased transport of glucose, amino acids and K+ into insulin sensitive cells.
Intermediate (Minutes)	Stimulation of Protein Synthesis
	Inhibition of protein degradation
	Activation of glycogen synthesis and glycolytic Enzymes
	Inhibition of Phosphorylase and gluconeogenic enzymes.
Delayed (hour)	Increase in MRANS for lipogenic and other enzymes.

The net effect of the hormone is storage of Carbohydrate, Protein, and fat. Therefore insulin is appropriately called the 'hormone of abundance' total daily release in man is about 50 IU out of a pancreatic store of 200-250 IU of insulin.

EFFECT OF INSULIN ON CARBOHYDRATE METABOLISM:

To facilitate rapid update storage and use of glucose by almost all tissues of the body mainly by muscles adipose tissue and liver.

Insulin Promotes muscle glucose uptake of metabolism:

Insulin facilitates the uptake of glucose by muscle and excess glucose is stored as glycogen.

Insulin Promotes liver uptake, Storage and use of Glucose:

Mechanism:

There is increase in the uptake of glucose by liver cells and also in the activity of enzymes that leads to glycogen synthesis. It in activates those enzymes that cause glycogen breakdown. Glucose is released from the liver, as blood glucose decreases Insulin converts excess glucose into fatty acids and inhibit neoglucogenesis in the liver.

II. EFFECT OF INSULIN ON PROTEIN METABOLISM & GROWTH:

1. Insulin promotes protein synthesis and storage. It facilitates the transport of amino acid into cell.
2. There is increase transcription of DNA and translation of messenger RNA.
3. Decrease catabolism of proteins is there
4. It decease the rate of gluconeogenesis
5. Insulin lack leads to protein depletion and increase the amino acids.
6. Insulin and growth hormone acts synergistically to promote growth.

EFFECTS OF INSULIN ON FAT METABOLISM:

Insulin promotes fat synthesis and storage mechanism. It increases the utilization of glucose by most of the body tissues which automatically decrease the utilization of fat thus, functioning as 'fat sparer'.

Insulin also promotes fatty acid synthesis. Almost all synthesis occur in liver cells and fatty acids are transported from liver by way of blood lipoproteins to the adipose tissue to be stored. It decreases the hormone sensitive lipase, thus preventing hydrolysis of triglycerides.

Insulin Deficiency leads to:

- ❖ Lipolysis of storage fat and release of free fatty acids.
- ❖ Increased synthesis of plasma cholesterol and phospholipids concentration.
- ❖ Excess use of fat during insulin lack causes ketosis and Acidosis.
- ❖ Excess acetoacetic acid is formed in the liver cells.
- ❖ Decrease utilization of aceto acetic acid in the peripheral tissues, thus so much of it is released from the liver that is cannot alebe metabolized by the tissue.

Role of Insulin in switching between carbohydrate and lipid metabolism:

Insulin promotes utilization of carbohydrate for energy and it will decrease the utilization of fat. Lack of insulin causes utilization of fat instead of glucose. The signal that controls the switching mechanism is principally blood glucose. So the role of insulin in the body is to control which these two foods (glucose & fat) will be used by the cells for energy.

BLOOD GLUCOSE REGULATION:

1. Liver has an important blood glucose buffer system.
2. Both insulin and glucagon functions as an important feedback control system for maintaining normal glucose concentration.
3. Decreased glucose stimulates sympathetic nervous system.
4. Growth hormone and cortisol are secreted in response of decreased blood glucose.

IMPORTANCE OF BLOOD GLUCOSE REGULATIONS:

- Glucose is the only nutrient for brain, retina and germinal epithelium of the gonads.
- It is important to maintain that blood glucose should not rise to high since.
- Increased osmotic pressure in extra cellular fluid leads to cellular dehydration.
- Increased glucose loss in urine
- Osmotic diuresis by the Kidney.
- Long term increase leads to vascular damage.

METABOLIC EFFECTS OF INSULIN AND GLUCAGONS:

Metabolic effects	Insulin	Glucagons
Secretion Stimulated	Hyper Glycemia	Hpoglycemia
Blood Glucose	Decrease	Increase
Liver glycogen	Increase	Decrease
Gluconeogenesis	Decrease	Increase
Protein Synthesis	Increase	Decrease
Fatty acid synthesis	Increase	Decrease
Triglyceride break down	Decrease	Increase
Glucose utilization	Increase	

CLASSIFICATION:

PRIMARY DIABETES MELLITUS:

1. Insulin dependent diabetes mellitus, immune dependent diabetes melltitus (IDDM) (or) type 1 (or) insulinopenic (or) juvenile onset diabetes (JOD) (1% to 20%).

In some individuals in later life a slow progression to insulin deficiency only occurs which is called latent auto immune diabetes of adults (LADA). This is associated with marked weight loss, un responsive hyperglycaemia with oral drugs. Pronenes to ketosis and presence of auto antibody.

2. Non insulin dependent diabetics mellitus. Non immune dependent Diabetics mellitus (NIDDM) (or) maturity onset diabetes mellitus (MOD) (or) Type II Diabetes (80% - 90%).

This is very commonly seen in adults and rarely in the yong. The cause is usually unknown. In most of these patients tissue insensitivity to insulin has

been found (genetic). This is at times more aggravated by ageing and visceral obesity of abdomen. There is also a lack of response of β cells to glucose and hyperglycaemia. Thus both these factors viz tissue insensitivity to insulin and lack of Betacell response to glucose are very important basic factors for the development of type II diabetes type II diabetes may be again to two types.

A. OBESE:

Here diabetes is secondary to extra pancreatic factors which local to insensitivity to endogenous insulin.

B. NON OBESE:

These patients generally show a blunted response (or) no response as regards insulin secretion after glucose load but albeit to other insulinogenic stimuli. Eg IX sulfonylurea, Glucagon (or) Secretin. They generally respond well to therapeutic oral hypoglycemic agents. In this group the following types are also seen.

I. MILD FORM OF TYPE I DIABETES:

Beta cell mass is contracted they become insulin dependent.

II. MATURITY ONSET DIABETES OF THE YOUNG (MODY)

There is a strong family history for Mody now five types of this syndrome have been identified.

SECONDARY DIABETES MELLITUS:

1. Non – Pancreatic endo crinal disorders

Acromegaly

Cushing's Syndrome

Thyrotoxicosis

Phaeochromocytoma

Glycagonoma

2. Pancreatic disease

Chronic Pancreatitis

Carcinoma of pancreas

Pancreatic Calculi.

Cystic fibrosis

Pancreatectomy

3. **Drug induced:**

   Steroids

   Thiazide diuretics

   Contraceptive pills etc.

4. **Miscellaneous:**

   Mumps

   Rubella

   Insulin receptor antibodies.

WHO CLASSIFICATION:

1. **Diabetes Mellitus:**

   (a) Insulin dependent diabetes mellitus – IDDM type I

   (b) Non insulin dependent diabetes mellitus- NIDDMType II

      (i) Obese

      (ii) Non obese

   (c) Mal nutrition related diabetes mellitus – MRDM.

   (d) Other types – Secondary

      Hormonal

      Drug induced

      Pancreatic

      Other abnormalities.

1. Impaired glucose tolerance – IGT
2. Gestational diabetes mellitus – GDM.

3. **DIFFERENCE BETWEEN TYPE I & TYPE II DIABETES**

S. No.		Type I	Type II
1.	Age	< 30 years	> 30 years
2.	Seasonal incidence	Present	Absent
3.	Autoimmunity	To islet cell, insulities, other autoimmune disease	Not present
4.	Heredity	HLA – DR3 (or) DR4 > 90%	NO HLA Connection
5.	Clinical	Markedly emaciated classical symptoms are present	Merkedly obese classical symptoms usually lacking.
6.	Serum Insulin	Less (or) Nil	May be less (or) more
7.	Kelo acidosis	Prone	Not so
8.	C-Peptide	Disappears	Remains
9.	Treatment	By insulin	Oral hypoglycaemic drugs rarely insulin.

AETIOLOGY:

Age: NIDDM occurs chiefly in middle aged individuals.

Sex : Both sexes suffer equally. But in lower age groups males and in middle age groups females are more affected.

Heredity:

If may run in families but there is complicating evidence for the made of inheritance. NIDDM has a greater hereditary component.

Stress and Strain:

Physical and mental stress (or) strain may be responsible of the disease as counter regulatory hormones are secreted in excess.

Infection:

Seasonal variations with winter and autumn peaks of increased incidence of diabetes have been described. These possibly indicate some environmental factors (or) infective factors in the causation of diabetes coxsackie B4 virus, rubella and encephalomia carditis may cause islet cell dysfunction

Obesity:

Two major mechanisms have been proposed for the tissue insensitivity to insulin in cases of obecity.

(i) Prolonged beta cell stimulation may lead to hyper insulin giving rise to receptor insensitivity.

They may be post receptor defect.

Histo Compatibility antigen (HLA –System)

About 95% of types 1 subject are having either HLA DR_3 (or) HLA DR_4.

Auto Immune Mechanism:

Diabetes is seen to be associated with various auto immune diseases such as myxoedema and Addison's disease.

Hyperglucagonaemia:

In IDDM hyperglucagonaemia is present.

Diet:

Bovine serum Albumin in milk may be an initiating factor in infancy when cow's milk is taken. Various nitrosoamines, coffee, gluten are also for development of IDDM.

HORMONAL ANTAGONISTS TO INSULIN

1. **Growth Hormone – Acromegaly**

Growth hormone hyper secretion affects the metabolism. So clinical diabetes mellitus (10%) occurs.

2. **Gluco carticoids – Cushing's syndrome**

Hyper fuction of the adrenal gland Cushing's syndrome is defined as the symptoms and signs associated with prolonged in appropriate elevation of free corticosteroid levels. Increased gluconeogenesis may lead to impaired glucose tolerance. So diabeties mellitus occurs.

3. Thyroid hormone – Hyper thryoidism:

In hyper thyroidism rapid rise of blood glucose follows a meal and the concentration exceeds the normal renal threshold, during this time glucose will be present in the urine.

4. Catecholamine excess:

Tumor is found in adrenal medulla excess of catecholamine produced. This causes glucose intolerance. So diabeties occurs.

PATHOGENESIS:

It is a complex affair and is still for from clear, Lack of insulin, presence of insulin antagonists, excessive neoglucogenesis, viral infection HLA system, heredity, auto immunity jointly (or) in proportion are possibly responsible for the development of diabeties.

Due to lack of insulin blood sugar level steadily rises and when it crosses the renal threshold level of 180mg/ 100cc glycosuria results. Renal threshould level however varies with age and pregnancy. Glucose increases the osmolarity of glomerular fillrate and also backs the obligatory volume of water during elimination. This result in profuse diruresis even up to 10 to 15 liters per day associated with hyponatraemia, hypokalaemia and hypomagnesaemia. Thus intense thirst, dehydration shock and crystalloid imbalance may develop.

Again as sugar is not burnt, adequately for energy requirement, fat is mobilized from the adipose tissues and large quantities of free acid circulate in the blood. Normally these are burnt in the liver. But as these are produced in large quantities of acetone which accumulate in the blood and ultimately appear in urine and breath.

Several hormones particularly the growth hormone may help in this process incompletely metabolized carbohydrate Eg. Pyruvic acid and lactic acid also accumulate in the blood. This condition is called diabetic keto acidosis which often leads to coma and death.

Liver is enlarged due to fat infiltration and blood contains enormous amount of neutral fat and various hormones may act as insulin antagonists. Apart from there, insulin antibodies may be produced in the blood. Lastly due to continuous loss of sugar in the urine the process of neoglucogenesis from protein maybe stimulated which may result in wasting of muscles and increased urinary loss of nitrogen.

CLINICAL FEATURES:

Onset is usually gradual but rarely there may be acute onset. There may be no symptom (or) sign and the disease may be diagnosed during routine investigation (or) examination. Sometimes patients may present features of complications of diabetes. The classical features of diabetes may be seen as follows:

Polyuria:

The amount of urine may be several litres in 24 hours. This is due to excessive sugar in the urine which acts as a diuretic.

Polydipsia (or) Excessive thirst:

Patient may consume several litres in 24 hours. This is due to polyuria and hyper osmolarity of blood. Blurred vision is also due to it.

Poly phagia (or) Excessive hungers:

Patient always feel hungry and may have a craving for carbohydrate food, sweet, honey, sugar rice etc. This symptom is due to non utilization of sugar for energy expenditure.

Rapid emaciation:

There may be rapid loss of weight. Initially it is due to loss of water, glycogen and triglyceride stores. Gradually reduced muscle mass.

Dryness of mouth and throat:

This is the effect of Poly uria.

Constipation:

The stool becomes hard and bowel movement may take place after every 2 to 3 days.

Itching:

This is an important symptom and is located in the anus (or) external genitalia. This is due to irritant action of sugar on the tissue and fungal (or) bacterial infections.

Signs:

Clinical signs may be absent. In IDDM cases emaciation may be a dominant feature. Other signs depend on the presence of complications and degree of dehydration degree of consciousness may vary depending on the hyper osmolarity, ketosis, electrolyte imbalance and dehydration. Loss of subcutaneous

fat and muscle wasting are important features of IDDM Hypotension is a serious sign with increased Chylomicrons and triglyceridemia, lipemia, liver is enlarged due to fat infiltration, chronic skin intection may be present. In females, candidal valvo vaginitis, hydramnios, toximeas of pregnancy, unexplained fetal loss on birth of large babies are seen.

Diagnosis:

Whenever diabeties is suspected the diagnosis should be confirmed by Glucose Tolerance Test (GTT) (or) more easily by fasting and post prandial blood sugar estimation. For the first, the patient should take adequate carbohydrate diet (250) gm) for about 7 days (or) least for 3 days prior to the test. After an overnight fast of 8 to 12 hours in the early morning the fasting samples of blood and urine are collected. After this the patients ingests 75gm of glucose (1.75 gm of glucose / kg of ideal body weight) dissolved in 250-300 cc of water. Following every half an hour of this blood samples and one hour of this urine samples are collected upto 2 ½ hours. Now sugar level is estimated in all samples and in urine test for acetone is also done.

Fasting blood sugar
Normal fasting blood sugar level - 80 – 120 mg%
(But true glucose level will be less than this)
Diagnostic fasting blood sugar level >140 mg% (7.8 mmol /L)
Post prandial blood sugar (2 hours after meals)
Normal post prandial blood sugar level < 140 mg (7.8 mmol / L)
Diagnostic post prandial blood sugar level 200 mg%
At risk for diabeties P.P.Sugar level 140 – 200 mg %

According to WHO

Fasting Blood Sugar

- Normal fasting Blood sugar level - <100 mg/ (6.1 mmol /L)
- Impaired fasting glucose level (IFG) 100 mg% to 126 mg % (7mmol / L)
- Diagnostic fasting blood sugar level > 126 mg%

GTT after 2 hours

- Normal blood sugar level < 140 mg% (7.8 mmol / L)
- Imparied glucose tolerance (IGT) 140 mg % to 200 mg%
- Diagnostic Blood sugar level > 200 mg % (110 / mmol / L)
- Note: GTT is only required in border line cases and for diagnosis.

Intravenous glucose tolerance test

It is done when there is gastro intestinal upsets (or) inadequate absorption of glucose is given IV and the fall of sugar level is observed in 1 ½ to 2 hours.

Cortisone glucose tolerance test

50 mg of cortisone is given to the patient 8 hours and at 2 hours before oral GTT. Sugar level is indicates the reserve capacity of beta cells or hyper glycaemia.

Intravenous talbutamide test

After giving an IV dose (or) 1 gm of sodium talbutamide, blood sugar levels are estimated at 0, 20, 30 & 40 minuts, as talbutamide stimulates insulin secretion from beta cells. This test indicates secretary capacity of insulin by beta cells.

Serum insulin level during GTT

Normal fasting insulin level 10 – 25 µu/cc

Normal 1 hour insulin level 50 – 130 µu/cc

Normal 2 hour insulin level < 10 µu / cc

Unresponsiveness (or) insensitivity of glucose at 1 & 2 hours > 100 µu/cc

Glycosylated Hb AI (C)

Normal level – 5 % to 7 %

The major form of HB AI (c) normally comprises 4% - 6% of total Hb. The remaining 2 % - 4% of Hb is phosphorylated glucose (or) fructose and termed as HbIa and HbIb respectively.

Serum Fructosamine level

Normal level 1.5 – 2.4 mmol / L. When the serum albumin level is 5 gm/dl.

When Hb AIc estimation become difficult due to abnormal Hb or Haemolytic state or in diabetic women recently becoming pregnant this assessment is advantageous.

Lipoprotein – abnormalities

In IDDM cases

Triglyceride	-	slight rise
LDL	-	slight rise
HDL	-	No change

Insulin Resistance cases

Triglyceride	-	High
LDL	-	Qualitative change in LDL particles.
HDL	-	Low

Urine:

Normally sugar is absent. When the blood sugar level croses the renal threshould level of 130 mg% sugar appear in the urine. But in presence of high renal threshold level three to no glycosuria. Again when the threshold is low, sugar is present in urine (Renal glycosuria). Threrefore glycosuria cannot be accepted as diagnostic of diabetes. Some drugs interfere with the determination of sugar in urine and false negative results are obtained. These drugs are vitamin C, Salicylate, methyldopa and leuodopa. Kelone bodies in urine at a time indicate diabetic keto acidosis.

Causes of Glycosuria:

- **Diabetes Mellitus:**

This is very common cause of glycosuria in elderly patient.

- **Renal glycosuria**

Low Renal threshold sugar comes out in the urine

- **Alimentary glycosuria**

After ingestion of 500gm of glucose sugar may appear in the urine.

Complications of Diabetes:

Virtually every tissue and organ is biochemically and structurally altered as a consequence of the hyperglycemia of diabetes. These for the complications of the disease. Two biochemical mechanisms appear to be involved in the development of many complication. In the first glucose reversibly bind to the body proteins. This is a non enzymatic event that can cause structural and

functional abnormalities of the involved proteins. The concentration of glycosylated hemoglobin in the blood is now used clinically as a measure of therapeutic control.

The second biochemical mechanism operates in the aorta, lens of the eye, Kidney and peripheral nerves. These tissues are endowed with an enzyme, aldose reductase that facilitates the accumulation of sorbital and fructose in cells of the hyperglycemic patient. As a result of the intracellular accumulation of sorbital and fructose an osmotic gradient is established and excessive amounts of water enter the cells from the extra cellular compartment. The cells then swell and are damaged.

ACUTE COMPLICATIONS:

Hypoglycaemia:

Hypoglycaemia, defined as a blood glucose concentration of less the 2.5 mmol/L, occurs commonly in diabetic patients treated with insulin and infrequently in those taking a sulphonylurea drugs. Severe hypoglycaemia result in severe morbidity ie coma, convulsion, brain damage, stroke, myocardial ischaemia, vitreous haemorrhage, hypothermia and accidents.

Symptoms of Hypoglycaemia:

Sweating, trembling, hunger, anxiety, confusion, drowsiness, speech difficulty nausea, tiredness and headache.

Diabetic ketoacidosis:

As a result of polyuria there will be dehydration, hyponatremia and hypopotassemia. The intra cellular water comes out in the extra cellular space as a result of hyperosmolarity of the extracellular fluid due to increase of glucose. Plasma volume is decreased, blood pressure falls and renal blood flow is diminished resulting in oliguria. The cellular glycogens and protein are catabolished.

Sugar is not burnt, fat is metablished from the adipose tissues and large quantities of free fatty acid circulate in the blood. Normally these are burnt in the liver, into carbondi oxide and water. But as these produced in large quantities.

Acetyl to enzyme – A – accumulates and after condensation forms acetoacetic acid and its derivatives beta hydroxybutyric acid and acetone, which accumulate in the blood and ultimately appear in urine and breath. Incompletely metabolised carbohydrate. Eg pyruvic acid and lactic acid also accumulate in the blood. This condition is called diabetic ketoacidosis.

When ketone bodies accumulate the plasma osmolarity is raised and the cells are further dehydrated ultimately ph of the blood falls. This acidosis stimulates the respiratory centre where by pulmonary ventilation is increased giving rise to air hunger. The breath contains the smell of acetone which is very diagnostic of this condition. The function of the brain cell is depressed and gradually Como supervenes.

CLINICAL FEATURE:

Patient is usually dehydrated, with sunken eyes dry skin and tongue and prominent malar bones. The ocular tension is low (Krauss's sign). There is kussmaul's air hunger with hissing respiration. Bp is usually low, pulse is low in volume, oliguria, abdomen may be regid and tender, vomiting, smell of acetone is present in the breath, diminished reflexes, Ketonuria, glycosuria, blood shows hyper glycaemia and reduced plasma bicarbonates, evidence of insection eg, boils, carbuncles and respiratory wact injection are very common patient may be couscious initially but gradually drowsiness and coma supervene.

NON-KETOTIC, HYPEROSMALAR COMA

In this condition due to very high blood sugar level dehydration occurs, leading to dehydration of the brain cells resulting in coma and convulsion. Marked elevation of BUN and creatinine is seen in this condition. But no ketoacidosis.

CHRONIC COMPLICATIONS:

OCULAR COMPLICATION:

1. **Diabetic Cataract**

   There may be senile cataract with clouding of the lens. Sometimes, true diabetic cataract may develop and in seen in juvenile cases.

2. **Diabetic Retinopathy**

   This consists of venous engorgement, Micro anaeurysm of the retina and hard exudates.

3. **Glaucoma develops in 6% cases.**

4. **Error of retractions**

   Due to increased sugar content of the retractive media may develop.

5. **Iritus rubeosa**

   The papillary margin of the iris may become red due to micro aneurysm and this may lead glaucoma after words.

6. Infection may develop complications of conjunctivae cornea, sclera and eyelid.

7. Pupillary changes.

SKIN COMPLICATIONS:

Skin infections are very common as sugar in tissues and sweat acts as a good media for bacterial growth, boils, carbuncles and fungal infections particularly monilial infection of vulva and penis are very common.

COMPLICATIONS DUE TO DRUGS:

Hypoglycaemia coma, Allergic reaction, insulin Lipodystrophy in the site of injection, immune insulin resistance. Cardio vascular complications with tolbutamide therapy and lactic acidosis with phenformin therapy may develop.

VASCULAR COMPLICATIONS:

Macro vascular complications are very common. Cerebral coronary, renal, limb arteries and abdominal aorta may be affected by the process of arteriosclerosis due to this there may be ischaemic heart disease with (or) without thrombosis, cerebro vascular accident (CVA), ischaemic limb with (or) without gantene, renal artery stenosis etc. longer duration of disease, advancing age, hyper insulenimia due to insulin resistance syndrome, obesity, Hyperlipidimia, particularly high triglyceride and low HDL, proteinuria and systolic hypertension will aggravate macro vascular complications.

RENAL COMPLICATIONS:

1. Renal arteriosclerosis.
2. Pyelo nephritis - This is very common and may lead to chronic renal failure.
3. Micro albuminuria
4. Papillitis, Necroticans- The renal papillae will show necrosis, ultimately leading to viaemia.
5. Kimmelstiel – Wilson syndrome (K.W. syndrome)
   This is develops in average 10 years duration clinically patient will present feature of nephritic syndrome.

NEUROLOGICAL COMPLICATION:

1. **Peripheral neuritis (30%)**
   It is very common in lower limbs. There may be poly neuropathy, Mononeuritis, painful neuropathy, Trophic ulcer may also develop.
2. **Autonomic imbalance**
   Impotency, postural hypotension, nocturnal diarrhea, loss of sweating, nocturnal sweating, cardiac irregularities, fixed pulse rate, resting tachycardia, sudden death, cold feet, pedal oedema, small pupil, delayed light reflex, resistance to mydriaties etc may develop.
3. **Diabetic amyotrophy**
   This consists of wasting, weakness of pelvic girdle muscle and thigh muscles.
4. Different cerebro vascular accidents.
5. Charcot's joint

Sexual and Genital Complications:

Impotence and frigidity may develop erectile dysfunction in males is particularly important. Balanitis and Balanoposthitis are common complications in males. These are due to secondary infection as urine contains heavy amount of sugar and nitrogenous materials. Leucorrhoea may develop in females.

