AN OPEN NON RANDOMIZED CLINICAL TRIAL OF PERUNGAYA CHOORANAM IN MADHUMEGAM (DIABETES MELLITUS TYPE-II)

The dissertation submitted by

Dr. K. NITHYA (Reg. No. 321511107)

Under the Guidance of

Prof. Dr. N. ANBU, M.D.(S)

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements
For the award of the degree of

SIDDHA MARUTHUVA PERARIGNAR

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I – MARUTHUVAM

POST GRADUATE DEPARTMENT OF MARUTHUVAM

THE GOVERNMENT SIDDHA MEDICAL COLLEGE

CHENNAI – 106

OCTOBER - 2018

CERTIFICATE

This is to certify that the dissertation entitled **"AN OPEN NON-RANDOMIZED CLINICAL TRIAL OF PERUNGAYA CHOORANAM IN MADHUMEGAM"** is a bonafide work done by Dr. **K.NITHYA** Government Siddha Medical College, Chennai – 600106 in partial fulfillment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2015 -2018.

Name & Signature of the Guide

Name & Signature of the HOD

Name & Signature of the Principal

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CONTENTS

INTRODUCTION

INTRODUCTION

Every Traditional System of medicine was directly connected with our culture and tradition. Evolution of human society, Tamil civilization peoples obviously learn the cause of human sufferings not only disease, ageing, life problems it absolutely about the purpose of human life. Some peoples understood the purpose of human life and they lives accordingly to achieve enlightenment called Siddhars.

Siddhars, men of highly cultured intellectuals and spiritual faculties combined with supernatural powers so called sages, saints. They wrote their experiences in palm manuscripts to share knowledge to human society. They strongly believed that healthful body is essential to attain the immortal life.

To achieve the goal of human life every human needs healthy body. So they discover medicines to cure human sufferings. This medicine was collectively called as Siddha system medicine. This system of medicine imitate with our mother nature.

The Siddhar, Thirumoolar has written in his song

"உடம்பா லழியில் உயிராலழிவர்

திடம்பட மெய்ஞனம் சேரவும் மாட்டார்

உடம்பை வளர்க்கும் உபாயம் அறிந்தே

உடம்பை வளர்தேன் உயிர் வளர்தேனே"

- திருமூலர் [1]

Accordingly the physical and mental wellbeing is more important to lead a healthy life. Nutritious lifestyle comprises all day to day activities and food habits.

There are five basic primordial elements in nature viz. earth, water, fire, wind, ether and everything is made up of these elements. The food we intake consists of six taste viz. sweet, sour, salt, bitter, acrid and astringent. These six tastes are formed by the selective combination of those five primordial elements.

Moreover there are three humours viz., Vatham, Pitham and Kabam, which is responsible for the physical and mental quality and also maintain the healthy body.

Any changes in the food habits and lifestyle brings imbalance of vatham, pitham and kabham which is the cause for the disease. Thus there is an interconnection between the five primordial elements, six tastes and three humours which are the basic fundamental of Siddha System.

The Siddha treatment peruses at the proper balance and equilibrium of the three humours through proper diet and perfect lifestyle.

In recent years the Siddha system has its dawn among worldwide for its natural inheritance, holistic approach, healthy lifestyle and preventive treatment.

Siddhar Therayar, one of the pioneers of the Siddha medical system, he explained excessive urination or decreased urination comes under by Neerinai perukkal noi and Nerinai arukkal noi. So Madhumegam is classified under "Neerinai perrukkal noi"[(2)].

Yugi Muni describes 20 types of Meganoigal, Madhumegam is one among them.

The word Madhumegam is very similar and closely resembles with the chronic metabolic disorder called Diabetes Mellitus-Type II in Modern medicine. This is mainly because of the Etymology that,

"Mellitus" means sweetness which means "Madhu".

"Diabetes" means "Passing like a Fountain"

In the world Non communicable diseases silently attacks more people every day. In this diabetes mellitus is the most common one.

Diabetes mellitus is a group of metabolic disorder in which a person has high blood sugar, either because the pancreas does not produce enough Insulin, or because cells do not respond to the Insulin that is produced. This high blood sugar produces the classical symptoms of Polyuria (frequent urination), Polydipsia (increased thirst) and Polyphagia (increased hunger) [(3)].

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals. The study conducted by ICMR revealed that a lower population is affected in Tamilnadu is 4.8 million [(4)].

WHO projects that diabetes will be the seventh leading cause of death in 2030 (who fact sheet November 2017) [(5)].

The sudden spurt in life style modification and vast urbanization has made India the Diabetic capital of the World.

Seeking the solution for this, our attention heads towards natural ways and medicines especially Siddha system of medicine. Siddha medicine is an unique one as it is not only a curative but also preventive and to achieve the salubrious body and mind.

Siddha medicine plays an effective role in treating Diabetes mellitus. At present most of the anti-diabetic medicines used for a long period, produce hypoglycemia and may bring many undesirable side effects too. So there is a need for the medicine which could be more potent and cause lower side effects to the mankind continues. So I consider my medicine ***"Perungaya Chooranam"*** as a unique preparation formulated by Siddhars against Diabetes mellitus.

AIM
AND
OBJECTIVES

AIM AND OBJECTIVES

AIM:

The Aim of this study is to evaluate the Clinical Efficacy and Safety of Siddha Medicine “Perungaya chooranam” in the management of Madhumegam.

OBJECTIVES:

1) To review the Siddha literary evidences dealing with Aetiology, Classifications, Signs & Symptoms, Diagnosis, Diet and Prognosis of Madhumegam in Siddha system of Medicine.
2) To study Madhumegam in various literatures in comparison with Diabetes Mellitus -Type II.
3) To understand the incidence of the disease with reference to Age, Sex, Thinaigal, Paruvakaalam, Socio Economic conditions, Diet and Family history.
4) To explore the unique diagnostic methods mentioned by Siddhars such as Envagai thervu, Mukkuttram, Udal thathukkal with specific reference by Naadi, Neerkuri and Neikuri.
5) To implement Siddha and utilize Modern parameters to diagnose and to confirm the severity & progress of the disease.
6) To evaluate the Bio-chemical analysis of the trial medicine.
7) To assess the Acute and Sub- Acute toxicity of the trial medicine.
8) To study the Anti- Diabetic activity of the trial medicine.
9) To evaluate the Clinical study of the trial medicine.
10) To analyze the Biostatic analysis of the trial medicine.

REVIEW OF LITERATURE

SIDDHA ASPECT

SIDDHA ASPECT

"தக்க தாரணி மானிடத்தோர்கள்
பக்கமாசலம் பத்திருவகையுமே
நக்க நாயகன் நாயகிக்கே சொல்
மிக்க நந்தி விளம்பி விதித்ததே"

- தேரையர் வாகடம் [6]

According to Therayar Vagadam, "The universe consist of two essential existences that is, matter and energy which Siddhar's referred to as Shiva and Sakthi". Shiva explained Megarogam to Sakthi. Here Nandhi explains its symptoms to the world for the benefit of the human kind.

"ஆமப்பா மனிதர் செய்த கன்மத்தாலே
அரகரா மேகமென்ற ராசாவாலே"

- அகத்தியர் [7]

This clearly indicates that the existence of this disease is as old as human race.

"மிகினும் குறையினும் நோய்செய்யும் நூலோர்
வளி முதலா எண்ணிய மூன்று"

- திருக்குறள் [8]

All the diseases are due to alteration of three vital humours and seven physical Constituents.The factors, which affect this equilibrium of vital humours are,

- Unavu Marupadugal (Altered diet habit)
- Kala marupadugal (Seasonal variations)
- Thega vanmai (Depending upon immunological status)

According to Siddhars, the imbalance of tridosha causes totally 4448 diseases to human beings. Among them, Megarogam is considered to be the emperor of diseases.

Madhumegam has its description in various literatures like Yugi Vaithya Chindamani, Agasthiyar Gunavagadam.

Earlier, diseases were classified only according to Mukkutram. Yugi Munivar classified the diseases according to cause, signs and symptoms and also explained about the prognosis, treatment and diet, which is now followed by the modern world. Madhumegam comes under Neerinai perukkal Noi, mentioned in Therayar Maha Karisal.

"நீரிருவினைக் குணத்தை நீயறி விரித்துச் சொல்வாம்
நீரினைப் பெருக்கலொன்று நீரினை யருக்க லொன்று"

- தேரையர் மகா கரிசல் [9]

VERUPEYARGAL (SYNONYMS)

Neerizhivu, Enippuneer, Vegumoothiram, Thithippuneer, Miguneer [10]

Neerizhivu	- Excess of urination.
Enippu Neer/ Thithippu neer	- The urine is sweet in taste.
Vegu Moothiram	- Frequency and large quantity of urine passed
Pramegam	- Sanskrit name for Madhumegam

IYAL (DEFINITION)

Madhumegam is a clinical condition characterized by frequent passage of urine more than the normal resulting in deterioration and diminution of the seven thathus.

இனிப்பான இனிப்பல்ல ஈ வந்தாடும்
ஒருதுளிவாய் விட்டார்கைப் பிணியாய் தோன்றும்

- குரு நாடி [11]

The above quote describes that ant and flies are attracted to the site of voided urine and when the urine is heated, it gives honey odour.

"தண்மையாய்ச் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கும் பீசமுங்கோ சமுங்க டுக்கும்
அண்மையா யடிக்கடிக்கு நீரிறங்கு
மடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையான யடியதனிற் றான்பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனிகன்றும்
பண்மையாய்ப் பஞ்சவாண் டதனிற் கொல்லும்
பகிர்கின்ற மதுமேகப் பாங்கு தானே"

- யூகி வைத்திய சிந்தாமணி [12]

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

"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 [13]

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

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

NOI VARUM VAZHI-AETIOLOGY:

"மேகமெனும் நீரிழிவு விதத்தை
விளம்புகிறேன் முன்செய்த கர் மந்தன்னால்
தாகமுடன் மதுபதார்த்தங்கள் நன்றாய்த்
தான்புசித்த லாலுஞ்சிற்றினத்தின் மங்கை
போகமதி கரித்தலா லுட்டின்ந்தான்
போதவே மிஞ்சுதலால் தயிர்மோர் நெய்பால்
ஏகமாய்ப் புசித்தலாற் கொழுத்த வூனை
யென்று முண்ண லுவந்நீரைக் குடித்தலாலே
ஆசையுடன் சிறுவழுதலங்காய் தன்னை
யதிகமா யுண்பதால் நடையலைச்சல்
போதவே யிருத்தலிரா கண்விழித்தல்
தேசமெங்கு திரிதலா மிவைகளாலே
சிரந்தனிற்சூ ட்திகங் கொண்டுடனே ரத்தம்
சோஷிதே யதிகமாய் மேகந்தோன்றித்
தொல்லை செய்யும் நீரிழிவும் இருபதாமே"

- சரபேந்திர மேகநிவாரண போதினி ஏன்னும் நிரிழிவுநோய் மருத்துவம் [14]

DIET HABITS :

"கோதையர் கலவி போதை
கொழுத்தமீ னீறைச்சி போதை
பாதுவாய் நெய்யுண் பாலும்
பரிவுட ண்பீ ராகில்
சோதபாண் டுருவ மிக்க
சுக்கில பிரமே கந்தான்
ஒதுநீ ரிழிவு சேர
உண்டென வறிந்து கொள்ளே"

- அகத்தியர் 1200 [15]

"உற்பவிக்கும் பால்நெய்யா லிறைச்சி கள்ளால்
உரிசையாய் மீன்றன்னா ல்வருவி ருத்த
மற்பவிக்கும் பதார்த்தத்தால் மதுர வஸ்தால்
மந்தங்கள் தனிற்பொசித்தல் வேகாப் பண்டம்
குற்பவிக்குங் குளிந்தவன்ன மங்கை கோஷ்டி
குறித்த நித்திரைதவிர் தலக்கினி மந்தம்
தற்பவிக்குஞ் சரீரந்தான் மிகப்பருக்கல்
சஞ்சலந்தான் பயன்படுதல் தரிக்கும் நோயெ"

- யூகி வைத்திய சிந்தாமணி [16]

Excessive intake of food, rich in carbohydrate and fat, red meat, sweet food, Raw food and sleeplessness induces mathumegam, which was quoted by Agathiyar and Yugi Munivar.

SEXUAL INDULGENCE:

"கன்னி மயக்கத்தால் கண்டிடு மேகமே"

- நாடி நூல் [17]

"கிரந்திப் புண்ணிரண மேகக்
கீசக னென்னுந் துன் மார்க்கன்
அருந்ததி யென்னும் பாஞ்சாலி
யன்னையைக் கண்ணுற்றானே"

- தேரையர் மருத்துவ பாரதம் [18]

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

OBESITY :

"தற்பவிக்குஞ் சரீரந்தான் மிகப்ப ருக்கல்
சஞ்சலந்தான் பயன்படுதல் தரிக்கும் நோயே"

- யூகி வைத்திய சிந்தாமணி [19]

Obesity is one of the main cause of Madhumegam.

PSYCHOSOMATIC CAUSE:

"இயம்பவே ஆறுகுணம் பின்னஞ் செய்தல்
ஏற்றமாய் மாற்றான்பெண் சங்கம் செய்தல்
பயம்பவே பாலகர்களுக் கொளித்து தின்னல்
பழமை சலம்போறவ ழிதனை தடுத்தல்
அயம்பவே ஆலயத்திற் சலம்விட்டோர்க்கும்
ஆதியாம் வேதத்தைத் தூஷித் தோர்க்கும்
துயம்பவே சூரியனை வணங்கா தார்க்கும்
சுருக்காக மேகம்வந்ததுற் பவிக்குந் தானே"

- யூகி வைத்திய சிந்தாமணி [22]

According to Yugi Vaidya Chinthamani, Megaroham may occur due to not giving proper respect to Guru, Father, Mother, Vedas and God Suriyan.

HEREDITARY:

"முறைகேட்கின் ஒன்பது முயற்சியால் வந்தது
தறை கேட்கிற கருப்பத்திற் றுவங்கிய மேகங்கள்
பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறை போற்றுங் கருப்பத்தில் வளர்ந்தது மேகமே"

- அகத்தியர் வைத்திய காவியம் [21]

Thirumoolar have noted in his literatures that Hereditary is one among the causes of the disease Madhumegam. At present, researches have also found that genetic factors play an important role in Madhumegam.

EXCESS STIMULATION OF MOOLATHARAM:

"சரியானமேகத்தா லபான வாயு
தான் புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெரிதான மேகத்தா லத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தச வாய்வால் மந்தம் கொண்டு
பெருந்தீனி மலபந்தம் உதான வாயு
விரிவாகித் தேகமெல்லாம் விட நீராலே
மெய்யழிந்த தேகமென்ற திருபதாச்சே"

- நோய் நாடல் (22)

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

In the Madhumegam 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.

DEEDS:

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

- தேரையர் மருத்துவ பாரதம் (23)

From the above poem, the diseases also occur as a result of bad deeds committed in previous or this birth.

MURKURIGUNAM (PREMONITORY SYMPTOMS):

Premonitory symptoms of Madhumegam are polyuria, polyphagia, polydipsia. Madhumegam 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 thirst is excreted as excessive quantity of urine (poly uria). In spite of abnormal consumption of food, stamina continues to decrease.

PODHU KURIGUNANGAL (GENERAL SIGNS & SYMPTOMS)

"கூறான மேகமது விருப துக்குங்
குணந்தனைச் சிவன்சொல்லத் தேவி கேட்கத்
தாறான தாகமொடு சோக மேக்கந்
தரியாமல் நீரழித லிருமல் மூச்சு
ஆறான அருசிசர்த்தி சித்தப் பிரமை
யடிக்கடித்வ் தண்ணீர்தா னன்னங் கேட்டல்
ஈறான விடுப்புக்குக் கடுப்பு காணல்
எலும்புழற்ற லழற்றலோ டெரிவுண் டாமே"

"எரிவோடு சரீரமெலா மறைபட் டாற்போ
லெழிலுடம்பு நோதல்நித் திரையில் லாமை
வரிவொடு வாயுமைத் தவும்ப ரித்தல்
மனதுசஞ் சலப்படுதல் காற்று வேண்டல்
மெரிவோடு மேல் மூச்சு மிகவுண் டாதல்
விக்கலொடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளிறுண் டாதல்
தேகமெத்த வாலோபப் படுதல் காணே"

- யூகி வைத்திய சிந்தாமணி [24]

COMMON SYMPTOMS:

Thirst, Polydipsia, Anorexia, Polyuria, Dyspnoea, Burning sensation, Sleeplessness, Giddiness, Flatulence, Anaemia, Loss of weight, Cough, Pain in the hip, Hiccough.

NOI VAGAIKAL - (CLASSIFICATION):

Megarogam is classified into twenty varieties to quote from Agasthiar.

"உட்டிண ரோகத்தாலும் உறும்பெரும்பசியினாலுங்
கட்டவிழ் கோதை மாதர்கலவிமட்டிலா மையலாலு
முட்டறா நாலுமாறு மும்மூன்று மொன்று மென்று
திட்டமாய் வருவதென்று திருமாமுனி யருளிச் செய்தார்"

- அகத்தியர் [25]

Yugi Munivar classifies the same as,

"வசனித்த மேகமது யிரண்டு பத்து
வாதத்திற் பிறந்தசலம் நாலே யாகும்
பிசனித்த பித்தத்திலு ற்றப வித்த
பேரான சலந்தானு மாறு மாகும்
தெசனிந்த சேட்டுமத்திலுற்ப வித்த
சீரான சலந்தானும் பத்தேயாகும்"

- யூகி வைத்திய சிந்தாமணி [26]

According to Theraiyar

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

- தேரையர் வாகடம் [27]

NOI VAGAIGAL:

BOOKS	NOI ENN	VALI	AZHAL	IYAM
யூகி வைத்திய சிந்தாமணி	**20**	**4**	**6**	**10**
அகத்தியர் 1200	**20**	**4**	**6**	**10**
தேரையர் வாகடம்	**20**	**4**	**6**	**10**
தன்வந்திரி வைத்தியன்	**20**	**4**	**6**	**10**
சரபேந்திர நீரிழிவு ரோக சிகிச்சை	**20**	**4**	**6**	**10**
யூகி வைத்திய காவியம்	**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 these twenty different kinds, four varieties are caused by Vali, six varieties are caused by Azhal, ten varieties are caused by Iyam.

Madhumegam comes under the classification of Azhal.

CLASSIFICATION OF MEGAM:

According to Yugi Vaidhya Chinthamani, Yugi described

four types under the Vatha premeham

six types under the pitha prameham and

ten types under Kaba prameham**.**

VAADHA NEER VAGAIGAL:

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

- யூகி வைத்திய சிந்தாமணி (28)

VALI – 4

1. Neimananeer
2. Pasumana neer
3. Seezhmana neer
4. Sathaimana neer

PITHA NEER VAGAIGAL:

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

- யூகி வைத்திய சிந்தாமணி (29)

AZHAL – 6

1. Yanai kozhupu mana neer
2. Katrazhai mana neer
3. Chunna mana neer
4. Innipu neer
5. Palingu neer
6. Muyal kurithi neer

IYA NEER VAGAIGAL:

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

- யூகி வைத்திய சிந்தாமணி [30]

IYAM – 10

1. Iaya Neer
2. Thuimai Neer
3. Moolai neer
4. Ilaneer
5. Kal neer
6. Thavala Neer
7. Kazhu neer
8. Then neer
9. Uppu neer
10. Kavichi Neer

NOIKURI KUNANGAL – (CLINICAL FEATURES)

Polyuria, Polyphagia, Polydypsia, perspiration, exhaustion, insomnia, giddiness and loss of weight are seen even at normal consumption of food.

COMMON SIGN AND SYMPTOMS OF PITHA PRAMEHAM:

"அறியவே பித்தசல மாறுக்குந்தான்
அங்கமதிற் செய்கின்ற குணத்தைக் கேளாய்
தறியவே சரீரம் வற்றி யெரிவுங்டாகும்
சடத்திலுந் நீரிலுந்தான் கவிச்சுண்டாகும்
தெறியவே சீப்போலுங் கற்றாழை போலும்
சேல் போலும் 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 தண்ணீந் தாகம்
சக்தியொடு சரீரமெல்லாந் தளர்ச்சி யாகும்
தாக்கடா மலசஞ்சலந்தான் மிகவுண்டாகும்
சமகுணந்தான் பித்த சல மாறு மாச்சே"

- யூகி வைத்திய சிந்தாமணி [31]

As per the above poem, polyuria, polyphagia, polydipsia, fever, angular stomatitis, burning sensation all over the body, loss of weight, are common signs and symptoms of pitha prameham.

MUKKUTRA IYAL:

VALI :

Sites of Vadha:

Below Naval, Urinary bladder, intestines, Pelvis, umbilical cord, thigh, bone, skin, nerve endings, joints, Musculature, hair root.

Properties:

Dryness, Lightness, Clearness, Coolness, Mobile, Formless.

Function:

- Praanan (Uyirkaal) :

This controls knowledge, mind and five sense organs, which are useful for breathing and digestion.

- Abaanan (Keezh nokku kaal) :

This is responsible for all down ward movements such as passing urine, stools, semen, menstrual flow etc

- Samaanan (Nadukkaal) :

This aids in proper digestion.

- Viyaanan (paravukaal):

This is responsible for all movements of all parts of the body.

- Uthaanan (Mel Nokkukaal) :

Responsible for all upward visceral movements, such as vomiting, eructation and nausea.

- Naagan :

Responsible for opening and closing the eyes.

- Koorman :

Responsible for vision and yawning.

- Kirukaran :

Responsible for salivation, nasal secretion and appetite.

- ` Devathatthan :

Responsible for Laziness, sleeping and anger.

- Thananjeyan :

Produces bloating of the body after death. It escapes on the third day after death bursting out of the cranium.

In Madhumegam

- 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 : Normal
- Samanan : Poly Phagia
- Nagan : Normal
- Koorman : Diabeic 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, eyes and skin.

Properties:

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

Function

1. Anal Pittham : It promotes appetite and helps in digestion.

2. Ranjagam : It gives colour to the blood.

3. Praasagam : It gives complexion to the skin.

4. Aalosagam : It brightens the eyes.

5. Saathagam : It controls the whole body. It has the property to fulfil all the activities which the mind desires.

In Madhu megam

Anala Pitham - Excess hunger

Ranjaga pitham - Pallor sometimes

Alosagapitham - Dimness of vision

Saathaga pitham- Lassitude

Prasaga 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 and stable.

Function:

- Avalambagam :

  Lies in the lungs, controls the heart and other kabhams.

- Kilethagam :

  Lies in the stomach, makes the food moist, soft and helps in digestion.

- Pothagam :

  Responsible for identifying taste.

- Tharpagam :

  Present in the head and responsible for the coolness of eyes.

- Santhigam :

  Responsible for lubrication and free movements of joints.

In Madhumegam

Avalambagam - Normal

Tharpagam - Burning sensation in the eyes

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 Madhumegam:

Saaram : Tiredness, General weakness

Senneer : Pallor

Oon : Emaciation

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 (PATHOGENISIS):

The disease Megaroham, due to external (or) internal causes affects 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.

PINIARI MURAIMAI- (DIAGNOSIS):

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

- **Poriyal Arithal** (or) understanding by the five organs of perception (Mei, Vai, Kann, Mooku, Sevi).
- **Pulanal Arithal** (or) understanding by the sense objects (Uraithal, Suvaithal, Parthal, Mugarthal and Kettal)**.**
- **Vinadal** (or) Interrogation.

Tools used by Siddha Physicians:

- Kaandal (Perception)
- Karuthal (Inference)
- Urai (The instruction of the inspired)

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

ENN VAGAI THERVU (EIGHT TOOLS OF DIAGNOSIS):

Naa:

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. In Madhumegam, 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 and hair etc.

Vatha Udal - Black and Whitish colour

Pitha Udal - Yellow or Reddish colour

Kabha Udal - White or Golden colour

Thontha Udal - Mix of two udal colours.

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

Mozhi:

Observation of speech and voice.

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

Vizhi:

Colour, character, vision should be observed.

In uncontrolled Madhumegam cataract set in last.

In longstanding cases, the Madhumegam affects retina and causes diabetic retinopathy which is the major cause of blindness.

Sparisam:

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 Madhumegam, increased tendency for fungal infection like moniliasis and vulvities.

In Madhumegam the skin is dry and pale.

Malam:

Quantity, colour, smell, froth should be observed.

In Madhumegam, constipation sometimes yellowish loose stool are passed.

Moothiram:

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

Urine:

Colour : In Madhumegam it is clear and white.

Specific Gravity : In Madhumegam, urine is thick in consistency like honey.

Smell : Honey like smell

Froths : In Madhumegam the urine is frothy at the time of urination.