Pulmonary complications:

Tuberculosis is very common in diabetes other infective complications like pneumonia, bronchopneumonia, pleurisy etc.

Effects on pregnancy and neonates:

There may be miscarriages and abortions toxaemias of pregnancy, hydramios etc Herculian child may be born of diabetic mothers due to secretion of excess of growth hormone.

TREATMENT:

1. Diet regime
2. Diet and oral hypoglycaemia agents
3. Diet and insulin
4. Special treatment for complications.

Diet regime:

Obese diabetics are to be given a reducing diet. On the other hand lean and thin diabetics should given a weight gaining diet. In case of mild diabetes with obesity diet control alone is required.

- ❖ Ideal calorie requirement is 20 calories per kg of body weight
- ❖ Protein requirement is 1 – 1.5 gm per kg of body weight for adults & 2-3gm per kg of body weight for children.
- ❖ Carbohydrate requirement is 2 gm per kg body weight.

In practice 40% to 60% of the total calorie should come from carbohydrate. Total fat should be < 35% of the energy take. Low fat products should be encouraged. Regarding fat it is better to prescribe unsaturated fat to keep the cholesterol level under control. A well balanced nutritions diet remains the fundamental key in the therapy of diabetes.

CALCULATION:

- ❖ Ideal weight of an adult is 60 kg.
- ❖ Therefore, the total calorie requirement will be 60 x 20 = 1200 kilo calorie.
- ❖ Protein requirement = 60 x 1 = 60 g which is gives 60 x 4.1` = 240 kilo calories.

- Carbohydrate requirement = 40 % of 1200 kilo calories 40% of 1200 kilo calories = 480 kilo calories. i.e. 120 gm of carbohydrate give = 480 kilo carlories (430 -4 = 120)
- Thus protein and carbohydrate will make about (240 + 430) – 720 kilo carlories.
- Fat will yield about 1200 – 720 = 480 kilo calories which may be obtained from 480 ÷ 9.1 = 52 gm of fat.
- Thus a patient of about 60 kg will require

Protein - 60gm
Fat - 52 gm
Carbohydrate- 120 gm

Many patients require between 1800 and 2500 kilo calories and women require a little less than this. For growing children, pregnant mothers (or) hard working labourers' provisions for additional calories should always be made.

Fibre Diet:

Plant components eg. Cellulose, gum and pectin are not digested and are termed dietary fibre. In soluble fibres eg, cellulose (or) hemi cellulose which are present in bran increase intestinal transit and other soluble fibres eg, Gums, pectin, etc which present in beans, apple skin and oat meal try to retard intestinal absorption. Therefore the later fibres will minimize glucose absorption and hence minimize hyperglycaemia. Moreover, these high soluble fibres have some cholesterol lowering property.

Salt

Salt in the diet should not be 6gm/day but it should be less in case of hypertention.

Vitamins and antioxidants;

These should be quite adequate in diet menu of diabetic patients.

Nutritional Approach:

Lifestyle modifications are the cornerstone of management of diabetes mellitus and include the prescription of a healthy diet, regular exercise, the management of stress, and avoidance of tobacco

The approach consists of provisions of adequate calories for maintaining or attaining standard weight for age sex and height.

General consensus on proportion of food constituents are

Carbohydrate 60 – 65 % (complex form have more fibre)

Fat 20 – 25 % (saturated 7.8 % polyunsaturated 7-8% Mono unsaturated 7.7%) Proteins 10 – 15 %

Disease like Madhumegam has very important role in diet and Nutrition. Diseases are largely due to irregular dietary habits. The body requires no medicine no it new food is eaten only after the food that has already eaten before is fully digested and the food agrees with body.

Diet:

The aims of dietary management are to achieve and maintain ideal body weight, euglycemia and desirable lipid profile, prevent and postpone complications related to diabetes and to provide optimal nutrition during pregnancy, lactation, growth, old age and associated conditions e.g. hypertension and catabolic illnesses.

The dietary recommendations should be individualized according to person's ethnicity, cultural and family background, and personal preferences and associated co-morbid conditions. It should be flexible in variety and preparation of food choices and timing of meals according to person's daily routine.

Dietary Recommendations:

Total Calorie Intake:

The calorie requirements of diabetic person depend on physical activity and nutritional status as in a normal individual, unless there is glycosuria. Individual with > 120% of ideal weight is considered overweight and < 90% of ideal weight is underweight. The ideal body weight (IBW) is calculated by formula:

IBW = (height in cm - 100) x 0.9

The caloric intake of person with diabetes should be altered gradually, preferably not more than 500 Kcal per day.

Total calorie distribution:

a) Carbohydrate (55-60% of total calorie requirement)

- Avoid sugar, honey, jaggery and sweets.
- Restrict processed refined food like maida-based products.
- Main source should be cereals, mixed coarse grains, whole pulses, salads and soybeans.

- Roots and tubers should be used sparingly.

b) Fibers

Traditional Indian diet is rich in fibers. Fiber rich foods include whole grains (ragi, jowhar, barley, oats etc.), whole pulses, soybean, green leafy vegetables and fenu-greek seeds.

c) Protein (10-15% of total calorie requirement):

Protein from vegetable sources, low fat milk and milk products, fish and lean meat is preferable.

d) Fat (20-25% of total calorie requirement):

- Saturated fat - <7% of total caloric intake (including ghee and butter)
- Rest should be in from of MUFA and PUFA
- N6/N3 ratio = 5-10
- Trans-fatty acid (hydrogenated vegetable oils) should be avoided.
- Dietary cholesterol should be minimal and in any case should not exceed 300mg per day.
- Use more than one edible oil.
- Oils containing linoleic acid (n-6) only such as ground nut, sesame, cotton seed, rice bran and safflower should be used along with oils containing a-linoleic acid (n-3) such as soyabean, mustard, canola etc.

e) Fruits:

Whole fruits are recommended in moderation (1-2 servings), however, very sweet fruits and fruit juices should be avoided.

Artificial sweeteners:

Use of artificial sweeteners in limited quantity is acceptable but they are to be avoided During pregnancy and lactation.

f) Common salt:

Up to 6g/day is permitted. Restrict pickles, papad, chatni and salty processed foods.

Alcohol δ Tobacco

- Alcohol intake is best avoided and if used, must be in moderation. It may exacerbate

Neuropathy, dyslipidemia, obesity and may worsen the control of diabetes.

- Smoking and the use of tobacco in any form should be prohibited.

DIABETIC DIET REGIME

DIABETES PATIENTS WERE ADVISED TO TAKE FOOD IN THEFOLLOWING MANNER

Time	Food regime
Morning 6.00	Coffee/Tea/ Milk(without sugar)/1cup
Morning 8.30	Idly -2/dosai-2/chappathi -2/pongal-1 cup
Morning 11. 00	Butter milk -1cup/lemon juice/vegetable soup/ Any fruit -1
Afternoon 1.00	Rice -2 cup/chappathi -3/sambar/ kerai koottu/poriyal/ rasam/butter milk
Evening 4.00	Moong dal -1 cup/any pulses/coffee/tea
Evening 6.00	any fruit-1
Night -8.00	Chappathi-2/rice -1 cup/idly -2/dosai -2
Before bedtime	milk without sugar -1 cup

EATING RULES FOR DIABETICS

People suffering from diabetes should follow certain rules during eating.

- For diabetics, water or any other liquid is recommended only after half an hour after a meal. But if a person wishes, he can drink water or any liquid half an hour before eating but not during eating.
- Food should not be eaten hurriedly by diabetic person. Food should be eaten very slowly and chewed properly.
- Diabetic patient should not keep fast as missing of meal which may be highly dangerous.
- Diabetics should never eat to a full stomach.
- Diabetic patient should not smoke or drink alcohol while eating.
- In a people with diabetes, the raw vegetables and raw fruits should not be taken together.

- After a principal meal, they should not do any heavy work.
- Diabetics should take 5-6 small, frequent meals rather than 3 big meals.
- They should take 15-20 glasses of water daily to avoid constipation.
- Diabetic persons should discard the skin of chicken pieces as it contains fat.
- If a diabetic feel giddy, it indicates that the blood glucose level has fallen, a condition known as hypoglycemia. So, in this condition they must immediately eat energy giving food like apple or drunken fruit juices.
- They should have normal eating at correct times and at regular time intervals.
- During eating, if sodium is to be restricted then they should avoid spinach, peas, carrot.

மேகம் இருபதுக்கும் பத்தியம்

பசுமேக மிருபதுக்கு பத்தி யந்தான்
பாங்காக வுரைத்திடவே பசுவின் வெண்ணெய்
பொரிவாக எருமை மோர் பொன்னாங்காணி
பேர்பெற்ற சிறுகீரை முசுட்டை யாகும்
ஆரிவாக அவரையொடு புடல் முருங்கை
அதிசரமாங் கண்டு சருக் கரையு மாகும்
மரிவாக மாதளையாம் பேரீத் தாகும்
மகாவிளம் பழமுந்திரிப் பழமு மாமே
ஆமேபே யன் வாழைப் பழமுங் கச்சல்
அத்தியிடப் பிஞ்சுசிறு பயறுபழஞ் சோறு
பாமேபழஞ் சோற்றுநீர் வெந்தயஞ் சீரகமாம்
பாகல் பீரிக் கங்காய்கரு வேப்பிலை கொத்தமலி
நேமேநெற் பொறி எள்ளு முசுமுசுக்கை யாகும்
நேரான நல்லெண்ணெய் புண்ணக்குளுந்து
தாமேக மிருபதுக்கும் பத்ய வர்க்கம்
சாற்றினார் சிவன்றானுந் தாய்க்குத் தானே.

யூகி வைத்திய சிந்தாமணி – 156

The following foods are preferable to the diabetics

கீரை வகைகள் (Greens)	**காய்கறிகள் (Vegetables)**
❖ பொன்னாங்கண்ணி	அவரை
❖ சிறுகீரை	புணல் முருங்கை
❖ முசுட்டை	வாழை கச்சல்
❖ கொத்த மல்லி	அத்தி பிஞ்சு
❖ கருவேப்பிலை	பாகல்
❖ முசுமுசுக்கை	பீர்க்கு

கொழுப்பு பொருட்கள் (Fat)	**விதை (Seed)**
பசுவின் வெண்ணெய்	வெந்தயம்
எருமை மோர்	சீரகம்
நல்லெண்ணெய்	எள்

மாவு பொருள் (Carbohydrate**)**	**விதை (Seed)**
பழஞ்சோறு	சிறுபயிறு
பழஞ்சோறு நீர்	உளுந்து
நெற்பொறி	

The few fat substance has curative nature against Madhumegam that is

Cow’s butter cures prameg

Buffalo’s butter milk cures thrist

Gingelly oil cures disease of eyes, ear, skin, infection like scabies, ulcer etc.

In generally oil, lipid peroxidation is reduced and vascular complications risks are lessened.

Most of the above foods used for Neerizhivu are ‘Sathva gunam” This clearly implies in Neerizhivu patients the stress level is very high and to reduce that stress improve tolerance of disease Sathvaguna foods are used.

PHYSICAL EXERCISES

The need for walking is emphasized by Therayar as

"நண்பு பெற வுண்ட பின்பு குறுநடையுங் கொள்வோம்"

தேரன் பிணியணுகா விதி

.

Regular physical exercise has important physiological and psychological benefits for all diabetes. It should be followed 5 days a week for duration of 40 minutes each day and its severity should achieve 50 – 70 % of individual maximum oxygen up take.Diabetics may undertake any one of the simple exercises like **'jogging, swimming and cycling etc….'**

Dr. M.M.Ahuja.

Simple sustained exercise like walking atleast 30 minutes is very important for diabetics.

Dr. Mani S.B.

Brisk walking for only 30 minutes a day was just effective as 1 ½ hours per week of vigorous exercise in reducing the risk of heart diseases (30-40% reduction).

– 'Rastrya Ratna' Dr. T.Subramanian

Benefits of Exercise:

- Exercise helps in weight reduction. Appropriate exercise and diet control together are sufficient to control blood glucose levels in many mild type 2 DM patients thus avoiding drug treatment.
- Exercise leads to lowering of blood pressure, which is commonly elevated in diabetes.
- Exercise help in reducing blood levels of VLDL & LDL and increase the blood level of HDL cholesterol exercise improves blood circulation in the legs (Many long standing diabetics suffer from poor blood circulation in the legs.)
- The ideal time to perform exercise is in the morning after a light snack. This ill prevent hypoglycemic reactions
- "Exercise training has been reported to improve whole body glucose metabolism in man and experimental animals.
- Insulin mediated glucose uptake has significantly increased in skeletal muscle.
- The glucose transporter isoform responsible for insulin and contraction – stimulated glucose uptake in skeletal muscle is termed GLUT 4. The exercise

training increases GLUT 4 concentration in skeletal muscles and decreases hyperglycaemia."

– **Endocrine journal (Japan)**

– **Endorcrine Society Vol 49 Oct 2002**

Evaluation of the people with diabetes before exercise:

Before beginning an exercise programme, the individual with diabetes mellitus should undergo detailed medical evaluation with appropriate diagnostic studies. Strenuous physical activity including weight lifting should be avoided in the presence of coronary artery disease, proliferative diabetic retinopathy, nephropathy, and autonomic neuropathy.

❖ Person with diabetes and coronary artery disease should be prescribed appropriate exercises after cardiac evaluation.

❖ In proliferative diabetic retinopathy, strenuous activity like diving, weight lifting or bending may precipitate vitreous hemorrhage or retinal detachment. Walking may be continued.

❖ Peripheral neuropathy results in loss of protective sensation in the feet. It is advised to wear proper footwear. The person with diabetes should be taught to monitor for blisters and other damages to the feet before and after exercise.

❖ Autonomic neuropathy may limit an individual's exercise capacity and increase the risk of adverse cardiovascular events during exercise.

❖ Person with diabetes and overt nephropathy often have reduced capacity to exercise.

YOGA:

Yogic Physical exercise makes the muscles healthy and strong. It also tones up all the involuntary organs of the body which are concerned with the processes as digestion, evacation, circulation, respiration and secretion and through them, the autonomic nervous system which regulates their activities.

-Yogic Aganas for health & Vigour.

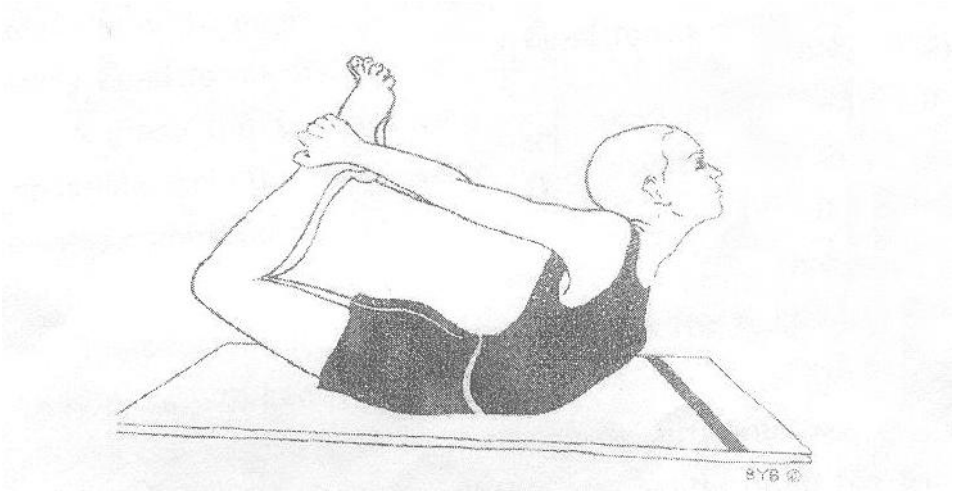
Gpuhzhahkk (Pranayanam)-breathing excersice :

Breathing is regulated with inspiration, expiration and retention of air in the ratio of 1: 2: 4

On practicing pranayamam regularly, supply adequate oxygen to nerves cells. The cells of the brain and spinal cord consume much more oxygen. It makes the mind alert and improves concentration.

V.G. Rali

Specific Asanas for Diabetes – Dhanurasana

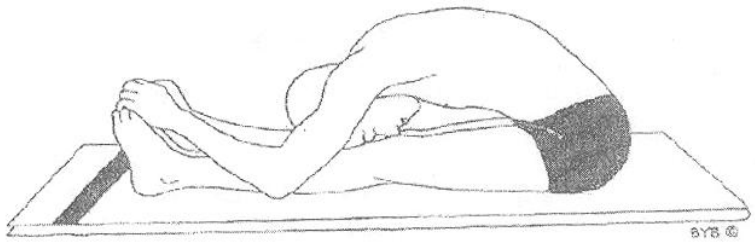


தனுராசனம் (Dhanurasana)- Bow pose

- Lie flat on the Abdomen with the legs and feet together and the arms and hands beside the body.
- Bend the knees and bring the heels close to the buttocks.
- Clasp the hands around the ankles.
- Place the chin on the floor.
- This is the starting position.
- Tightens the leg muscles and push the feet away from the body. Arch the back, lifting the thighs, chest and head together.
- Keep the arms straight.
- In the final position the head is tilted back and the abdomen supports the entire body on the floor. The only muscular contraction is in the legs; the back and arms remain relaxed.
- Hold the final position for as long as it is comfortable and then slowly relaxing the leg muscles, lower the legs, chest and head to the starting position
- Release the pose and relax in the prone position until the respiration returns to normal.

Benefits: The pancreas and adrenal glands are toned balancing the secretions. It is recommended in Yoga therapy for diabetes.

பச்சிமோத்தாசனம்(Pachimottanasana) -Back stretching pose



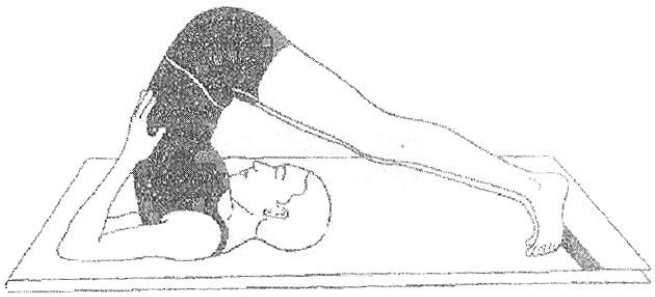
- Sit on the floor with the legs outstretched, feet together and hands on the knees.
- This is the starting position.
- Relax the whole body
- Slowly bend forward from the hips, sliding the hands down the legs. Try to grasp the big toes with the fingers and thumbs. If this is impossible, hold the heels, ankles or any part of the legs that can be reached comfortably.
- Move slowly without forcing or jerking.
- Hold the position for a few seconds. Relax the back and leg muscles allowing them to gently stretch.
- Keeping the legs straight and utilizing the arm muscles, not the back muscles, begin to bend the elbows and gently bring the trunk down towards the legs, maintaining a firm grip on the toes, feet or legs.
- Try to touch the knees with the forehead. Do not strain.
- This is the final position.
- Hold the position for as long as is comfortable and relax.
- Slowly return to the starting position.

Benefits: It tones and massages the entire abdominal and pelvic region including the liver, pancrease, Spleen, Kidneys and adrenal glands.

ஆரைமச்சயேந்திராசனம்(Arai Matsyendrasana) :

- Sit with the legs stretched out in front of the body.
- Bend the right leg and place the right foot flat on the foot on the outside of the left knee.
- The toes of the right foot should face forward.
- Bend the left leg and bring the foot around to the right buttock. The outside edge of the foot should be in contact with floor.
- Pass the left arm through the space between the chest and the right knee, and place it agains the outside of the right leg.
- Hold the right foot or ankle with the left hand, so that the right knee is close to the left armpit.
- Sit up as straight as possible.
- Raise the right arm in front of the body and gaze at the fingertips.
- Slowly twist to the right, simultaneously moving the arm, trunk and head.
- Use the left arm as a lever against the right leg to twist the trunk as far as possible without using the back muscles.
- Follow the tips of the fingers of the right hand with the gaze and look over the right shoulder.
- Do not strain the back.
- Bend the right elbow and place the arm around the back of the waist. The back of the right hand should wrap around the left side of the waist.
- Alternatively, it can be placed as high as possible between the shoulder blades with the fingers pointing up. This arm position enforces the straightness of the spine.
- Reverse the movements to come out of the posture and repeat on the other side.

ஹலாசனம (Halasana)- Plough pose



- ❖ Lie flat on the back with the legs and feet together. Place the arms beside the body with the palms facing down.
- ❖ Relax the whole body.
- ❖ Raise both legs to the vertical position, keeping them straight and together, using only the abdominal muscles.
- ❖ Press down on the arms and lift the buttocks, rolling the back away from the floor. Lower the legs over the head.
- ❖ Do not force the toes to touch the floor.
- ❖ Turn the palms up, bend the elbows and place the hands behind the ribcage to support the back as in sarvangasana.
- ❖ Relax and hold the final pose for as long as is comfortable.
- ❖ Return to the starting position by lowering the arms with the plams facing down, and then slowly lower the back and buttocks to the floor.
- ❖ Raise the legs to the vertical position. Using the abdominal muscles, lower the legs to the starting position, keeping the knees straight

Benefits: It promotes the production of insulin by the pancreas. It boosts the immune system.

General Asanas:

- ❖ Surya Namaskara
- ❖ Savasana
- ❖ Theraiyar clearly explains mild sunrays are much beneficial to our body to prevent diseases.

சவாசனம்-Savasanam:

- By doing savasanam, the whole body is relaxed and rejunuvate the body.
- This Asana being practiced in the world for relaxation techniques.
- Effect of Yoga Asanas on nerve conduction in Type II Diabetes.
- Yoga asanas included "Suryanamskar, Konasan, Padmasan pranayam,

Paschimottansan, Ardhmatsyendrasan, Shavasan. The yoga exercises were performed for 30-40 minutes every day 40 days in above sequence. The subjects were prescribed certain medicines and diet.

Yoga asanas have a beneficial effect on glycaemic control and improve nerve function in mild to moderate type II diabetes with such clinical neuropathy.

-Indian Journal physiology & Pharmacology 2002: 46 (3)

Varun Malhotra etal

Stress Management:

Diagnosis of diabetes mellitus is a stressful situation in life of an individual and appropriate management requires a holistic approach that includes behavioral modification to develop positive attitude and healthy life style. A satisfactory treatment plan should include special attention to person with diabetes, quality of life, coping skills, optimal family support and a healthy workplace environment. Appropriate support and counseling is an essential component of the management at the time of diagnosis and throughout life.

PREPARATION OF TRIAL DRUGS

SERRANKOTTAI

PURIFICATION METHOD:

Take serronkottai and boil it in Tarmarind leaf decoction, Butea monosperma flower decoction, Cow dungand Aleo Vera juice respectively.

SERRONKOTTAI THIRAVAGAM:

INGREDIENTS:

Serronkottai seed- 2 ser (560 gm)

PREPARATION:

Take two ser purified serrankottai seed and smash into pieces.Then place it in a pot and add 1 pathakku (10.7 lit) water, soak the content for two days.Then transper distillation apparatus and heat it to get thiravagam.

DOSE:

10 drops

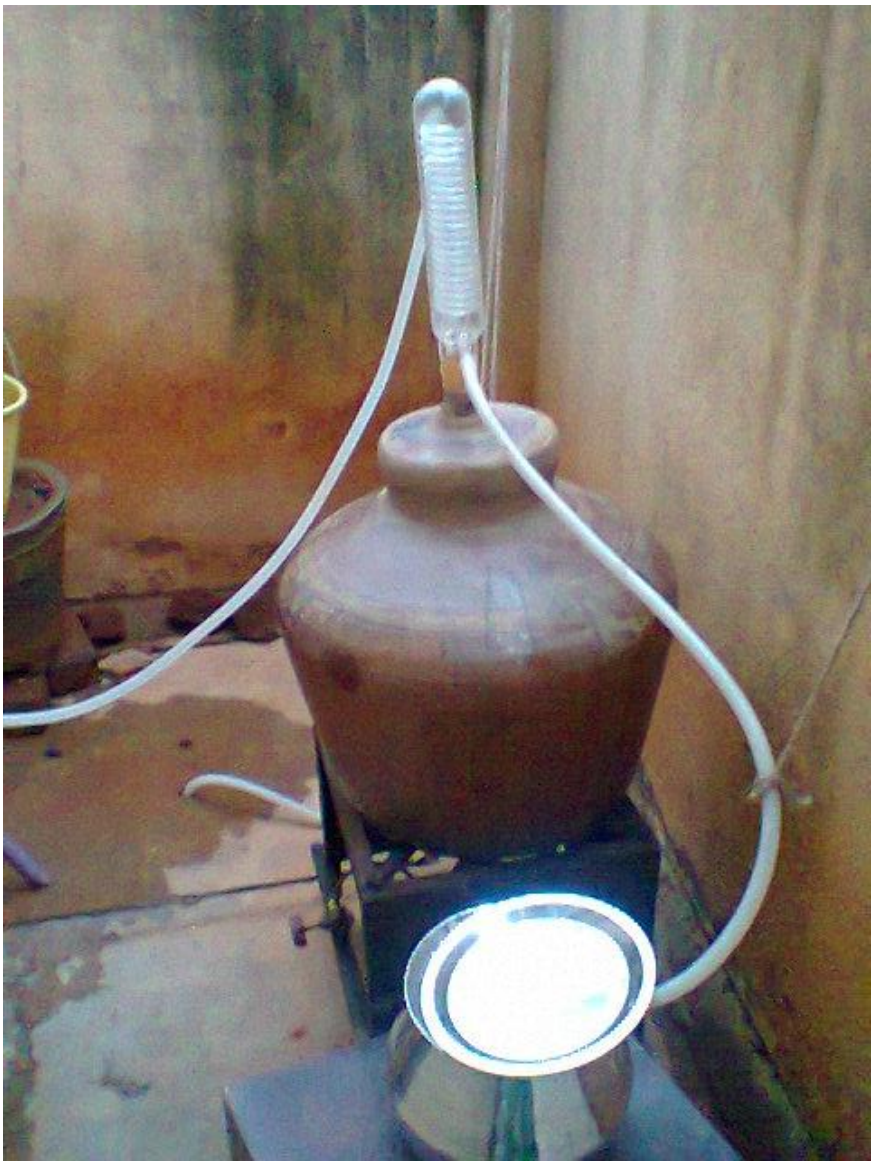
ADJUVANT:

Water

REFERENCE:

Yagopu vaidhya chinthamani-700.

THIRAVAGA VAALAI



MATERIALS AND METHODS

(PROTOCOL)

STUDY DESIGN:

The open clinical trial on Neerizhivu was conducted at the OPD section of post graduates department of pothu maruthuvam, Government Siddha Medical College, Arignar Anna Hospital Chennai 600106 during the period of 2011-2013.

SAMPIE SIZE:

During this dissertation work on Neerizhivu (Diabetes Mellitus) totally 40 patient of both sexes in the age group above 40 are taken.

INCLUTION CRITERIA:

- Above 30 years of age
- Both sexes
- Non Insulin dependent diabetes mellitus
- Poly uria
- Poly Phagia
- Poly dipsia
- Nocturia
- General weight loss
- General weakness
- Peripheral Neuritis

Exclusion CRITERIA:

- Insulin dependent diabetes mellitus.
- Patients with hyperglycemia due to secondary causes like pancreatic pathology.
- Patient with cardio vascular diseases.'
- Tuberculosis
- Diabetic Nephropathy
- Diabetic Retinopathy

TRIAL DRUG, DOSAGE, DURATION:

INTERNAL DRUG:

SEERANKOTTAI THIRAVAGAM -10 drops/tid with Water.

TRIAL PERIOD: 48 DAYS.

INVESTIGATION:

The investigations are carried out promptly and regularly before and after treatment.

All patients are subjected to routine clinical investigation which include

- Urine sugar-fasting and post – prandial
- Albumin and deposits in urine
- Total count, differential count, Erythrocyte sedimentation rate, Haemoglobin and urea, cholesterol in blood.
- Blood Sugar, fasting and post prandial investigations like glucose tolerance test, glycosylated Haemoglobin (HbA1c).

Siddha system of clinical diagnosis

- Poriyal Therthal : Mei, Vai, Kann, Mooku, Sevi
- Pulanal Therthal : Unarthal, Suvaithal, Parthal, Mugarthal, Kettal.
- Venaathal :
- Mukkutra Nilaiga : Vali, Azhal, Iyam
- Ezhu Udal Kattugal: Saaram, Senneer, Oon Kozhuppu, Enbu, Moolai, Sukkilam.
- Envagai Thervu : Naa, Niram, Mozhi, Vizhi, Naadi, Sparisam, Malam, Moothiram.

Case sheet proforma:

Patients will be treated with clinical signs and symptoms of Neerizhivu

Complaints and duration

History of past illness

Personal History

Personal Habits

Family History

Systemic Examination

Laboratory investication

Prognosis of the disease and management.

PROPERTIS OF TRIAL DRUGS

SERRONKOTTAI:

Botanical name - Semecarpus anacardium

Family - Anacardiaceae

Part used - Seed

Tast (suvai) - Kaippu, viruviruppu

Nature (thanmai) - Veppu

Pirivu - Kaarppu.

Action:

Alterative
Caustic

பொது குணம் :

சேரிரண்டு சேங்கொட்டை யிடித் துச்சாலில்
ஜெலம்பதக்கு விட்டதையும் கலந்துமூடி
ஊரியநாள் ரெண்டதன்பின் வாலைவைத்து
ஊற்பனமா யெரித்துவிடில் திராவகத்தை
சீராக வாங்கியிதைக் குப்பியிட்டுத்
தினங்கொண்டால் வாதபித்தயெரிவுசோபம்
நீரிழிவு வயிற்றிரைச்சல் நீங்காத்தாகம்
நிறுத்துமென்று யாகோப்பு நிசஞ்சொன்னாரே.

Chemical Constituent:

- Anacardic acid
- Anacardol
- Semecarpo
- Catechol
- Bhilawanol
- Cardol

SERRANKOTTAI TREE

SERRANKOTTAI

RESULTS AND OBSERVATION:

The factors considered for observation for the purpose of the study comprised of the following.

- Age – Wise
- Gender – wise classification
- Distribution of thinai
- Paruvakalam (seasonal incidence)
- Occupation
- Socio economic status
- Dietary habits
  - Family history
  - Body weight
  - Classification of results according to Vali, Azhal & Iyam
  - Ezhu udal kattugal
  - Enn vagai thervugal
  - Classification on the basis of Neikuri
  - Naadi
  - Signs and symptoms
  - Urine

    Fasting

    Post prandial
- Blood Sugar

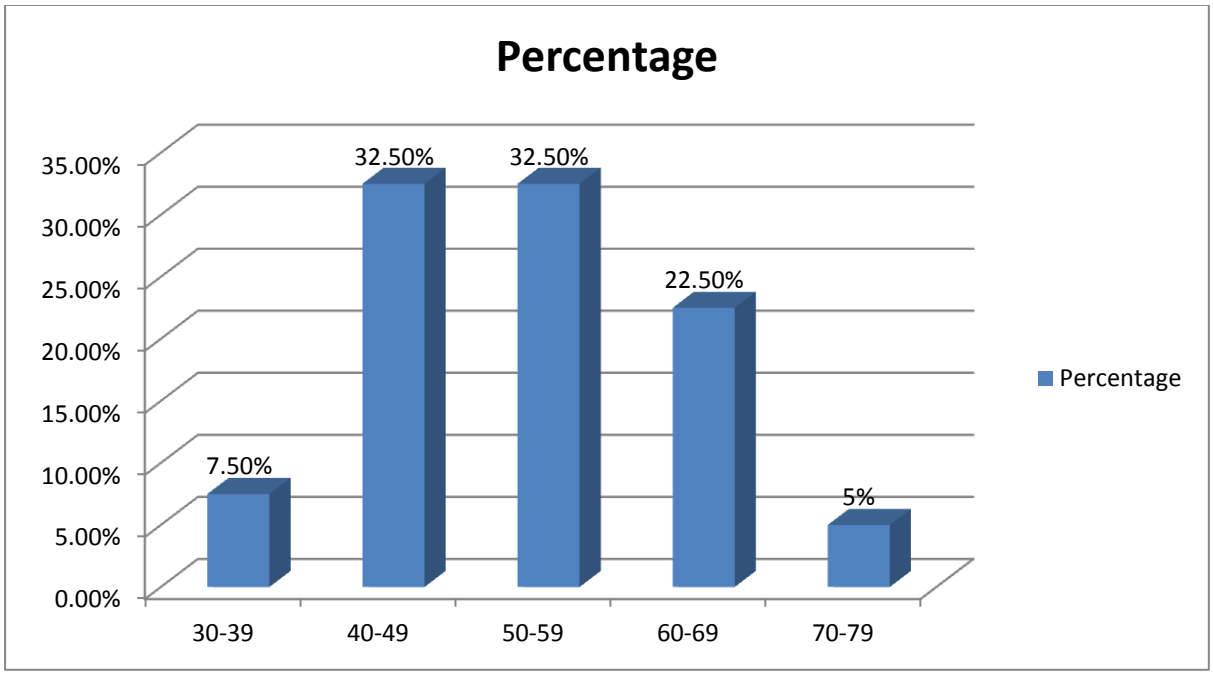
  Fasting

  Past prandial
- Glycosylated Hb (HbA1c)
- Efficacy of medicine

TABLE – 1

AGE WISE CLASSIFICATION:

AGE	NO OF PATIENTS	PERCENTAGE
30-39	3 Patients	7.5%
40-49	13 Patients	32.5%
50-59	13 Patients	32.5%
60-69	9 Patients	22.5%
70-79	2 Patients	5%

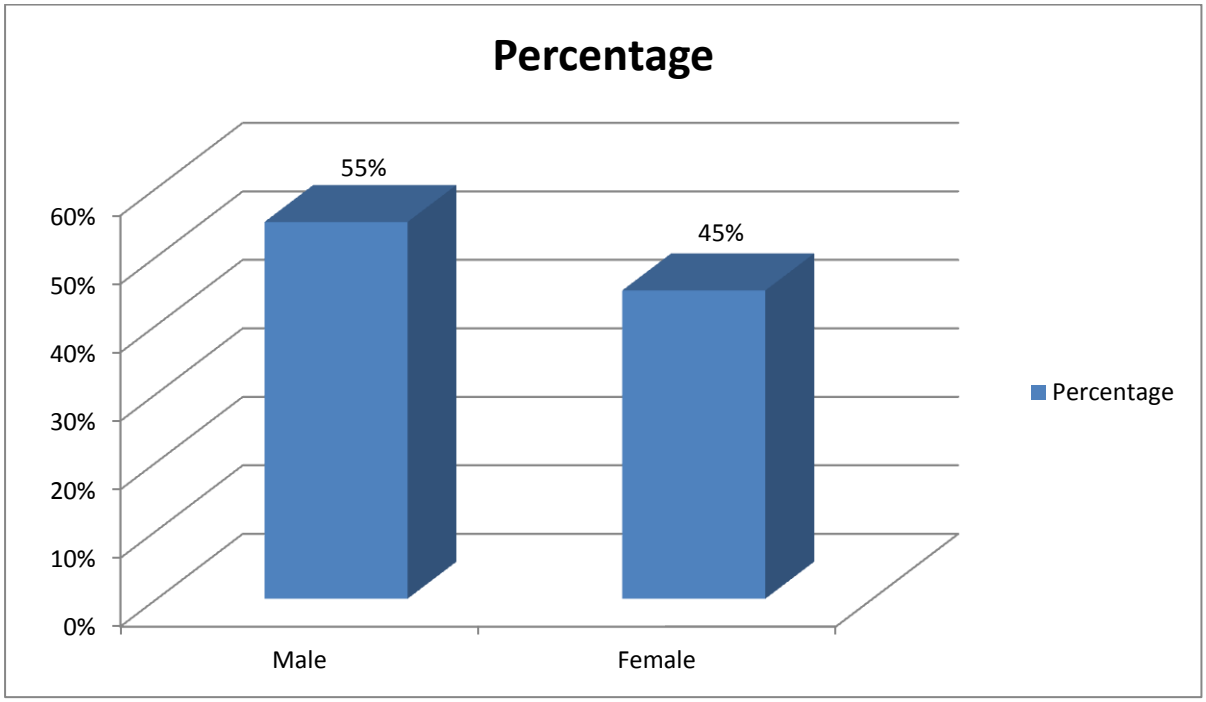


It is observed from the above analysis that the incidence of Neerizhivu is more in the age group of 40-49, 50-59, and 60-69 in each of thes groups which clearly shows that Neerizhivu set automatically in the process of ageing ie.Pithakalam.

TABLE - 2

GENDER WISE CLASSIFICATION

GENDER	NO OF PATIENTS	PERCENTAGE
Male	22	55%
Female	18	45%

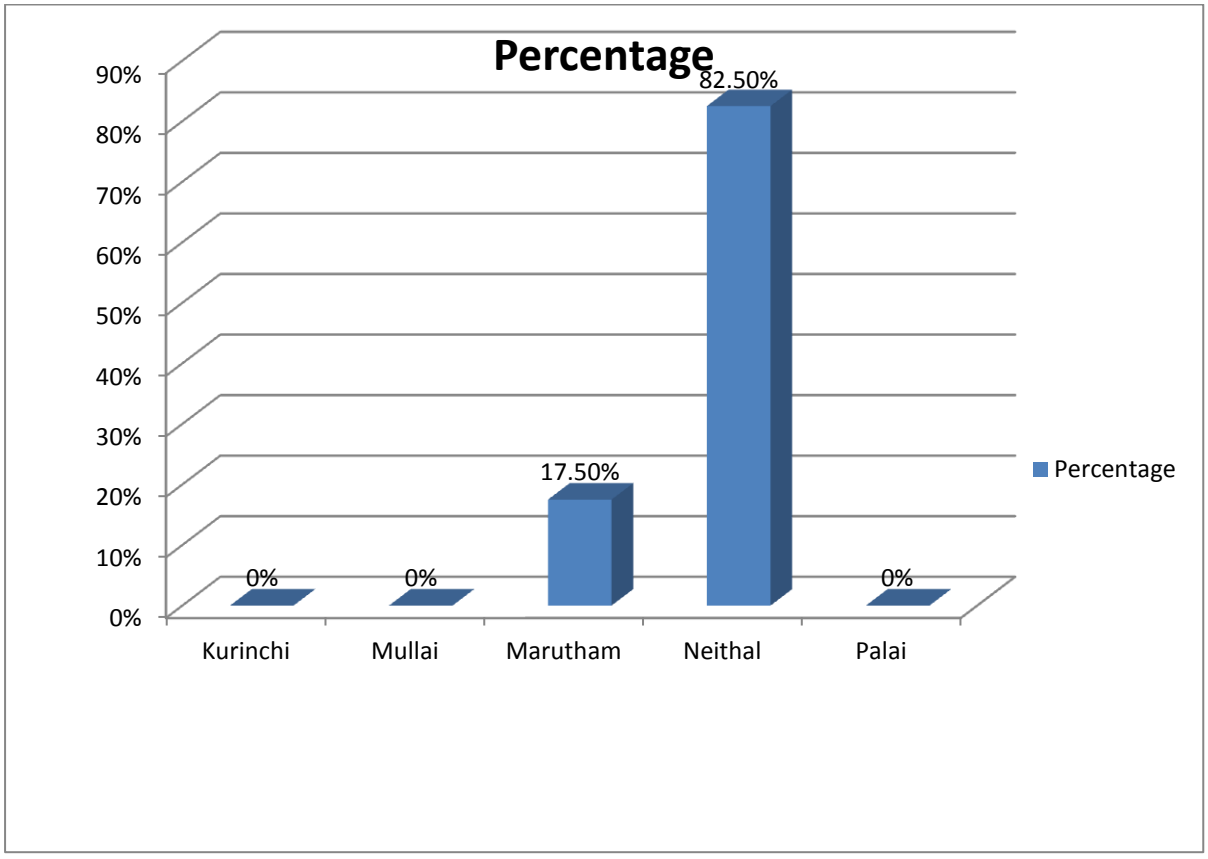


From the pie chart the study done on the basis of patients who come for treatment of Neerzhivu at the hospital reveals that majority of them male are 55% and female 45% usually the NIDDM occurs both sexes.

TABLE - 3

DISTRIBUTION OF THINAI

THINAI	NO OF PATIENTS	PERCENTAGE
Kurinchi	-	-
Mullai	-	-
Marutham	7	17.5%
Neithal	33	82.5%
Palai	-	-

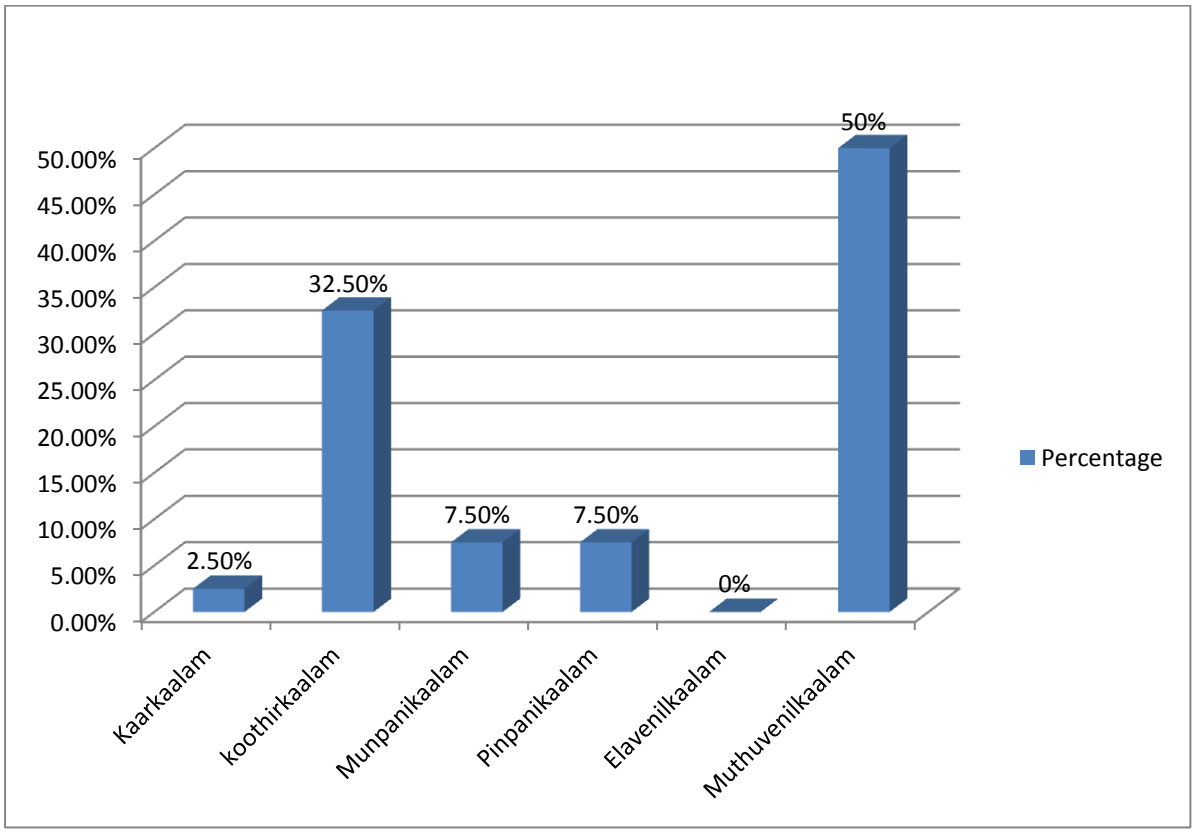


From the pie-chart as above it may be observed that people living in Neithal are prone to Neerizhivu while compared to others.This is very evident from the fact that 82.5% of the patients with Neerizhivu are from this region. This indicates that the food habits of people in these regions which have a tropical climate and humid temperature play a signicant.

TABLE - 4

SEASONAL INCIDENCE (PARUVA KAALAM)

PARUVA KAALAM	NO OF PATIENTS	PERCENTAGE
Kaar kaalam (Mid Aug to Mid Oct)	1	2.5%
Koothir kaalam (Mid Oct to Mid Dec)	13	32.5%
Munpani kaalam (Mid Dec to Mid Feb)	3	7.5%
Pinpani kaalam (Mid Feb to Mid Apr)	3	7.5%
Elavenil Kaalam (Mid Apr to Mid June)	-	-
Muthuvenil kaalam (Mid June to Mid Aug)	20	50%

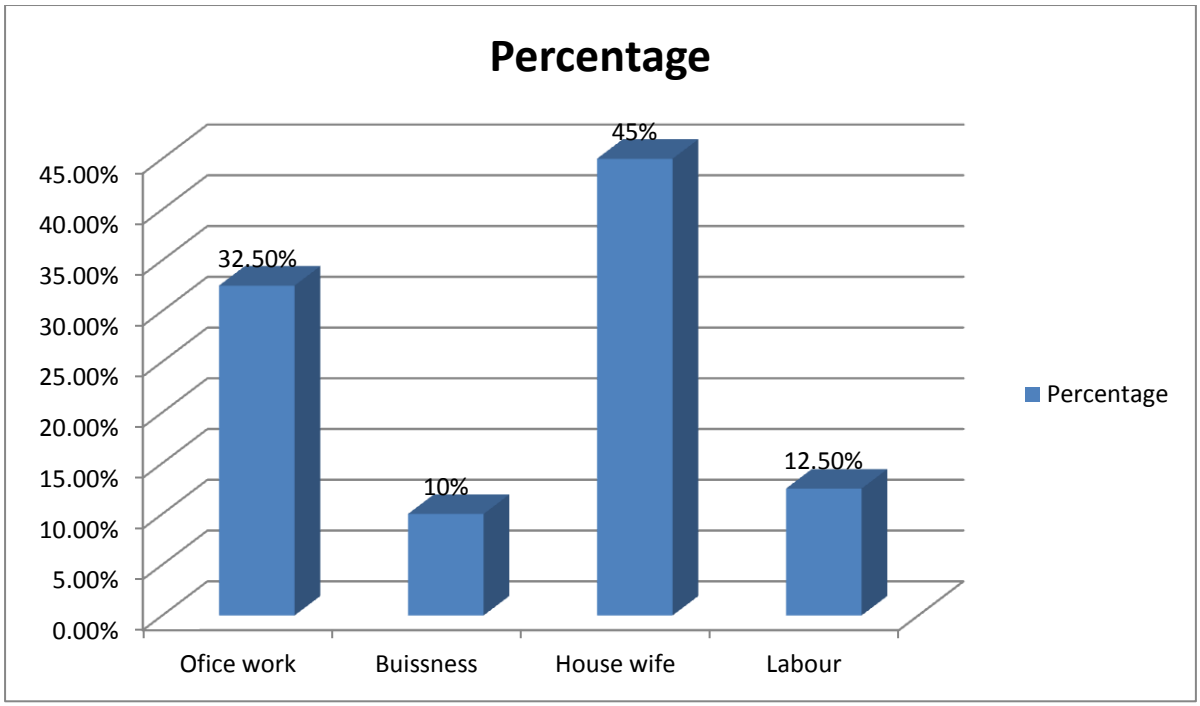


The chart clearly indicates that seasonal variations do not have a serious impact.In almost throughout the year Neerizhivu sets in.