Deposits : In Madhumegam few epithelial cells are present in urine.

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

Disturbing Polyuria at night (nocturia) and Glucosuria (the presence of sugar in urine) are present.

NEER NIRAKKURI:

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

- நோய் நாடல் [32]

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. The patient must have sound sleep on the previous night. The urine

is collected on the dawn of the next day in a glass container and closed immediately to prevent contamination. This specimen must be examined with in one and half hours. This procedure should be follow strictly to get accurate observation of Neerkuri and Neikuri**.**

NEIKKURI:

"நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடு விடுத்
தென்றுறத் திறந்தொலி யேகாதமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுந் செய்தியை யுணரே"

- நோய் நாடல் (33)

The diagnosis and prognosis of deranged Mukkutrams are studied on the basis 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 மொழிவதென் கபமே"

- நோய் நாடல் (34)

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

NAADI:

Pulse is the confirmatory diagnosis.

In Madhumegam,

"இருமியே பித்தமும் வாதமும் கூடில்
மருவுசல மேகம் வாருதி போலாகும்
உருவம் வேறாகு முண்டவுடன் காந்திடும்
உருகவே வூனோடு உறிஞ்சி இனிக்குமே"

- திருமூலர் (35)

Coupling of Azhal and vadha naadi causes excessive urine as vast as sea, loss of weight and polyphagia .

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

"பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மேவந் தோன்றியே பொருந்திமெடுயில்"

- திருமூலர் நாடி [36]

When all that three nadi's runs in low volume, Diabetes develops.

MADHUMEGA GUNAM:

"இனிக்கின்ற வாதத் திடைசேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்
கனிக்கின்ற மேனி கரைந்து வெளுப்பேறும்
தனிக்கும் மதுமேகந் தப்பாது ஐயமே"

- திருமூலர் [37]

The coupling of the Vali and Iya naadi, causes increase in urination, and diabetes develops.

AVATHAIGAL – (COMPLICATIONS):

"காணவே முதலவத்தை சரீரந் தானும்
கனமாகப் பருத்திறுகி நீர்த்து வாரம்
வேணவே வேண்டாக்கி யகலம் பண்ணும்
மிக்க விரண்டாமவத்தை விளம்பக் கேளாய்
மூணவே மூத்திரப்பீ டையுமாச் சுக்ல
முகமழுகித் தேஜசுதான் மிகவே குன்றும்
நாணவே மூன்றாகு மவத்தைக் குத்தான்
நாவரளும் வாயுவது மீற்ய்ந் தானே

தானான நாலவத்தை யங்க தாகம்
சன்னியது பாதமுண்டா மைந்த வத்தைத்
தேனான நீர்பெருகுந் தாது நஷ்டம்
நிலையாறா மவத்தையுடற் கிடைகொள் ளாது
மூனான மூர்ச்சைவரு மேழ வத்தை
மிக்கவரோ சகஞ் சுவாசந்தேக சாட்டியம்
ஏனான எட்டாவ தவத்தை தானே
எழுகிரந்தி பிளவையுந்தான் மிகவுண் டாமே

உண்டாகு மொன்பதா மவத்தைக் கேளாய்
ஒழுக்கான அதிசாரங் கிருமி யுண்டாம்
பண்பான பத்தாந்தா னவத்தை கேளாய்
பாரமாம் சயங்கண்டு பரத்துக் கேகும்
வெண்டாகு மேகந்தா னிருப துக்கும்
விளங்கியதோர் தசவவத்தை விபரஞ் சொன்னோம்
அண்டாகுஞ் சாத்யவ சாத்யமி ரண்டும்
அறிந்து கொண்டு அடவாகவவிழ்தஞ் செய்யெ"

- யூகி வைத்திய சிந்தாமணி

1. Obesity and enlargement of urethral orifice.
   There is obstruction in urinary flow.
   In, Modern literature obesity is the diabetogenic factor and it will produce insulin resistance.
2. Polyuria resulting in gradual diminition of sukkila thathu. Impotence may occur.
   Dryness of the skin occur due to dehydration.
3. Intense thirst and dryness of mouth due to polyuria
   Flatulance and acute abdomen also common in diabetes due to diabetic keto acidosis.
4. Delirium (Janni) may supervenses after dehydration.
   It is one of the acute complication of diabetes mellitus in diabetic keto acidosis and non-ketotic hyperosmolar coma.
5. Polyuria and loss of sukkila thathu are seen.
   Impotence and retrograde ejaculation are common in diabetic patients due to autonomic nervous system involvement.
6. Restlessness is present.
   There is Kussmaul's air hunger with hissing type of respiration are seen.
7. Ageusia, dysphagia, exhaustion are seen.
8. Abscess and carbuncles are formed.
9. Diarrhoea nocturnal in type.
   Recurrent infection may occur.
10. Tuberculosis and acute infections may lead to death.

The above complications occur one by one in unidentified or improper treatment of Madhumegam.

Other complications of Madhumegam:

Meganeer Kattigal (Diabetic Carbuncle)

1. Madaku Katti
2. Ammaiodu Katti
3. Valai Kann Katti
4. Athomuga Katti
5. Paisura Katti
6. Kadalai Katti
7. Kadugu Katti
8. Thirathi Katti
9. Nilapoosani Katti
10. Megavithirathi Katti

PROGNOSIS (தீரும் தீராதவை):

"செய்யவே வச்சரமாந் தண்ட மான
செய்மான முதுகுதண்டைப் பற்றி நிற்கும்
பெய்யவே பெருநரம்பில் மேகந்தானும்
பிறக்கும்மென்றே தானறி ந்து வாதந்தன்னால்
பிய்யவே பிறந்தசலம் நால சாத்தியம்
பித்தத்திற் பிறந்தசல மாறும் யாப்யம்
பையவே சேட்டுமத்திற் பிறந்தசலம் பத்தும்
பரமனுரைத் தார் சாத்யம் பராப ரிக்கே"

- யூகி வைத்திய சிந்தமணி [38]

"வழியும் வாதம் நான்காமே மாறா தவிடிநதந் தன்னாலே
பொழியும் வாதம் நில்லாது போமே மருந்தை பொய்யெனவே"

- தேரையர் வாகடம் [39]

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.

ஏந்தெந்த ரோகங்களில் சிறுநீர் அதிகரித்தாலும் குறைந்தாலும் தீது ?

"வெப்பு பிணியதனில் வெம்மேகத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் – செப்பும்
கிராணியிற் பாண்டில் குளிர்நீர் சுருங்கிற்
பிராணன் பிரியுமெனப் பேசு"

- கண்ணுசாமியம் (40)

Excess of urination in Megaroham causes death.

"துதிப்பான மேகத்தில் நீரிழிவு மாகா
தோன்றிய நீரிழிவு தண்ணீர் வாதமுமாகா"

- சதக நாடி (41)

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

"மேகத்தில் நீரிழிவு மேவுமதில் வாதநோய்
வேக வயித்துள் வயிற்றுளைவு – சோக விக்கல் பன்னு விக்கல்
தன்னில் பகைளைப்புப் பாங்க தனிற்
பின்னளை யாகாது பேசு"

- நோய் நாடல் (42)

In Megaroham complications associated with carbuncle, Morbid thirst, excessive body heat, shock and sweat occurs and the prognosis will be bad.

"வேர்வைதனிற் கபமும் மேவுமதில் விக்கல் நோய்
கார்முகில் நேர் கூந்தலா ய்கண்டு மேல் – சீர்கொள்
மருத்துவத்திற்றேர்ந்த மதியுடையாராவி
தரித்திரா தென்பர்சரி"

- நோய் நாடல்

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

மேகம் இருபதுக்கும் பத்தியம் :

"மேக மிருபதுக்கும் பத்தி யந்தான்
பாங்காக 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 பீர்க் கங்காய்கரு வேப்பிலை கொத்தமல்லி
நேமேநெற் பொறி எள்ளு முசுமுசுக்கை யாகும்
நேரான நல்லெண்ணெய் புண்ணக்குளுந்து
தாமேக மிருபதுக்கும் பத்ய வர்க்கம்
சாற்றினார் சிவன்றானுந் தாய்க்கும் தானே"

- யூகி வைத்திய சிந்தாமணி (43)

The following foods are preferred for Madhumegam:

பொன்னாங்காணி, சிறுகீரை, கொத்தமல்லி, முசுமுசுக்கை, பீர்க்கு, கருவேப்பிலை, பாகல், அவரை, முருங்கை, வெந்தயம், சீரகம், எள், பசுவின்வெண்ணெய், எருமை மோர், சிறுபயறு, உளுந்து இவை மேகம் இருபதுக்கும் பத்தியம் ஆகும்.

MARUTHUVAM:

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.

"உற்றா னளவும் பிணியளவும் காலமுங்
கற்றான் கருதிச் செயல்"

- திருக்குறள்

A physician must have good knowledge about the patient, the disease, the duration of the disease and seasons in which symptoms get aggravate.

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 Kappu (Prevention), Neekam (Treatment) and Niraivu (Restoration).

KAPPU:

Prevention is better than cure

- 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.

NEEKAM:

For the disease Madhumegam, PERUNGAYA CHOORANAM - 2gm twice a day is given.

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 causes impairment of dasavayu which in turn affect the seven udal thathukkal.

LINE OF TREATMENT:

″பகர்பித்த விந்தையலாது மேகம் வராது″

- நோய் நாடல் (44)

So the treatment must be done to correct Azhal and then to correct the other two kutram Vali and Iyam.

PHYSICAL EXCERCISES :

″நண்பு பெற வுண்ட பின்பு குறுநடையுங் கொள்வோம்″

- பதார்த்த குண சிந்தாமணி (45)

The need for walking is emphasized by Theraiyar.

Exercise forms an important component along with drugs and diet management of Diabetes Mellitus – Type II.

Patients should be encouraged to take regular physical activity in form of brisk walking, gardening, swimming and cycling for 30 minutes daily to improve insulin sensitivity and maintain blood pressure.

YOGA FOR MADHUMEGAM:

YOGA:

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

- Yoga Aganas for Health & Vigour

Yoga is primarily the process of self-culture. Yoga according to Thirumandiram is the attainment of spiritual, psychological and physical perfection.

"உடம்பா லழியில் உயிராலழிவர்
திடம்பட மெய்ஞனம் சேரவும் மாட்டார்
உடம்பை வளர்க்கும் உபாயம் அறிந்தே
உடம்பை வளர்தேன் உயிர் வளர்தேனே"

- திருமூலர் [1]

As a body is said to be divine, Thirumoolar advised that each and every individual aspiring for self realisation should practice yoga. It is a science that helps to lead a pure and healthy life, the practice of yoga prevents the decay of tissues by increasing with abundant energy force.

"இயமம் நியமம் எண்ணிலா ஆதனம்
நயமுறு பிராணாயாமம் பிரத்தியாகாரம்
சயமுறு தாரணை தியானம் சமாதி
அயமுறு அட்டாங்க மாவது மாமே

- திருமூலர் [46]

Asanas are nothing but a kind of yoga. There are innumerable types asanaas described in Siddha text. Each and every yogasana is indicated for a definite effect in a particular region of the body by stimulating the internal organs to function in a normal way.

The following Asanaas will help to manage madhumegam:

1. Dhanurasanam
2. Paschimottanasanam
3. Pawanamukthasanam
4. Ardha Matsyendrasanam

1. Dhanurasanam

Dhanurasanam has been named after the shape the body takes while performing it that of a bow. Dhanu means bow.

Practice:

1. Lie on your stomach with your feet hip-width apart and your arms by the side of your body.
2. Fold your knees, take your hands backwards and hold your ankles.
3. Breathing in, lift your chest off the ground and pull your legs up and back.
4. Look straight ahead with a smile on your face.
5. Keep the pose stable while paying attention to your breath. Your body is now curved and taut as a bow.
6. Continue to take long deep breaths as you relax in this pose. But bend only as far as your body permits you to. Do not overdo the stretch.
7. After 15 -20 seconds, as you exhale, gently bring your legs and chest to the ground. Release the ankles and relax.

Benefits:

Helps regulate the pancreas and is recommended for people with diabetes.

2. Paschimottanasanam

"**Paschima**" means your "**back**" and "**Uttana**" means "**stretching**". This asan coversthe stretching of the whole body from head to heels so it is called as Paschimottanasnam.

Practice:

1. Sit down straight with your legs together by stretching in front of you. keep your head neck and spine erect.
2. Place the palms on your respective knees.
3. Now bend your head and trunk slowly forward to catch the toes with the thumb, index and middle fingers without bending knees.
4. Take a deep breath and exhale slowly. Try to touch your head to your both knees as shown in above image.
5. Bend the arm and try to touch the elbow on the floor.
6. Exhale completely and holding out your breath stay in this posture for a few seconds.
7. After few seconds slowly return to your starting position.
8. Breathe normally.
9. Repeat this for 3-4 times.

Benefits:

1. It acts as a stress reliever.
2. Reduces fatty deposits in the abdomen.
3. Remove anxiety, anger and irritability.
4. It induces the pancreas and helps in preventing diabetes.

3. Pawana muktasanam

Pawana muktasanam knows as Wind Removing Pose. It is beneficial to curen gas problems and poor digestion. Regular practice of Pawanamuktasanam helps to stimulate bowel movement which is very necessary for removing waste material.

Practice:

1. Lie flat on your back and keep the legs straight and relax breathe deeply and rhythmically.
2. Inhale slowly and lift the legs and bend in the knee. Bring upwards to the chest till your thigh touches to stomach
3. Hug your knees in place and lock your fingers.
4. Try to touch the knee with your nose tip. This is not easy in first time. But regular practice you can do this. Hold this position for 20 to 30 seconds. You can extend it till 1 minute as per your capability.
5. Now exhale slowly and come back to the original position that is Shavasana (Lie straight)
6. This is very beneficial for stomach abs. The results are very impressive.
7. Practice 3 to 5 cycles each day.

Benefits:

1. Cures acidity Indigestion and Constipation.
2. Very good for all abdominal organs.
3. Regular practice cures gastrointestinal problems.
4. It induces the pancreas and helps in preventing diabetes.

4. Ardha matsyendrasana

Ardha means Half, Matsyendra means King of fish.

Practice:

1. Sit with the legs straight and relax the whole body.
2. Place the sole of the right foot flat on the floor on the outside of the left knee.
3. Bend the left leg and lay the left heel beside the right buttock. Both buttocks remain on the floor. The back is upright and relaxed.
4. Bring the left arm to the outside of the right knee and grasp the right ankle.

5. Turn the upper body as far as possible to the right, place the right arm across the back and look over the right shoulder. Breathing normally remains for a few minutes in this position and relax the whole body.

6. Then slowly return to the starting position.

Benefits:

1. Promotes mobility of the spine and hips.
2. The twist aids release of tension from the deep layers of muscle in the back.
3. The breath is also deepened in this position.
4. Function of the kidneys and pancreas is stimulated and the ability to concentrate is improved.

MODERN ASPECT

MODERN ASPECT

DEFINITION:

Diabetes Mellitus is a group of metabolic disorder characterized by Hyperglycemia resulting from defects in Insulin secretion, Insulin action, or both. The chronic Hyperglycemia of Diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart and blood vessels.

Several pathogenic processes are involved in the development of Diabetes. These range from autoimmune destruction of the β-cells of the Pancreas with consequent Insulin deficiency to abnormalities that result in resistance to Insulin action. The basis of the abnormalities in Carbohydrate, Fat and Proteins metabolism in Diabetes is deficient action of Insulin on target tissues. Deficient Insulin action results from inadequate Insulin secretion or diminished tissue responses to Insulin at one or more points in the complex pathways of hormone action. Impairment of Insulin secretion and action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycemia [(47)].

EPIDEMIOLOGY:

The application of Epidemiology to the study of Diabetes Mellitus has provided valuable information on several aspects of this disease such as its natural history, prevalence, incidence, morbidity and mortality in diverse populations around the world. Identification of the cause of the disease and the possible preventive measures that could be instituted to arrest or delay the onset of this disease which has reached epidemic proportions in both the developed and the developing nations. Unfortunately, the improvement in outcomes for individual patients with Diabetes has not resulted in similar improvements from the public health perspective.

Diabetes is fast gaining the status of a potential Epidemic in India with more than 62 million Diabetic individuals currently diagnosed with the disease. The prevalence of Diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that, by 2030 Diabetes Mellitus may afflict up to 79.4 million individuals in India, while China (42.3 million) and the United States (30.3 million) will also see significant increase in

those affected by the disorder [48]. The WHO-ICMR national NCD risk surveillance study reported an overall frequency of self-reported diabetes of 4.5% with urban population scoring (7.3%), over the rural areas (3.1%) [49]

India currently faces an uncertain future in relation to the potential burden that Diabetes may impose upon the country. Many influences affect the prevalence of disease throughout a country, and identification of those factors is necessary to facilitate change when facing health challenges [50].

ETIOLOGY:

- Type I Diabetes occurs when your immune system, the body's system for fighting infection, attacks and destroys the Insulin-producing beta cells of the Pancreas. Type I Diabetes is caused by genes and environmental factors, such as viruses, that might trigger the disorder.
- Type II Diabetes is the most common form of Diabetes and it is caused by Several factors, including lifestyle factors and genes.
- Gestational Diabetes is a type of Diabetes that develops during pregnancy, and is caused by the hormonal changes of pregnancy along with genetic and lifestyle factors [51].

OTHER CAUSES:

GENETIC MUTATIONS:

Monogenic Diabetes is caused by mutations, or changes, in a single gene. These changes are usually passed through families, but sometimes the gene mutation happens on its own. Most of these gene mutations cause Diabetes by making the Pancreas less able to make Insulin. The most common types of monogenic Diabetes are Neonatal Diabetes and Maturity-Onset Diabetes of the Young (MODY). Neonatal Diabetes occurs in the first 6 months of life.

Cystic Fibrosis produces thick mucus that causes scarring in the Pancreas. This scarring can prevent the Pancreas from making enough Insulin.

Hemochromatosis causes the body to store too much iron. If the disease is not treated, iron can build up in and damage the Pancreas and other organs.

HORMONAL DISEASES:

Some hormonal diseases cause the body to produce too much of certain hormones, which sometimes cause Insulin resistance and Diabetes.

Cushing's syndrome occurs when the body produces too much Cortisol and often called the "Stress hormone." Acromegaly occurs when the body produces too much growth hormone. Hyperthyroidism occurs when the thyroid gland produces too much thyroid hormone.

DAMAGE OR REMOVAL OF THE PANCREAS:

Pancreatitis, pancreatic cancer and trauma can all harm the beta cells or make them less able to produce Insulin, resulting in Diabetes. If the damaged Pancreas is removed, Diabetes will occur due to the loss of the beta cells [(52)].

PATHOPHYSIOLOGY:

There is a direct link between Hyperglycaemia and physiological and behavioural responses. Whenever there is Hyperglycaemia, the brain recognizes it and sends a message through nerve impulses to Pancreas and other organs to decrease its effect.

TYPE I DIABETES MELLITUS:

Type I Diabetes is characterized by autoimmune destruction of Insulin producing cells in the Pancreas by CD4+ and CD8+ T cells and macrophages infiltrating the Islets. Several features characterize Type I Diabetes Mellitus as an autoimmune disease.

1. Presence of immuno-competent and accessory cells in infiltrated Pancreatic Islets.
2. Association of susceptibility to disease with the class II (immune response) genes of the major histocompatibility complex (MHC; Human Leucocyte Antigens HLA).
3. Presence of Islet cell specific autoantibodies.
4. Alterations of T cell mediated immune regulation, particularly in CD4+ T cell compartment.
5. The involvement of monokines and TH1 cells produce inginterleukins in the disease process.
6. Response to immunotherapy

7. Frequent occurrence of other organ specific auto- immune diseases in affected individuals or in their family members.

Approximately 85% of patients have circulating islet cell antibodies, and the majorities also have detectable anti-Insulin antibodies before receiving Insulin therapy. Most Islet cell antibodies are directed against Glutamic Acid Decarboxylase (GAD) within pancreatic β cells.

TYPE II DIABETESMELLITUS:

The two main pathological defects in Type II Diabetes are impaired Insulin secretion through a dysfunction of the pancreatic β-cell, and impaired Insulin action through Insulin resistance. In situations where resistance to Insulin predominates, the mass of β-cells undergoes a transformation capable of increasing the Insulin supply and compensating for the excessive and anomalous demand.

In absolute terms, the plasma Insulin concentration (both fasting and meal stimulated) usually is increased but although relative to the severity of Insulin resistance, the plasma Insulin concentration is insufficient to maintain normal Glucose Homeostasis. Keeping in mind the intimate relationship between the secretion of Insulin and the sensitivity of hormone action in the complicated control of Glucose Homeostasis, it is practically impossible to separate the contribution of each to the etiopathogenesis of Diabetes Mellitus type II.

Insulin resistance and Hyper Insulinemia eventually lead to Impaired Glucose Tolerance. Except for Maturity Onset Diabetes of the Young(MODY), the mode of inheritance for Type II Diabetes Mellitus is unclear. MODY, inherited as an autosomal dominant trait, may result from mutations in Glucokinase gene on chromosome 7p. MODY is defined as Hyperglycaemia diagnosed before the age of twenty-five years and treatable for over five years without Insulin in cases where Islet cell antibodies (ICA) are negative.

INSULIN RESISTANCE:

The primary events are believed to be an initial deficit in Insulin secretion and in many patients relative Insulin deficiency in association with peripheral Insulin resistance. Resistance to the action of Insulin will result in impaired Insulin mediated glucose uptake in the periphery (by muscle and fat), incomplete suppression of hepatic glucose output and impaired triglyceride uptake by fat. To overcome the Insulin resistance, Islet cells will increase the amount of Insulin secreted. Endogenous

glucose production is accelerated in patients with Type II Diabetes or Impaired Fasting Glucose. Because this increase occurs in the presence of Hyper Insulinemia, at least in the early and intermediate disease stages, hepatic Insulin resistance is the driving force of Hyperglycemia of Type II Diabetes [53].

THE PANCREAS:

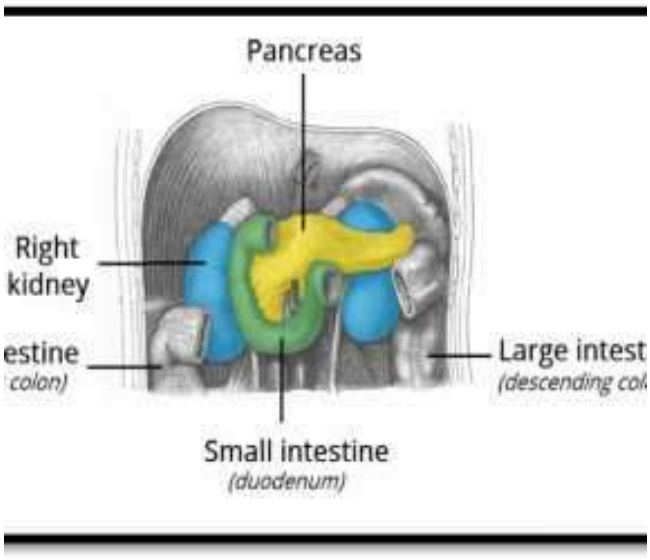
The Pancreas is an Abdominal Glandular organ, with a Digestive (Exocrine) and Hormonal (Endocrine) function.

ANATOMICAL POSITION:

The Pancreas is an oblong-shaped and flattened organ, about the size of a hand. Aside from the tail, it is a Retroperitoneal structure (lies behind the peritoneal cavity), located deep within the upper abdomen in the epigastrium and left hypochodrium regions.

Within the abdomen, the Pancreas is surrounded by other viscera and vessels.

- Stomach – lies Anteriorly and Superiorly.
- Duodenum – situated Anteriorly and Medially, curving around the head of the Pancreas.
- Spleen – located Posteriorly and Laterally. It is connected by ligaments to the tail of the Pancreas.
- Vasculature – the Aorta and Inferior vena cava pass Posteriorly to the head of the Pancreas.



ANTERIOR VIEW OF ABDOMEN:

ANATOMICAL STRUCTURE:

The Pancreas is typically divided into five parts:

1. **Head:**

This is the widest part of the Pancreas. It lies within the C-shaped curve created by the Duodenum, and is connected to it by connective tissue.

2. **Uncinate process**:

This is a projection arising from the lower part of the head and extending medially to lie beneath the body of the Pancreas. It lies posterior to the superior mesenteric vessels.

3**. Neck:**

It is located between the head and body of the Pancreas. It overlies the superior Mesenteric vessels which form a groove in its posterior aspect.