TABLE – 5

OCCUPATION:

OCCUPATION	NO OF PATIENTS	PERCENTAGE
Profossionals	13	32.5%
Buissness	4	10%
House wife	18	45%
Labour	5	12.5%

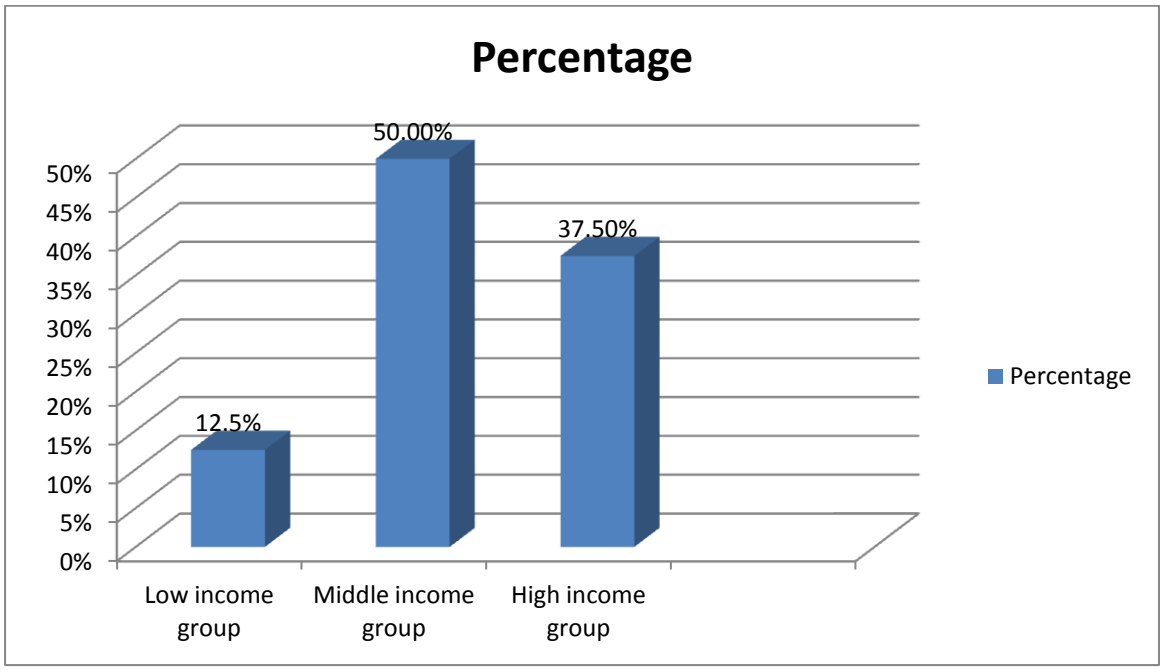


The chart it may be noted that the incidence of Neerizhivu is more on house wife i.e.45% of the total patients,32.5%on the office worker and 12.5% on the labor community.

TABLE – 6

SOCIO ECONOMIC STATUS:

INCOME	NO OF PATIENTS	PERCENTAGE
Low income group Below 10000/month	5	12.5%
Middle income group 10000-20000/month	20	50%
High income group Above 20000/month	15	37.5%

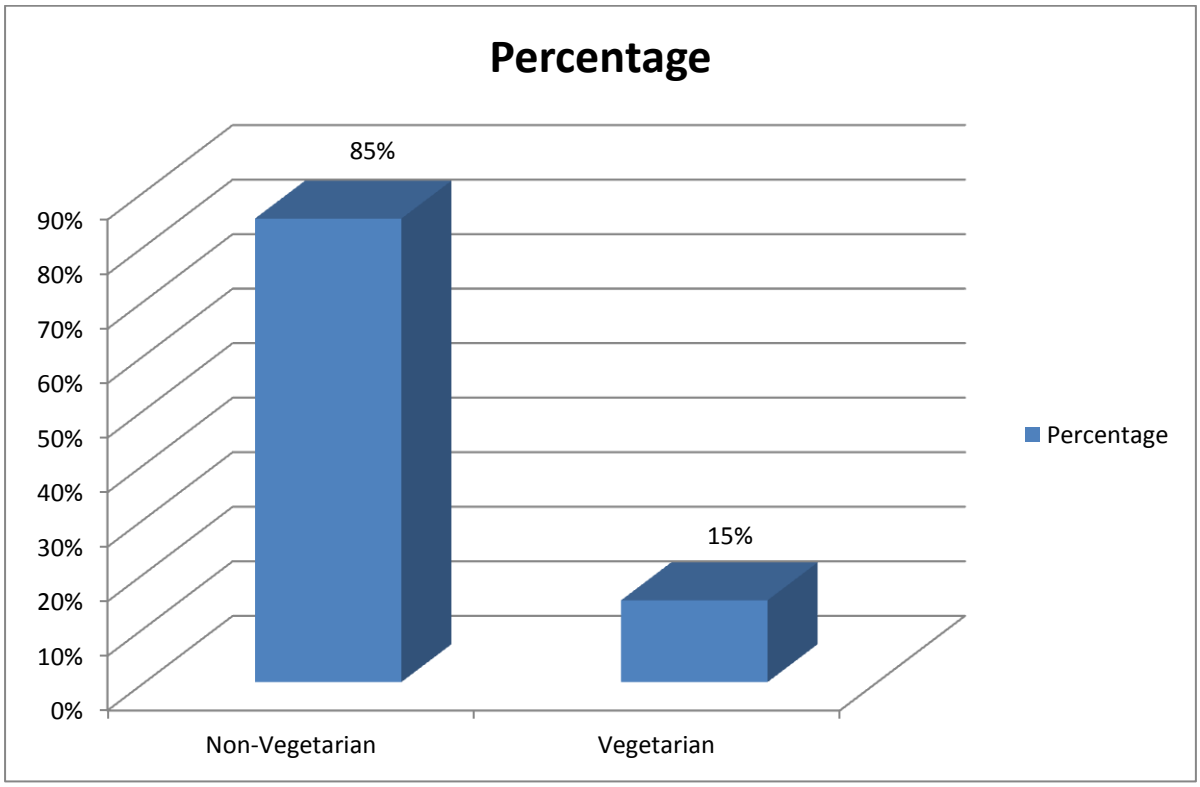


From the above table middle income group are prone to Neerizhivu.

TABLE – 7

DIETARY HABITS:

DIETARY HABITS	NO OF PATIENTS	PERCENTAGE
Non-vegetarian	34	85%
Vegetarian	6	15%

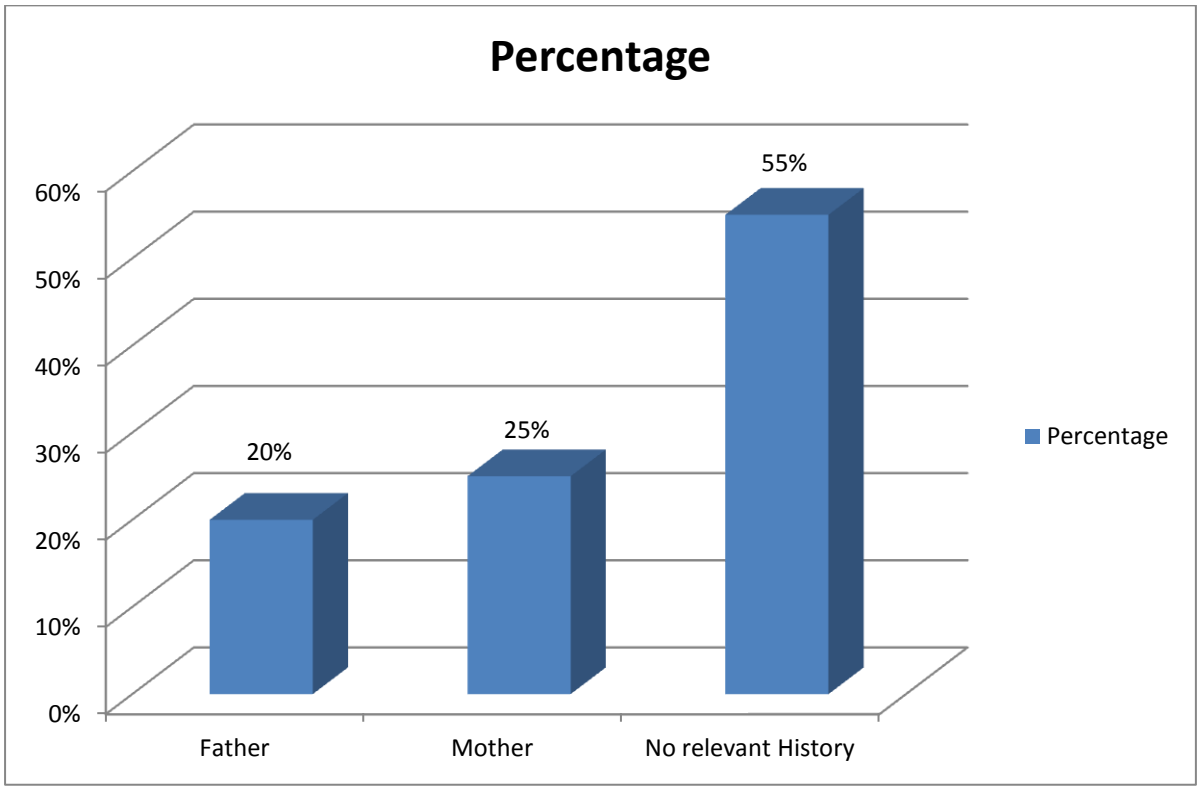


From the above table clearly shows that non- vegetarians people more prone to Neerizhivu**.**

TABLE – 8

FAMILY HISTORY:

FAMILY HISTIRY	NO OF PATIENTS	PERCENTAGE
Father	8	20%
Mother	10	25%
No relevant History	22	55%

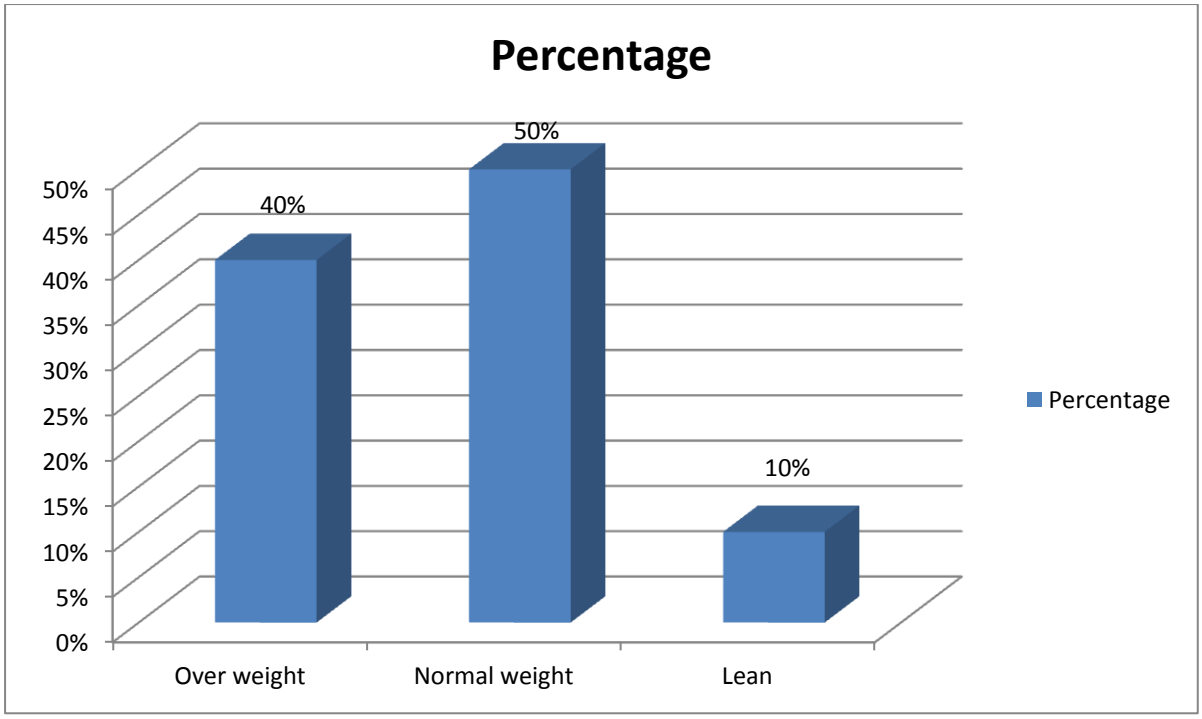


It is distinctly seen from the above table that no relevant history also plays an important role in Neerizhivu.

TABLE – 9

BODY BUILD :

WEIGHT	NO OF PATIENTS	PERCENTAGE
Over weight	16	40%
Normal weight	20	50%
Lean	4	10%



Shows that the role of normal weight people is more prone to Neerizhivu.

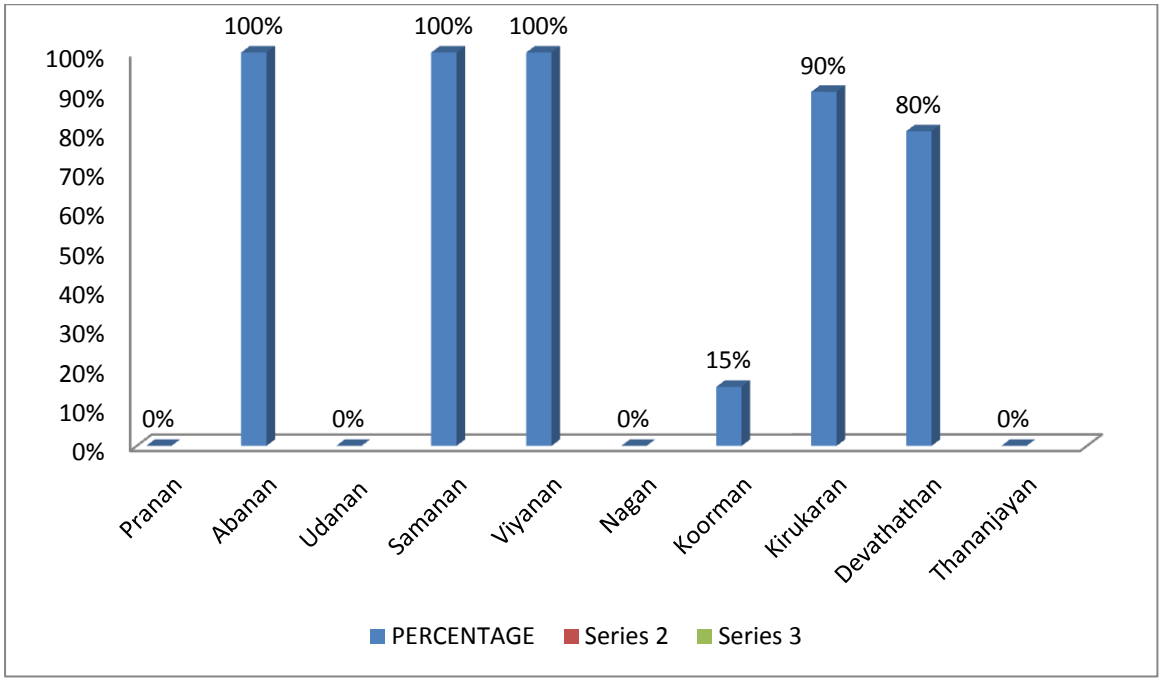
The body mass index (BMI) in adult are derived from the formula, weight (Kg)/ height (m) 2.T-In overweight people the incidence of Neerizhivu is more he acceptable normal range of BMI is 20 to 25.

The BMI between 25.0 to 29.9 are classified as overwigt.Obesity is taken to start at a BMI of 30.0 to 39.9Davidsons Textbook of Medicine P.256

TABIL – 10

VALI:

S.NO	PARTICULARS	NO OF PATIENTS	PERCENTAGE
1	Pranan	0	0%
2	Abanan	40	100%
3	Udanan	0	0%
4	Samanan	40	100%
5	Viyanan	40	100%
6	Nagan	0	0%
7	Koorman	6	15%
8	Kirukaran	36	90%
9	Devathathan	32	80%
10	Thananjayan	0	0%

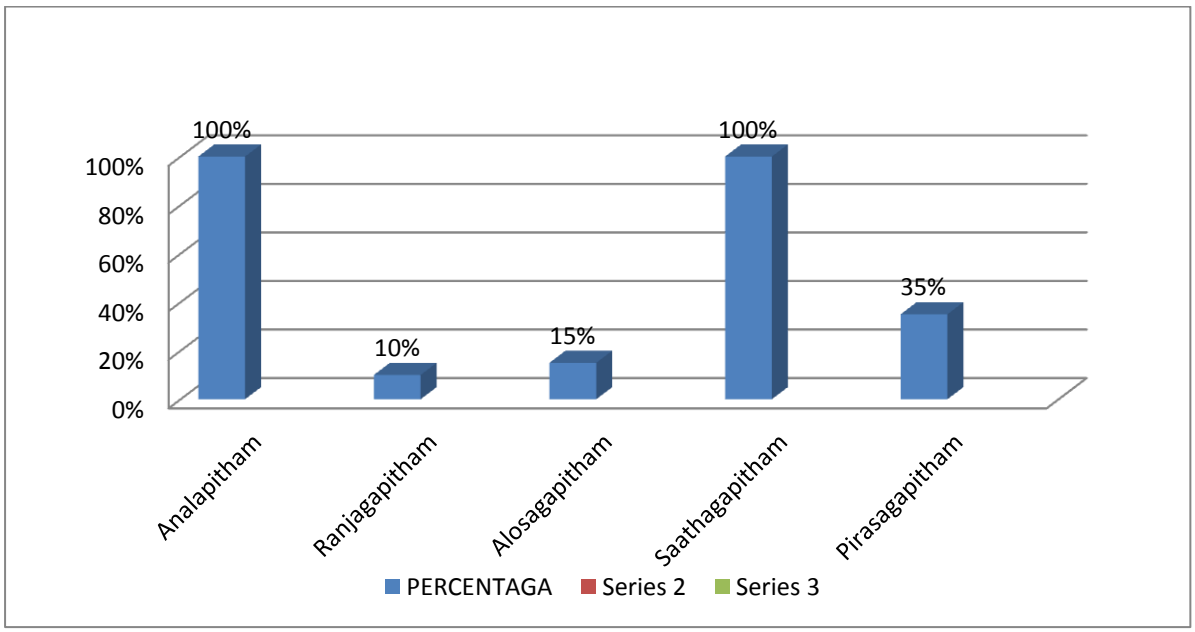


In Vali Abanan, Samanan, Viyanan are affected in all patients i.e. 100%. Koorman are affected 15% Kirukaran are affected in 90%, Devthathan are affected in 80% patients are affected.

TABLE – 11

AZHZL:

S.NO	PARTICULARIS	NO OF PATIENTS	PERCENTAGE
1	Analapitham	40	100%
2	Ranjagapitham	4	10%
3	Alosagapitham	6	15%
4	Saathagapitham	40	100%
5	Pirasagapitham	14	35%

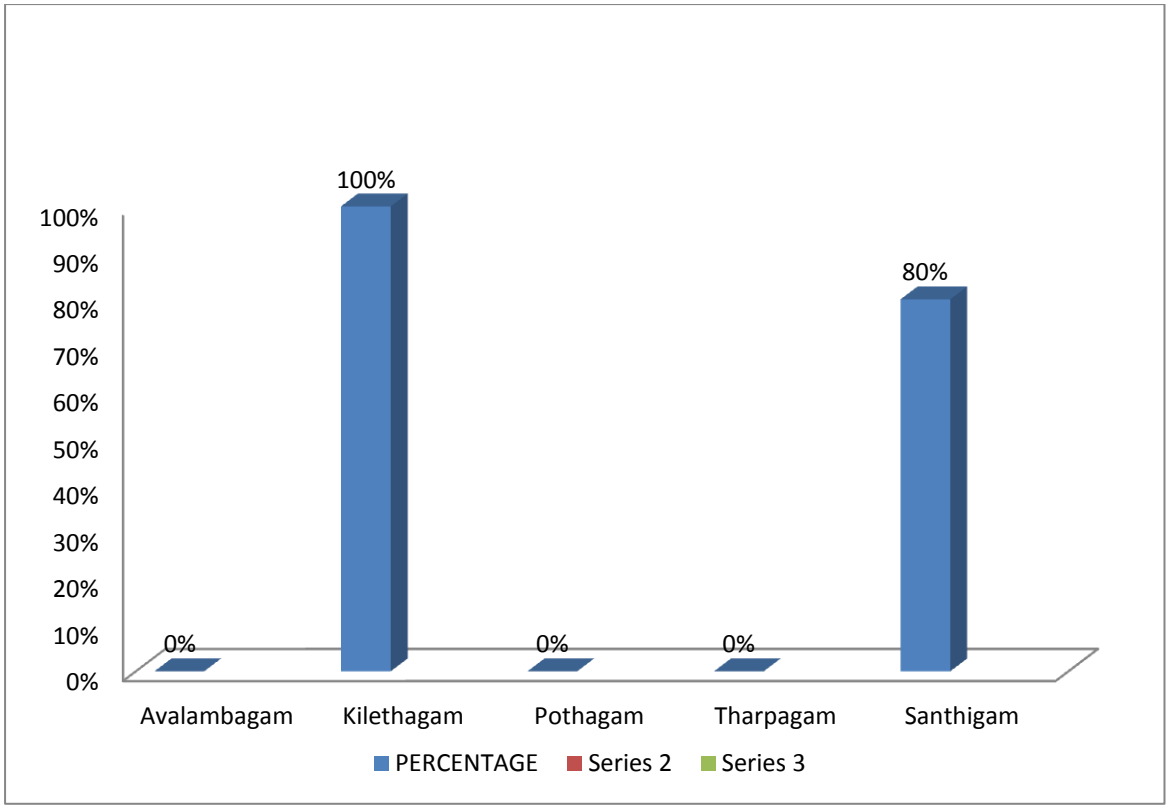


In Azhal: Anal pitham, Saathaga pitham are affected in 100% patients,Ranjaga pitham 10%, Alosaga pitham 20 % patients affected.

TABLE – 12

IYAM:

S.NO	PARTICULARS	NO OF PATIENTS	PERCENTAGE
1	Avalambagam	0	0%
2	Kilethagam	40	100%P
3	Pothagam	0	0%
4	Tharpagam	0	0%
5	Santhigam	32	80%

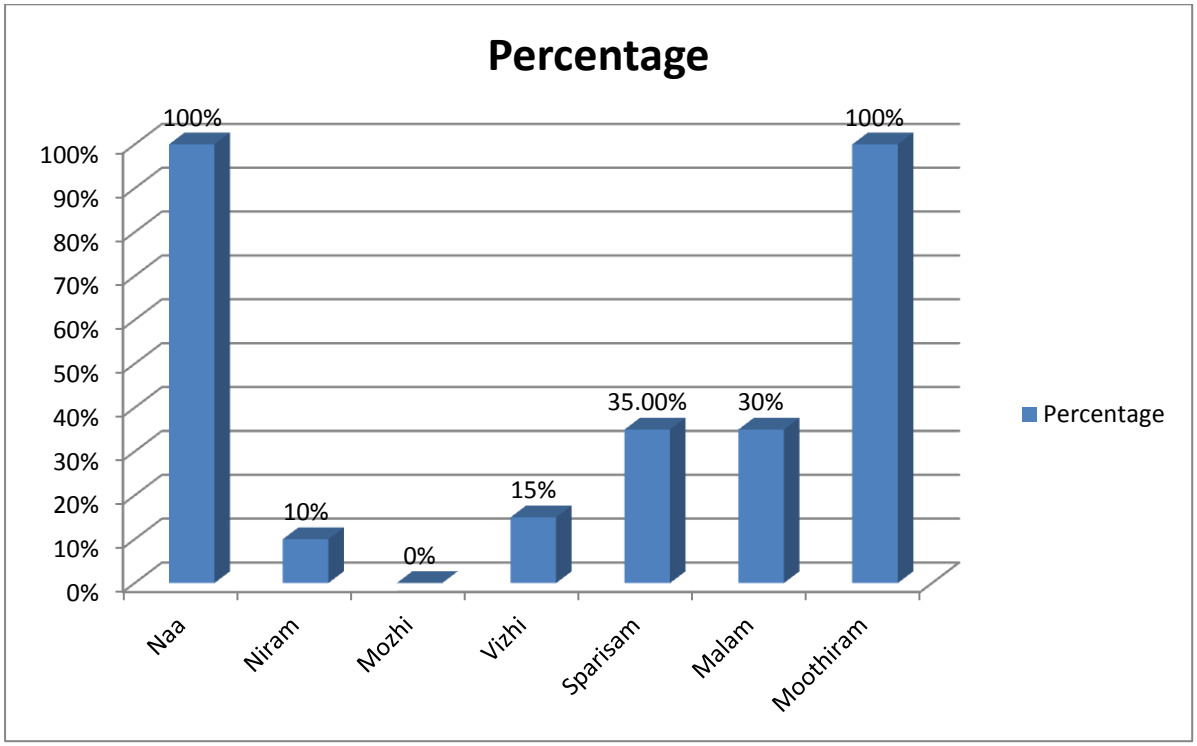


In Iyam: Kilethagam are affected in 100% patients and Santhigam 80% patients affected.

TABLE – 13

ENN VAGAI THERVUGAL – BEFORE TREATMENT:

THERVUGAL	NO OF PATIENTS	PERCENTAGE
Naa	40	100%
Niram	4	10%
Mozhi	0	0%
Vizhi	6	15%
Sparisam	14	35%
Malam	12	30%
Moothiram	40	100%

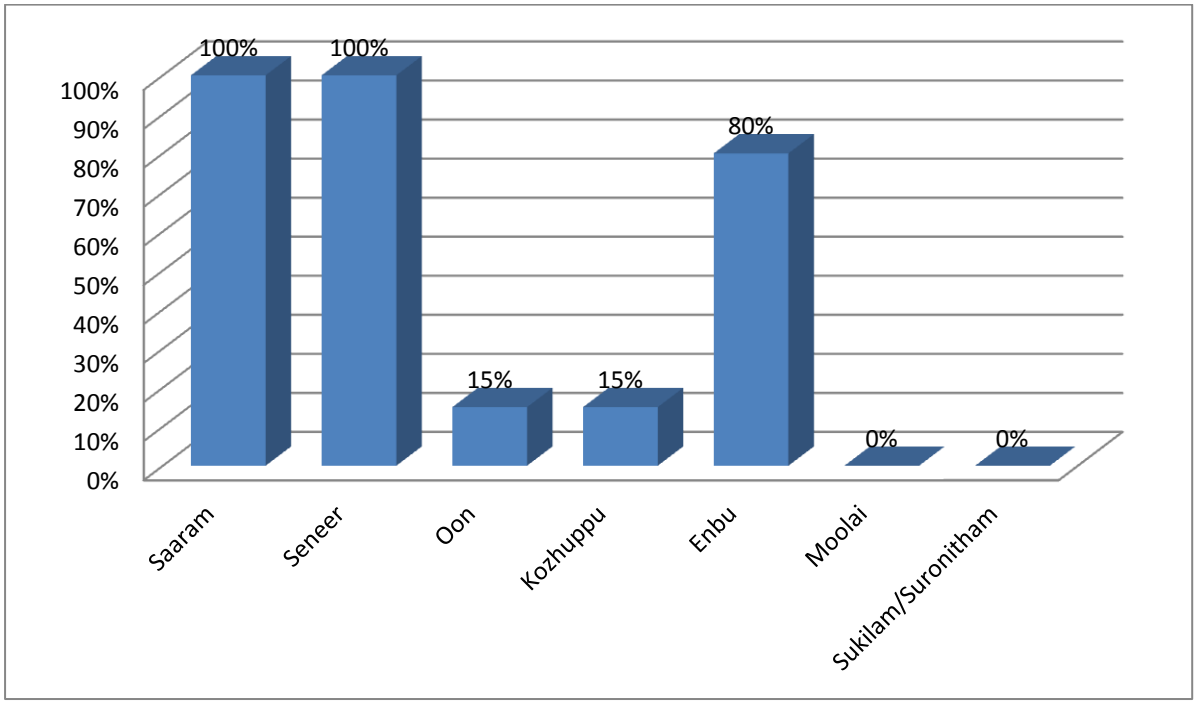


On analyzing the facts of diagnosis, Naa, Moothiram had 100% impact.

TABLE – 14

EZHU UDAL KATTTUGAL:

EZHU UDAL KATTTUGAL	NO OF PATIENTS	PERCENTAGE
Saaram	40	100%
Seneer	40	100%
Oon	6	15%
Kozhuppu	6	15%
Enbu	32	80%
Moolai	0	0%
Sukilam/Suronitham	0	0%

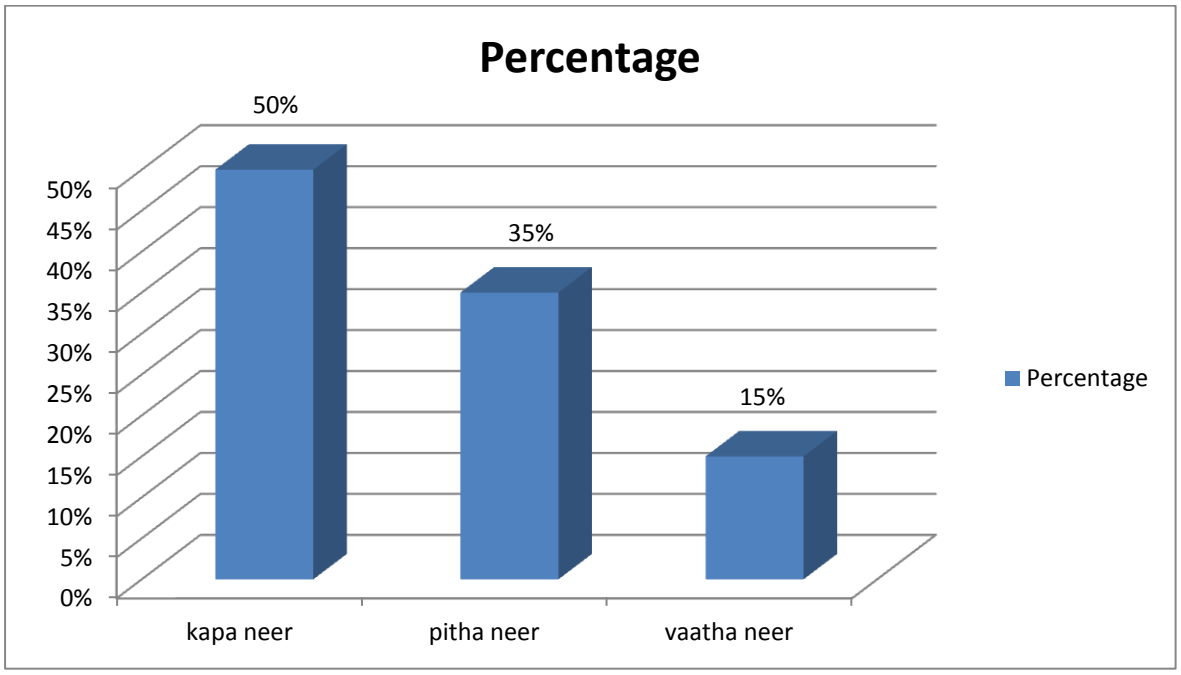


From the above chart we can observe that Saaram and senner are affected in all patients i.e.100%.Kozhuppu, Oon are affected to the to the extended 15%%. Enbu are affected 80% respectively.

TABLE – 15

CLASSIFICATION OF THE BASIS OF NEIKURI:

NEIKURI	NO OF PATIENTS	PERCENTAGE
Kapa neer	20	50%
Pitha neer	14	35%
Vaatha neer	6	15%

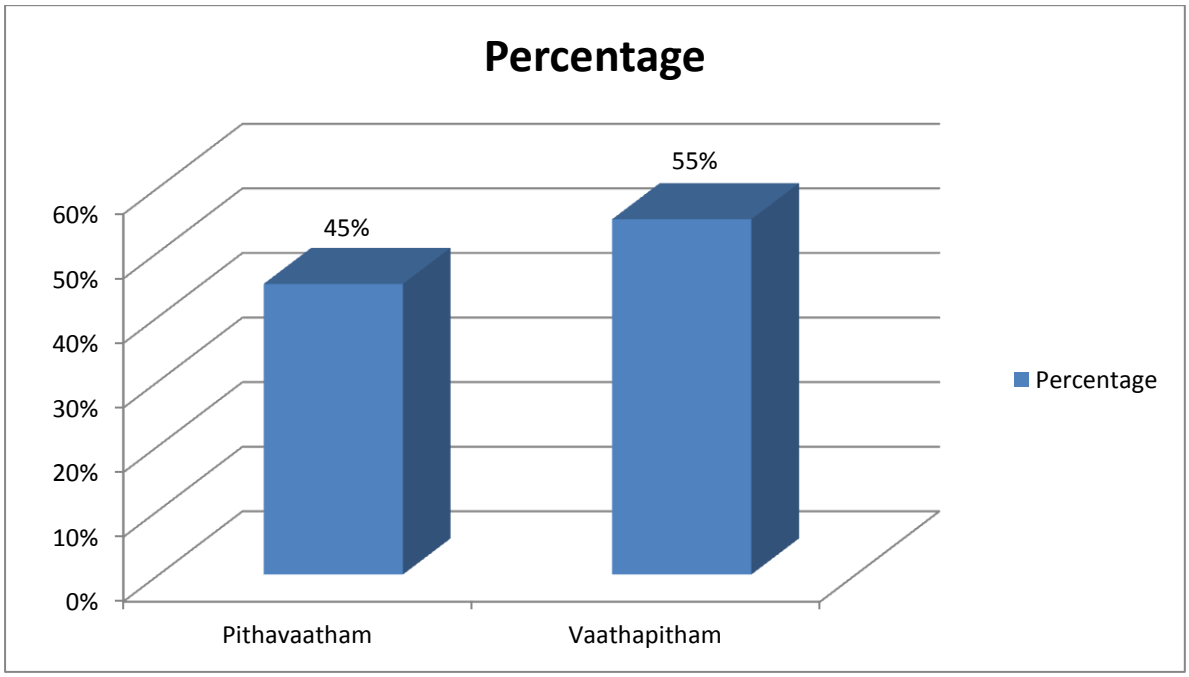


In respect of 50% of the patients when oil is dropped in the urine it look like Kapha Neer, In the balance 35% PithaNeer,which indicate kapham is prominent.

TABLE – 16

NAADI:

NAADI	NO OF PATIENTS	PERCENTAGE
Pithavaatham	18	45%
Vaathapitham	22	55%

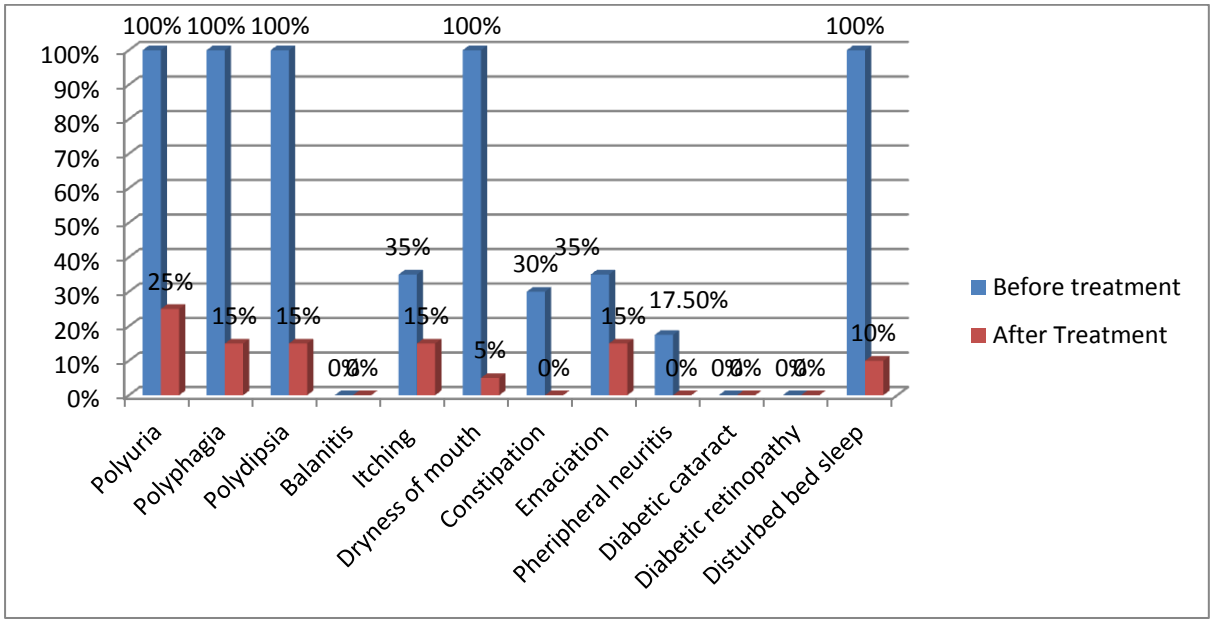


In 55% of the patients with Neerizhivu Vaatha pitham Naadi was prominent and 45% of the cases Pitha vaatham was there.

TABLE – 17

BEFORE AND AFTER TREATMENT OF SIGNS AND SYMPTOMS:

SIGNS AND SYMPTOMS	NO OF CASES BEFORE TREATMENT	PERCENTAGE	NO OF CASES AFTER TREATMENT	PERCENTAGE
Polyuria	40	100%	10	25%
Polyphagia	40	100%	6	15%
Polydipsia	40	100%	6	15%
Balanitis or prunitis valvae	0	0%	0	0%
Itching all over the body	14	35%	6	15%
Dryness of the mouth and throat	40	100%	2	5%
Constipation	12	30%	0	0%
Emaciation	14	35%	6	15%
Peripheral neuritis	7	17.5%	0	0%
Diabetic cataract	0	0%	0	0%
Disturbed bed sleep	40	100%	4	10%



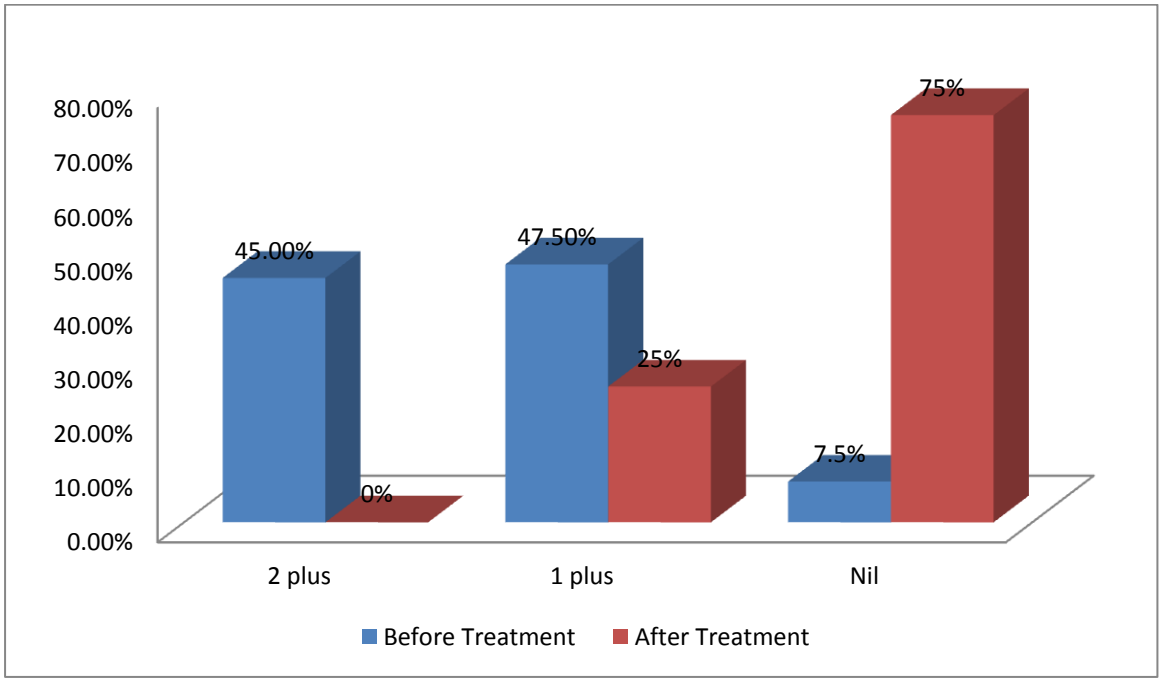
In respect of the patients with Neerizhivu the clinical symptoms of polyurea, poly phagia, Dryness of mouth and throat, poly dipsia and disturbed sleep were present in all cases i.e 100% before treatment.

The clinical signs and symtoms were improved after treatment showing only 25% of the have poly urea,15% of the people have polyphagia, 15% of the people have polydipsia,15% of the people have itching all over the body and 10%have disturbed sleep.

TABLE – 18

URINE SUGAR - FASTING:

URINE SUGAR - FASTING	BEFORE TREATMENT	PERCENTAGE	AFTER TREATMENT	PERCENTAGE
++	18	45%	0	0%
+	19	47.5%	10	25%
NIL	3	7.5%	30	75%

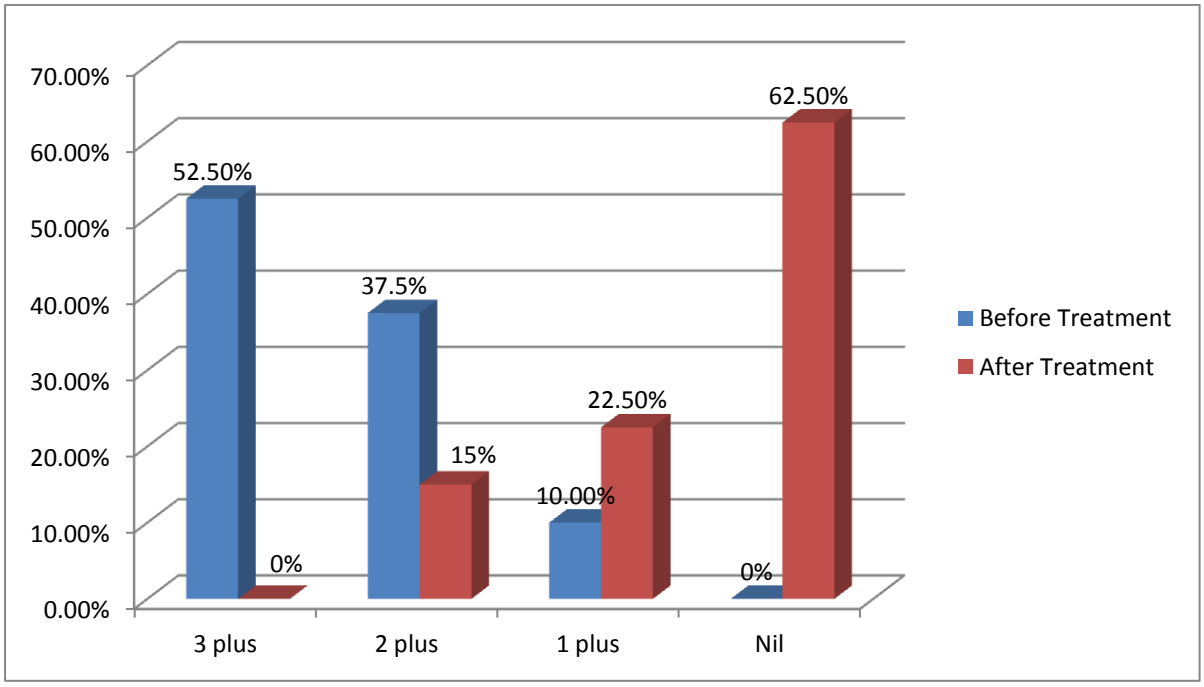


From the above chart it may be observed thst the Urine sugar position on fasting; after treatment had improved drastically it was nil in 75% of the cases after treatment.

TABLE – 19

URINE SUGAR – POST PRANDIAL:

URINE SUGAR – POST PRANDIAL	BEFORE TREATMENT	PERCENTAGE	AFTER TREATMENT	PERCENTAGE
+++	21	52.5%	0	0%
++	15	37.5%	6	15%
+	4	10%	9	22.5%
NIL	0	0%	25	62.5%

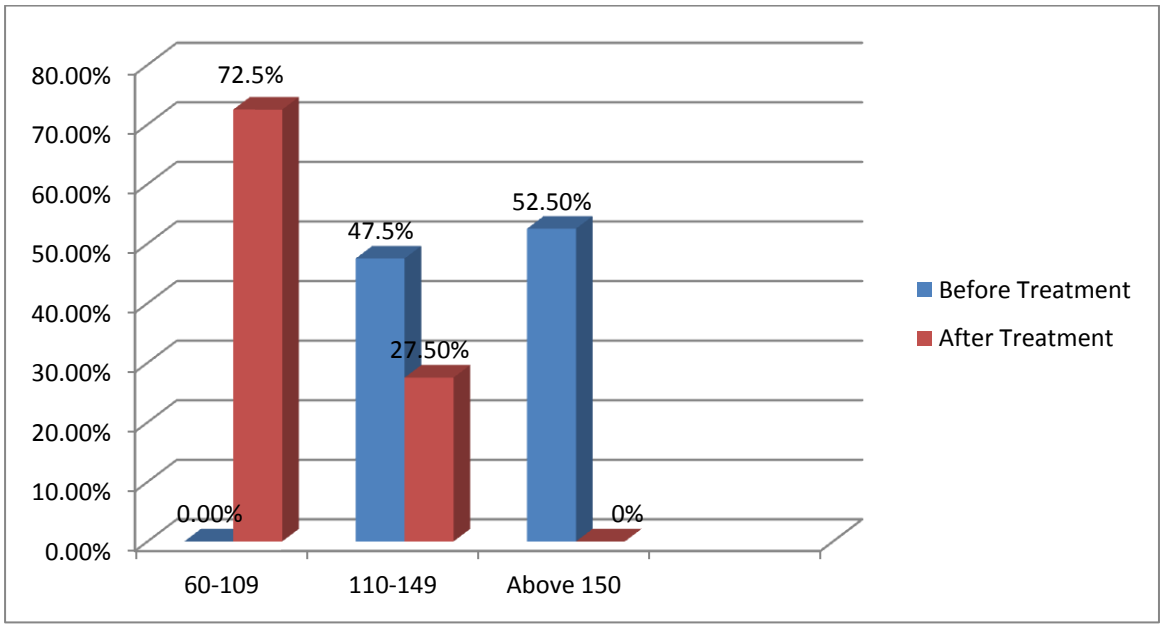


It may be noted that the post prandial urine sugar position after treatment had improved drastically it was nil in 62.5% of the cases after treatment.