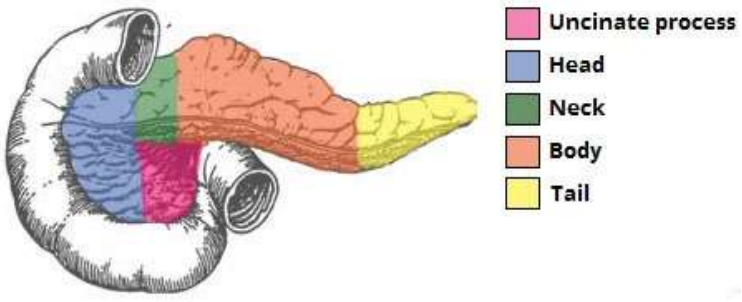
4. **Body:**

The body is centrally located, crossing the midline of the human body to lie behind the Stomach and to the left of the Superior Mesenteric vessels.

5. **Tail:**

The left end of the Pancreas that lies within close proximity to the hilum of the Spleen. It is contained within the Spleno -renal ligament with the Splenic vessels. This is the only part of the Pancreas that is intraperitoneal.

PARTS OF THE PANCREAS



THE DUCT SYSTEM:

The Exocrine compartment is classified as a serous gland. It is composed of approximately a million ‘Berry-like’ clusters of cells called Acini, connected by short intercalated ducts.

Intercalated duct cells beginning within Acini are called Centroacinar cells. The intercalated ducts drain into a network of intralobular collecting ducts, which in turn drain into the main Pancreatic duct.

The Pancreatic duct runs the length of the Pancreas and unites with the common bile duct, forming the Hepato-pancreatic Ampulla of Vater. This structure opens into the Duodenum.

Secretions into the Duodenum are controlled by a muscular valve, the sphincter of Oddi. It surrounds the Ampulla of Vater, acting as a valve.

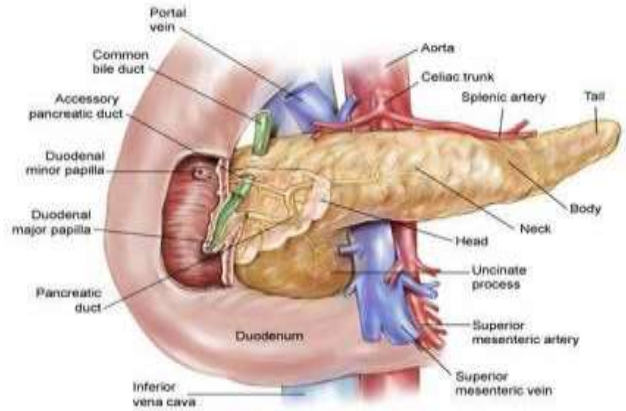
EXOCRINE PANCREAS SECRETING INTO THE DUODENUM:

VASCULATURE:

The Pancreas is supplied by the pancreatic branches of the Splenic artery. The head is additionally supplied by the superior and inferior Pancreatico-duodenal arteries which are branches of the Gastro-duodenal and superior Mesenteric arteries, respectively.

Venous drainage of the head of the Pancreas is into the superior Mesenteric branches of the Hepatic portal vein. The Pancreatic veins draining the rest of the Pancreas do so into the splenic vein.

THE ARTERIAL SUPPLY AND VENOUS DRAINAGE OF THE PANCREAS



LYMPHATICS:

The Pancreas is drained by lymphatic vessels that follow the arterial supply. They empty into the Pancreatico- Splenal nodes and the pyloric nodes, which in turn drain into the superior Mesenteric and Celiac lymph nodes.

CELLS AND SECRETIONS OF THE PANCREATIC ISLETS:

The Pancreatic Islets each contain four varieties of cells:

1. The alpha cell produces the hormone Glucagon and makes up approximately 20 percent of each Islet. Glucagon plays an important role in blood Glucose regulation; low blood glucose levels stimulate its release.

2. The beta cell produces the hormone Insulin and makes up approximately 75 percent of each Islet. Elevated blood Glucose levels stimulate the release of Insulin.

3. The delta cell accounts for four percent of the Islet cells and secretes the peptide hormone Somatostatin. It is also released by the hypothalamus (as GHIH). Pancreatic Somatostatin inhibits the release of both Glucagon and Insulin.

4. The Pancreatic polypeptide cell accounts for about one percent of Islet cells and secretes the pancreatic polypeptide hormone. It is thought to play a role in appetite, as well as in the regulation of Pancreatic Exocrine and Endocrine secretions. Pancreatic polypeptide released following a meal may reduce further food consumption; however, it is also released in response to fasting.

REGULATION OF BLOOD GLUCOSE LEVELS BY INSULIN AND GLUCAGON:

Glucose is required for cellular respiration and is the preferred fuel for all body cells. The body derives glucose from the breakdown of the carbohydrate containing foods and drinks. Glucose not immediately taken up by cells for fuel can be stored by the liver and muscles as Glycogen, or converted to Triglycerides and stored in the adipose tissue. Hormones regulate both the storage and the utilization of glucose as required. Receptors located in the Pancreas sense blood glucose levels, and subsequently the pancreatic cells secrete Glucagon or Insulin to maintain normal levels.

GLUCAGON:

Receptors in the Pancreas can sense the decline in blood glucose levels, such as during periods of fasting or during prolonged labour or exercise. In response, the alpha cells of the Pancreas secrete the hormone Glucagon, which has several effects:

It stimulates the liver to convert its stores of glycogen back into glucose. This response is known as Glycogenolysis. The glucose is then released into the circulation for use by body cells.

It stimulates the liver to take up amino acids from the blood and convert them into glucose. This response is known as Gluconeogenesis.

It stimulates Lipolysis, the breakdown of stored Triglycerides into free Fatty acids and Glycerol. Some of the free glycerol released into the bloodstream travels to the liver, which converts it into glucose. This is also a form of Gluconeogenesis.

These actions increase blood glucose levels. The activity of Glucagon is regulated through a Negative feedback mechanism; rising blood glucose levels inhibit further Glucagon production and secretion.

INSULIN:

The primary function of Insulin is to facilitate the uptake of glucose into body cells. Red blood cells, as well as cells of the brain, liver, kidneys, and the lining of the small intestine, do not have Insulin receptors on their cell membranes and do not require Insulin for glucose uptake. Although all other body cells do require Insulin if they are to take glucose from the bloodstream, skeletal muscle cells and adipose cells are the primary targets of Insulin.

The presence of food in the intestine triggers the release of gastrointestinal tract hormones such as glucose-dependent insulin tropic peptide (previously known as Gastric inhibitory peptide). This is in turn the initial trigger for Insulin production and secretion by the beta cells of the Pancreas. Once nutrient absorption occurs, the resulting surge in blood glucose levels further stimulates Insulin secretion.

Precisely how Insulin facilitates glucose uptake is not entirely clear. However, insulin appears to activate a Tyrosine kinase receptor, triggering the phosphorylation of many substrates within the cell. These multiple biochemical reactions converge to support the movement of intracellular vesicles containing facilitative glucose transporters to the cell membrane. In the absence of Insulin, these transport proteins are normally recycled slowly between the cell membrane and cell interior. Insulin triggers the rapid movement of a pool of glucose transporter vesicles to the cell membrane, where they fuse and expose the glucose transporters to the extracellular fluid. The transporters then move glucose by facilitated diffusion into the cell interior[(54)].

CLASSIFICATION

The new classification is primarily based on etiologies. The staging of pathophysiology by degree of deficiency of insulin is also adopted. The previous terms, insulin-dependent diabetes mellitus (IDDM) and non-insulin dependent diabetes mellitus (NIDDM), are abandoned. Instead, the terms type 1 and type 2 diabetes mellitus are used for etiological classification. The etiologic classifications of Diabetes Mellitus are listed below.

TYPE I DIABETES:

Type I Diabetes Mellitus (Juvenile Diabetes) is characterized by beta cell destruction caused by an autoimmune process, usually leading to absolute Insulin deficiency. Type I is usually characterized by the presence of anti–glutamic acid decarboxylase, Islet cell or Insulin antibodies which identify the autoimmune processes that lead to beta cell destruction.

IMMUNE-MEDIATED DIABETES:

This form of Diabetes, which accounts for only 5–10% of those with Diabetes, previously encompassed by the terms Insulin-Dependent Diabetes. Type I Diabetes or Juvenile-onset Diabetes, results from a cellular-mediated autoimmune destruction of the β-cells of the Pancreas.

In this form of Diabetes, the rate of β-cell destruction is quite variable, being rapid in some individuals (mainly infants and children) and slow in others (mainly adults). Some patients, particularly children and adolescents, may present with ketoacidosis as the first manifestation of the disease. Others have modest fasting hyperglycemia that can rapidly change to severe Hyperglycemia and/or ketoacidosis in the presence of infection or other stress.

IDIOPATHIC DIABETES:

Some forms of Type I Diabetes have no known etiologies. Some of these patients have permanent insulinopenia and are prone to ketoacidosis, but have no evidence of autoimmunity. Although only a minority of patients with Type I Diabetes fall into this category most are of African or Asian ancestry. Individuals with this form of Diabetes suffer from episodic Ketoacidosis and exhibit varying degrees of insulin deficiency between episodes.

TYPE II DIABETES MELLITUS:

This form of Diabetes, which accounts for approximately 90–95% of those with Diabetes, previously referred to as Non–Insulin-Dependent Diabetes, Type II Diabetes, or Adult-Onset Diabetes, encompasses individuals who have Insulin resistance and usually have relative (rather than absolute) insulin deficiency.

Most individuals with Type II Diabetes exhibit intra-abdominal (visceral) obesity, which is closely related to the presence of Insulin resistance. In addition, hypertension and dyslipidemia (high triglyceride and low HDL-cholesterol levels, postprandial hyperlipidemia) often are present in these individuals.

This is the most common form of Diabetes Mellitus and is highly associated with a family history of Diabetes, older age, obesity and lack of exercise. It is more common in women, especially women with a history of Gestational Diabetes, and in blacks, Hispanics and Native Americans.

GESTATIONAL DIABETES MELLITUS (GDM):

Gestational Diabetes Mellitus is an operational classification (rather than a pathophysiologic condition) identifying women who develop Diabetes Mellitus during gestation. Women who develop Type I Diabetes Mellitus during pregnancy and women with undiagnosed asymptomatic Type II Diabetes Mellitus that is discovered during pregnancy are classified with Gestational Diabetes Mellitus (GDM). In most women who develop GDM, the disorder has its onset in the third trimester of pregnancy.

OTHER SPECIFIC TYPES:

MONOGENIC DIABETES:

Types of Diabetes Mellitus of various known etiologies are grouped together to form the classification called “Other Specific Types”. This group includes persons with genetic defects of beta-cell function (this type of Diabetes was formerly called MODY or Maturity-onset Diabetes in youth) or with defects of insulin action. Persons with dysfunction associated with other endocrinopathies (e.g.Acromegaly), and persons with pancreatic dysfunction caused by drugs, chemicals or infections and they comprise less than 10% of DM cases [(55)].

RISK FACTORS:

The risk factors for Type I Diabetes are still being researched. However, having a family member with Type I Diabetes slightly increases the risk of developing the

disease. Environmental factors and exposure to some viral infections have also been linked to the risk of developing Type I Diabetes.

Several risk factors have been associated with Type II Diabetes and include:

- Family history of Diabetes
- Overweight
- Unhealthy diet
- Physical inactivity
- Increasing age
- High Blood pressure
- Ethnicity
- History of Gestational Diabetes
- Poor nutrition during pregnancy
- Impaired Glucose Tolerance (IGT) is a category of higher than normal blood glucose, but below the threshold for diagnosing Diabetes.
- Changes in diet and physical activity related to rapid development and urbanization has led to sharp increases in the numbers of people developing Diabetes.
- Pregnant women who are overweight, have been diagnosed with IGT, or have a family history of Diabetes are all at increased risk of developing Gestational Diabetes Mellitus (GDM). In addition, having been previously diagnosed with Gestational Diabetes or being of certain ethnic groups puts women a increased risk of developing GDM [(56)].

CLINICAL FEATURES:

Most of the symptoms are similar in both types of Diabetes but they vary in their degree and develop more rapidly in Type I Diabetes and more typical. Some of the clinical features and symptoms are listed below.

- Glycosuria:

  When blood glucose level is above 180mg/dl, glucose appears in urine. It is the renal threshold for glucose.

- Osmotic Diuresis:

  The excess glucose in renal tubules decreases re absorption of water result in diuresis. This leads to polyuria, polydipsia.

- 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, Dryness of mouth:

  Polyuria decreases water content in the body stimulates taste centre and in turn increases water intake.

- Polyphagia and Predilection for sweet food:

  This symptom is due to non utilization of sugar for energy expenditure.

- Asthenia:

  This is due to proteins depletion and increased utilization of proteins for energy.

- Emaciation:

  It is due to loss of water, glycogen and triglyceride and proteins stores and gradually reduced muscle mass occurs.

- Pruritis vulva , Balanitis, Genital Candidiasis, Skin Sepsis (Boils):

  This is due to irritant action of sugar on the tissue and fungal or bacterial infections.

- Constipation:

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

- Nausea, Headache, Blurring of Vision,
- Mood change, Irritability, Difficulty in concentrating, Apathy, Unhealed wounds.
- Frequent changes in Refractive error and Cataract [57].

COMPLICATIONS:

People with Diabetes have an increased risk of developing a number of serious health problems. Consistently high blood glucose levels can lead to serious diseases affecting the heart and blood vessels, eyes, kidneys, nerves and teeth. In addition, people with Diabetes also have a higher risk of developing infections. In almost all high-income countries, Diabetes is a leading cause of cardiovascular disease, blindness, kidney failure, and lower limb amputation. Maintaining blood glucose

levels, blood pressure, and cholesterol at or close to normal can help delay or prevent Diabetes complications. Therefore people with Diabetes need regular monitoring.

CARDIOVASCULAR DISEASE:

Affects the heart and blood vessels and may cause fatal complications such as Coronary Artery Disease (leading to heart attack) and Stroke. Cardiovascular Disease is the most common cause of death in people with Diabetes. High blood pressure, high cholesterol, high blood glucose and other risk factors contribute to increasing the risk of cardiovascular complications.

DIABETIC NEPHROPATHY:

This is caused by damage to small blood vessels in the kidney's leading to becoming less efficient or to fail altogether. Kidney disease is much more common in people with Diabetes than in those without Diabetes. Maintaining near normal levels of blood glucose and blood pressure can greatly reduce the risk of kidney disease.

DIABETIC NEUROPATHY:

Diabetes can cause damage to the nerves throughout the body when blood glucose and blood pressure are too high. This can lead to problems with digestion, erectile dysfunction, and many other functions. Among the most commonly affected areas are the extremities, in particular the feet. Nerve damage in these areas is called Peripheral Neuropathy, and can lead to pain, tingling, and loss of feeling. Loss of feeling is particularly important because it can allow injuries to go unnoticed, leading to serious infections and possible amputations. People with Diabetes carry a risk of amputation that may be more than 25 times greater than that of people without Diabetes. However, with comprehensive management, a large proportion of amputations related to Diabetes can be prevented. Even when amputation takes place, the remaining leg and the person's life can be saved by good follow-up care from a multidisciplinary foot team. People with Diabetes should regularly examine their feet.

DIABETIC RETINOPATHY:

Most people with Diabetes will develop some form of Eye disease (Retinopathy) causing reduced vision or blindness. Consistently high levels of blood glucose, together with high blood pressure and high cholesterol, are the main causes of Retinopathy. It can be managed through regular eye checks and keeping glucose and lipid levels at or close to normal.

FOOT DAMAGE:

Nerve damage in the feet or poor blood flow to the feet increases the risk of various foot complications. Left untreated, cuts and blisters can develop serious infections, which often heal poorly. These infections may ultimately require toe, foot or leg amputation.

SKIN CONDITIONS:

Diabetes may leave you more susceptible to skin problems, including bacterial and fungal infections.

HEARING IMPAIRMENT:

Hearing problems are more common in people with Diabetes.

ALZHEIMER'S DISEASE:

Type II Diabetes may increase the risk of Alzheimer's disease. The poorer your blood sugar control, the greater the risk appears.

COMPLICATIONS OF GESTATIONAL DIABETES:

Most women who have Gestational Diabetes deliver healthy babies. However, Untreated or Uncontrolled blood sugar levels can cause problems for mother and baby.

I. PREGNANCY COMPLICATIONS:

Women with any Type of Diabetes during pregnancy risk a number of complications if they do not carefully monitor and manage their condition. To prevent possible organ damage to the foetus, women with Type I Diabetes or Type II Diabetes should achieve target glucose levels before conception.

II. SUBSEQUENT GESTATIONAL DIABETES:

Once you've Gestational Diabetes in one pregnancy, you're more likely to have it again with the next pregnancy [58].

DIAGNOSIS:

Diabetes Mellitus refers to a condition in which circulating blood glucose is chronically elevated. In Anaemia, there are many possible causes for a high blood glucose. Equally, there can be wide variety of possible consequences, such that a physician specializing in Diabetes must have some familiarity with almost every system in the body. Investigation of Diabetes itself can be divided into the study of its causes, natural history, epidemiology, genetic basis, pathophysiological mechanism

and biochemical consequences, and each of its many complications requires investigating along similar lines. This entry provides a brief introduction to the way in which research findings have over the years been translated into the clinical investigation of Diabetes [59].

Blood:

1. Blood sugar estimation:

a. Blood sugar estimation is mandatory for confirming the diagnosis of Diabetes.

b. Both fasting and postprandial blood sugar levels are estimated.

2. Criteria for diagnosis of Diabetes Mellitus:

a. Random blood sugar>200mg/dl on two occasions,

b. Fasting Plasma Glucose>126mg/dl and sustained elevation of plasma glucose concentration>200mg/dl after an oral glucose load of 75gm at 2 hours.

3. Screening by Fasting Glucose test:

Fasting blood glucose determination is a screening test for Type II Diabetes Mellitus. It is recommended for all above age of 45 yrs and must be tested every 3 yrs and relatively earlier in overweight persons. A fasting plasma glucose value above 126mg/dl is certainly indicative of Diabetes Mellitus.

GLYCOSYLATED HAEMOGLOBIN (HbA1C):

Measurement of blood glucose level in Diabetics suffers from variation due to dietary intake of the previous day. Glycated Haemoglobin (HbA1C) provides an accurate and objective measure of glycaemic control over a period of weeks to months. In Diabetes the slow non-enzymatic covalent attachment of glucose to Haemoglobin (glycation) which takes place over 90-120 days that is lifespan of red blood cells. So it gives an estimate of Diabetic control for the preceding 3-4 months. There is an increase in the amount of the HbA1 relative to Non-Glycated Adult Haemoglobin.

Advantages of HbA1C:

1. No dietary preparation or fasting is required

2. Increased (HbA1C) value certainly means Diabetes Mellitus, but normal value does not rule out IGT

3. It is not used to diagnose Diabetes but it gives idea about poor control and development of micro vascular complications.

Normal value:

Below 5.7%

HbA1C diminished in

1. Anaemia
2. During pregnancy
3. Uraemia
4. Hemoglobinopathies
5. Blood transfusions.

C-PEPTIDE ASSAY:

C-peptide is released into circulation during conversion of pro-insulin to insulin which is more sensitive than insulin assay.

OTHER INVESTIGATIONS:

1. Lipid profile
2. Liver function test
3. Blood urea
4. Serum Creatinine

URINE:

Glucose:

a. Testing the urine for glucose with dipsticks is a common screening procedure

b. For detecting Diabetes.

c. Performed on urine passed 1-2 hrs after meal to maximize sensitivity.

d. Glycosuria warrants further assessment by blood testing.

Common cause for Glycosuria:

Low renal threshold which also occur in pregnancy, starvation, raised intracranial tension (cerebral tumors, hemorrhage and head injury) and alimentary glycosuria.

Renal Glycosuria:

Normal renal threshold for glucose is below 180 mg/dl but glucose still appears in urine due to low renal threshold. It is benign condition unrelated to Diabetes Mellitus and runs in families, also in pregnancy.

Alimentary Glycosuria:

A rapid and transitory rise in blood glucose level after meal above the normal renal threshold is called lag storage curve or alimentary glycosuria and returns to normal after 2 hrs.

Ketones:

Test for Ketone bodies is for assessing severity of Diabetes and not diagnosis of disease.

If Glycosuria and Ketonuria are present, diagnosis of diabetes is certain, Ketonuria also seen in fasting, strenuous exercise, diet rich in fat and low in carbohydrate.

Proteins:

Micro albuminuria or Proteinuria in absence of urinary tract infection is an indicator of development of Diabetic Nephropathy and other macro vascular complications [(60).]

MANAGEMENT:

The management of Type I and II Diabetes Mellitus (DM) requires addressing multiple goals, with the primary goal being glycemic control. Maintaining glycemic control in patients with Diabetes prevents many of the micro vascular and macro vascular complications associated with Diabetes. This chapter presents a review of the prevalence, screening, diagnosis, and management of these complications.

MICROVASCULAR:

Micro vascular complications of Diabetes are those long-term complications that affect small blood vessels. These typically include Retinopathy, Nephropathy and Neuropathy.

Diabetic Retinopathy is divided into two main categories:

- Non-proliferative Retinopathy
- Proliferative Retinopathy.

Non-proliferative Retinopathy is the development of Micro aneurysms, venous loops, Retinal Hemorrhages, hard exudates and soft exudates.

Proliferative Retinopathy is the presence of new blood vessels, with or without vitreous Hemorrhage. It is a progression of Non-Proliferative Retinopathy.

Diabetic Nephropathy is defined as persistent Proteinuria, which is characterized by

Progressive decline in renal function resulting in end-stage of renal disease.

Diabetic Neuropathy is a heterogeneous condition associated with nerve pathology. The condition is classified according to the nerves affected and includes focal, diffuse, sensory, motor, and autonomic Neuropathy.

MACROVASCULAR:

Macro vascular complications of Diabetes are primarily diseases of the coronary arteries, peripheral arteries, and cerebro vasculature. Early macro vascular disease is associated with atherosclerotic plaque in the vasculature supplying blood to the heart, brain, limbs, and other organs. Late stages of macro vascular disease involve complete obstruction of these vessels, which can increase the risks of Myocardial Infarction (MI), stroke, claudication and gangrene. Cardiovascular Disease (CVD) is the major cause of morbidity and mortality in patients with Diabetes.

The aim of treatment is to achieve normal blood glucose levels, to alleviate symptoms and to prevent complications.

The four pillars of Diabetic management are,

1. Diet
2. Exercise
3. Drugs –Oral hypoglycaemic agents and Insulin therapy by regular monitoring of glycaemic control.
4. Early detection and treatment of complications [61].

1. DIET:

It is the cornerstone of management of Diabetes. The objective is to have good glycaemic control and to provide a nutritious and balanced diet. In Type II Diabetes the calories need to be restricted in order to avoid obesity.

Total Caloric Intake:

It depends on body weight, degree of physical activity and presence of Co-morbid illness.

Body Mass Index (BMI):

It determines the total caloric requirement

BMI= Weight (in kg) / Height in m^2

BMI Normal Range**:**

21-25

The calories are derived from three principal sources like carbohydrates, proteins and fats.

Carbohydrates:

The amount of carbohydrate recommended in the diet is upto 50-60%. Whole grains, ragi, wheat, millets, oats, brown rice which have low glycaemic index are recommended.

Proteins:

Recommended amount is 12-20% of total calorie intake. Dhals or grams with outer skin and sprouts, lean meat, fish, egg white and chicken are preferred.

Fat:

It should be 20-24% of total intake. Sunflower oil, gingely oil, safflower oil, olive oil rich in Mono and Polyunsaturated fats are advised. Palm oil, coconut oil and vanaspathi should be avoided.

Salt:

Dietary salt should be less than 6g/day.

Milk and Milk Products:

Contribute to 40-45% of total fat content of vegetarian diet. Skimmed milk, unsweetened yogurt, curd, buttermilk are recommended.

Vegetables:

Fiber rich in greens, brinjal, cauliflower, gourds, grains, legumes, cereals and salads are advised.

PHYSICAL ACTIVITY:

Exercise forms an important component along with drugs and diet management in Type II Diabetes Mellitus. Patients should be encouraged to take regular physical activity in form of brisk walking, jogging, swimming, gardening and cycling for 30 minutes daily. This improves peripheral utilization of glucose, insulin sensitivity, prevents complications of Diabetes and assist in maintaining lipid profile and blood pressure, improves and muscle strength and beneficial for mental state of the individual [(62).]

TRIAL MEDICINE

PREPARATION OF TRIAL MEDICINE

Medicine Name - *Perungaya Chooranam*

Ingredients:

Ferula asafetida	- 4.2gm
Rock salt	- 8.4gm
Cuminum cyminum *Acorus calamus* *Zingiber officinale* *Terminalia chebu* *Plumbago zeylanica* *Costus speciosus*	- 12.6gm

Procedure:

The above ingredients are made into fine powder separately and sieved using cloth.

Dosage : 2 gm/ Bd

Adjuvant : Lukewarm water

Duration : 90 Days

Text Reference: Sarabenthira Vaithiya Rathna Vali[63]

LITERATURE REVIEW OF TRIAL MEDICINE

PERUNGAYA CHOORANAM

1. PERUNGAYAM

Botanical name - *Ferula asafoetida*

Family - Apiaceae

Nature (Suvai) - Kaippu, karakarappu

Thanmai - Veppam

Pirivu - Kaarppu

Parts used - Gum resin

Phyto chemicals : ferulic acid

umbel – liferone

asaresinotannols
2- butyl propenyl disulfide
Glucose
Galactose
L - arabinose
Glucuronic acid
Valeric acid [64]

Action:

Carminative
Stimulant
Laxative
Expectorant

General characters:

"தந்தவே தந்த மூலதெழும்பிணி
சருவகாளம் விருச்சிங்கீடம்மா
மந்தம்வாதம் உதாவர்த்தம் அல்குல்நோய்
மார்ப்பணங்கட்ட குன்மம்ம கோதரம்"

-தேரன் குணவாகடம் [65]

2. SEERAGAM

Botanical name - *Cuminum cyminum*
Family - Apiaceae
Nature (Suvai) - Kaarppu, Inippu
Thanmai - Thatppam
Pirivu - Inippu
Parts used - Fruit

Phyto chemicals :

α – Pinene
Limonene
Linalyl acetate
α - terpineole
p – menthstriene
2 – allylphenol

Benzaldehyde p – isopropyl

Cuminaldehyde [66]

Action :

Carminative

Astringent

Stomachic

Astringent

General characters :

"பித்தமெனும் மந்திரியைப் பின்னப் படுத்தியவன்

சத்துருவை யுந்துறந்து சாதித்து – மத்தனெனும்

ராசனையு மீவென்று நண்பைப் பலப்படுத்தி

போசனகு டாரிசெயும் போர்"

-தேரன் வெண்பா [67]

3. **VASAMBU**

Botanical name - *Acorus calamus*

Family - Araceae

Nature (Suvai) - Kaarppu

Thanmai - Veppam

Pirivu - Kaarppu

Parts used - Rhizome

Phyto chemicals :

β-asarone

sesquiterpenes

choline

flavones

acoradin

2,4,5-tri-MeObenzaldehyde

2,5-di-MeO-benzoquinone

Galangin

Calameone

acolamone

isoacolamone [68]

Action : Stimulant

Stomachic

Anti -periodic

Carminative

Emetic

General Characters:

"பாம்பாதி நஞ்சற் புதப்புண் வலிவிடபாகங் குன்மம்

சூம்பாரி ரத்தபித் தம்முக நாற்றம்வன் சூலைசன்னி

வீம்பாம்பை காசம் பீலிகஞ் சிலிபதம் வீறிருமல்

தாம்பாங் கிருமி யிவையேகு மாசிவ சம்பினைய".

-தேரன் குணவாகடம் [69]

4. CHUKKU :

Botanical name - *Zingiber officinale*

Family - Zingiberaceae

Nature (Suvai) - Kaippu, kaarppu

Thanmai - Veppam

Pirivu - Kaarppu

Parts used - Underground dried stem

Phyto chemicals : Gingerol

shogaols

dihydrogingerol

gigerdione

hexahydrocurcumin

desmethyl hexahydrocurcumin

α-zingiberene

β-sesquiphellandrene [70]

Action :

Stomachic

Carminative

Anti Vatha

General characters:

"சூலை மந்தம் நெஞ்செரிப்பு தோடமேப்பம்மழலைமூலம்

இரைப் பிருமல் மூக்குநீர் வாலக

தோடமதி சாரந் தொடர்வாத - குன்மநீர்த்

தோடம் ஆமம்போக்குஞ் சுக்கு"

- அகத்தியர் குணவாகடம் [71]

5. KADUKKAI:

Botanical name - *Terminalia chebula*

Family - Combretaceae

Nature (Suvai) - Thuvarppu

Thanmai - Veppam

Pirivu - Inippu

Parts used - Fruit

Phyto chemicals - Gallic acid
Chebulagic acid
punicalagin
chebulanin
corilagin
ellagic acid
chebulinic acid
palmitic acid
oleic acid
linoleic acid
1,6-di-o-galloyl-D- glucose
Anthraquinones [72]

Action :

Stomachic
Digestive
Laxative
Antioxidant

General characters:

″தாடை கழுத்தக்கி தாலு குறியிவிடப்

பீடை சிலிபதமுற் பேதிமுடம் – ஆடையெட்டாத்

தூலமிடி புண்வாத சோணிகா மாலையிரண்

டாலமிடி போம்வரிக்கா யால்″ (73)

6. CHITHIRA MOOLA VER PATTAI

Botanical name - *Plumbago zeylanica*

Family - Plumbaginaceae

Nature (Suvai) - Kaarppu, Viruviruppu

Thanmai - Veppam

Pirivu - Kaarppu

Parts used - Root bark

Phyto chemicals - Plumbagin
Citranone
Elliptone
ß-sitosterol-glucoside
bakuchiol
phenols
isoaffinetin
saponaretin
flavanoids
psorealen
iso-orientin (74)

Action : Antiperiodic
Diaphoretic

General characters:

″கட்டிவிர ணங்கிரந்தி கால்கள் அரையாப்புக்

கட்டிச்சூ லைவீக்க்கங் காழ்மூலம்-முட்டிரத்தக்

கட்டுநீ ரேற்றங் கனத்த பெருவயிறும்

அட்டும் கொடிவேலி யாம்″

- அகத்தியர் குணவாகடம் (75)

7. KOSHTAM

Botanical name - *Costus speciosus*

Family - Costaceae

Nature (Suvai) - Kaippu

Thanmai - Veppam

Pirivu - Kaarppu

Parts used - Rhizome

Phyto chemicals :

Tigogenin
diosgenin
a -amyrin stearate
b-amyrin
lupeol

Action :

Stomachic
Stimulant
Tonic
Expectorant
Diaphoretic [76]

General characters :

"நாட்டிலுறு வெட்டை நடுக்கம் எனுநோய்கள்
கொட்டமெனச் சொன்னால் குலையுங்காண் -கூட்டிற்
சுரதோடந் தொண்டைநோய் தோலாத பித்தம்
பரதேசம் போமே பறந்து."

-அகத்தியர் குணவாகடம் [77]

8. INDUPPU

Chemical name : Impure Sodium chloride

Actions :

Stomachic
Laxative
Purgative
Carminative [78]

General properties:

"அட்டகுன்மம் மந்தம் அசிர்க்கரஞ்சூர் சீதபித்தத்

துட்டவையும் நாடிப்புண் டோடங்கன் – கெட்டமலக்

கட்டுவிட வித்தையக் காமியநோய் வங்கரப்பான்

விட்டுவிட விந்துப்பை வின்" [79]

PERUNGAYA CHOORANAM- INGREDIENTS

Perungayam

Kadukkai

Induppu

Seeragam

Chukku

Chithira moola ver pattai

Vasambu

Koshtom

PERUNGAYA CHOORANAM

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY DESIGN

The clinical trial on Madhumegam (Diabetes Mellitus – Type II) was decided to conduct as an open label study.

STUDY CENTER:

The entire study was conducted on patients at Out Patient Department of Govt Siddha Medical College, Chennai in the premises of Arignar Anna Government Hospital for Indian Medicine and Homeopathy, Arumbakkam, Chennai-106, during the period of 2016-2018.

DATA COLLECTION:

Literary evidence from various,

- Siddha books
- Modern book
- Medical journal
- Internet

POPULATION:

The Population consists of Diabetes accompanied by Polyuria, Polyphagia, Polydypsia, generalized tiredness, Fatigue, Itching all over the body and satisfying the inclusion and exclusion criteria mentioned below.

SAMPLE SIZE:

40 patients.

INCLUSION CRITERIA:

- Newly identified Type II Diabetic cases only.
- Subject within 30-60 years.
- Blood Glucose (F) – 126mg/dl to 140 mg/dl
- Blood Glucose (pp) - 180mg/dl to 280 mg/dl

- HbA1C - 6.5% to 8%
- Polyuria
- Polyphagia
- Polydypsia
- Nocturia
- Fatigue

EXCLUSION CRITERIA (BASED ON CLINICAL HISTORY):

- H/O Insulin Dependent Diabetes Mellitus (IDDM).
- H/O Cardiovascular Disease.
- H/O Diabetic Nephropathy.
- H/O Diabetic Retinopathy.
- Pregnant women, lactating mothers, T.B affected individuals.

DURATION OF TREATMENT:

- 90 days.

Patients were followed under the guidance and supervision of the HOD, Professor, Reader, Lecturer and Assistant Lecturer of Maruthuvam, PG Department, GSMC, Chennai-106.

The patients were carefully studied for their history, clinical examinations, investigations and management.

EVALUATION OF CLINICAL PARAMETERS:

The history includes past, personal, family, occupation, dietary habits and associated history.

CLINICAL INVESTIGATION:

Blood:

- Blood sugar (Fasting, Post Prandial)
- Glycaemic control: HbA1C
- Blood urea
- Serum creatinine

- Serum cholesterol
- BMI (Body Mass Index)
- Lipid profile
- C – Peptide assay

Urine:

- Urine Sugar (Fasting, Postprandial)

SIDDHA ASSESSMENT:

- Envagaithervugal
- Neerkuri
- Neikkuri

A case sheet format was prepared based on the Siddha methodology like Envagaithervugal, Mukkutram, Nilam, Kaalam, Udalthathukkal including Neerkuri and Neikuri. Individual case sheet was maintained for each patient at Outpatient Department.

RESULTS AND OBSERVATION

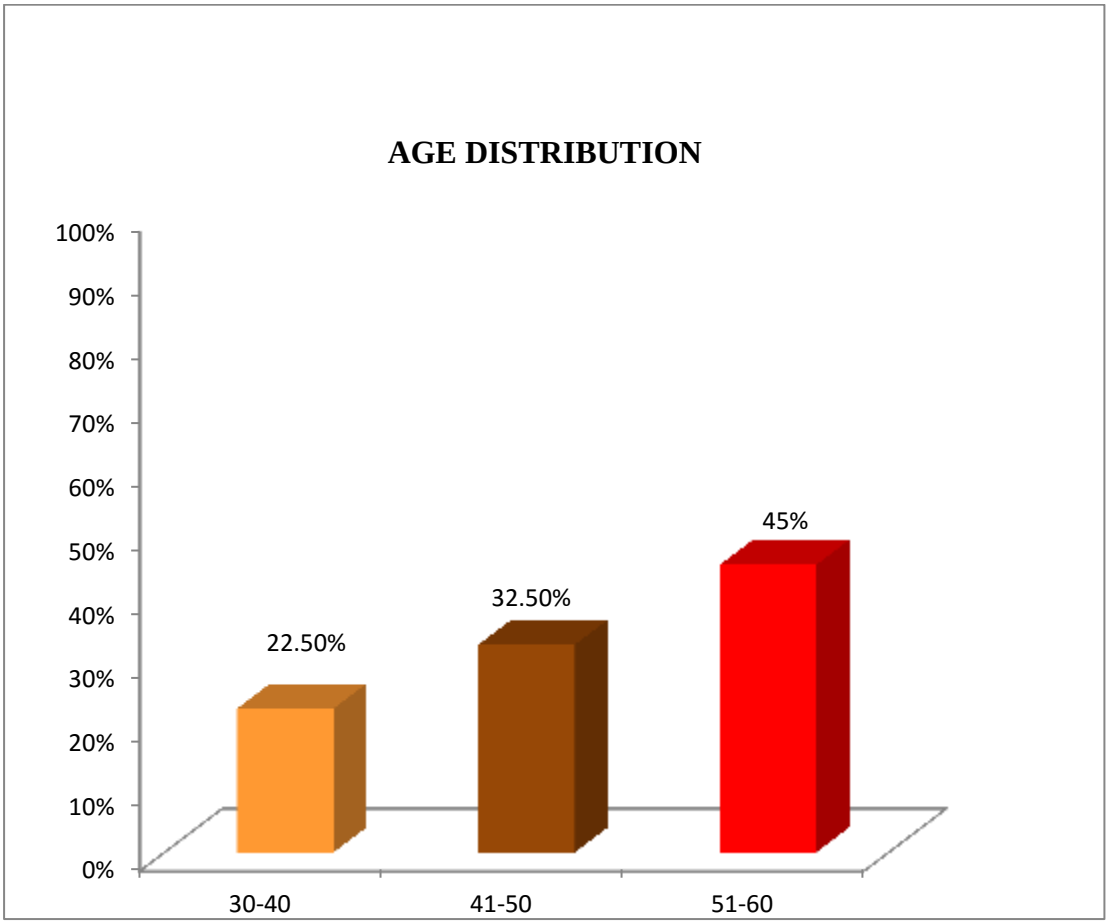
RESULTS AND OBSERVATION

The study on Madhumegam was carried out with 40 patients in the Out Patient Department PG Maruthuvam, Govt Siddha Medical College attached to Arignar Anna Govt Hospital of Indian Medicine, Chennai-106, during the period 2016-2018 were analyzed. The observation were made and tabulated with following criteria.

1. Age Distribution
2. Sex Distribution
3. Occupational Status
4. Socio economic Status
5. Dietary Habits
6. Family History
7. Kaalam
8. Paruva Kaalam
9. Thinai
10. Duration of illness
11. Mukkutram - Vali, Azhal, Iyam
12. Ezhu Udalthathukkal
13. Ennvagai Thervugal
14. Naadi
15. Neikuri
16. Clinical features
17. Clinical Prognosis
18. Urine sugar - Fasting, PostPrandial
19. Blood Sugar - Fasting, PostPrandial
20. HbA1C Level
21. Grading of Results

1. AGE DISTRIBUTION :

AGE	NO. OF CASES	PERCENTAGE
30-40	9	22.5%
41-50	13	32.5%
51-60	18	45%

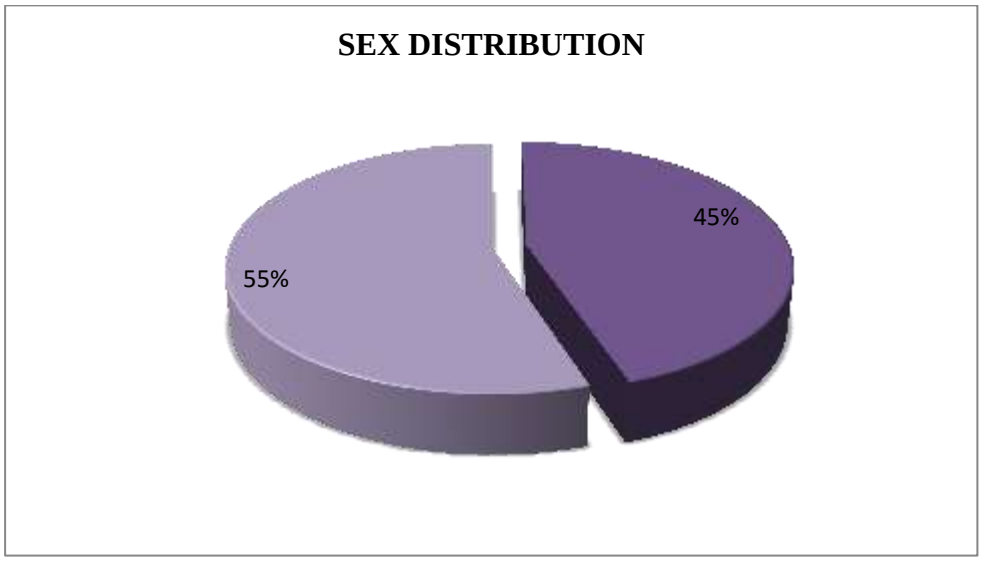


Inference:

From selected 40 cases, 9 patients (22.5%) were between 30 – 40 years, 13 patients (32.5%) were between 41 – 50 years and 18 patients (45%) were between 51 – 60 years old.

2. SEX DISTRIBUTION:

SEX	NO.OF CASES	PERCENTAGE
MALE	18	45%
FEMALE	22	55%

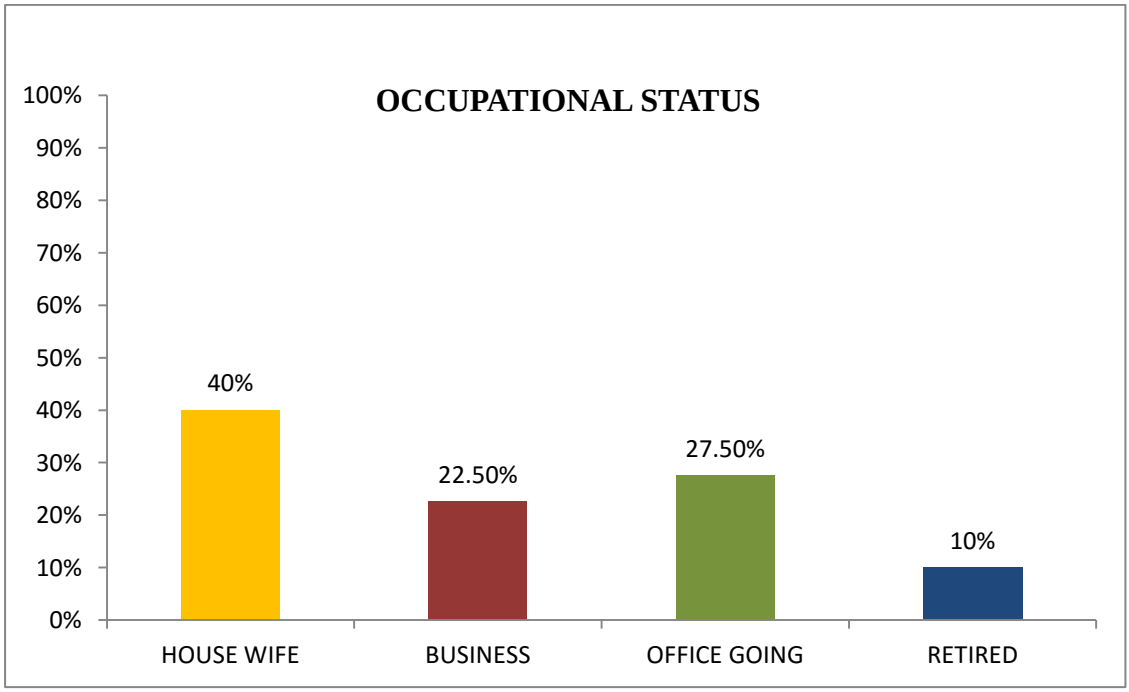


Inference:

Out of 40 patients, 18 cases (45%) were male and 22 cases (55%) were female.

3. OCCUPATIONAL STATUS

OCCUPATION	NO. OF CASES	PERCENTAGE
HOUSE WIFE	16	40%
BUSINESS	9	22.5%
OFFICE GOING	11	27.5%
RETIRED	4	10%

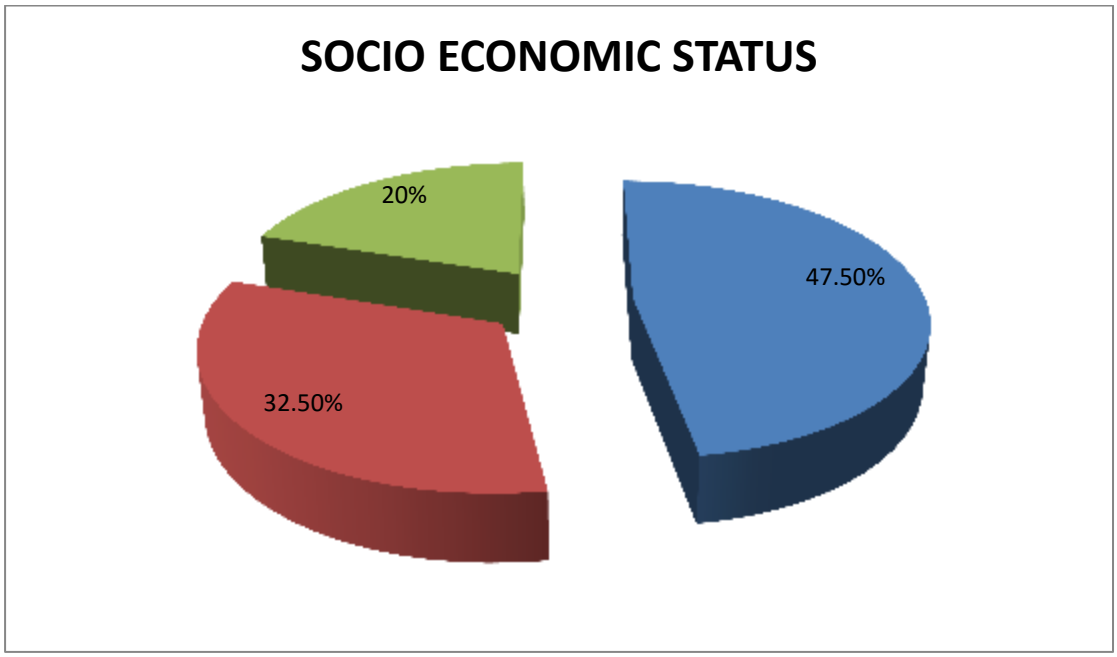


Inference:

From selected 40 cases, 16 patients (40%) were housewives, 9 patients (22.5%) are doing business, 11 patients (27.5%) are office goers and 4 (10%) are retired.

4. **SOCIO ECONOMIC STATUS**:

SOCIO ECONOMIC STATUS	NO. OF CASES	PERCENTAGE
LOW INCOME (Below 2 lakhs)	19	47.5%
MIDDLE INCOME (Up to 2 lakhs)	13	32.5%
HIGH INCOME (More than 5 lakhs)	8	20%

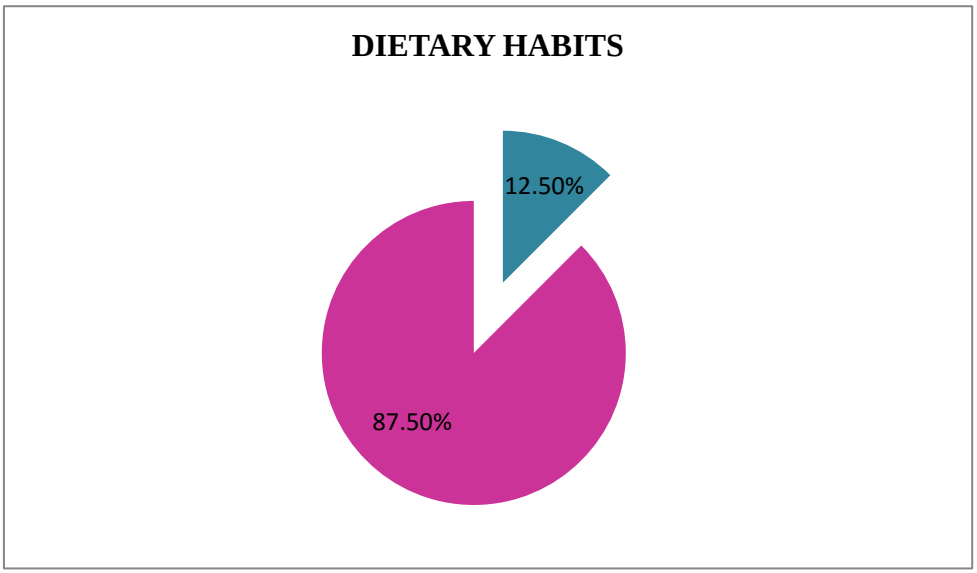


Inference:

Regarding Socio Economic Status 10 Patients (25%) were from lower income group, 24 patients (60%) were from Middle income group and 6 Patients (15) were from High income group.