TABLE – 20

BLOOD SUGAR – FASTING:

BLOOD SUGAR – FASTING (mg)	BEFORE TREATMENT	PERCENTAGE	AFTER TRAETMENT	PERCENTAGE
60-109	0	0%	29	72.5%
110-149	19	47.5%	11	27.5%
Above 150	21	52.5%	0	0%

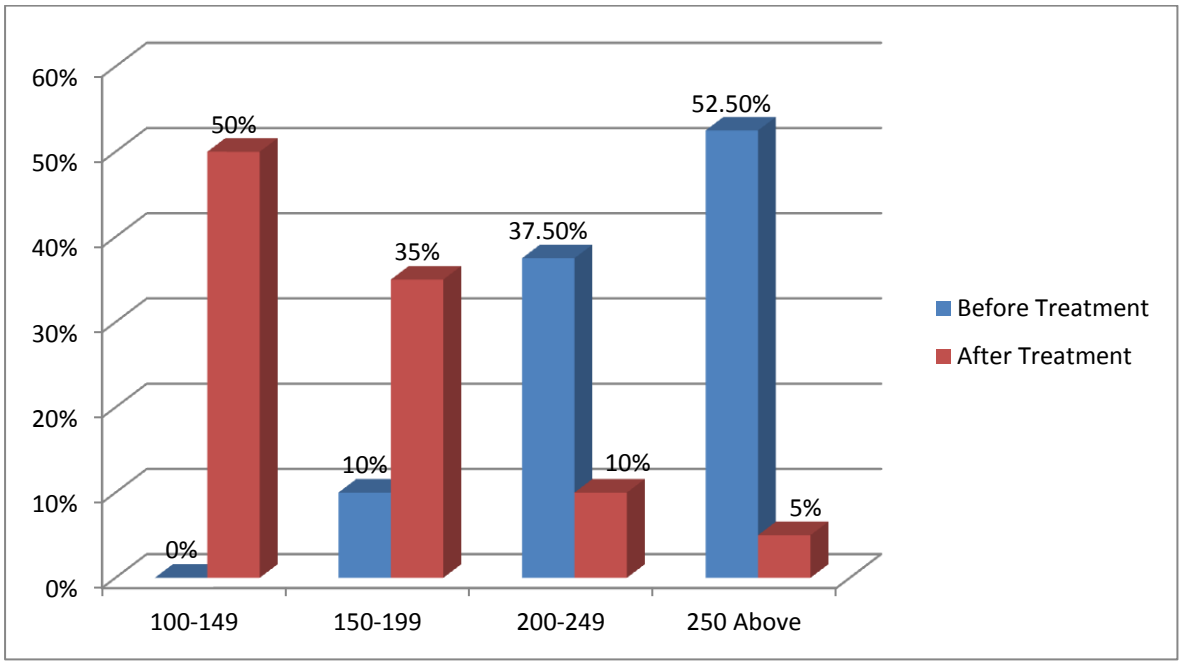


Fasting blood sugar has control in 72.5% of the cases.

TABLE – 21

BLOOD SUGAR – POST PRANDIAL:

BLOOD SUGAR – POST PRANDIAL(mg)	BEFORE TREATMENT NO OF CASES	PERCENTAGE	AFTER TREATMENT	PERCENTAGE
100-149	0	0%	20	50%
150-199	4	10%	14	35%
200-249	15	37.5%	4	10%
250 Above	21	52.5%	2	5%

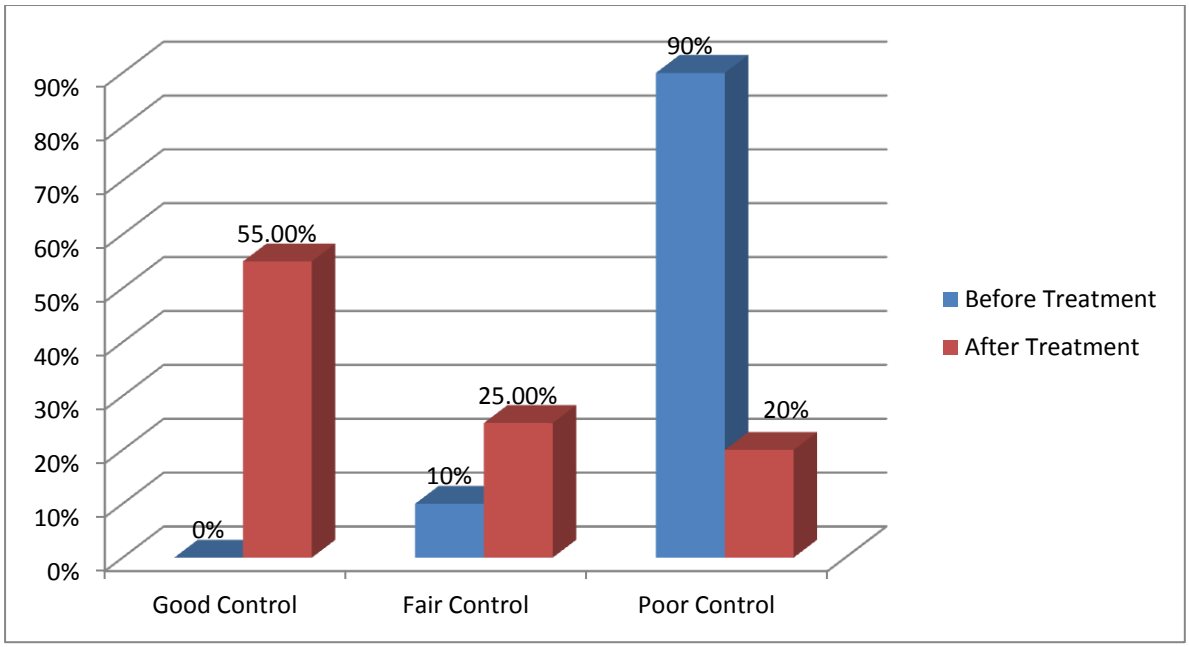


The blood sugar post prandial level has control 50% of the cases.

TABLE – 22

ASSESSMENT OF HbA1c

	HbA1C			
	BEFORE TREATMENT		AFTER TREATMENT	
Good control 5_7%	**0**	**0%**	**22**	**55%**
Fair control 7_8%	**4**	**10%**	**10**	**25%**
Poor control Above 8%	**36**	**90%**	**8**	**20%**

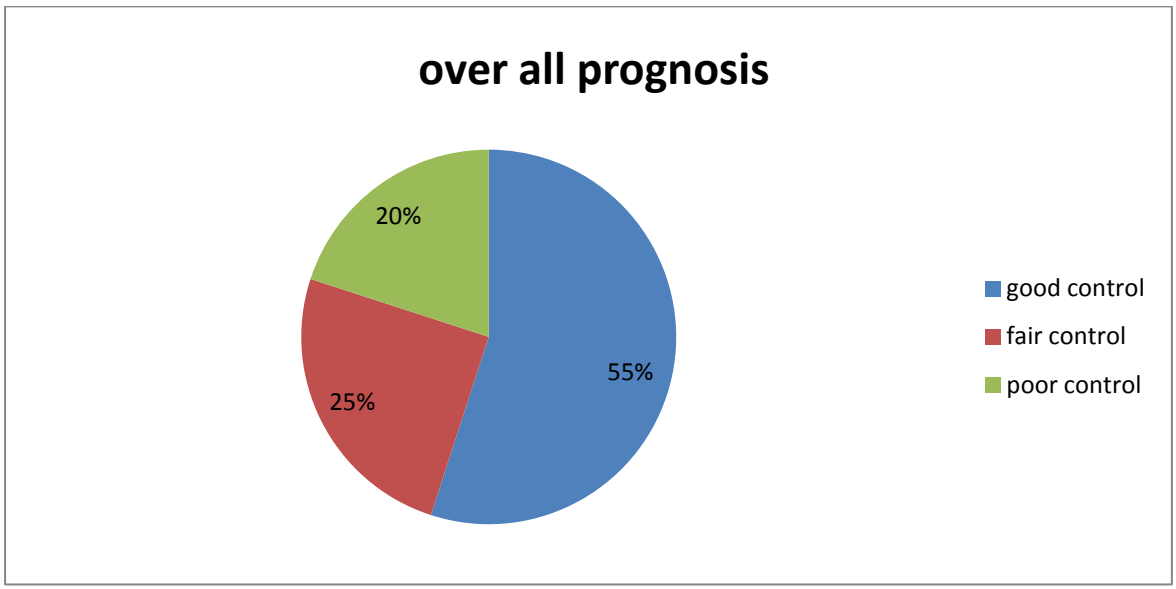


The HbA1(C) results shows good control in 55% of the cases, fair control in 25% of the cases, poor control in 20% of the cases.

TABLE- 23

OVER ALL PROGNOSIS

S.NO	PROGNOSIS	PERCENTAGE
1.	Good control	55%
2.	Fair control	25%
3.	Poor control	20%



INFERENCE:

Over all prognosis showed in 55% of cases.

DISCUSSION

The open clinical trial on Neerizhivu was conducted at the OP/IP section of PG-Pothu maruthuvam department attached to Govt. A. A. H. OF INDIAN MEDICINE, CHENNAI-106 during the period 2011-2013.

Neerizhivu was supposed to be associated with rich community of people of white collar job.But now a days the disease spread its wing to entire human kind.changing food habit life style and moderinization of world plays vital role in the health aspect of humanity patient must be actively involved in his own management and should develop confidence to bring about adjustment in day to day management.A small group of 20 patient in OPD and 20 patient in IP with complaints of Neerizhivu were selected.

The diagnosis was again established and confirmed with the help of envagai thervugal and modern Bio chemical investigations these cases were given the trial drug with strict diet regimen.During the course of treatment the patient was subjected to routine blood sugar and urine sugar and blood pressure asscesments.

AGE WISE ANALYSIS:

It shows that the Neerizhivu common in the age 40-49-32.5%, 50-59-32.5%, 60-69-22.5% ie.Pithakalam and usually the non insulin dependent diabetes mellitus occurs only in the middle and old aged groups.

GENDER WISE:

The gender wise classification shows that the Neerizhivu in the male 55%, Female 45%, usually the non insulin dependent diabetes mellitus occurs both sexes equally.

THINAI:

95% of people living in Neithal are prone to Neerizhivu. The reason is that the land is closer to sea and food crops grown in that area is Alkaline.Also neithal land makes people obese which are one of the causes of Neerizhivu.

SEASONAL INCIDENCE:

There is no serious impact of seasonal variation in Neerizhivu.Muthuvenil kaalam 50%, koothir kaalam 32.5%, munpani 7.5%, pinpani 7.5%, kaarkaalam 2.5%

OCCUPATION:

Incidence of Neerizhivu is common sedentary worker in female is more among housewife 45% due to modernization and invention of electrical electronic kitchen equipment and they lack physical exercise. In male it more among office workers 32.5%.

SOCIO ECONOMIC STATUS:

People belonging to all groups are affected by Neerizhivu.In olden Day it was said that rich community who lack exercise are prove to Neerizhivu.But recent research indicates that the poor were also prove to Neerizhivu.

DIETARY HABITS:

Non vegetarians are more prove to Neerizhivu.People who are habituated to fast food and jung food are more prove as they are rich in calories.

FAMILY HISTORY:

Family history has no relevant history in 55% of cases.It shows that though genetics play an important role in Neerizhivu. Non –Hereditary factors also plays an major role

MUKKUTRAM CLASSSIFFICATION:

IN VALI:

Abanan, viyanan and samanan are affected.

Action of Abanan:

1. Regulation of excretion and feaces.
2. Its derangement leads to polyuria and constipation.

Action of viyanan:

1. Exists all over the body lives in skin and active 72000 naadi.
2. Helps in movement of all organs and sensation.
3. In Neerizhivu viyanan is affects which leads to pain all over the body and peripheral neuritis.

Action of samanan:

1. Help in digestion and absorption of food.
2. Strengthen the body.
3. Samanan is affected in the turn metabolism of carbohydrate, protein and fat are affected.

IN AZHAL:

Action of Analapitham:

1. Action is exactly between the stomach and small intestine which means the pancreatic action is mainly maintained by Analapitham.

2. It derangement causes Madhumegam.

3. Analapitham is affected greatly in all patients.

IN IYAM:

1. The function of Kilethagam is to make the contents of the stomach ready for digestive process.

2. If the function get deranged the initial phase of metabolism gets affected.

3. Kilethagam affected in all patients.

EZHU UDAL KATTUGAL

1. In Neerizhivu saaram and seneer are affected in all patients.

2. Affected saaram leads to emaciate and loss of interest in general activities.

3. Decrese in saaram causes dryness of the skin and mouth.

4. Affected seneer leads to nervousness, dryness, and paling and change in texture of the skin.

ENN VAGAI THERVUGAL:

-It shows Naa and Moothiram affected in100%, Vizhi affected in 15%.

-Sparisam is affected in 35% which causes dry skin and peripheral neuritis.

-Malam is affected in 30%, Niram is affected in 20%.

NEIKURI:

When oil is dropped in urine of the affected patient, it looks like pearl 50%0f cases,35% it spreads slowly and snake like 15%.

NAADI:

In all the patient pitha thondhanaadi is prominent.

I.e. Vatha pitham 55%

Pitha vatham 45%

SIGNS AND SYMPTOMS:

The main symptoms of Neerizhivu are polyurea; polyphagia and polydipsia are present in all cases.

-It has marked improvement in all the above symptoms clinically and their condition was good.

Urine sugar fasting and urine sugar postprandial has become normal in 75% and 62.5% of the cases respectively.

Blood sugar fasting and post prandial has improved in 72.5% and 50% of the cases respectively.

HbA1(C) - 5-7% in 55% of cases shows the valuable management and control of Neerizhivu.

TREATMENT:

MUKKUTRA THEORY

- The treatment is based on the derangement of Mukkutram which again is based on the panchabootham theory.
- Incidence of Neeerizhivu and treatment are also based on these primary principles of siddha medicine.
- The fusion of Neer and Mann gives raise to sweetness.If there is excess of these boothas in the body.It is excreted in the urine, which gives sweetness to urine that is Neerizhivu iyam is primarily dearranged.
- The fusion of Thee +Thee gives raise to pitham,excess of these boothas in the body,it is excreted in the urine, dryness of mouth and derangemend of Sukkilam and suronitham.
- The Medicine chosen to treat Neerizhivu kaippu suvai (Kattru+vinn) which has the property to control Polyurea and excess of fat and balance pitha kutram and kaba kutram.According to the above concept the drug act on the –rinciple of Ethirurai.
- The trial medicine chosen to treat Neerizhivu has Kaippu suvai which is used settle down the pitha kuttrum there by the drug plays an important role on Neerizhivu.
- The drug also subjected to pharmacological and toxicological tests in rat models.The results revealed that the Seeronkottai thiravagam had very effective results and no toxicity was absorbed during the toxicity study.
- The biostatistical report of the clinical trial shows significant result.

SUMMARY

Yugi munivar classified megarogam into 20 varieties in which Neerizhivu is one among them.Disease Neerizhivu come under pitha neer classification which is very specific and correlates with maturity onset diabetes mellitus i.e.non insulin dependent,which is a chronic metabolic disorder.Currently Neerizhivu is considered as one of the worst life style disorders faced by civilized world.

The clinical diagnosis of all the cases of Neerizhivu were done on basis of signs and symptoms explained by yugi in siddha text yugi vaidhya chinthamani -800,Noi Naadal and Noi muthal Naadal and modern concept whereas need.

The disease Neerizhivu has been thoroughly studied by selecting totally 40 patients were treated both OP & IP Department of pothu maruthuvam, Arignar Anna Hospital for period of 2 years.

Patients were examind and investigated with Siddha and Modern concept.

The medicine administered were,

- Seerankottai thiravagam -10 drops with water twice daily after food.
- Encouragingly the patients responded to the medicine showing gradual decrease in signs and symptoms.
- No hypoglycemia was observed during the study.
- HbA1(c) - 5% to 7% in 55% of cases shows the valuable management and control of Neerizhivu.
- Seeronkottai thiravagam also significantly prevents associated symptoms like high bood pressure peripheral neuritis, cholesterol in the blood.
- The drug shows no contra indications and side effect.
- The drug also subjected to Pharmacological and toxicological tests in rat models. The result revealed that the Seeronkottai thiravagam had very effective result. There were no signs of toxicity as could be judged by the absence of undesirable clinical manifestations.
- The biostatistical report of the clinical trial shows significant result.

CONCLUSION

- The drugs selected in this study are very effective and easily available.
- Rasam (mercury) is very specific in treating Megarogam. The drug Seeronkottai is equal to rasam in the action, so this drug specifically treats Neerizhivu.
- The drug acts as Kayakarpam.
- Route of administration is easy.
- As the drug is in Thiravagam from absorption is very quick.
- The drug is a safe and effective one for Neerizhivu.
- The drug has anti-hyperglycemia activity in rat model.
- The drug is economically viable and without any untoward side effect.
- The drug used to prepare the medicine is easily available herb, which even helps the lay man support from this disease.
- The pharmacological study revealed that the Seerankottai thiravagam yield ed good results in rat models.
- The statistical analysis proved significance of clinical improvement with the treatment.
- As the result of this dissertation work is highly encouraging and the medicine being a cost effective one for the treatment of NEERIZHIVU, further detailed study will definitely fruitful to the society.

ANNEXURE –I

CHEMICAL ANALYSIS OF TRIAL MEDICINES

Preparation of Sodium Carbonate extract: 2 gm of the sample is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No.	Experiment	Inference	
		Drug	**Drug**
1	Test for Acid Radicals		
a.	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b.	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2.	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	white precipitate is obtained.	Present

3.	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Yellow Precipitate is obtained.	Absent
4.	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absent
5.	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hydrochloric acid	Absence of Rotten egg smelling	Absent
6.	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absent
7. a.	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absent
b.	5 drops of clear solution is added with 2ml of dilute sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of KMNO4 solution discolourisation.	Absent
8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent

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.	Absence of Green tinged flame	Absent
II.	**TEST FOR BASIC RADICALS**		
10.	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution	Absence of Yellow precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absence of bluish green coloured flame	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent
12.	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Absence of Blood red colour.	Absent
b.	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Absence of Blood red colour	Absent
14.	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate	Absent
15.	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Absence of White precipitate.	Absent
16.	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent

17.	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown precipitate	Absent
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absence of Yellow precipitate	Absent
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absence of Yellow precipitate.	Absent
22.	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23.	**Test of reducing Sugar** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Green colour is obtained.	Present
24.	**Test of the alkaliods** 2ml of the extract is treated with 2ml of potassium iodide solution	Absence of Red colour	Absent
25.	**Test for proteins: (biuret test)** Take 2 ml of solution and 2ml of 5% sodium hydroxide, mix and add 2 drops of copper sulphate.	violet colour is obtained	present

RESULTS

- The given sample contains.
- Drug- serrankottai thiravagam– 2ml
- Chemicals present:
- Chloride
- Reducing sugar
- Protein.

ANNEXURE.-II

ACUTE AND SUB ACUTE TOXICITY STUDY ON SERANKOTTAI THIRAVAGAM

Animals

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house and those animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28^{0}C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

ACUTE TOXICITY STUDY-OECD 425 GUIDELINES

Acute oral toxicity test for the Serankottai Thiravagam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal.

Observation of toxicity signs: General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes

were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

SUB-ACUTE TOXICITY

In a 28-days sub acute toxicity study, twenty four rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Serankottai Thiravagam (p.o.) for 28 days at a dose of 0.5, 1.0 and 1.5ml/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity. The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at 4^{o}C for 10 min to separate the serum and used for the biochemical assays.

Hematological and blood biochemical analyses:

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis glucose, creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

Necropsy:

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

Statistical analysis

Values were represented as mean ± SEM. Data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparison test using GraphPad Instat-V3 software. $P<0.05$ were considered significant.

RESULTS

Animals treated with Serankottai Thiravagam 1ml/kg onwards shown significant toxic clinical signs during the dosing period of 28 days. All animals from control and all the treated dose groups not survived throughout the dosing period of 28 days and it was found four animal dead after 12days of treatment in moderate and high dose. Results of body weight determination of animals of control and different dose groups exhibited body weight loss throughout the dosing period of 28 days. During dosing period, the quantity of food consumed by animals from different dose groups was found to be comparable and normal with that of control animals.

Ophthalmoscopic examination of animals in control and Serankottai Thiravagam treated group revealed remarkable abnormality in liver and kidney. Urine analysis data of control group and treated group of animals determined in week 4 did not reveal any significant abnormalities except pH changes. Comparison of organ weights of treated animals with respective control animals on day 28 was found to be altered. Gross pathological examination of animals in the Serankottai Thiravagam treated group revealed abnormalities.

The results of haematological investigations conducted on day 28, revealed changes in the values of different parameters investigated when compared with those of respective controls; Results of Biochemical investigations revealed the few significant changes in the values of different parameters studied when compared with those of respective controls; however, the values obtained were within biological and laboratory limits.

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Table 1: Dose finding experiment and its behavioral Signs of Toxicity

Dose ml/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	+	+	-	+	-	+	-	+	+	+	-	-	-	+	+	+	+	+	+	+
2	+	+	-	+	-	+	-	+	+	+	-	-	-	+	+	+	+	+	+	+

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9.
2. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of rats exposed to Serankottai Thiravagam for 28days.

Dose (ml/kg/day)	Days				
	1	**7**	**14**	**21**	**28**
Control	141.59±5.19	146.34±5.00	148.26±4.96	153.68±5.42	162.22±6.12*
0.5	152.25±4.87	154.02±4.33	156.22±5.00	141.12±4.56	134.61±4.51*
1	155.14±5.00	157.67±5.10	155.09±4.24	143.56±4.88	132.25±5.02**
1.5	144.42±5.36	146.28±4.42	142.31±4.20	136.62±4.75	130.18±4.99

Values are mean ± S.E.M. (Dunnet 't' test). *P>0.05; **P>0.01. N=6.

Table 3. Food intake of rats exposed to Serankottai Thiravagam for 28days.

Dose (ml/kg/day)	Days (gms/rats)				
	1	7	14	21	28
Control	42.75±2.85	44.33±2.95	47.15±2.55	46.24±2.20	48.26±3.21
0.5	40.22±2.46	45.52±2.61	41.26±2.67	40.51±2.15	41.52±3.00
1	42.44±2.25	45.50±2.46	42.44±2.26	40.22±2.43	40.44±2.82
1.5	42.38±2.16	45.14±2.34	40.52±2.79	38.12±2.45	45.22±2.61

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$. N=6.

Table 4. Water intake of rats exposed to Serankottai Thiravagam for 28days.

Dose (ml/kg/day)	Days(ml/rat)				
	1	7	14	21	28
Control	40.00±2.45	48.22±2.52	45.60±3.36	51.20±2.18*	52.10±3.22*
0.5	42.25±2.28	46.04±2.67	52.41±3.00*	48.22±2.80	52.31±2.74*
1	41.96±2.24	47.18±3.00	52.62±2.88*	50.12±2.66	50.85±3.00
1.5	45.20±2.25	51.34±3.28	54.12±2.54	56.00±2.54*	54.12±2.64

Values are mean ± S.E.M. (Dunnet 't' test). *P<0.05.. N=6.

Table 5. Hematological parameters after 28days treatment with Serankottai Thiravagam.

Parameter	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Red blood cell (mm^3)	5.10±0.42	5.57±0.48	5.12±0.36	5.18±0.40
HB (%)	16.48±0.36	15.81±0.45	14.22±0.34**	15.12±0.31*
Leukocyte ($x10^3$/Cu.mm)	8.00±1.4	8.24±1.12	8.00±0.94	7.16±1.52
Platelets(K/µl)	426±14.11	451±17.00	412±18.33	424±20.24
MCV (gl)	54.33±4.15	54.54±4.24	54.00±4.12	54.02±4.18
Neutrophil	15.17±1.08	15.37±0.88	15.18±0.74	15.26±3.42
Lymphocyte	76.24±2.46	78.02±2.19	80.10±2.15	82.00±2.92
Monocyte	1.32±0.28	1.41±0.30	1.37±0.21	1.66±0.32
Eosinophil	1.00±0.12	2.02±0.11**	2.00±0.10**	3.14±0.12**
Basophil	0±0.00	1±0.01**	2±0.01**	1±0.01**
ESR(mm)	1±00	2±0.01**	3±0.02**	4±0.04**
PCV	52.66±2.74	52.14±2.45	54.18±3.00	53.10±3.05

Values are mean ± S.E.M. (Dunnet 't' test). *P<0.05; **P<0.01. N=6.