5. DIETARY HABITS:

DIETARY HABITS	NO. OF CASES	PERCENTAGE
VEGETARIAN	5	12.5%
MIXED	35	87.5%

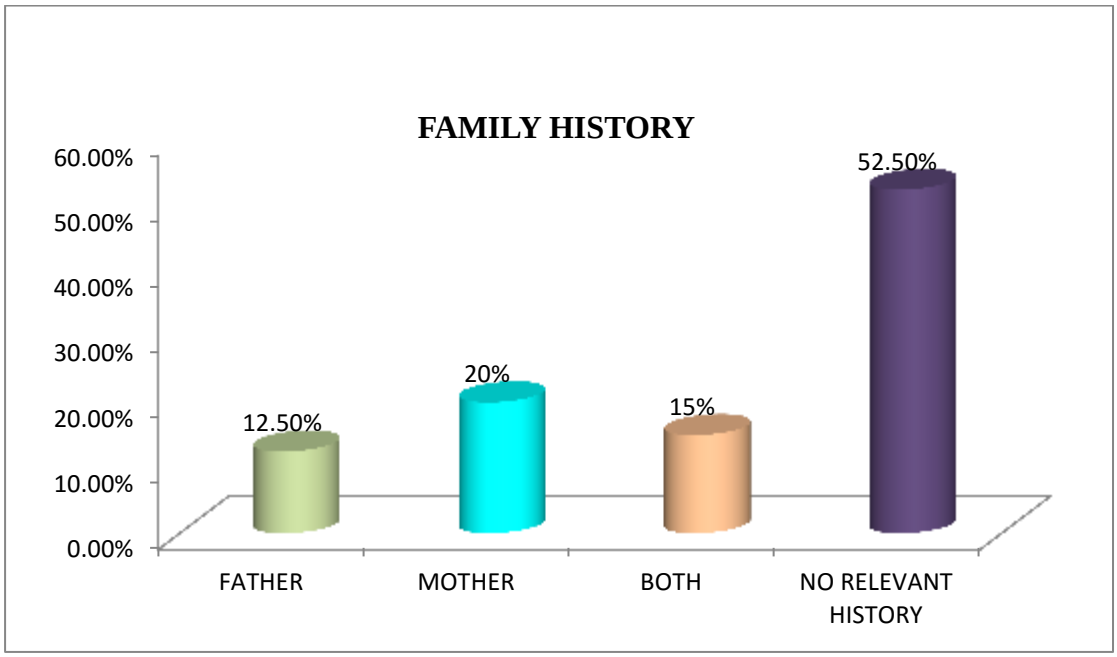


Inference:

Regarding Diet, out of 40 patients, 5 patients (12.5%) were taking Vegetarian diet and 35 patients (87.5%) were taking mixed diet.

6. FAMILY HISTORY:

FAMILY HISTORY	NO. OF CASES	PERCENTAGE
FATHER	5	12.5%
MOTHER	8	20%
BOTH	6	15%
NO RELEVANT HISTORY	21	52.5%

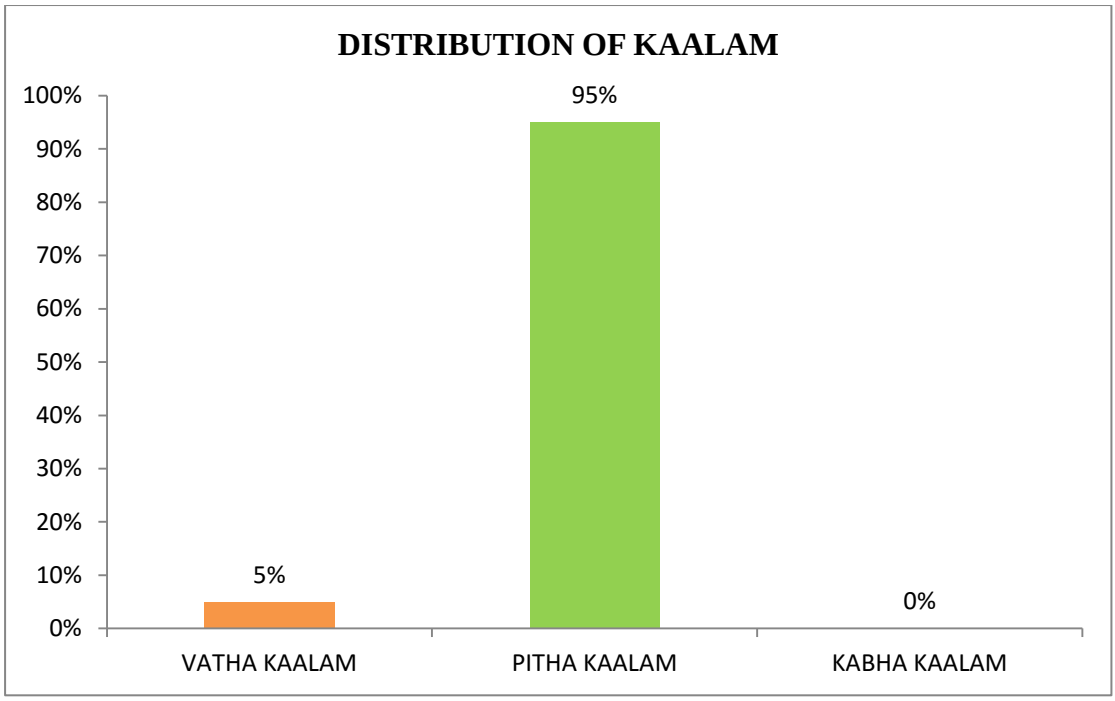


Inference:

Regarding family history 5 patient (12.5%) fathers had diabetic, 8 patient (20%) mothers had diabetic, 6 patient (20%) parents both had diabetic and 21 patients (62.5%) had no relevant family history.

7. DISTRIBUTION OF KAALAM:

KAALAM	NO. OF CASES	PERCENTAGE
VAATHA KAALAM (0 – 33)	2	5%
PITHA KAALAM (34 – 66)	38	95%
KABHA KAALAM (67- 100)	NIL	0%

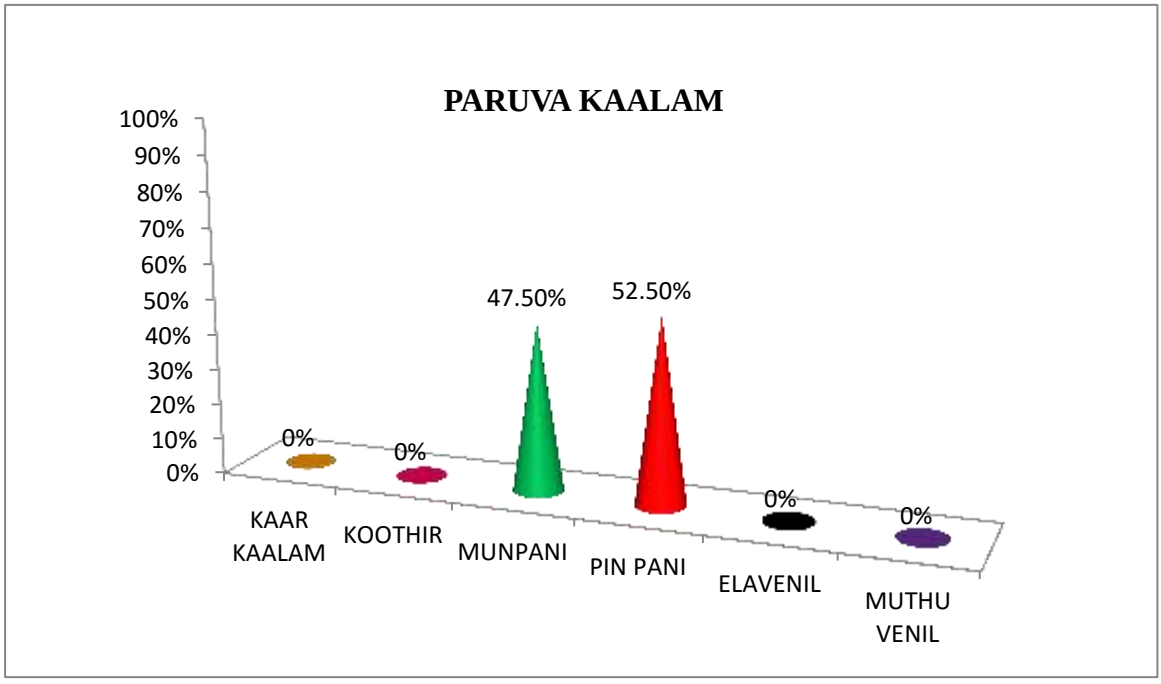


Inference:

Out of 40 patients, 2 patients (5%) were comes under Vaatha Kaalam, 38 patients (95%) were comes under Pitha Kaalam and no patients were under Kabha Kaalam.

8. PARUVA KAALAM:

PARUVA KAALAM (SEASONS)	MONTH	NO.OF CASES	PERCENTAGE
KAAR KAALAM	Aavani, Purattasi (Mid Aug- Mid Oct)	NIL	0%
KOOTHIR KAALAM	Iyppasi, Karthigai (Mid Oct – Mid Dec)	NIL	0%
MUN PANI KAALAM	Margazhi, Thai (Mid Dec- Mid Feb)	19	47.5%
PIN PANI KAALAM	Maasi, Panguni (Mid Feb – Mid Apr)	21	52.5%
ELAVENIL KAALAM	Chithirai, Vaigasi (Mid Apr – Mid June)	NIL	0%
MUTHU VENIL KAALAM	Aani,Aadi (Mid June- Mid Aug)	NIL	0%

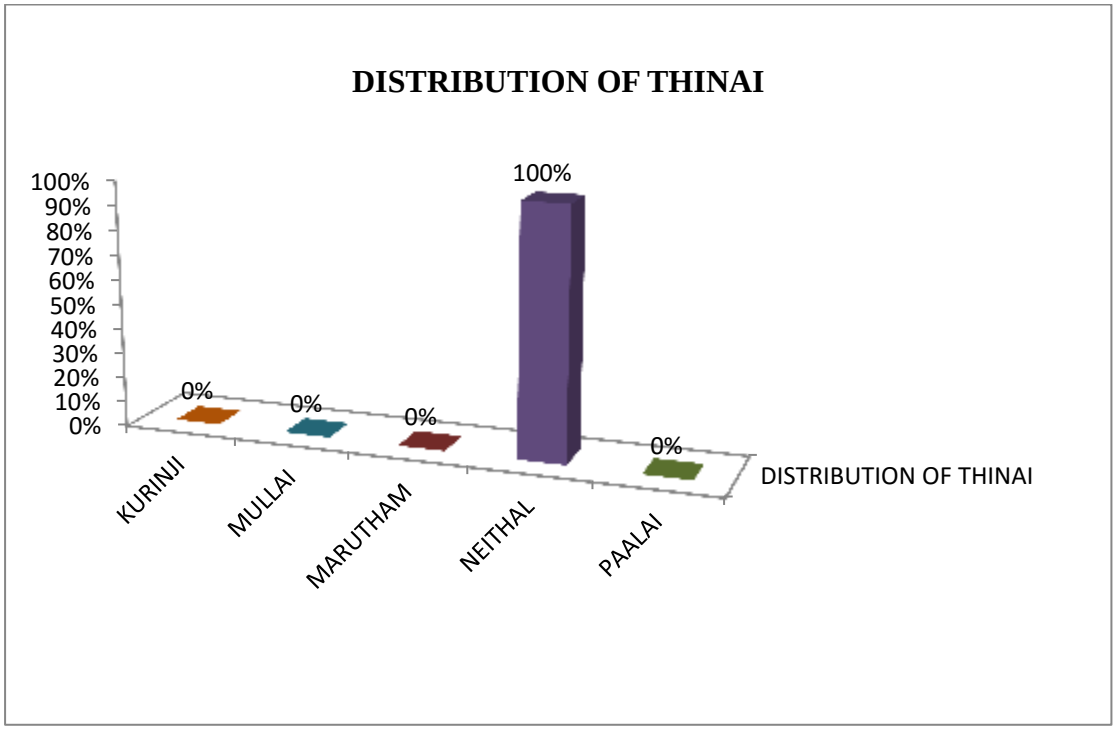


Inference:

From selected 40 patients, 19 patients (47.5%) were noted in Mun Pani Kaalam, 21 patients (52.5%) were noted in Pin Pani Kaalam.

9. DISTRIBUTION OF THINAI:

THINAI	NO. OF CASES	PERCENTAGE
KURINJI	0	0%
MULLAI	0	0%
MARUTHAM	0	0%
NEITHAL	40	100%
PAALAI	0	0%

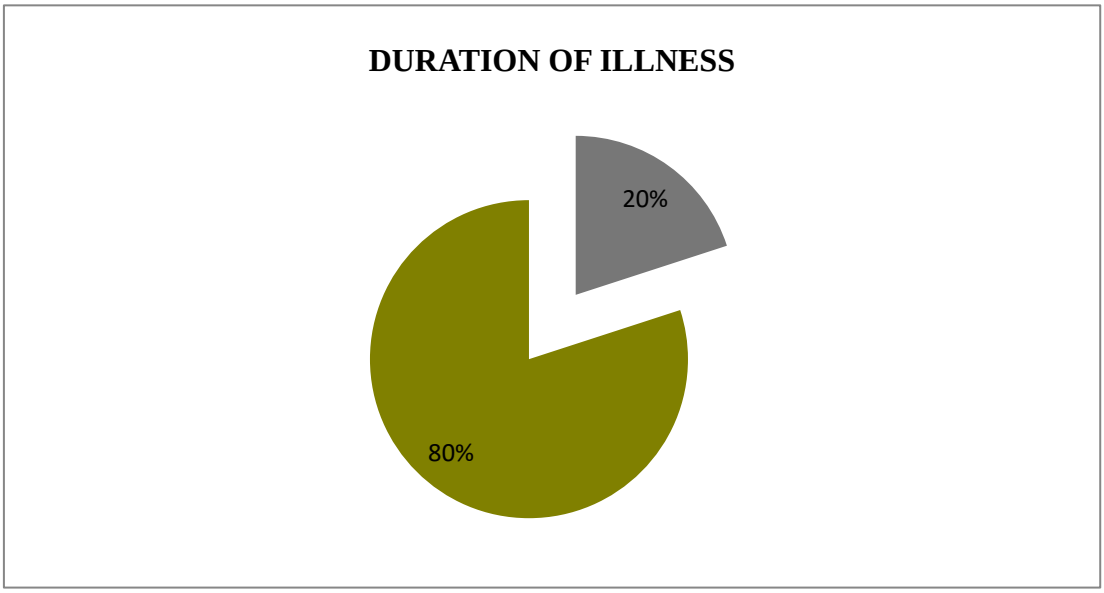


Inference:

All the 40 patients were from Neithal Nilam.

10. **DURATION OF ILLNESS:**

DURATION OF ILLNESS	NO. OF CASES	PERCENTAGE
NEWLY IDENTIFIED PATIENTS	8	20%
3 – 6 MONTHS	32	80%



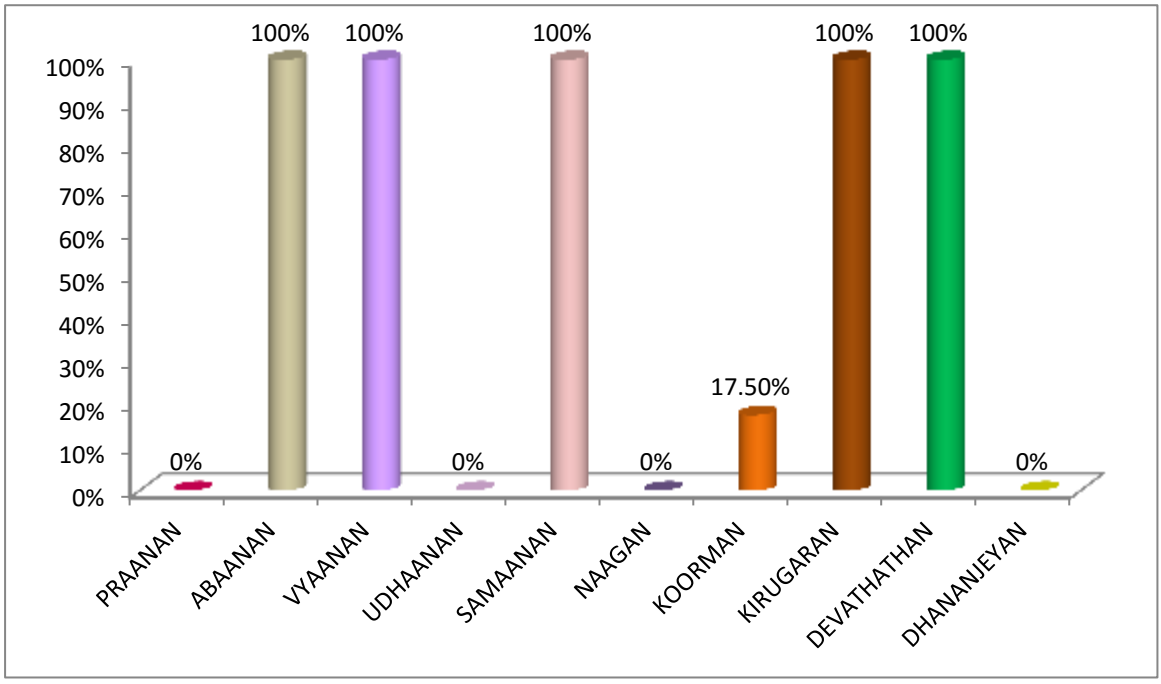
Inference:

Out of 40 patients, 8 patients (20%) were comes under newly identified category and 32 patients (80%) were comes under to 3 – 6 months category.

11. REFERENCE OF MUKKUTTRAM:

A. Affected Vali:

CLASSIFICATION OF VALI	NO. OF CASES	PERCENTAGE
PRAANAN	0	0%
ABAANAN	40	100%
VYAANAN	40	100%
UDHAANAN	0	0%
SAMAANAN	40	100%
NAAGAN	0	0%
KOORMAN	7	17.5%
KIRUGARAN	40	100%
DEVATHATHAN	40	100%
DHANANJEYAN	-	-

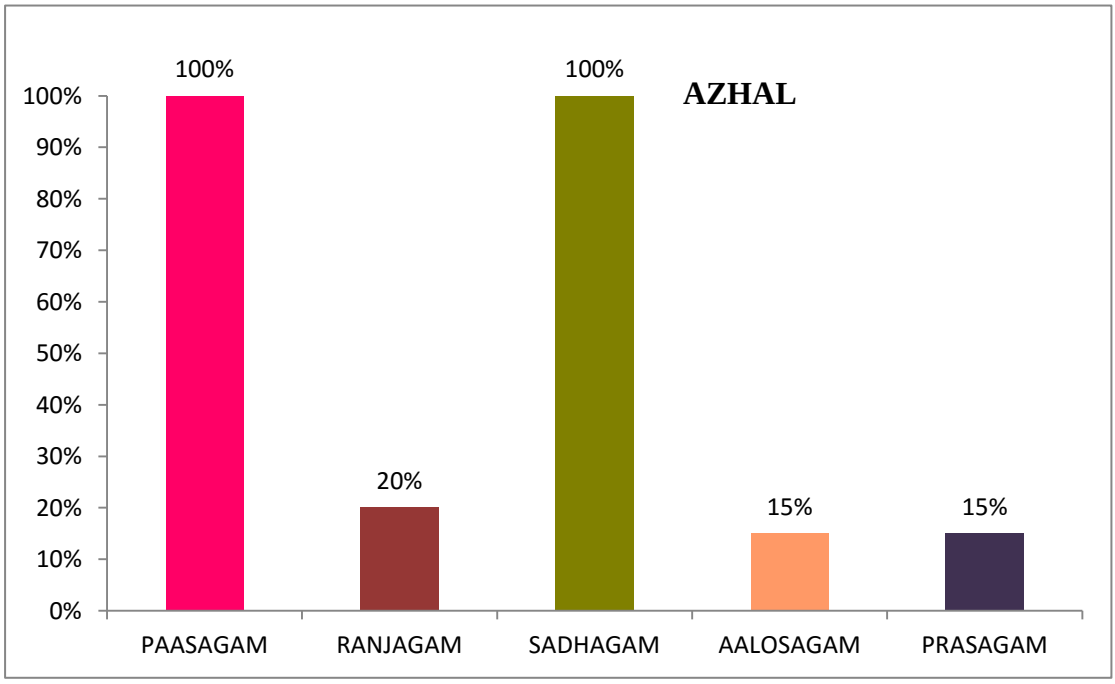


Inference:

From the selected 40 patients, all the 40 patients were affected with Abaanan, Vyaanan, Samaanan, Kirugaran and Devathathan. Koorman was affected in 7 patients (17.5%).

B. Affected Azhal:

CLASSIFICATION OF AZHAL	NO. OF CASES	PERCENTAGE
PAASAGAM (ANAL PITHAM)	40	100%
RANJAGAM	8	20%
SADHAGAM	40	100%
AALOSAGAM	7	17.5%
PRASAGAM	6	15%

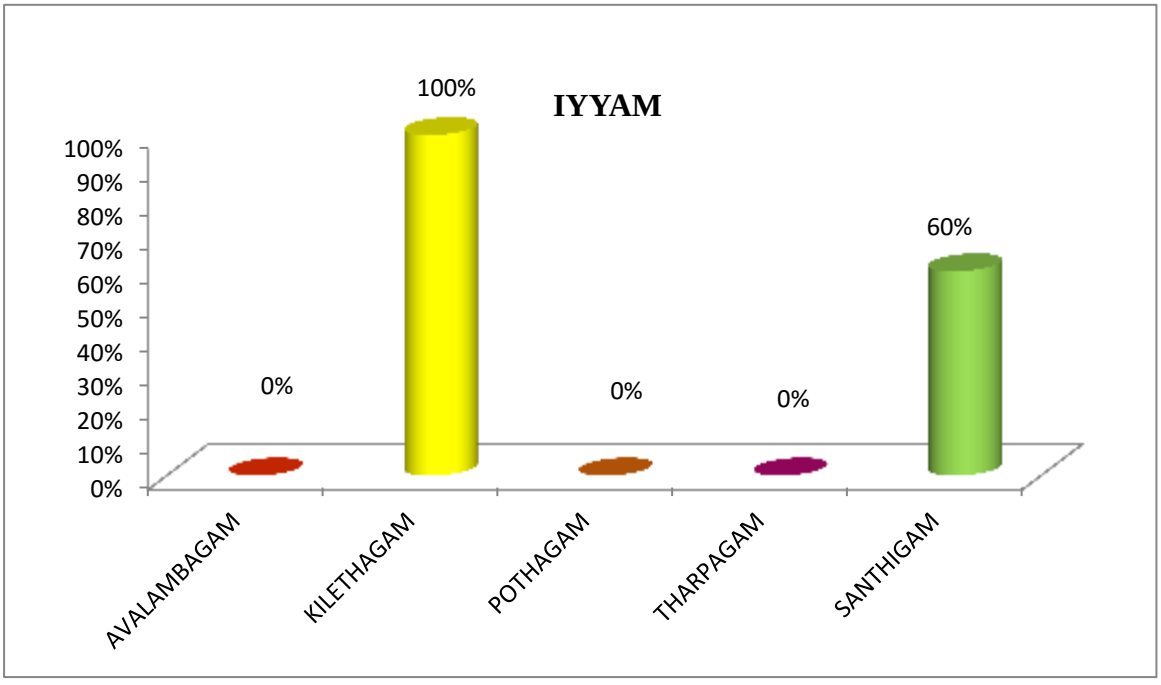


Inference:

Out of 40 patients, all the 40 patients were affected with Paasagam and Saadhagam. Ranjagam was affected in 8 patients (20%), Aalosagam was affected in 7 patients (17.5%) and Prasagam was affected in 6 patients (15%).

C. Affected Iyyam:

CLASSIFICATION OF IYYAM	NO. OF CASES	PERCENTAGE
AVALAMBAGAM	0	0%
KILETHAGAM	40	100%
POTHAGAM	0	0%
THARPAGAM	0	0%
SANTHIGAM	24	60%

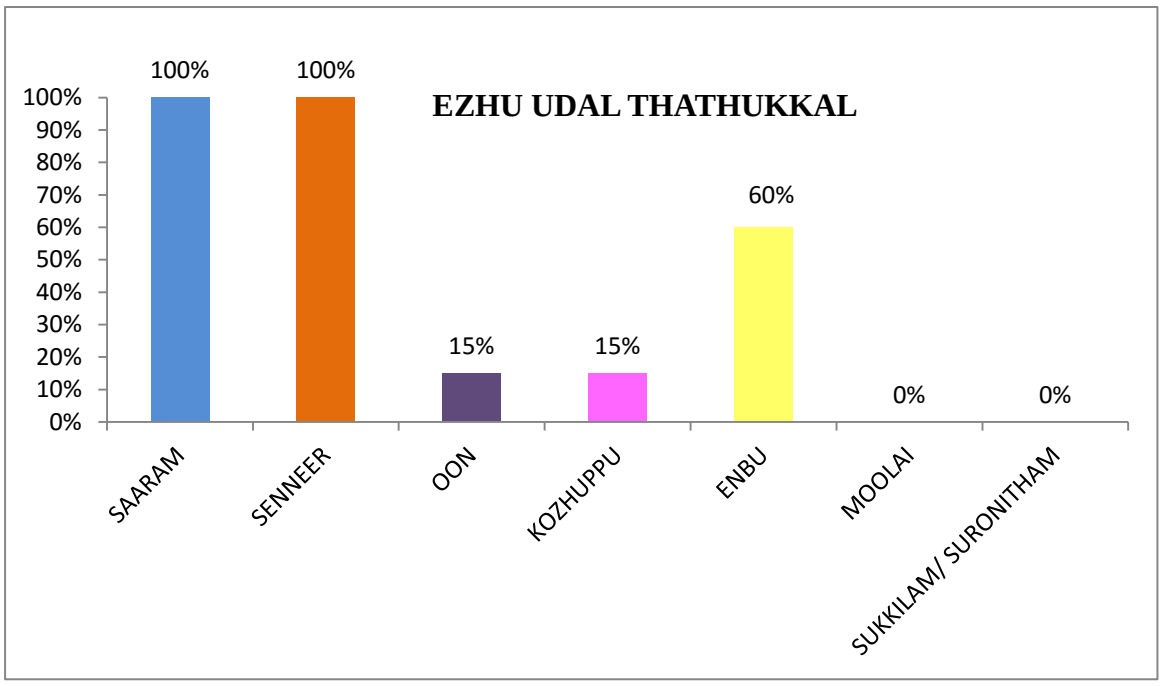


Inference:

Out of 40 patients, all patients were affected with Kilethagam and Santhigam was affected in 24 patients (60%).

12. EZHU UDAL THATHUKKAL:

EZHU UDAL THATHUKKAL	NO. OF CASES	PERCENTAGE
SAARAM	40	100%
SENNEER	40	100%
OON	6	15%
KOZHUPPU	6	15%
ENBU	24	60%
MOOLAI	0	0%
SUKKILAM/SURONITHAM	0	0%

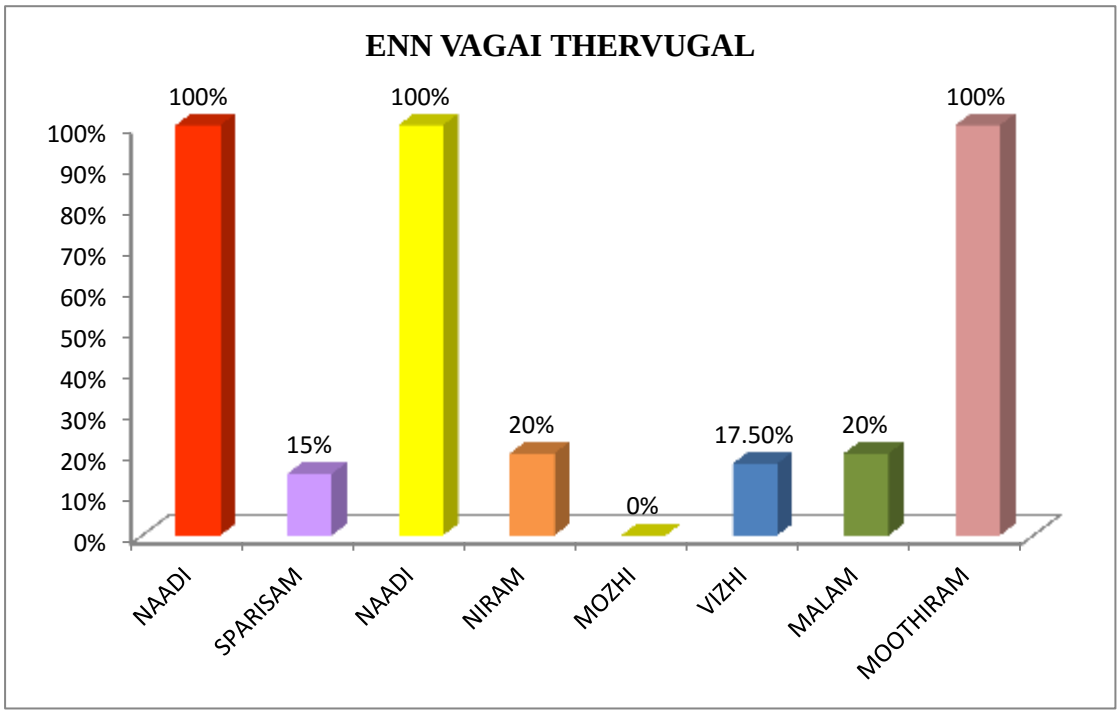


Inference:

From the above chart, we observe that Saaram and Senneer were affected in all the patients (100%), Oon & Kozhuppu were affected to the extent of 15% and Enbu was affected in 24 patients (60%).

13. ENN VAGAI THERVUGAL:

ENN VAGAI THERVUGAL	NO. OF CASES	PERCENTAGE
NAADI	40	100%
SPARISAM	6	15%
NAA	40	100%
NIRAM	8	20%
MOZHI	0	0%
VIZHI	7	17.5%
MALAM	8	20%
MOOTHIRAM	40	100%

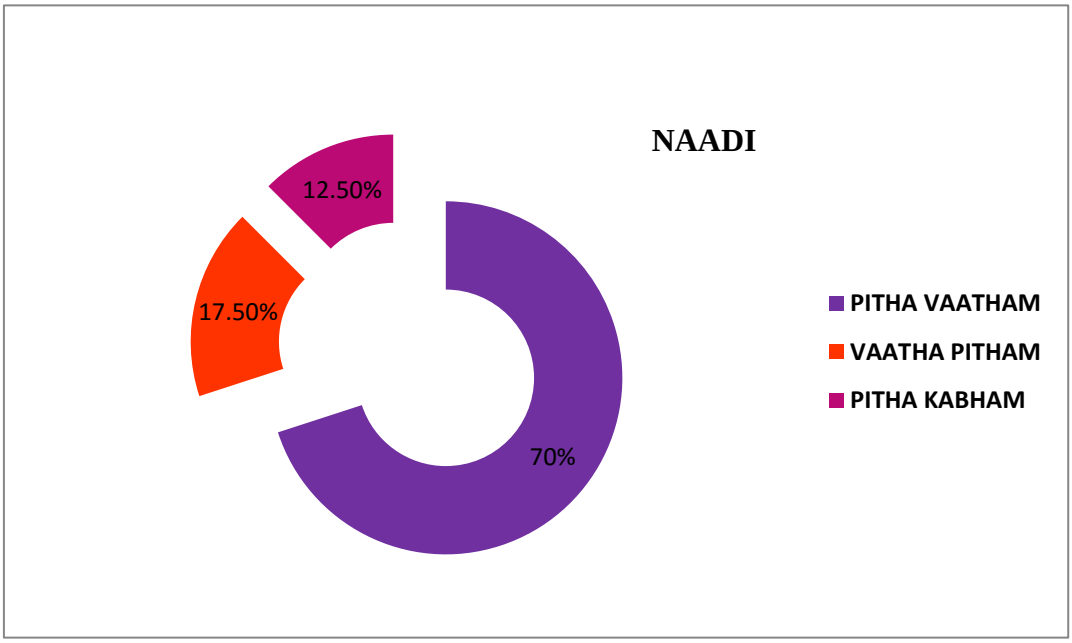


Inference:

Regarding Enn Vagai Thervu, all the 40 patients were affected with Naadi, Naa, Moothiram, 8 patients (20%) were affected with Malam and Niram. Vizhi was affected in 7 patients (17.5%) and Sparisam was affected in 6 patients (15%).

14. NAADI:

NAADI	NO. OF CASES	PERCENTAGE
PITHA VAATHAM	28	70%
VAATHA PITHAM	7	17.5%
PITHA KABHAM	5	12.5%

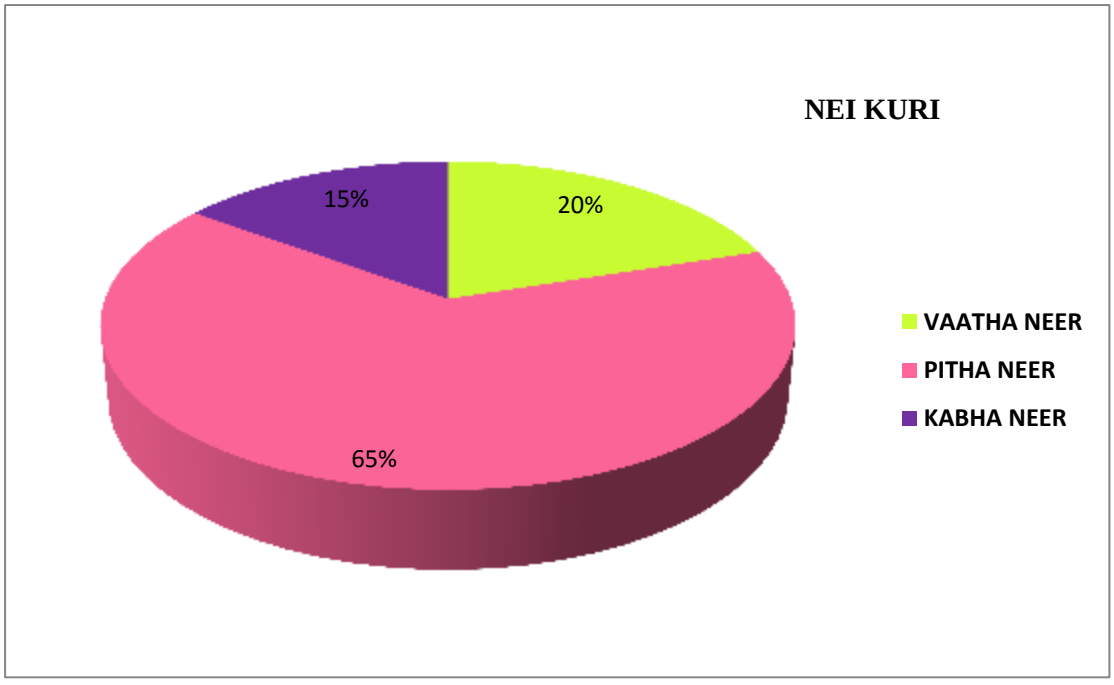


Inference:

Out of 40 patients, 28 patients (70%) had Pitha Vaatha naadi, 7 patients (17.5%) had Vaatha Pitha naadi and 5 patients (12.5%) had Pitha Kabha naadi.

15. NEI KURI REFERENCE:

NEI KURI	CHARACTER OF URINE	NO. OF CASES	PERCENTAGE
VAATHA NEER	SPREADS LIKE SNAKE	8	20%
PITHA NEER	SPREADS LIKE RING	26	65%
KABHA NEER	FLOAT LIKE PEARL	6	15%

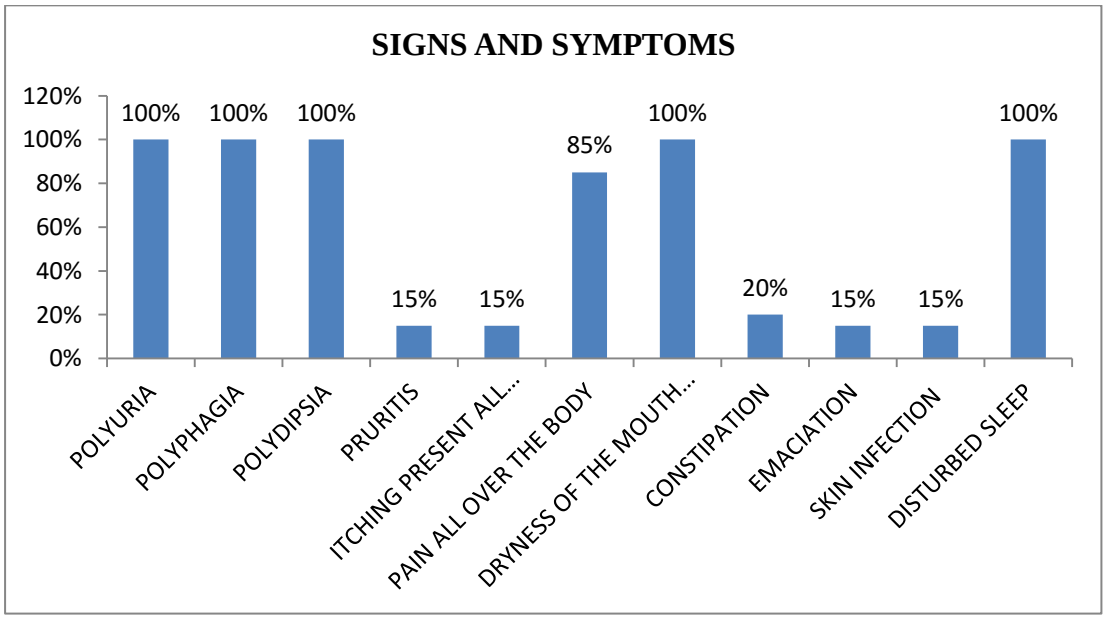


Inference:

Out of 40 patients, 8 samples (20%) show Vaatha Neer, 26 samples (65%) show Pitha Neer and 6 samples (15%) show Kabha Neer.

16. CLINICAL FEATURES:

SIGNS & SYMPTOMS	NO. OF CASES	PERCENTAGE
POLYURIA	40	100%
POLYPHAGIA	40	100%
POLYDIPSIA	40	100%
PRURITIS VULVAE/ BALANITIS	6	15%
ITCHING PRESENT ALL OVER THE BODY	6	15%
PAIN ALL OVER THE BODY	34	85%
DRYNESS OF MOUTH & THROAT	40	100%
CONSTIPATION	8	20%
EMACIATION	6	15%
SKIN INFECTION	6	15%
DISTURBED SLEEP	40	100%

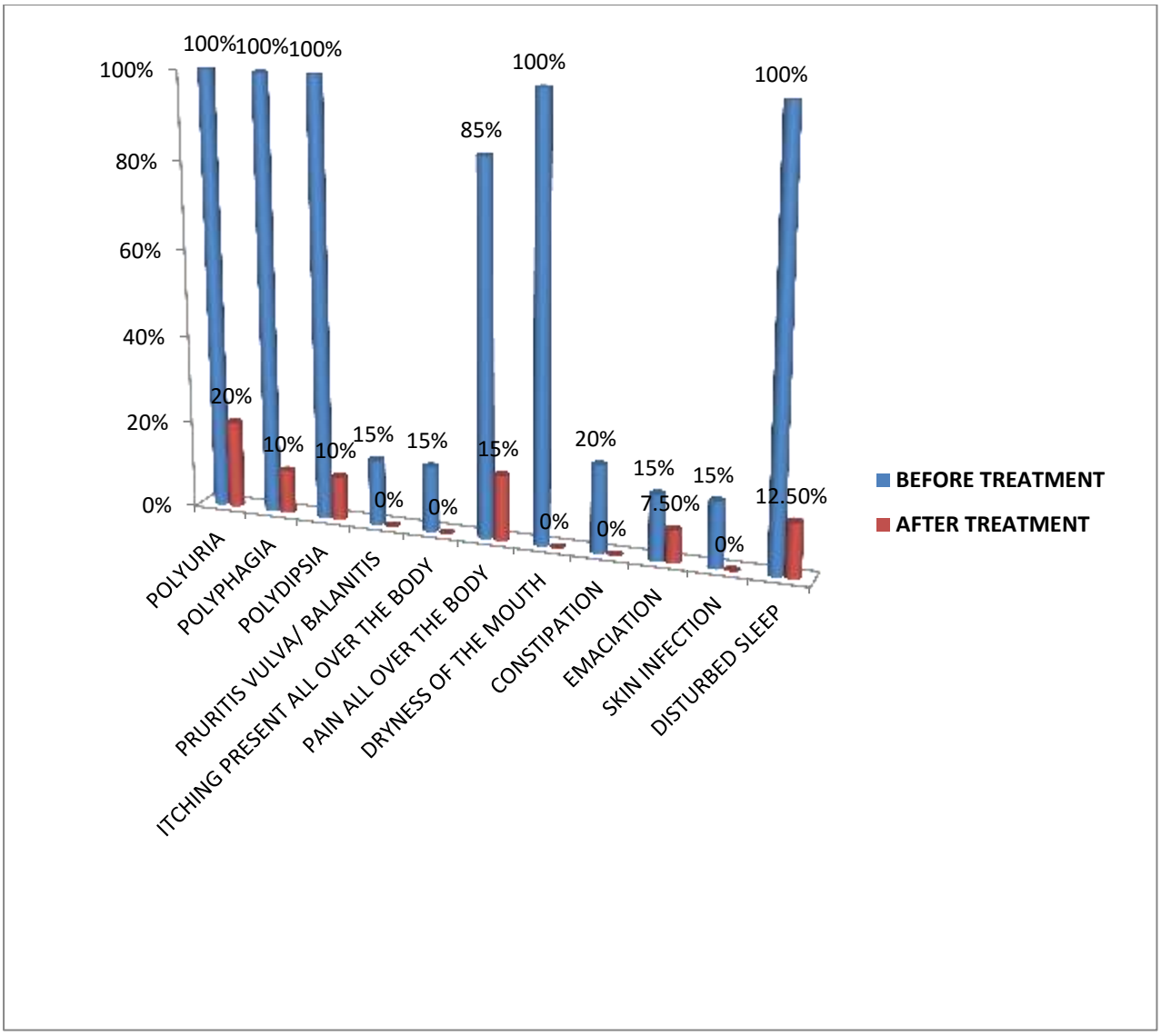


Inference:

In respect of the patients with Madhumegam, the clinical symptoms of Polyuria, Polyphagia, Polydipsia, Dryness of Mouth & Throat and Disturbed sleep were present in all cases. Pruritis Vulvae / Balanits, Itching present all over the body, Skin infection and Emaciation were present in 6 cases (15%). Constipation was present in 8 patients (20%) and Pain present all over the body was in 34 cases (85%).

17. CLINICAL PROGNOSIS:

SIGNS & SYMPTOMS	BEFORE TREATMENT		AFTER TREATMENT	
	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE
POLYURIA	40	100%	8	20%
POLYPHAGIA	40	100%	4	10%
POLYDIPSIA	40	100%	4	10%
PRURITIS VULVAE/ BALANITIS	6	15%	0	0%
ITCHING PRESENT ALL OVER THE BODY	6	15%	0	0%
PAIN ALL OVER THE BODY	34	85%	6	15%
DRYNESS OF MOUTH & THROAT	40	100%	0	0%
CONSTIPATION	8	20%	0	0%
EMACIATION	6	15%	3	7.5%
SKIN INFECTION	6	15%	0	0%
DISTURBED SLEEP	40	100%	5	12.5%

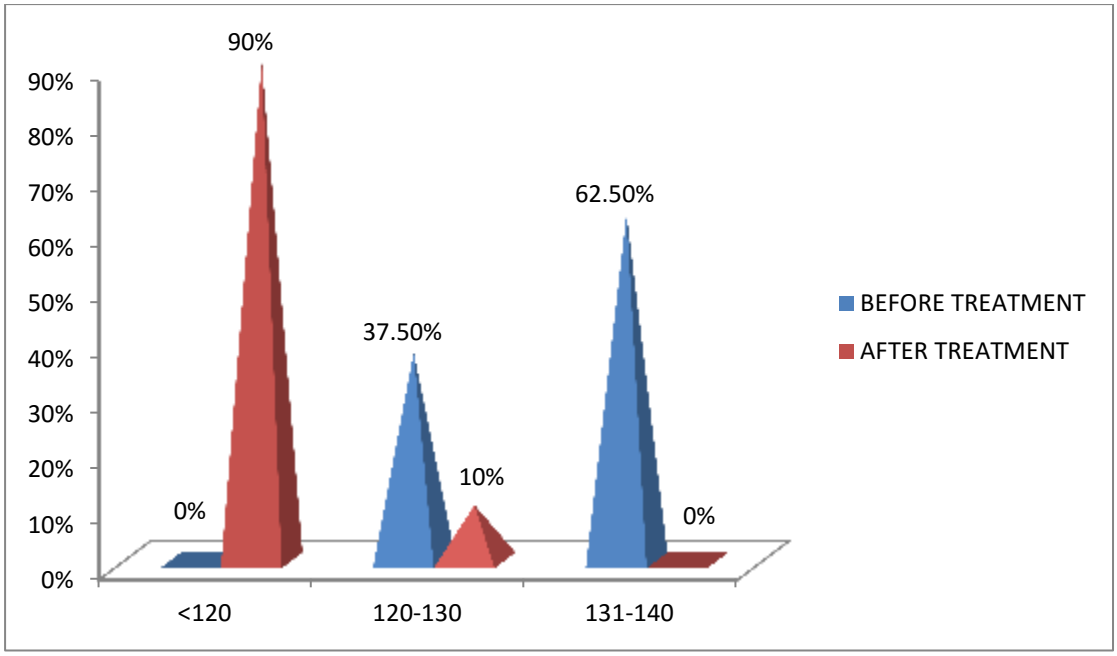


Inference:

The clinical signs & symptoms were improved. After treatment, 20% of peoples had Polyuria, 12.5% had Polyphagia, 10% of people had Polydypsia, 15% had pain all over the body, 12.5% had disturbed sleep and 7.5% had emaciation. The symptoms of Pruritis Vulvae/ Balanitis, Itching all over the body, dryness in Mouth & Throat, skin infection and constipation were completely relieved.

18(A). BLOOD SUGAR (FASTING):

BLOOD SUGAR mg/dl	BEFORE TREATMENT (NO. OF CASES)	PERCENTAGE	AFTER TREATMENT (NO. OF CASES)	PERCENTAGE
<120	0	0%	36	90%
120-130	15	37.5%	4	10%
131-140	25	62.5%	0	0%

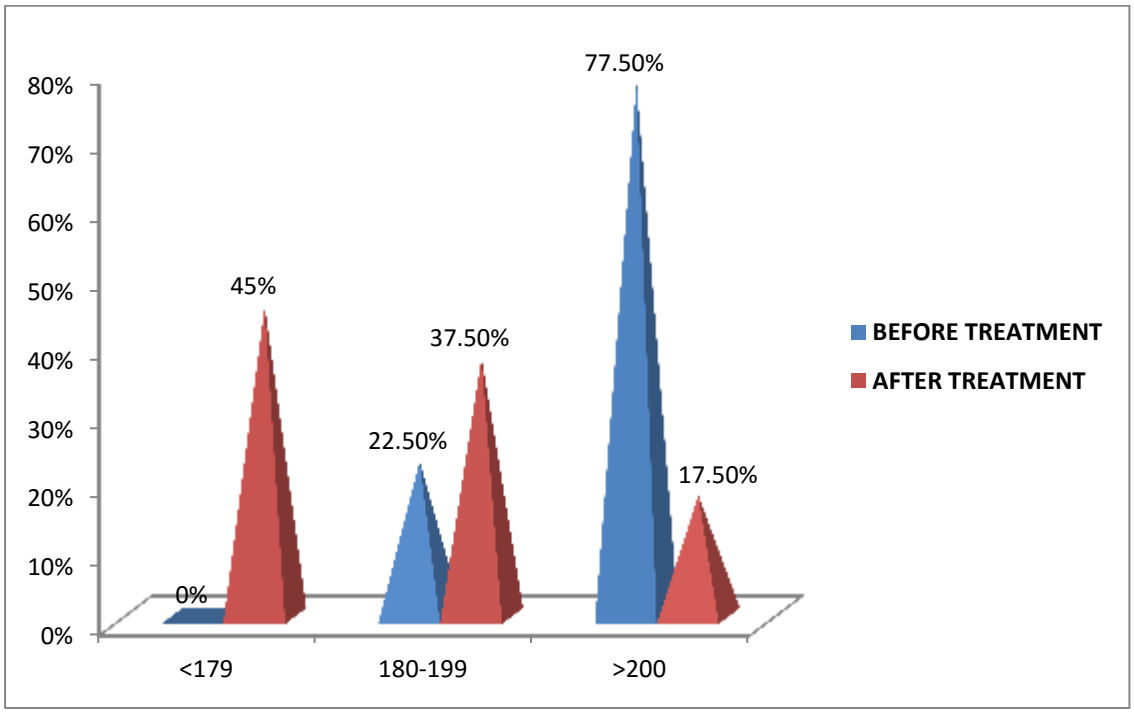


Inference:

Fasting blood sugar had controlled by 90% of cases.