Table 6. Effect of treatment with Serankottai Thiravagam biochemical parameters

Dose (mg/kg)	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Total Bilirubin (mg/dL)	0.28±0.02	0.30±0.04	0.30±0.03	0.32±0.04
Bilirubin direct (mg/dL)	0.25±0.04	0.22±0.03	0.25±0.04	0.26±0.05
ALP (U/L)	104.85±4.56	114.20±4.78	110.15±4.22	112.25±5.00
SGOT (U/L)	110.18±4.33	112.54±4.41	117.20±5.12	112.22±5.44
SGPT(U/L)	34.11±2.52	34.17±2.46	34.44±2.95	35.10±2.43
Total Protein(g/dl)	7.30±1.48	6.88±1.00	7.31±0.90	7.10±0.84
Albumin(g/dl)	2.43±0.22	2.35±0.31	2.82±0.24	3.00±0.25
Globulin(g/dl)	5.00±0.28	5.15±0.42	5.00±0.36	4.72±0.32

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$ Vs Control N=6.

Table-7 RFT

Dose (mg/kg)	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Urea (mg/dL)	4.52±1.48	4.44±1.30	5.00±1.85	4.96±1.64
Creatinine (mg/dL)	0.70±0.04	0.70±0.05	0.78±0.05	0.82±0.05
Uric acid (mg/dL)	3.74±0.15	5.13±0.18**	4.55±0.12**	6.12±0.14**
Na m.mol	114.12±5.00	115.02±4.78	115.89±4.70	112.64±3.99
K m.mol	6.42±2.41	6.25±1.44	6.52±1.23	6.34±2.11
Cl m.mol	104.1±4.00	106.20±4.64	105.48±4.75	104.44±5.00

Values are mean ± S.E.M. *P<0.05; **P<0.01. V*s.* Control N=6.

Table-8. Lipid Profile

Dose (mg/kg)	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Total cholestrol(mg/dL)	76.11±5.78	73.04±5.42	70.26±6.20	66.21±4.82
HDL(mg/dL)	38.51±4.10	34.18±4.56	36.24±5.00	32.12±2.78
LDL(mg/dL)	41.00±4.12	42.41±3.38	36.20±2.58	40.45±4.77
VLDL(mg/dl)	24.18±2.55	25.42±2.41	24.28±2.44	24.82±2.64
Triglycerides (mg/dl)	24.10±2.00	25.12±2.15	25.05±2.84	25.14±2.45
Blood glucose(mg/dl)	76.88±4.45	81.22±3.88	72.42±4.00	69.78±4.45

Values are mean ± S.E.M. (Dunnet 't' test). ^{ns}P>0.01 V*s* Control N=6.

Table-9 Urine Analysis

Parameters	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Colour	Yellow	Yellow	Brown	Brown
Transparency	Clear	turbid	cloudy	Turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.2	>8.2	>8.4
Protein	Nil	1+	2+	2+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	+ve
Ketones	-ve	-ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Normal	Normal	Normal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

Table 10. Effect of Serankottai Thiravagam on organ weight

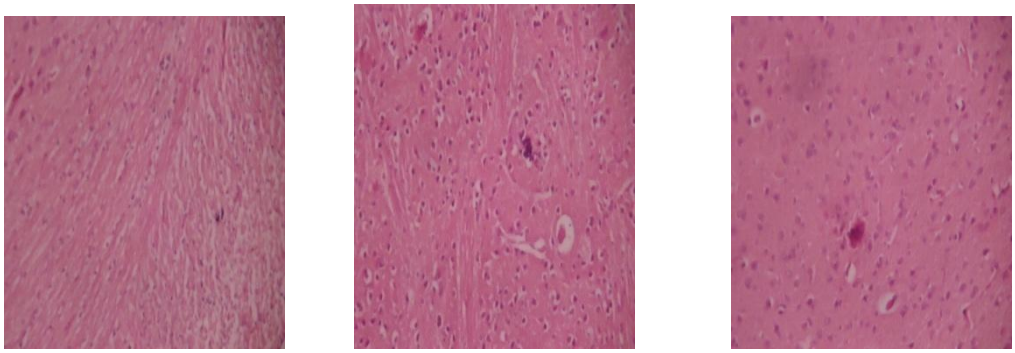
Dose (mg/kg)	Control	0.5ml/kg	1ml/kg	1.5ml/kg
Liver (g)	3.00±0.10	3.10±0.12	2.92±0.09	2.58±0.10*
Heart (g)	0.30±0.04	0.30±0.04	0.29±0.02	0.30±0.02
Lung (g)	0.44±0.12	0.44±0.10	0.45±0.10	0.44±0.12
Spleen (g)	0.45±0.04	0.46±0.04	0.46±0.05	0.47±0.04
Ovary (g)	1.62±0.28	1.64±0.20	1.66±0.18	1.64±0.21
Testes (g)	2.32±0.12	2.34±0.11	2.30±0.10	2.33±0.12
Brain (g)	1.22±0.14	2.20±0.12**	2.20±0.12**	2.10±0.10**
Kidney (g)	0.81±0.05	0.78±0.05	0.75±0.05	0.72±0.04
Stomach (g)	1.12±0.11	1.14±0.12	1.10±0.10	1.12±0.11

Values are mean ± S.E.M. (Dunnet 't' test). *P<0.05; **P<0.01 Vs Control. N=6.

SUBACUTE TOXICITY OF SEERONKOTTAI THIRAVAGAM

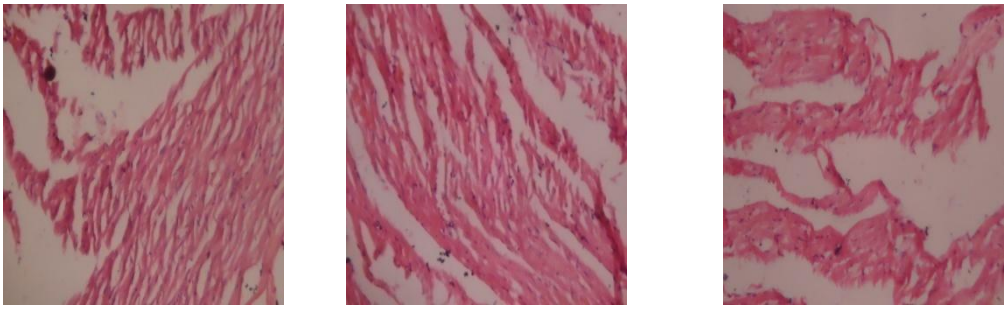
BRAIN

HIGH DOSE LOW DOSE MID DOSE



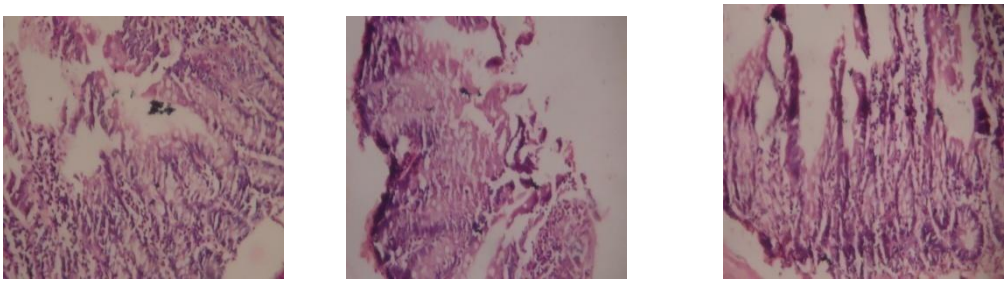
HEART

HIGH DOSE LOW DOSE MID DOSE



INTESTINE

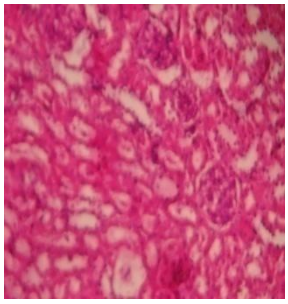
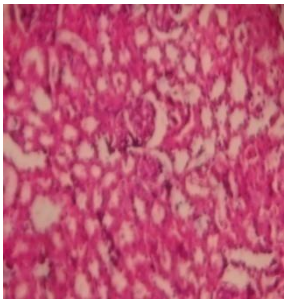
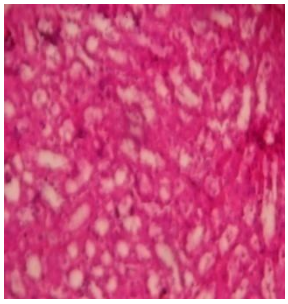
HIGH DOSE LOW DOSE MID DOSE



SUBACUTE TOXICITY OF SEERONKOTTAI THIRAVAGAM

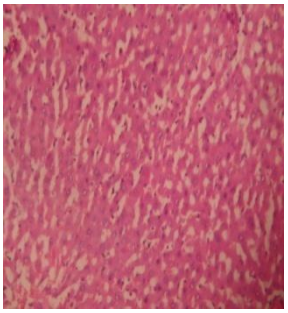
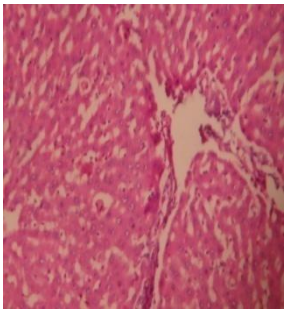
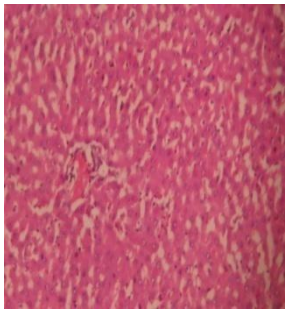
KIDNEY

HIGH DOSE LOW DOSE MID DOSE

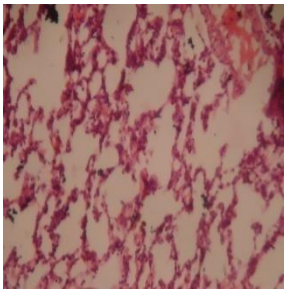
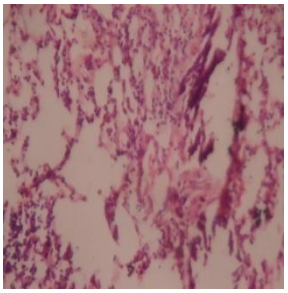
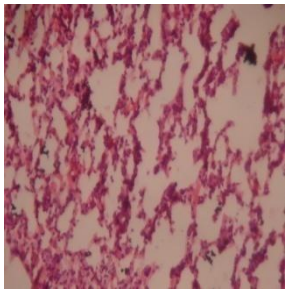
LIVER

HIGH DOSE LOW DOSE MID DOSE

LUNGS

HIGH DOSE LOW DOSE MID DOSE

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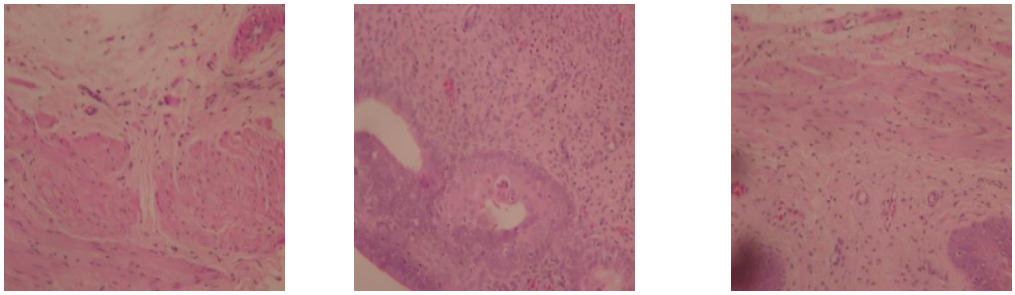
SUBACUTE TOXICITY OF SEERONKOTTAI THIRAVAGAM

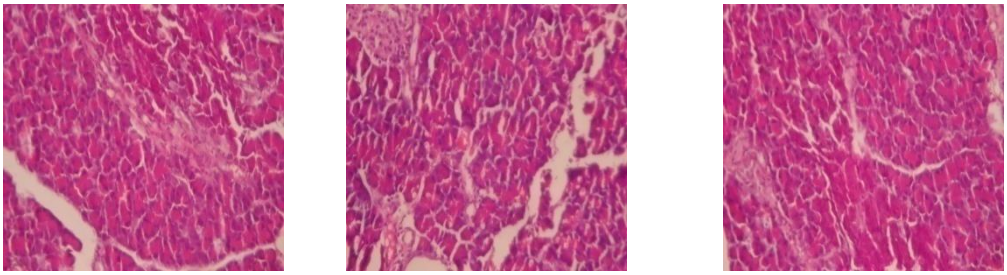
OVARY

HIGH DOSE LOW DOSE MID DOSE



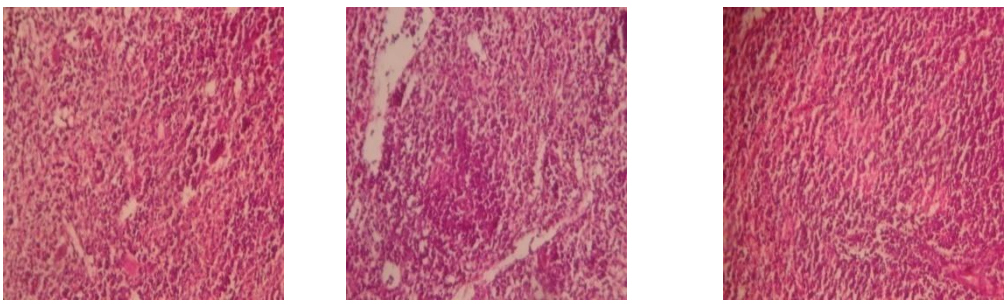
PANCREAS

HIGH DOSE LOW DOSE MID DOSE



SPLEEN

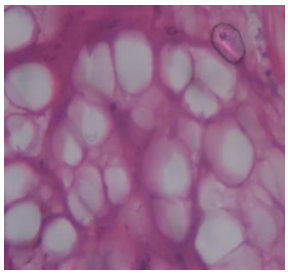
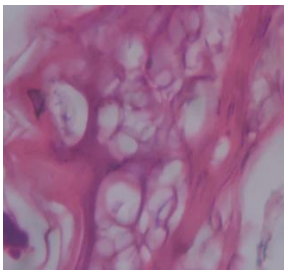
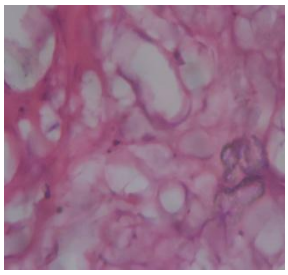
HIGH DOSE LOW DOSE MID DOSSE



SUBACUTE TOXICITY OF SEERONKOTTAI THIRAVAGAM

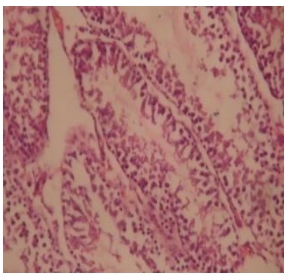
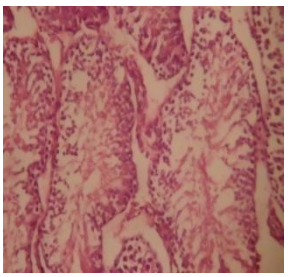
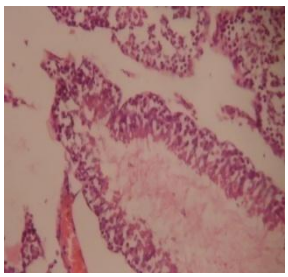
STOMACH

HIGH DOSE LOW DOSE MID DOSE

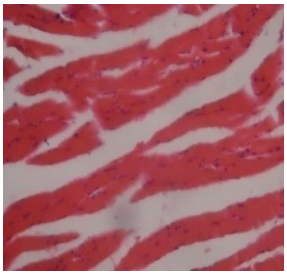
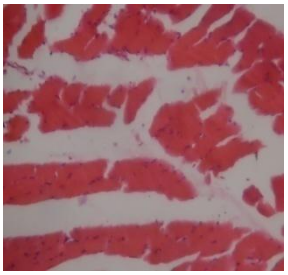
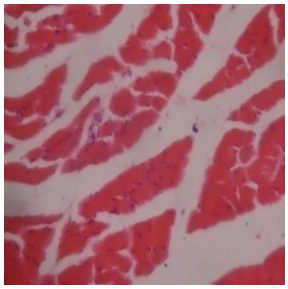
TESTIS

HIGH DOSE LOW DOSE MID DOSE

BONE

HIGH DOSE LOW DOSE MID DOSE

ANNEXURE-III

ANTIDIABETIC ACTIVITY OF SERANKOTTAI THIRAVAGAM IN ALLOXAN INDUCED DIABETIC RATS

INTRODUCTION:

Diabetes mellitus consists of a group of syndromes characterized by hyperglycemia; altered metabolism of lipids, carbohydrates, and proteins; and an increased risk of complications from vascular disease. Apart from currently available therapeutic options for diabetes like oral hypoglycemic agents and insulin. Nowadays, many Indian systems of medicines have been recommended for the treatment of diabetes. Diabetes mellitus occurs throughout the world; however, it is more common in the more developed countries. Diabetes is in the top 10, perhaps in the top 5, of the most significant diseases in the developed world and is still gaining significance. Therefore, it is advised to allow such remedial measures as supplements to other modes of therapy. Alloxan causes diabetes through its ability to destroy the insulin-producing beta cells of the pancreas. *In vitro* studies have shown that alloxan is selectively toxic to pancreatic beta cells, leading to the induction of cell necrosis. The cytotoxic action of alloxan is mediated by reactive oxygen species, with a simultaneous massive increase in cytosolic calcium concentration, leading to a rapid destruction of beta cells. The objective of our present study is to prove the antidiabetic activity of the Serankottai Thiravagam by using alloxan induced diabetic rats.

MATERIALS AND METHODS

Animals

Wistar albino rats (8–10 weeks) of both sexes were obtained from the animal house of School of Pharmacy, Vels University, Chennai. Before and during the experiment, rats were fed with standard diet (Sai durga foods, Bangalore). After randomization into various groups and before initiation of experiment, the rats were acclimatized for a period of 7 days under standard environmental conditions of temperature, relative humidity, and dark/light cycle. Animals described as fasting were deprived of food and water for 16 hours ad libitum. The present study was conducted

after getting experimental protocol approval from Institutional AnimalEthicsCommittee(IAEC).(No. XIII/VELS/PCOL/06/2000/CPCSEA/IAEC/08.08.2012)

Drugs, Chemicals and stock solution

Alloxan (Loba chemie, Mumbai, India), and diagnostic kits (Biolab diagnostics, Mumbai, India) were used in this study. Other chemicals used were of analytical grade and were obtained from local suppliers. The drug Serankottai Thiravagam was diluted with saline. The drug was administered continuously for 21 days orally using an oral feeding tube. The results were compared with that of the standard drug Glibenclamide which was also given continuously for 21 days.

Oral Glucose Tolerance Test

Rats were divided into five groups containing six animals in each group. All animals fasted before treatment. Group I was kept as vehicle control which received only saline p.o., group II received glucose only (2g/kg, p.o.), group III received Serankottai Thiravagam 1ml/kg, and group IV received Serankottai Thiravagam 2ml/kg. The rats of group V were treated with Glibenclamide. Blood samples were collected by puncturing the retro orbital sinus just prior to drug administration, and 30, 90 minutes after loading glucose. Serum glucose level was measured immediately.

Acute Oral Toxicity Studies

Acute oral toxicity study was performed as per OECD-425 guidelines. Mice (n = 6) of either sex selected by random sampling technique were used for acute toxicity study. The animals were kept fasting for overnight providing only water, after which the Serankottai Thiravagam in normal saline was administered orally at the different dose levels in up and down dosing schedule according to body weight by gastric intubation and observed for 14 days.

Experimental Design

Five groups of rats, six in each received the following treatment schedule.

Group I: Normal control (saline).

Group II: Alloxan treated control (150mg/kg.ip).

Group III: Alloxan (150mg/kg.i.p) + Serankottai Thiravagam 1ml/kg, p.o,

Group IV: Alloxan (150mg/kg.ip) + Serankottai Thiravagam 2ml/kg, p.o

Group V: Alloxan (150mg/kg.ip) + Standard drug, Glibenclamide (5mg/kg, p.o).

Serankottai Thiravagam and standard drug glibenclamide (5mg/kg) and saline were administered with the help of feeding cannula. Group I serve as normal control, which received saline for 14 days. Group II to Group V are diabetic control rats. Group III to Group V (which previously received alloxan) are given a fixed dose Serankottai Thiravagam (1ml/kg, p.o), (2ml/kg, p.o) and standard drug glibenclamide (5mg/kg) for 14 consecutive days.

Induction of Diabetes in Experimental Animals

Rats were made diabetic by a single intraperitoneal injection of alloxan monohydrate (150mg/kg). Alloxan was first weighed individually for each animal according to the body weight and then solubilized with 0.2ml saline just prior to injection. Two days after alloxan injection, rats with plasma glucose levels of >150mg/dl were included in the study. Treatment with Serankottai Thiravagam was started 48 h after alloxan injection.

Collection of Blood Sample and Blood Glucose Determination

Blood samples were drawn from tail tip of rat at weekly intervals till the end of study. Fasting blood glucose estimation and body weight measurement were done on day 1, 7, and 14 of the study. Blood glucose estimation was done by one touch electronic glucometer using glucose test strips. On day 14, blood was collected from retro-orbital plexus under mild ether anesthesia from overnight fasted rats and fasting blood sugar was estimated.

Serum was separated and analyzed for serum cholesterol, serum triglycerides, serum HDL, serum LDL was estimated. The whole pancreas from each animal was removed after sacrificing the animal and was collected in 10% formalin solution, and immediately processed by the paraffin technique. Sections of 5μ thickness were cut and stained by haematoxylin and eosin for histological examination.

Statistical Analysis

All the values of body weight, fasting blood sugar, and biochemical estimations were expressed as mean ± standard error of mean (S.E.M.) and analyzed for ANOVA and Dunnet's *t*-test. Differences between groups were considered significant at $P < 0.01$.

RESULTS AND DISCUSSION

The OECD guidelines AOT-425 was followed for estimation of acute toxicity Study in mice. Mortality in the acute oral toxicity test was seen in the dose 2ml/kg. The animals showed severe toxic signs and abnormal behavior like itching, restlessness and aggressiveness etc. Hence, as per the siddha literature recommendations the main stock was further diluted ten times with saline and from this diluted stock solution 1ml and 2ml/kg dose was considered for the further pharmacological evaluation. To ascertain a scientific base for the usefulness of this drug in the treatment of diabetes, it was decided to evaluate experimental design of antidiabetic activity by following glucose tolerance test and the alloxan-induced model.

After alloxon administration, there was severe hyperglycemia in all the animals when compared with the normal animals. The upper bound dose 2ml/kg doses of Serankottai Thiravagam significantly lowered the elevated blood glucose level when compared with that of diabetic control. It was observed that the standard drug glibenclamide lowered the blood glucose level significantly bringing it nearly back to normal, whereas Serankottai Thiravagam significantly (P<0.05) decreased fasting blood serum glucose in the diabetic rats after five days of treatment compared with the initial blood serum glucose levels. However, the sugar control effect of the Serankottai Thiravagam was incomparable to that of the reference drug. In the present investigation, statistical analysis revealed that the 21 days treatment with standard drug Glibenclamide showed significant decrease in glucose, cholesterol, triglyceride VLDL, LDL and increase in body weight and HDL level, thereby exhibited significant antidiabetic activity.