18(B). BLOOD SUGAR (POST PRANDIAL):

BLOOD SUGAR mg/dl	BEFORE TREATMENT (NO. OF CASES)	PERCENTAGE	AFTER TREATMENT (NO.OF CASES)	PERCENTAGE
<179	0	0%	18	45%
180-199	9	22.5%	15	37.5%
>200	31	77.5%	7	17.5%

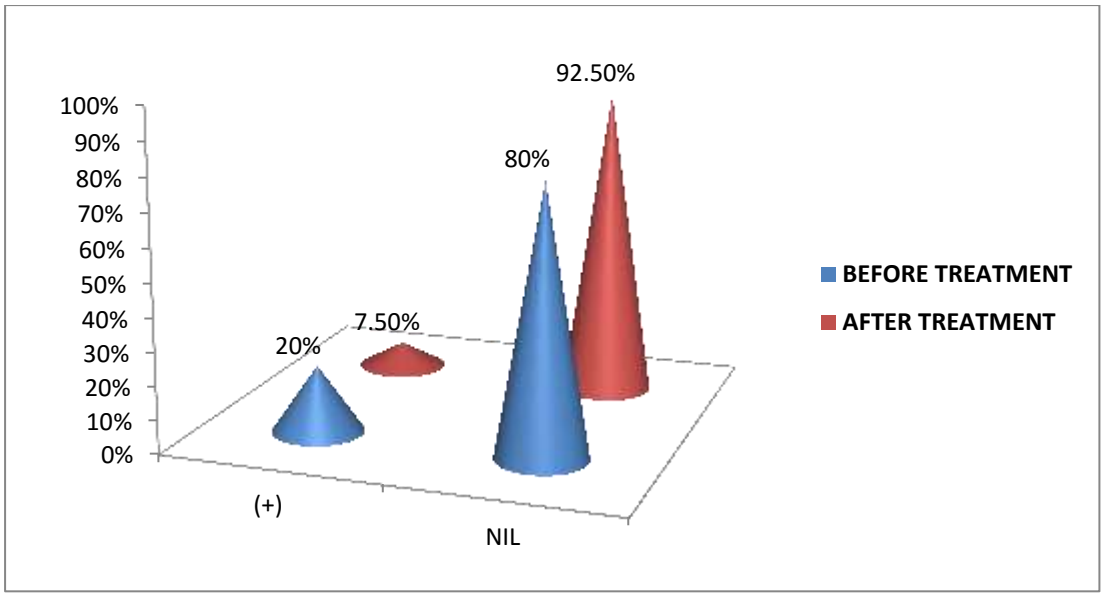


Inference:

The Blood Sugar Post Prandial had controlled by 82.5% of cases.

19 (A). URINE SUGAR (FASTING):

URINE SUGAR	BEFORE TREATMENT (NO. OF CASES)	PERCENTAGE	AFTER TREATMENT (NO. OF CASES)	PERCENTAGE
(+)	8	20%	3	7.5%
NIL	32	80%	37	92.5%

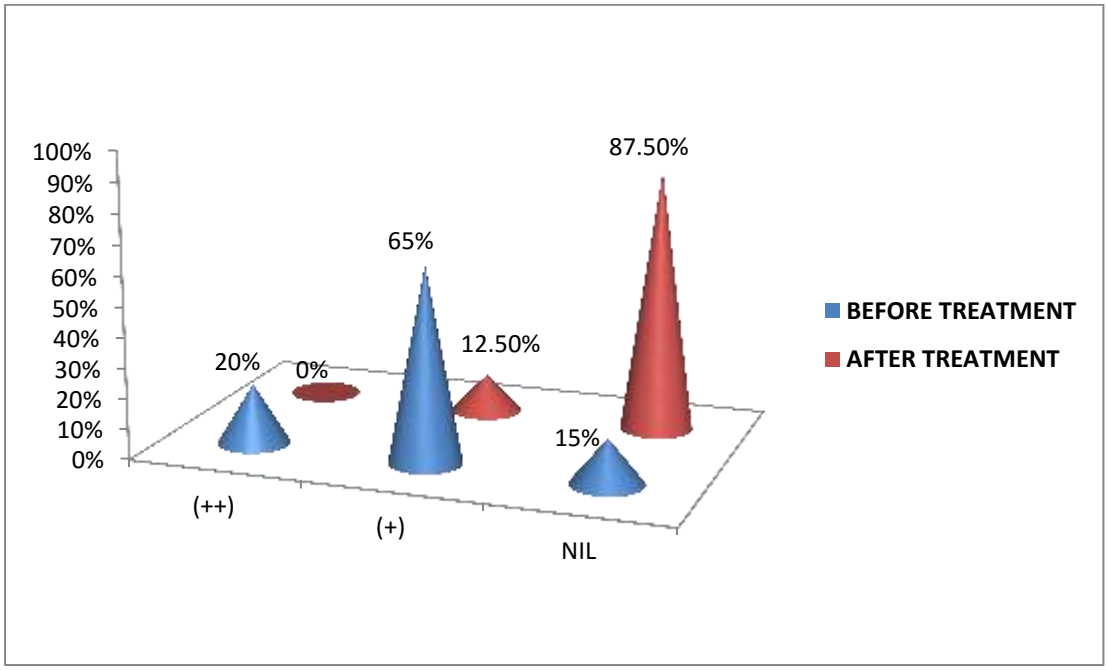


Inference:

From the above chart it may be observed that urine sugar results on fasting after treatment had improved drastically and it was nil in 92.5% of cases after treatment.

19(B). URINE SUGAR (POST PRANDIAL):

URINE SUGAR	BEFORE TREATMENT (NO. OF CASES)	PERCENTAGE	AFTER TREATMENT (NO. OF CASES)	PERCENTAGE
(++)	8	20%	0	0%
(+)	26	65%	5	12.5%
NIL	6	15%	35	87.5%

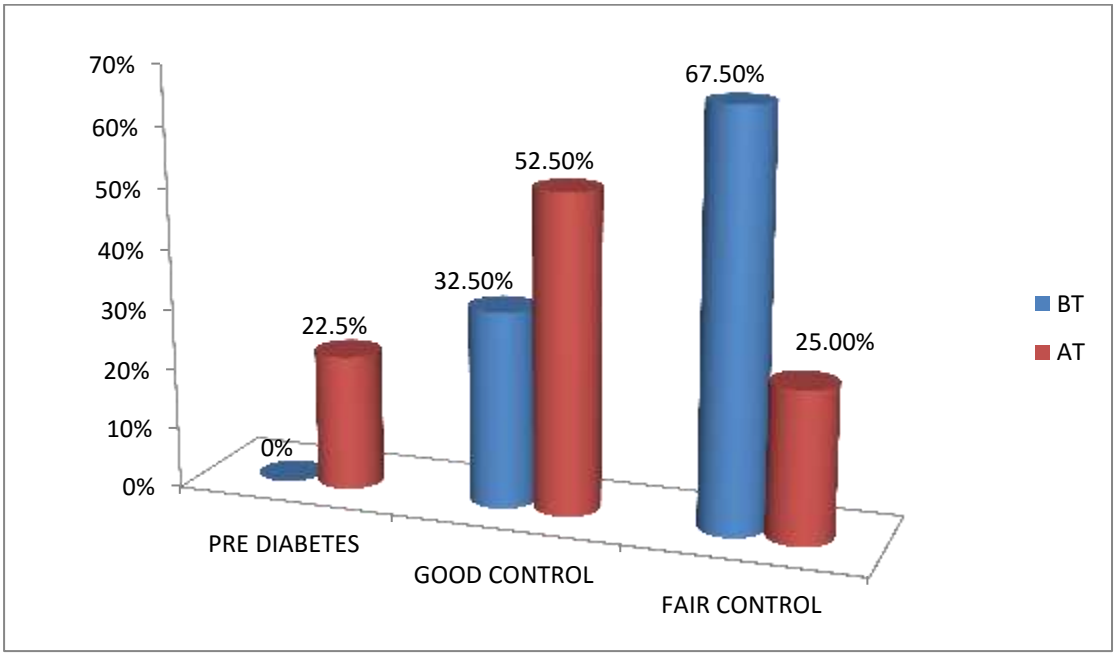


Inference:

It may be noted that urine sugar result on post prandial after treatment had improved drastically. It was nil in 87.5% of cases after treatment.

20. HbA1C LEVEL:

HbA1C	BEFORE TREATMENT (NO.OF CASES)	PERCENTAGE	AFTER TREATMENT (NO.OF CASES)	PERCENTAGE
PRE-DIABETES 5.7- 6.4	0	0%	9	22.5%
GOOD CONTROL 6.5- 7.0	13	32.5%	21	52.5%
FAIR CONTROL Above 7 - 8	27	67.5%	10	25%

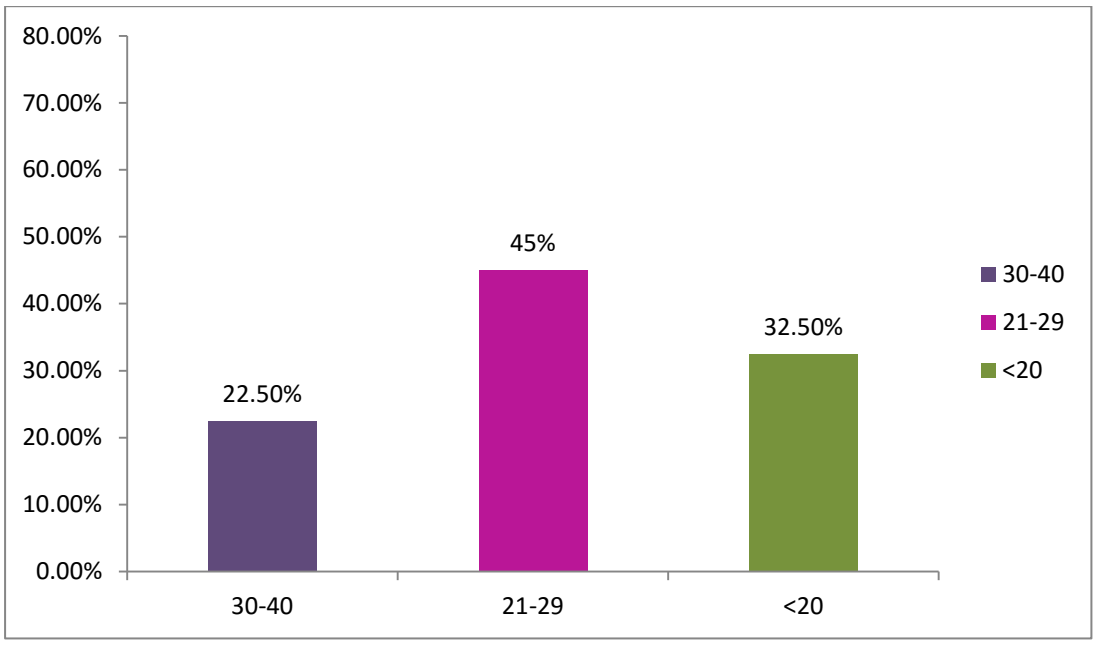


Inference:

HbA1C level had good control in 75% of cases and fair control in 20% of cases after treatment.

21(A). BASED ON REDUCTION IN BLOOD SUGAR FASTING:

BLOOD SUGAR LEVEL (F)	PROGNOSIS	NO. OF CASES	PERCENTAGE
30 – 40 mg/dl	GOOD	9	22.5%
21 – 29 mg/dl	MODERATE	18	45%
< 20 mg/dl	MILD	13	32.5%

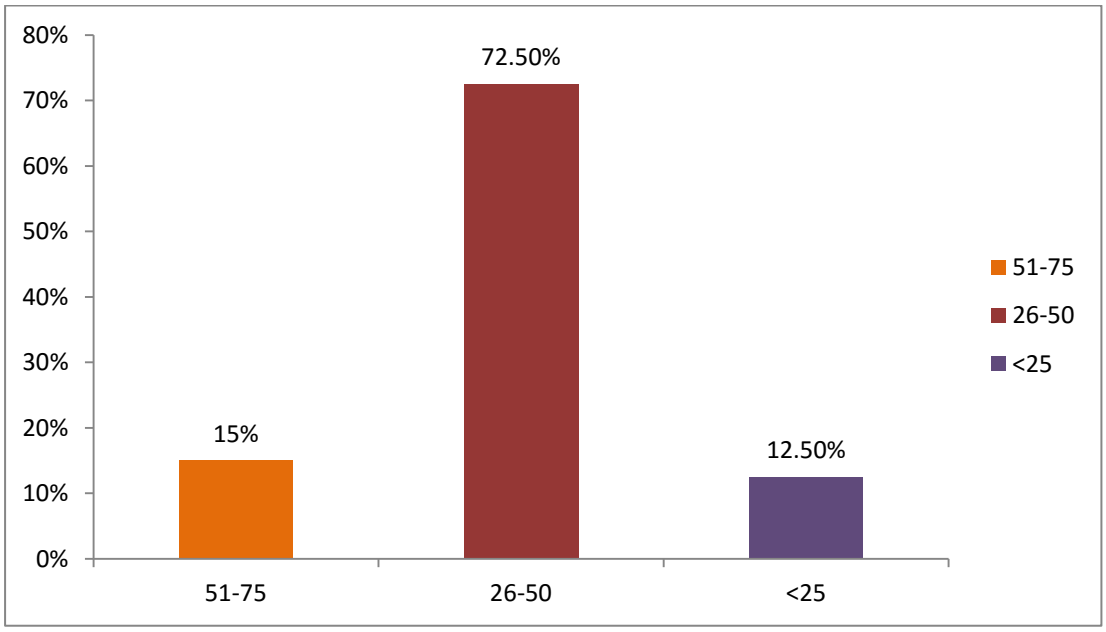


Inference:

Out of 40 patients, 9 patients (22.5%) shows good result, 18 patients (45%) shows moderate results and 13 patients (32.5%) shows mild results.

21(B). BASED ON REDUCTION IN BLOOD SUGAR POST PRANDIAL:

BLOOD SUGAR LEVEL (PP)	PROGNOSIS	NO. OF CASES	PERCENTAGE
51 - 75 mg/dl	GOOD	6	15%
26 - 50 mg/dl	MODERATE	29	72.5%
< 25 mg/dl	MILD	5	12.5%



Inference:

Out of 40 patients, 6 patients (15%) shows good result, 29 patients (72.5%) shows moderate results and 5 patients (12.5%) shows mild results.

BLOOD SUGAR LEVEL:

S.NO	OUT PATIENT NO.	AGE/SEX	BLOOD SUGAR LEVEL			
			BEFORE TREATMENT		AFTER TREATMENT	
			F (mg/dl)	PP (mg/dl)	F (mg/dl)	PP (mg/dl)
1	8677	48/M	127	192	106	162
2	8693	35/F	133	186	119	141
3	3820	60/M	139	240	126	169
4	9305	46/M	132	214	127	180
5	1002	48/F	134	184	106	152
6	1126	41/M	135	245	109	205
7	1196	52/M	140	258	130	208
8	1208	40/M	128	244	98	192
9	1319	44/F	133	220	111	181
10	1543	52/F	131	200	105	166
11	1732	46/M	126	229	95	180
12	1811	33/M	129	242	107	196
13	3166	37/F	128	219	100	179
14	4732	58/F	127	208	94	177
15	5015	52/F	134	239	112	189
16	5177	55/M	138	257	113	221
17	5202	54/F	129	218	100	165
18	6323	43/M	133	230	108	195
19	8184	48/F	140	268	122	211
20	9328	50/M	133	222	99	181
21	1593	57/F	127	203	114	179
22	1786	59/F	135	275	123	242

23	1822	45/F	132	229	113	197
24	3204	55/M	129	228	93	177
25	3678	52/M	130	247	107	198
26	7406	60/F	128	191	102	180
27	4653	47/M	126	219	97	178
28	4900	52/M	133	182	119	163
29	4966	42/F	128	260	107	216
30	5401	56/M	137	219	113	182
31	5386	59/F	128	214	110	175
32	6241	37/F	128	186	93	159
33	7418	45/F	132	213	117	183
34	7762	36/M	134	233	111	190
35	7644	53/F	138	251	106	212
36	8101	32/M	126	183	98	170
37	7887	37/F	137	268	107	199
38	7712	55/F	136	225	129	176
39	8754	60/F	136	188	100	163
40	9576	40/F	132	184	95	153

BMI CHART OF THE PATIENTS

			BEFORE TREATMENT				AFTER TREATMENT			
S.NO	**OP. NO.**	**AGE /SEX**	**WT(kg)**	**HT (cm)**	**BMI**	**H/L/ O**	**WT(kg)**	**HT (cm)**	**BMI**	**H/L/O**
1	8677	48/M	72	170	24	H	70	170	24	H
2	8693	35/F	50	164	18	L	55	164	20	H
3	3820	60/M	75	170	27	O	73	170	25	O
4	9305	46/M	60	162	22	H	58	162	22	H
5	1002	48/F	59	158	23	H	56	158	22	H
6	1126	41/M	68	173	22	H	65	173	22	H
7	1196	52/M	80	175	26	O	77	175	25	O
8	1208	40/M	62	160	24	H	60	162	23	H
9	1319	44/F	65	170	22	H	63	170	22	H
10	1543	52/F	63	163	24	H	60	163	23	H
11	1732	46/M	50	164	18	L	55	164	20	H
12	1811	33/M	60	162	22	H	58	162	22	H
13	3166	37/F	65	163	24	H	62	163	23	H
14	4732	58/F	70	189	24	H	74	189	21	H
15	5015	52/F	65	163	24	H	62	163	23	H
16	5177	55/M	75	170	26	O	73	170	25	O
17	5202	54/F	70	170	24	H	67	170	23	H
18	6323	43/M	62	162	23	H	60	162	23	H
19	8184	48/F	68	163	26	O	66	163	25	O
20	9328	50/M	61	165	22	H	59	165	22	H
21	1593	57/F	62	160	24	H	60	162	23	H
22	1786	59/F	70	165	26	O	69	165	25	O

23	1822	45/F	58	160	23	H	57	162	22	H
24	3204	55/M	50	163	18	L	54	163	20	H
25	3678	52/M	56	157	22	H	54	157	22	H
26	7406	60/F	58	160	23	H	57	162	22	H
27	4653	47/M	58	163	22	H	56	163	21	H
28	4900	52/M	50	163	18	L	55	163	21	H
29	4966	42/F	65	170	22	H	63	170	22	H
30	5401	56/M	61	165	21	H	59	165	22	H
31	5386	59/F	50	164	18	L	55	164	20	H
32	6241	37/F	70	170	24	H	67	170	23	H
33	7418	45/F	72	170	24	H	70	170	24	H
34	7762	36/M	80	175	26	O	74	175	24	H
35	7644	53/F	50	163	18	L	53	163	20	H
36	8101	32/M	65	170	22	H	63	170	22	H
37	7887	37/F	70	165	26	O	68	165	25	O
38	7712	55/F	69	165	25	O	70	165	26	O
39	8754	60/F	62	162	23	H	62	162	23	H
40	9576	40/F	62	157	25	O	59	157	24	H

H – HEALTHY L – LEAN O - OVERWEIGHT

BIO CHEMICAL ANALYSIS OF THE PATIENTS

S.NO	O.P. NO.	AGE/ SEX	BLOOD SUGAR LEVEL (mg/dl)				URINE SUGAR LEVEL				HBA1C (%)		C-PEPTITE ASSAY (ng/dl)	
			BT		AT		BT		AT					
			F	PP	F	PP	F	PP	F	PP	BT	AT	BT	AT
1	8677	48/M	127	192	106	162	NIL	NIL	NIL	NIL	7.7	7.1	2.20	2.15
2	8693	35/F	133	186	119	141	NIL	(+)	(+)	NIL	8	7.2	2.89	2.90
3	3820	60/M	139	240	126	169	(+)	(+)	(+)	NIL	7.8	7.0	4.40	4.92
4	9305	46/M	132	214	121	180	NIL	(+)	NIL	NIL	7.3	6.8	2.40	2.28
5	1002	48/F	134	184	106	152	NIL	NIL	NIL	NIL	7.6	7.1	3.69	3.70
6	1126	41/M	135	245	109	205	NIL	(+)	NIL	NIL	7.4	6.3	2.65	2.50
7	1196	52/M	140	258	130	208	(+)	(++)	(+)	(+)	7.9	7.6	3.50	3.40
8	1208	40/M	128	244	98	192	NIL	(+)	NIL	NIL	6.6	6.1	2.20	1.90
9	1319	44/F	133	220	111	181	NIL	(+)	NIL	NIL	6.7	5.8	3.8	3.62
10	1543	52/F	131	200	105	166	NIL	NIL	NIL	NIL	7.1	6.5	2.80	2.68
11	1732	46/M	126	229	95	180	NIL	(+)	NIL	NIL	6.8	6.0	1.90	1.97
12	1811	33/M	129	242	107	196	NIL	(+)	NIL	NIL	6.7	6.5	2.36	2.58
13	3166	37/F	128	219	100	179	NIL	(+)	NIL	NIL	7.3	6.9	3.66	3.50
14	4732	58/F	127	208	94	177	NIL	(+)	NIL	NIL	7.0	6.5	2.44	1.19
15	5015	52/F	134	239	112	189	NIL	(+)	NIL	NIL	7.5	6.5	3.80	3.97
16	5177	55/M	138	257	113	221	(+)	(++)	NIL	(+)	8	7.4	5.18	4.71
17	5202	54/F	129	218	100	165	NIL	NIL	NIL	NIL	6.8	5.9	1.89	1.85
18	6323	43/M	133	230	108	195	NIL	(++)	NIL	NIL	6.7	6.2	3	2.93
19	8184	48/F	140	268	122	211	(+)	(++)	NIL	(+)	7.8	6.8	3.40	3.28
20	9328	50/M	133	222	99	181	NIL	(+)	NIL	NIL	7.4	7.2	3.86	3.82
21	1593	57/F	127	203	114	179	NIL	(+)	NIL	NIL	6.9	6.5	2.50	1.89
22	1786	59/F	135	275	123	242	(+)	(++)	(+)	(+)	8.0	7.9	4.88	4.70

23	1822	45/F	132	229	113	197	NIL	(+)	NIL	NIL	7.1	6.8	3.22	3.38
24	3204	55/M	129	228	93	177	NIL	NIL	NIL	NIL	7.0	6.5	2.75	2.70
25	3678	52/M	130	247	107	198	NIL	(+)	NIL	NIL	7.4	6.8	3.08	3
26	7406	60/F	128	191	102	180	NIL	(+)	NIL	NIL	7.5	6.9	1.93	1.88
27	4653	47/M	126	219	97	178	NIL	(+)	NIL	NIL	7.8	6.6	3.1	3.18
28	4900	52/M	133	182	119	163	NIL	NIL	NIL	NIL	6.8	6.0	3.0	2.80
29	4966	42/F	128	260	107	216	NIL	(+)	NIL	NIL	7.9	7.2	2.10	2.0
30	5401	56/M	137	219	113	182	NIL	(+)	NIL	NIL	7.2	6.8	1.88	1.89
31	5386	59/F	128	214	110	175	NIL	(+)	NIL	NIL	6.9	6.2	2.24	2.12
32	6241	37/F	128	186	93	159	NIL	(+)	NIL	NIL	8	6.8	4.05	4.13
33	7418	45/F	132	213	117	183	(+)	(++)	(+)	NIL	6.6	5.8	3.25	3.16
34	7762	36/M	134	233	111	190	NIL	(+)	NIL	NIL	7.7	6.7	3.60	3.45
35	7644	53/F	138	251	106	212	NIL	(++)	NIL	NIL	7.5	6.6	3.26	3.46
36	8101	32/M	126	183	98	170	NIL	(+)	NIL	NIL	7.6	6.6	2.06	2.12
37	7887	37/F	137	268	107	199	NIL	(+)	NIL	NIL	7.2	6.7	1.80	1.66
38	7712	55/F	136	225	129	176	NIL	(++)	NIL	(+)	8.0	7.9	4.48	5.02
39	8754	60/F	136	188	100	163	NIL	(++)	NIL	NIL	7.8	7.2	3.32	3.38
40	9576	40/F	132	184	95	153	NIL	(+)	NIL	NIL	7.0	6.5	2.70	2.65

BT – BEFORE TREATMENT, AT- AFTER TREATMENT, N – NIL, F- FASTING, PP – POST PRANDIAL

LABORATORY INVESTIGATION REPORT OF THE PATIENTS

S.NO	O.P.NO	AGE/SEX	BLOOD UREA (mg/dl)		SERUM CREATININE (mg/dl)	
			BT	AT	BT	AT
1	8677	48/M	28	22	0.6	0.56
2	8693	35/F	19	24	0.5	0.62
3	3820	60/M	26	23	0.71	0.63
4	9305	46/M	22	16	0.54	0.60
5	1002	48/F	16	19	0.9	0.9
6	1126	41/M	21	22	0.65	0.52
7	1196	52/M	38	29	0.89	0.95
8	1208	40/M	21	23	0.88	0.81
9	1319	44/F	27	29	0.77	0.64
10	1543	52/F	18	16	0.93	0.86
11	1732	46/M	42	36	0.92	0.90
12	1811	33/M	32	28	0.68	0.70
13	3166	37/F	20	23	0.59	0.72
14	4732	58/F	34	35	0.81	0.83
15	5015	52/F	16	24	0.9	0.88
16	5177	55/M	47	40	0.86	0.81
17	5202	54/F	26	22	0.74	0.73
18	6323	43/M	21	20	0.65	0.66
19	8184	48/F	28	19	0.59	0.63
20	9328	50/M	31	33	0.98	0.96
21	1593	57/F	28	25	0.66	0.84
22	1786	59/F	45	40	0.83	0.81
23	1822	45/F	37	29	0.79	0.86
24	3204	55/M	28	24	0.71	0.66