Alloxan, a beta cytotoxin, induces ‘chemical diabetes’ in a wide variety of animal species by damaging the insulin-secreting cells of the pancreas. Though not routinely used anymore, the oral glucose tolerance test is the gold standard for making the diagnosis of type 2 diabetes. It is still commonly used for diagnosing gestational diabetes also. Literature sources indicate that alloxan treated rats are hyperglycemic. The use of alloxan (150 mg/kg b.w.) produced a partial destruction of pancreatic β-cells even though the animals became permanently diabetic. Thus, these animals have surviving β -cells and regeneration is possible. The acute oral toxicity study of Serankottai Thiravagam showed mortality at 2ml/kg. Since the Serankottai Thiravagam was identified with remarkable toxicity at the higher dose in the acute toxicity study. Hence the stability and tolerance was observed at 0.2ml/kg dose level. Administration of diabetogenic agent alloxan 150mg/kg, i.p. lead to elevation of fasting blood glucose levels, which was maintained

over a period of 2 weeks. Alloxan caused body weight reduction (P<0.01), which is reversed by Serankottai Thiravagam at the dose (2ml/kg) is more effectively after 5days of treatment.

The control rats had the blood glucose level 74.52 ± 2.42mg/dl while untreated diabetic rats showed 215.72±10.14mg/dl blood glucose level. On day 5, 10 and 14 of treatment at 2ml dose of Serankottai Thiravagam reduced the blood glucose level to 184.40±10.26, 172.14±8.65 and 158.00±9.00mg/dl (P<0.01) respectively. In present investigation, it was observed that Serankottai Thiravagam can reverse the effects of Alloxan induced diabetes to a significant level.

Histopathological changes in pancreas

Normal - Normal acini and normal cellular population in the islets of Langerhans in pancreas of normal untreated rats.

Diabetic Control- Severe damage to the islets of Langerhans and reduced dimensions of islets results damage of pancreas in alloxan-treated diabetic control rats.

Serankottai Thiravagam 1ml/kg-The moderate damage to the islets of Langerhans and reduced dimensions of islets.

Serankottai Thiravagam 2ml/kg- partial restoration of normal cellular population and enlarged size of β-cells with hyperplasia were seen.

Standard-

Restoration of normal cellular population size of islets with hyperplasia by Glibenclamide was seen.

CONCLUSION

This study results indicates that Serankottai Thiravagam have significant anti-hyperglycemic activities in alloxan-induced hyperglycemic rats with changes in body weight. Hence the above discussion reveals that Serankottai Thiravagam at high dose (2ml/kg) is moderately effective. The knowledge of the system of diabetes mellitus, as the history reveals, existed with the Indians since prehistoric age. 'Madhumeha' is a disease in which a patient passes sweet urine and exhibits sweetness all over the body, i.e., in sweat, mucus, breathe, blood, etc. In the present study, diabetic rats had lower body weights, high blood glucose level as compared to the normal rats.

Treatment with Serankottai Thiravagam not significantly enhanced the average body weights of rats which indicate muscle wasting resulted due to hyperglycemic condition. So it can be concluded that the Serankottai Thiravagam have moderate antidiabetic effects in alloxan-induced diabetic rats. The possible mechanism for this

action might be due to the inhibition of the enzyme glycogen phosphorylase, an enzyme that catalyzes the process of glycogenolysis. This might be the cause for depletion of glucose and lipid parameters such as total cholesterol and triglyceride in hyperglycemic condition. Thus the claim made by the traditional Indian siddha systems of medicine regarding the use of Serankottai Thiravagam in the treatment of diabetes stands confirms.

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Table 1.Oral Glucose Tolerance Test

Treatment (dose / kg body weight)	Blood glucose (mg/dl)		
	Fasting	**30 min**	**90 min**
Normal	72.4 ± 2.4	81.20 ± 2.1**	86.4 ± 2.8**
Glucose; 2g.	72.9 ± 2.6	168.24 ± 1.8	226.04 ± 6.4
SKT (1ml/kg)+Glucose	74.1 ± 2.5	96.20 ± 3.4**	90.12 ± 4.2**
SKT-II (2ml/kg)+Glucose	73.6 ± 2.2	84.42 ± 2.32**	81.00 ± 2.3**
Glibenclamide (5mg/kg)	71.5 ± 2.4	96.18 ± 4.12**	92.15 ± 6.1**

Values are as mean ± S.E.M **P <0.01; Vs group II; n=6

Table.2. Measurement of Body weight changes after Serankottai Thiravagam treatment

Drug treatment	Periodical Weight changes					
	Day0	**Day1**	**Day2**	**Day4**	**Day8**	**Day14**
Normal	158.32± 2.12	161.30± 3.00	163.15±2.52[b],*	164.88±3.45[a],**	168.22±4.18[a,]**	172.70±4.18[a,]**
Diabetic control	160.41± 2.16	156.72± 2.42	152.8±2.20	148.11±2.33	122.34±2.12	113.40±3.20
SKT -I 1ml/kg	158.67± 2.44	160.12± 2.30	158.82±2.54	155.28±4.63	141.00±3.15[a,]**	133.12±3.24[a,]**
SKT -II 2ml/kg	159.52± 2.50	161.40± 2.25	157.17±3.28	143.20±3.46[a]	133.72±3.37[a]	121.10±2.30[a]
Glibenclamide (5mg/kg)	158.45± 2.34	161.76± 2.58	164.42±2.71*	170.16±2.28**	172.53±2.93**	182.12±2.95**

Values are as mean ± S.E.M [a]P <0.001; [b]P <0.05 Vs Normal, **P <0.01; *P <0.05 Vs Diabetic Control; n=6

Table: 3. Fasting serum Glucose concentration is normal and Alloxan-induced diabetic rats

Treatment	Fasting serum Glucose concentration (mg/dl) measured at regular intervals			
	Day 1	**Day 5**	**Day 10**	**Day 14**
Normal	74.52 ± 2.42[a,**]	72.37 ± 3.04[a,**]	74.30 ± 4.10[a,**]	72.23±2.32[a,**]
Diabetic control	215.72±10.14	226.14 ± 11.4	254.25 ±9.12	292.56±12.88
SKT -I 1ml/kg	211.25 ± 8.02[a]	196.22 ± 12.13[a]	182.72 ± 10.00[a,**]	180.21±11.32[a,**]
SKT -II 2ml/kg	222.18 ± 9.24[a]	184.40 ± 10.26[a,*]	172.14 ± 8.65[a,**]	158.00±9.00[a,**]
Glibenclamide (5mg/kg)	230.62 ± 10.11[a]	162.12±8.21[a,**]	158.4 ± 9.14[a,**]	128.65±7.16[a,**]

Values are as mean ± S.E.M [a]P <0.001; [b]P <0.05 Vs Normal, **P <0.01; *P <0.05 Vs Diabetic Control; n=6

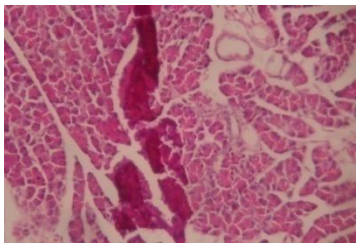
Table: 4. Lipid profile in normal and effect of Serankottai Thiravagam in Alloxan-induced diabetic rats.

Treatment	Dose	Parameters (mg/dl)			
		Total Cholesterol	Triglycerides	HDL	LDL
Normal control	10mg/kg of vehicle	75.60±9.00	70.46±5.88	35.10±8.72	36.22±2.16 [a,**]
Diabetic control	-	82.10±8.92	74.20±10.22	27.02±2.28	138.14±5.21
SKT –I	(1ml/kg)	79.01±6.46	80.62±9.12	36.81±5.62	79.92±5.19 [a,**]
SKT –II	(2ml/kg)	72.18±5.81	74.40±6.51	34.00±3.13	68.10±5.45 [a,**]
Glibenclamide	(5mg/kg)	84.2±6.22	72.51±7.09	40.52±10.00	34.18±5.10

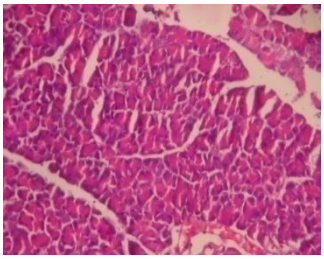
Values are as mean ± S.E.M; [a]P <0.001; [b]P<0.01; [c]P<0.05 Vs Normal; [d]P <0.001 Vs Diabetic; n=6

HISTOPATHOLOGICAL IMAGES OF PANCREASE OF DIFFERENT GROUP

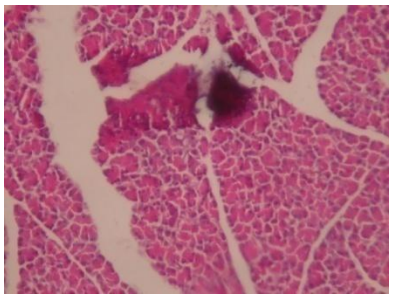
DIABETIC CONTROL



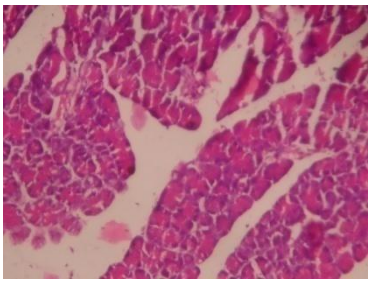
NORMAL



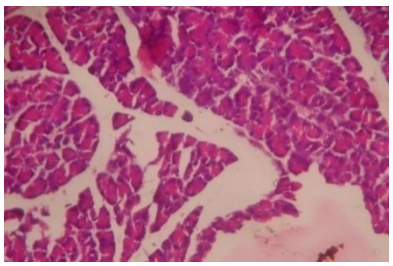
SKT – I



SKT – II



STANDARD



ANNEXURE-V

BIO –STATISTICS

ANTIDIABETIC EFFEICACY OF SEERONKOTTAI THIRAVAGAM ON HUMAN SUBJECTS -A CLINICAL STUDY

INTRODUCTION:

Neerizhivu (Diabetes mellitus) is the commonest endocrine disorder that affects more than 100 million people worldwide. It is caused by deficiency or ineffective production of insulin by pancreas which results in increase or decrease in concentrations of glucose in the blood. It is found to damage many of the body systems, particularly the blood vessels and nerves. The study was carried out in post-graduate's department of Maruthuvam, Government Siddha Medical College attached to Aringnar Anna Hospital of Indian Medicine, Chennai -106.

METHODS

For carrying out the study, clinical protocol was set and was approved by the institutional ethical committee. This study was performed under the supervision of physicians. Inclusion and exclusion criteria were formed for the study. Written consent was taken from the patients. Initial postprandial blood glucose level was estimated at the time of enrolment in the study and then after each week during the entire period of the study. At the end of the study, the initial and final readings were compared.

Inclusion Criteria:

Type II diabetic patients with fasting plasma glucose level equal to or greater than 140 mg/dl of blood without any detectable/visible complications. Type II diabetic patients taking oral hypoglycemic agents with history of inadequate control of blood glucose with these agents. The patients were of either sex (male or female) above 30 years.

Exclusion Criteria:

Pregnant or nursing patients, Smokers, Patients with GIT, hepatic, cardiovascular, renal or endocrine disorder (other than diabetes mellitus) which can interfere with the absorption, metabolism and excretion of the study drug Seeronkottai Thiravagam. Patients with any complication of diabetes mellitus. Patients suffering from type 1 (IDDM) diabetes mellitus.

Subjects:

The selected subjects were medically examined and given code numbers and were asked to present themselves on a specified date for sample collection. Initial postprandial blood glucose level (PPBGL) was estimated at the time of enrolment in the study and then after each week during the entire period of the study.

Blood Sample:

Blood samples (3-5 ml) were drawn from each patient and control subject by vene-puncture through plastic disposable syringes. The blood samples were collected in clean oven dried glass bottles which were previously rinsed with 1% sodium fluoride, 3% potassium oxalate solution to prevent coagulation and glycolysis. The plasma was separated after centrifugation. Any sample showing haemolysis was discarded. After separation of plasma, it was transferred to clean, previously acid rinsed, washed and oven dried glass bottles with plastic caps. The plasma glucose estimation was done immediately on the same day by O-toluidine method.

General Plan of Study:

Seeronkottai thiravagam dried in shadow, were powdered and its decoction was used for the study. A suitable dose was decided by initial randomized study in the first week. The study was performed in two different groups for a period of 16 weeks. Each group was having 20 NIDDM patients.

Group I - In Patients received only Seeronkottai thiravagam 10 ml twice daily.

Group II - Out Patients Seeronkottai thiravagam 10 ml received twice daily.

Drop Outs: No dropouts recorded in the study.

Compliance: All participants in the study were showing the compliance and were following the instruction regarding the diet and exercise.

Untoward Effects: Some of the patients they complained about the flatulence. It may be due to the bulking effect of FG where as some others complained about the headache which may be psychological.

RESULTS

During this dissertation work on Neerizhivu (Diabetes Mellitus)-20 patients were examined as in-patients in the hospital and 20 patients in the out patients department.

Objective parameters analysed were

- Blood Sugar – Fasting before and after treatment.
- Blood sugar – post prandial –Before and after treatment.
- Assessment of HbA1c-Beore and after treatment.

There were significant changes in PPBGL of group II as compared to all other groups. The order of decrease in PPBGL was Group II>I, whereas there were no any significant results seen in control patients. The statistical results are summarized in the Table 1 and Fig.

P value and statistical significance:

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 64.20

95% confidence interval of this difference: From 55.03 to 73.37

Intermediate values used in calculations:

t = 14.1625

df = 39

standard error of difference = 4.533

FASTING

GROUPS	MEAN	SD	SEM
BEFORE TREATMENT	169.18	46.86	7.41
AFTER TREATMENT	104.98	25.41	4.02

P value and statistical significance:

The two-tailed P value is less than 0.0001

By conventional criteria; this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 97.38

95% confidence interval of this difference: From 86.56 to 108.19

Intermediate values used in calculations:

t = 18.2148

df = 39

standard error of difference = 5.346

POST PRIANDIAL

GROUPS	MEAN	SD	SEM
BEFORE TREATMENT	271.68	51.31	8.11
AFTER TREATMENT	174.30	48.70	7.70

P value and statistical significance:

The two-tailed P value is less than 0.0001

By conventional criteria; this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 3.075

95% confidence interval of this difference: From 2.501 to 3.649

Intermediate values used in calculations:

t = 11.2052

df = 19

standard error of difference = 0.274

Hb A1c

GROUPS	MEAN	SD	SEM
BEFORE TREATMENT	10.020	1.348	0.301
AFTER TREATMENT	6.945	0.938	0.210

ANNEXURE-V

CONSENT FORM

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

DATE: SIGNATURE

NAME

CONCENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigation to be performed to monitor and safeguard my body functions.

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

I exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of SERRONKOTTAI THIRAVAGAM for the treatment of NEERIZHIVU.

DATE: SIGNATURE

NAME

CASE SHEET

POST GRADUATE DEPARTMENT - BRANCH-I

(POTHU) MARUTHUVAM

GOVT. SIDDHA MEDICAL COLLEGE & ANNA HOSPITAL, CHENNAI-106.

CASE SHEET PROFORMA FOR "NEERIZHIVU"

WARD NO.	:	NATIONALITY	:
I.P. NO	:	RELIGION	:
BED NO	:	OCCUPATION	:
NAME	:	INCOME	:
AGE	:		
SEX	:	DOA	:
PERMANENT ADDRESS	:		
		DOD	:
		DIAGNOSIS	:
TEMPORARY ADDRESS:			
		MEDICAL OFFICER	:

COMPLAINTS AND DURATION:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS:

PERSONAL HISTORY & HABITS:

FOOD : VEG NON-VEG

MARRITAL STATUS : SINGLE MARRIED

FAMILY HISTORY

GENERAL EXAMINATION:

1. Body built :
2. Consciousness :
3. Nourishment :
4. Decubitus :
5. Anaemia :
6. Jaundice :
7. Cyanosis :
8. Clubbing :
9. Lymphadenopathy :
10. Oedema :
11. Jugular venous pulsations:
12. Enlarged vein :
13. Miscellaneous :

VITAL SIGNS:

14. Pulse rate :
15. Temperature :

16. Respiratory rate :
17. Heart Rate :
18. Blood Pressure. :

SIDDHA ASPECTS:

NILAM (PLACES):

1. Kurinchi (Moutains and their adjoining Areas) :
2. Mullai (Forest and their adjoining Areas) :
3. Marudham (Fertile and their adjoining Areas) :
4. Neithal (Sea and their adjoining Areas) :
5. Paalai (Desert and their adjoining Areas) :

PARUVA KAALAM (SEASONS) :

1. Kaar Kaalam (Aavani-Puratasi) Aug-sept. :
2. Koothir Kaalam (Iypasi-Karthigai) Oct-Nov. :
3. Munpani Kaalam (Maargazhi-Thai) Dec-Jan. :
4. Elavenil Kaalam (Chithirai-Vaikasi) Apr-May :
5. Mudhuvenil Kaalam (Aani-Aadi) Jun-Jul :

YAAKAI (UDAL) :

1. Vali udal :
2. Azhal udal :
3. Iyya Udal :
4. Kalappu Udal :

GUNAM :

1. Sathuva Gunam :
2. Rajo Gunam :
3. Thamo Gunam :

IYAMPORIGAL (SENSORY ORGANS):

1. Mei - Unarthal :
2. Vaai - Suvaiththal :
3. Kan - Parththal :
4. Mooku –Mugarthal :
5. Sevi – Kettal :

KANMENTHIRIYAM / KANMAVIDAYAM:

1. Kai - Koduththal :

2. Kaal - Nadaththal :
3. Vaai - Pesal :
4. Eruvai - Malam Kazhithal :
5. Karuvai – Aananthithal :

PIRA URUPUKALIN NILAI:

1. Irudhayam :
2. Puppusam :
3. Eraippai :
4. Kalleral :
5. Manneeral :
6. Kudal :
7. Siruneeragam :
8. Karuppai :
9. Moolai :

UYIR THATHUKKAL:

Vatham:

1. Pranan :
2. Abanan :
3. Viyanan :
4. Udhanan :
5. Samanan :
6. Naagan :
7. Koorman :
8. Kirukaran :
9. Devadathan :
10. Thanenjeyan :

PITHAM:

1. Anal Pitham :
2. Ranjaga Pitham :
3. Saadhaga Pitham :
4. Aalosaga Pitham :
5. Prasaga Pitham :

KAPHAM:

1. Avalambagam :
2. Kledagam :
3. Podhagam :
4. Tharpagam :
5. Santhigam :

UDAL THATHUKKAL:

1. Saaram :
2. Senneer :
3. Oon :
4. Kozhuppu :
5. Enbu :
6. Moolai :
7. Sukkilam / Suronitham:

Envagai Thervu:

1. Naa - Niram -
   Thanmai -
   Pulan -
2. Niram -
3. Mozhi -
4. Vizhi -
5. Sparisam -
6. Malam - Niram-

Erugal / Elagal-

Manam-

Nurai-

7. Moothiram a. Neerkuri:- a) Niram-

b) Manam-

c) Edai-

d) Nurai-

c) Enjal-

b. Neikuri:-

8. **Naadi** - Thani Naadi -
Kalappu Naadi -
Mukkutra Naadi -
Thondha Naadi -

MODERN ASPECTS:

1. Cardio Vascular System:
2. Respiratory system :
3. Central Nervous System:
4. Genito-Urinary System :
5. Gastro intestinal Tract :

Laboratory Investigation:

I. Blood : TC -
DC-
ESR-
Hb %-
Group-
Urea-
Creatinine-
Cholesterol-
VDRL-
Sputum for AFB-
Mantoux test-
Sugar - Random-
Fasting-
Post Prandial-
Glycosylated Haemoglobin (HbAIC)-
GTT -

2. Urine :
Albumin:
Sugar :
Deposits:

3. Motion:

   Ova-

   Cyst-

4. X-ray Chest : PA View
5. ECG :

CASE SUMMARY

FINAL DIAGNOSIS:

MEDICINE:

1. SEERANKOTTAI THIRAVAGAM-10drops/Bd With water

MEDICAL ADVICE:

Pathiam(Do's and Dont's):

PROGNOSIS CHART:

Signs and Symptoms:

S.No.	CLINICAL FEATURES	BEFORE TREATMENT	DURING TREATMENT		AFTER TREATMENT
			24days	48days	
1.	Polyurea				
2.	Polyphagia				
3.	Polydipsia				
4.	Balanitis / Pruritis vulva				
5.	Itching all over body				
6.	Pain all over body				
7.	Dryness of the mouth and throat				
8.	Constipation				
9.	Skin infection				
10.	Emaciation				
11.	Dry skin				
12.	Peripheral Neuritis				
13.	Ulcer in the foot				
14.	Diabetic cataract				

Medical officer

HOD

DISCHARGE CASE SHEET

POST GRADUATE DEPARTMENT, MARUTHUVAM (BRANCH-I)

GOVT. SIDDHA MEDICAL COLLEGE & HOSPITAL, CHENNAI-106.

"NEERIZHIVU"

IP No	:	OCCUPATION	:
WARD NO	:	INCOME	:
BED NO	:	NATIONALITY	:
NAME	:	RELIGION	:
AGE	:	DOA	:
SEX	:		
ADDRESS	:	DOD	:
		TOTAL NO. OF DAYS TREATED	:
		RESULT	:
		DIAGNOSIS	:
		MEDICAL OFFICER	:

S.No.	CLINICAL FEATURES	DURING ADMISSION	DURING DISCHARGE
1.	Polyurea		
2.	Polyphagia		
3.	Palydipsia		
4.	Balanitis / Pruritis vulva		
5.	Itching all over body		
6.	Pain all over body		
7.	Dryness of the mouth and throat		
8.	Constipation		
9.	Skin infection		
10.	Emaciation		
11.	Dry skin		
12.	Peripheral Neuritis		
13.	Ulcer in the foot		
14.	Diabetic cataract		

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VEL'S COLLEGE OF PHARMACY

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S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
4.	Evaluation of ovulation inducing activity for infertility and toxicological studies for Uppu parpam.	Dr. N. Kavitha	According to the protocol 36 rats were proposed, but while scrutinizing for pooling the final data, only 30 rats were sanctioned.	XIII/VELS/PCOL/04/2000/CPCSEA/IAEC/11.08.2012
5.	Analgesic activity of Karungali Ver Kudineer in rodents	Dr. S. Umera	Total number of animals proposed was 60 rats and mice. But only 60 mice and 18rats were sanctioned because, it was advised to share the control and standard group results. Since the similar pattern of the study has been planned in the same department, hence these data will serve as common.	XIII/VELS/PCOL/05/2000/CPCSEA/IAEC/11.08.2012
6.	A study on Serankottai Thiravagam for the treatment of Diabetes.	Dr. A. Chinnaswamy	Total number of animals proposed was 42 rats. But only 36 animals were sanctioned.	XIII/VELS/PCOL/06/2000/CPCSEA/IAEC/11.08.2012
7.	A study of Kanthaga parpam for the treatment of Arthritis (Kumbavatham).	Dr. G. Krishnaprakash	Total number of animals proposed was 36 rats and sanctioned.	XIII/VELS/PCOL/07/2000/CPCSEA/IAEC/11.08.2012
8.	Hypolipidemic activity of Kadukkai chooranam.	Dr. F. Priya	Total number of animals proposed was 48 rats, and it was advised to minimize the number to 40 rats only.	XIII/VELS/PCOL/08/2000/CPCSEA/IAEC/11.08.2012

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