25	3678	52/M	34	31	0.77	0.74
26	7406	60/F	18	17	0.92	0.84
27	4653	47/M	24	24	0.69	0.63
28	4900	52/M	33	29	0.58	0.61
29	4966	42/F	25	26	0.60	0.68
30	5401	56/M	37	30	0.86	0.95
31	5386	59/F	18	22	0.94	0.67
32	6241	37/F	35	32	0.86	0.95
33	7418	45/F	40	38	0.68	0.54
34	7762	36/M	32	30	0.57	0.60
35	7644	53/F	15	18	0.90	0.83
36	8101	32/M	26	29	0.50	0.69
37	7887	37/F	36	31	0.81	0.74
38	7712	55/F	21	27	0.63	0.66
39	8754	60/F	22	18	0.70	0.82
40	9576	40/F	35	23	0.92	0.87

BT – BEFORE TREATMENT AT – AFTER TREATMENT

LIPID PROFILE:

S. NO	OP. NO	BEFORE TREATMENT					AFTER TREATMENT				
		Total chol (mg/dl)	Sr.Trigl (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	Total chol (mg/dl)	Sr.Trigl (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
1	8677	180	105	62.2	86	26.4	175	102	63	81	25
2	8693	128	96	68	66	18.5	126	96.5	66.2	65.1	17.2
3	3820	196.2	142.5	80.5	98.2	36.4	180	138.6	76	90	35.4
4	9305	174	122	63.1	84	18.8	172	121	63	83	20.2
5	1002	166	122	72.4	78.5	29	168	118	72	76	29.3
6	1126	118	75	80	53.8	15.2	119	107	87	61.5	17
7	1196	224	168	66	96	37.5	198	150	71.5	94.7	37
8	1208	176	133	69	60	18	171	132.5	68	73.7	18.2
9	1319	204	139	62	59	31	192	131	63.1	59	30.8
10	1543	155	114	66	71.1	24	155.2	112	69.4	70.5	23
11	1732	181	137	72	98	31	180	137	72.3	95	31
12	1811	126	119	63	79	29.5	120	111	66	78.2	28
13	3166	141	120	81	110	33	152	124.6	82	97.6	33
14	4732	186	137	80	91	24.2	184.1	140	77	87.1	22.6
15	5015	173	125.5	77	88.2	22.8	172	125	78	85.8	21.5
16	5177	246	167	60.6	73	31	215	150	61	72.5	31
17	5202	148	112	72	68.6	22	160	110	72	66.5	23
18	6323	172	126	81.2	88	29	170	128.2	80.6	87.6	29
19	8184	193	150	64	114	31	190	146.2	66	103	30.6
20	9328	188	163	81	96	35.3	188.2	160	79.5	91.2	33.6
21	1593	170	117	47.7	98.9	23.4	169	115.7	59.4	91.8	23
22	1786	211	156	62	69.1	30	199	147	66	69.6	31.8
23	1822	175	90	78	91	28.2	172	102	72	90.3	25.9

24	3204	167.4	102	66	74.2	18.8	170	113	68.2	73.5	19
25	3678	163	121	62.5	33	41	161	123.2	66.5	80	35
26	7406	184	143	87.6	112	36	185	140.2	83.2	97.2	35.6
27	4653	177	123	64	91	25.4	177.5	123.2	66.5	80	35
28	4900	151	116	72.1	53.7	27	152	114	74.8	55.2	25.2
29	4966	169	120	37.7	107.3	24	170	121	56.6	102.3	26
30	5401	196	120	63.3	88.6	31	176	118	63	86	31.1
31	5386	237	139	73	89.5	28.2	200	138	70	85.1	26.5
32	6241	166	126	71	64.4	30.5	170	131.6	77.2	64	28
33	7418	174	136	69.4	57.5	24	174.2	135	70	59	23.3
34	7762	171	215	52	82	43	182	195.2	54.5	80.8	40
35	7644	174	166	68	101	33.2	180	152	69.4	98.7	32.8
36	8101	189.3	169.1	33	122	34	189	158	49.8	105	32
37	7887	200	144	70	66.2	32.5	197	144	71	67.5	33
38	7712	191	155	53	92	21.8	188.2	149.2	55	91	22
39	8754	164	128	62.7	81.8	19	174	130	66.2	83	23
40	9576	215	172	39	121	35	200	158	50	102	34.3

Total.Chol- Total Cholestrol, Sr. Trigl- Serum Triglycerides, HDL- High Density Lipoprotein,

LDL- Low Density Lipoprotein, VLDL- Very Low Density Lipoprotein

DISCUSSION

DISCUSSION

Diabetes Mellitus, a group of metabolic disorder in which a person has high blood sugar level, either because the pancreas does not produce enough Insulin, or because cells do not respond to the Insulin that is produced. This high blood sugar produces the classical symptoms of Polyuria (frequent urination), Polydipsia (increased thirst) and Polyphagia (increased hunger).

Madhumegam, is a clinical entity described by Yugimunivar in his "Yugi Vaidhya Chintamani 800" can be compared with Diabetes Mellitus. The classical symptoms are Polyuria, Polyphagia, Polydipsia, Itching all over the body and Pain all over the body.

Various Siddha literature has been studied and discussed for choosing the trial medicine for treating Madhumegam and finally choosen "Perungaya Chooranam", which was mentioned in "Sarabenthira Vaithiya Rathna Vali".

Authentication is a critical step for successful and reliable clinical applications and for further experimental studies on Siddha drugs.

DRUG AUTHENTICATION:

Authentication of given specimen is the basic starting point in developing a botanical product.

A sample of specimen is collected from raw drug store and its organoleptic characters; Microscopic and Macroscopic examination was conducted and authenticated by Botanist, CCRS, Chennai.

PHYSICOCHEMICAL ANALYSIS:

Physicochemical parameters includes

Loss on drying at 105^0 C - 3.68%

Total ash – 38.37%

Water soluble ash – 33.88%

Acid insoluble ash - Less than 1%

Water soluble extraction – 47.53%

Alcohol soluble extraction – 13.92%

These values of the given sample were compared with the standard values of Indian pharmacopoeia.

TOXICOLOGICAL STUDY:

Acute toxicity study of the drug Perungaya Chooranam was carried out as per OECD guideline (Organisation For Economic Co-Operation and Developement) Guideline-423. The experimental protocol was approved bt The Institutional Animal Ethics Committee of C.L. Baid Metha College of Pharmacy, Chennai, Tamil Nadu, India. IAEC reference no: IAEC NO: LI/10/CLBMCP/2017. The study was conducted with single oral dose administration of Perungaya Chooranam. In acute toxicity test the Perungaya Chooranam was found to be non-toxic at the dose level of 2000mg/kg body weight.

SUB – ACUTE TOXICITY STUDY:

Sub- acute toxicity study was carried out for 28 days as per OECD guidelines-407. The animals randomly divided into control group and drug divided groups for low and high doses. At the end of the studies the animals were sacrificed and the haematological parameters, biochemical parameters, urine parameters and the histopathology of the vital organs like brain, liver, heart, lung and kidney were carried out. The study result shows that the trial medicine was safe and did not produce any toxic effects.

PHARMACOLOGICAL EVALUATION:

The experimental protocol was approved by The Institutional Animal Ethics Committee of C.L Baid Metha College of Pharmacy, Chennai, Tamil Nadu, India.

IAEC NO: LI/10/CLBMCP/2017.

Pharmacological studies of the trial medicine Perungaya Chooranam showed ANTI-DIABETIC ACTIVITY on tested animals.

BIOCHEMICAL ANALYSIS:

Biochemical assays are needed to evaluate disease models and to drive biomarker analysis in translation medicine and clinical research.

Based on the analysis Perungaya Chooranam exhibits the properties of Chloride, Phosphate, Copper, Iron and Zinc.

IEC:

IEC has approved my trial medicine with the allowed sample size of 40 patients with combined gender.

IEC NO: GSMC-CH-ME-5/007/2016

CTRI:

The global mandate is to register all clinical trials prospectively, i.e. before the enrollment of the first patient. I have successfully registered my trial medicine by submitting the details and scientific data's to Clinical Trial Registry.

CTRI NO: CTRI/2018/03/012505

CLINICAL STUDY:

Clinical studies were conducted followed by CTRI registration with the sample size of 40 patients.

In my study, 40 patients with Madhumegam were selected in the Department of Maruthuvam, Government Siddha Medical College, attached to Arignar Anna Govt Hospital for Indian Medicine, Arumbakkam, Chennai - 106.

All necessary investigations were carried out to all patients and trial medicine was given. The results of before and after treatment of all the patients were analysed and discussed below.

Age distribution:

- From selected 40 cases, 9 patients (22.5%) were between 30 – 40 years, 13 patients (32.5%) were between 41 – 50 years and 18 patients (45%) were between 51 – 60 years old.
- Usually the non-insulin diabetes mellitus occurs only in the age group above 45 years – International Diabetic Monitor.

Sex distribution:

- Out of 40 patients, 13 cases (32.5%) were male and 27 cases (67.5%) were female. Recent studies show that more women are prone to diabetes than men.

Occupational status:

- From selected 40 cases, 16 patients (40%) were housewives, 9 patients (22.5%) are doing business, 11 patients (27.5%) are office goers and 4 (10%) are retired.
- Nowadays due to modernisation and invention of electrical and electronic kitchen equipments, the women lack physical exercise and results in more prone to Diabetes.

Socio-economic status

- Regarding Socio Economic Status 19 Patients (47.5%) were from lower income group, 13 patients (32.5%) were from Middle income group and 8 Patients (20%) were from High income.
- People belonging to lower group are more prone to Madhumegam. Recent research indicates that the poor are more prone to diabetes.
- Research was being conducted to analyze whether rapid changes in their lifestyle or the stress of poverty triggers diabetes.

Dietary Habits:

- Regarding Diet, out of 40 patients, 5 patients (12.5%) were taking Vegetarian diet and 35 patients (87.5%) were taking mixed diet.
- . Further it could also be noted that people who used fast and fried foods are more prone to diabetes as they have more calories of fat.

Family history:

- Regarding family history 5 patients (12.5%) father had diabetic, 8 patients (20%) mother had diabetic and 6 patient (15%) parents both had diabetic and 21 patients (62.5%) had no relevant family history.
- Genetics plays an important role in Madhumegam.

Paruvakaalam:

- From selected 40 patients, 19 patients (47.5%) were noted in Mun Pani Kaalam, 21 patients (52.5%) were noted in Pin Pani Kaalam.

Thinai:

- From the selected 40 patients all (100%) were from Neithal nilam
- Neithal nilam is more prone to Pitha diseases.

Body built:

- Regarding body built, 26 patients (65%) were having normal weight, 9 patients (22.5%) were overweight and 5 patients (12.5%) were lean.

Duration of illness:

- Out of 40 patients, 8 patients (20%) were under newly identified category and 32 patients (80%) were under 3 – 6 months category.

MUKKUTRAM CLASSIFICATION:

In Vatham:

1. Abanan affected in all patients (100%) causing Polyuria, Nocturia, Constipation.
2. Viyanan affected in all patients (100%) with Pain all over the body.
3. Samanan and Kirukaran affected in all patients (100%) causing Polyphagia.
4. Devethathan affected in all patients (100%) causing disturbed sleep, fatigue.
5. Koorman affected in 7 patients (17.5%) causing dimness of vision.

In Pitham:

1. Analagam affected in all patients (100%) causing polyphagia.
2. Sathagam affected in all patients (100%) causing lassitude.
3. Ranjagam, Aalosagam and Prasagam affected in (20%), (17.5%), (15%) patients causing pallor, dimness of vision and dry skin respectively.

In Kabham:

1. Kilethagam affected in all patients (100%) results in Polyphagia.
2. Santhigam affected in 24 patients (60%) causing joint pain.

EZHUUDALTHATHUKKAL:

1. Saaram affected in all patients results in tiredness, general debility.
2. Senneer affected in all cases causing pallor, dryness.
3. Oon and Kozhuppu affected in 6 patients (15%) each causing emaciation
4. Enbu affected in 24 patients (60%) causing joint pain.

ENVAGAITHERVUGAL:

1. Naa, Naadi and Moothiram affected in all 40 patients (100%).
2. Malam and Niram affected in 8 patients (20%) results in constipation and pallor.
3. Vizhi was affected in 7 patients (17.5%) causing dimness of vision, Sparisam affected in 6 patients (15%) causing dry skin.

NAADI:

1. 28 patients (70%) had Pitha Vatha naadi
2. 7 patients (17.5%) had Vatha Pitha naadi
3. 5 patients (12.5%) had Pitha Kaba naadi.

NEIKURI:

1. 8 samples (20%) show Vatha neer
2. 26 samples (65%) show Pitha neer
3. 6 samples (15%) show Kabha neer.

SIGNS AND SYMPTOMS:

1. Polyuria, Polyphagia, Polydipsia, dryness of the mouth & throat and disturbed sleep were present in all cases i.e 100%.
2. Pain all over the body in 34 patients (85%).
3. Itching and skin infection in 10 patients (25%)
4. Constipation in 8 patients (20%).
5. Emaciation in 4 patients (10%).

CLINICAL PROGNOSIS:

The clinical signs and symptoms were improved after treatment, 20% had polyurea, 10% had polyphagia, 10% of the people had polydipsia, 15% have pain all over the body, 7.5% had emaciation and 12.5% had disturbed sleep. Pruritis

vulvae/Balanitis, itching all over the body, skin infection, dryness of mouth and constipation were completely relieved.

LABORATORY ASSESSMENT:

Blood sugar Fasting:

- From the selected 40 patients, before treatment 25 patients of fasting sugar level were in the range of 131-140mg/dl and after treatment 36 patients were <126mg/dl.

Blood sugar Post Prandial:

- From the selected 40 cases before treatment 31 patients of post prandial sugar level were > 200 mg /dl and after treatment 18 patients post prandial sugar level were < 179 mg/ dl.

Urine sugar Fasting:

- From the selected 40 patients, before treatment 8 patients showed (+) and 32 patients showed Nil. After treatment 3 patients are reduced to (+) and 37 patients showed Nil Urine Sugar.

Urine sugar Post Prandial:

- From the selected 40 patients, before treatment 26 patients showed (+) and 8 patients showed (++). After treatment 35 patients postprandial urine sugar showed Nil.

HbA_1C:

- From the selected 40 cases all patients HbA1C level is in the range of 6.5-8 %. After treatment 30 patients had good control range of 5.7 - 7 %.

Investigations:

Investigations like TC, DC, ESR, Hb, Serum cholesterol and Blood urea, were examined and urine analysis for albumin, sugar and deposits were also examined. Lipid profile, C- peptide assay were examined.

SUVAI MUKKUTRAM THEORY:

Madhumegam is primarily due to derangement of Pitha kuttram. The trial medicine Perungaya Chooranam predominant with Thuvarppu suvai, it neutralizes the deranged Pitham.

BIO STATISTICAL ANALYSIS:

The p value is highly significant ($p<0.000$). So, there is significant reducing of fasting, post prandial blood sugar level (mg) and HbA1C level among the patients for the treatment of Madhumegam. Hence, it is concluded that treatment was effective and significant.

GRADING OF RESULTS:

Out of 40 patients, 75% of cases showed good result, 15% of the cases showed moderate result and 10 % showed minimal significance.

SUMMARY

SUMMARY

The clinical study on Madhumegam was carried out in the Post Graduate Department of Maruthuvam, Govt Siddha Medical College, Arignar Anna Hospital, Chennai-106, during the period of 2016 - 2018.

A total of 40 patients were treated in the Out Patient Department (OPD). The clinical and pathological assessment was carried out on the basis of both Siddha and Modern aspects.

All the 40 patients were treated with Perungaya Chooranam, 2g BD with lukewarm water for 90 days. The responses were assessed once in 7 days for all the patients.

- The peak incidence of Madhumegam was in the age group of 51- 60 yrs (45%) in both sexes.
- The prevalence was higher among the lower income (47.5%).
- The disease is more common in housewives (40%). So high incidence occurs in women.
- Regarding diet, the disease is seen among mixed dietary habits of about 80%.
- Regarding family history, 52.5% had no relavent history.
- Most of the patients were affected in Pitha kaalam (95%).
- In Vatham- Abaanan, Viyanan, Samanan, Kirukaran and Devathathan were affected in all the cases 100%, Koorman was affected in 7 cases (17.5%).
- In Pitham-Analagam and Sathagam 100%, Ranjagam 20%, Alosagam 17.5% and Prasagam 15% were affected.
- In Kabam- Kilethagam 100% and Santhigam 60% were affected.
- Among the Ezhu Udalthathukkal, Saaram, Seneer were 100% affected, Enbu (60%), Oon (15%), Kozhuppu (15%) were affected.
- Regarding Envagaithervugal - Naa, Naadi and Moothiram were 100% affected Niram, Malam (20%), Vizhi (17.5%), Sparisam (15%) were affected.
- Naadi – PithaVatha naadi (70%) was most commonly observed.
- In Neikuri examination, 65% Pithaneer were observed.
- The Toxicological study of Perungaya Chooranam revealed no toxicity.

- The pharmacological study shows Anti Diabetic Activity in Streptozotocin induced diabetic rats.
- Urine sugar Fasting and Postprandial became normal in 92.5% and 87.5% of patients respectively.
- Regarding Blood sugar level, fasting and post prandial blood sugar reduced in 90% and 82.5% of the cases respectively.
- HbA_1C level improved in 75% of cases which shows Good control in Madhumegam.
- The clinical trial shows that there is significant improvement in clinical manifestations of Madhumegam.
- The Biostatistical analysis of the clinical trial shows significant p-value <0.000 and hence the treatment was effective and significant.

CONCLUSION

CONCLUSION

i. Madhumegam is primarily due to derangement of Pitha kutram.

ii. The trial medicine Perungaya Chooranam predominating with Thuvarppu suvai, it neutralises the deranged Pitham by Ethirurai Maruthuvam.

iii. Perungaya Chooranam reveals no toxicity in animal models and hence proved to be safe in human subjects.

iv. From Preclinical Pharmacological studies, Perungaya Chooranam has Anti-Diabetic activity.

v. No adverse effect was reported during the clinical study.

vi. Perungaya Chooranam significantly reduced blood sugar level and also reduced clinical features of Madhumegam.

vii. Perungaya Chooranam is cost effective.

viii. Hence I conclude that Perungaya Chooranam will be a better Medicine that can be used in the treatment of Madhumegam.

ANNEXURES

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

69, Anna Salai, Guindy, Chennai - 600 032.

This Certificate is awarded to Dr/Mr/Mrs. K. NITHYA

For participating as ~~Resource Person~~ / Delegate in the Twentieth Workshop on

"RESEARCH METHODOLOGY & BIOSTATISTICS"

For AYUSH Post Graduates & Researchers

Organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University From 07th to 11th March 2016.

Dr.N.KABILAN, M.D.(S)
PROF & HEAD
DEPT.OF SIDDHA

Prof Dr.P.ARUMUGAM, M.D.,
REGISTRAR i/c

Prof. Dr.S.GEETHALAKSHMI, M.D., Ph.D.,
VICE CHANCELLOR

सिद्ध केंद्रीय अनुसन्धान संस्थान
(सी.सी.आर.एस., चेन्नई, आयुष मंत्रालय, भारत सरकार)
अण्णा सरकारी अस्पताल परिसर, अरुम्बाक्कम, चेन्नई - 600106
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Anna Govt. Hospital Campus, Arumbakkam, Chennai - 600106
E-mail: crisiddha@gmail.com Phone: 044-26214925, 26214809

02.06.2017

AUTHENTICATION CERTIFICATE FOR 17051828-34

Certified that the drugs submitted by Dr. K. Nithya, MD (S) II Year, Dept of Maruthuvam, Govt. Siddha Medical College, Arumbakkam, Chennai-106 are identified as:

SN	Botanical Name	Tamil Name	Part	Code
1.	*Ferula assa-foetida* L.	Perunkayam	Oleo-gum-resin	F17051828A
2.	*Cuminum cyminum* L.	Cirakam	Fruit	C17051829C
3.	*Acorus calamus* L.	Vacambu	Rhizome	A17051230C
4.	*Zingiber officinale* Roscoe.	Cukku	Rhizome	Z17051831O
5.	*Terminalia chebula* Retz.	Kattukai	Fruit	T17051832C
6.	*Plumbago zeylanica* L.	Chithiramoolam	Root	P17051833Z
7.	*Saussurea costus* (Falc.) Lipsch.	Kottam	Root	S17051834C

Continued in next page....

F17051828A

C17051829C

A17051230C

Z17051831O

T17051832C

P17051833Z

S17051834C

Dr. K.N. Sunil Kumar
Research Officer and HOD
Department of Pharmacognosy

Dr. M. Kannan
Research Officer (Siddha)
and In Charge

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், சென்னை - 600 106
सिद्ध केंद्रीय अनुसन्धान संस्थान,
अण्णा सरकारी अस्पताल परिसर, अरुम्बाक्कम, चेन्नई – 600 106
SIDDHA CENTRAL RESEARCH INSTITUTE
(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)
Anna Govt. Hospital Campus, Arumbakkam, Chennai – 600106
Phone: 044-2621 4925, Fax: 044-2621 4809

23.5.2017

CERTIFICATE

Certified that the sample submitted by Dr. K. Nithya, II year MD Student, Department of Maruthuvam, Government of Siddha Medical College, Chennai-600106 is identified as Indhuppu – Sodium Chloride.

(R. Shakila)
Research Officer (Chemistry) & Head,
Department of Chemistry

(Dr. P. Sathiyarajeswaran)
Assistant Director (Siddha) I/c

C.L.BAID METHA COLLEGE OF PHARMACY

(An ISO 9001-2000 certified institute)

Jyothi Nagar, Old Mahabalipuram Road

Thoraipakkam, Chennai – 600 097

CERTIFICATE

This is to certify that the project entitled, Pharmacological and Toxicological screeniung of Perungaya chooranam submitted in partial fulfilment for the degree of M.D. (siddha) was carried out at C.L.Baid Metha college of Pharmacy, Chennai-97, in the Department of Pharmacology during the academic year of 2017-2018. It has been approved by the IAEC No: LI/10/CLBMCP/2017

C.L. Baid Metha College of Pharmacy CHENNAI-97 Thoraipakkam

Dr.P. MURALIDHARAN

IAEC MEMBER SECERATARY

ACUTE ORAL TOXICITY STUDY OF *PERUNGAYA CHOORANAM* (OECD GUIDELINE – 423)

Introduction:

- The acute toxic class method is a stepwise procedure with the use of 3 animals of a single sex per step.
- Depending on the mortality and/or the moribund status of the animals, on average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance.
- This procedure is reproducible, uses very few animals and is able to rank substances in a similar manner to the other acute toxicity testing methods.
- The acute toxic class method is based on biometric evaluations with fixed doses, adequately separated to enable a substance to be ranked for classification purposes and hazard assessment.
- In principle, the method is not intended to allow the calculation of a precise LD50, but does allow for the determination of defined exposure ranges where lethality is expected since death of a proportion of the animals is still the major endpoint of this test.
- The method allows for the determination of an LD50 value only when at least two doses result in mortality higher than 0% and lower than 100%.
- The use of a selection of pre-defined doses, regardless of test substance, with classification explicitly tied to number of animals observed in different states improves the opportunity for laboratory to laboratory reporting consistency and repeatability.

Principle of the Test:

It is the principle of the test that based on a stepwise procedure with the use of a minimum number of animals per step, sufficient information is obtained on the acute toxicity of the test substance to enable its classification. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex. Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.

− no further testing is needed.

− dosing of three additional animals, with the same dose.

− dosing of three additional animals at the next higher or the next lower dose level. The method will enable a judgment with respect to classifying the test substance to one of a series of toxicity classes.

Methodology:

Selection of Animal Species

The preferred rodent species is the Wistar albino rat, although other rodent species may be used. Healthy young adult animals are commonly used laboratory strains should be employed. Females should be nulliparous and non-pregnant. Each animal, at the commencement of its dosing, should be between 6 to 8 weeks old and the weight (150-200gm) should fall in an interval within±20 % of the mean weight of any previously dosed animals.

Housing and Feeding Conditions

The temperature in the experimental animal room should be 22ºC ± 3ºC. Although the relative humidity should be at least 30% and preferably not exceed 70% other than during room cleaning the aim should be 50-60%. Lighting should be artificial, the sequence being 12 hours light, 12 hours dark. For feeding, conventional laboratory diets may be used with an unlimited supply of drinking water. Animals may be group-caged by dose, but the number of animals per cage must not interfere with clear observations of each animal.

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 7 days prior to dosing to allow for acclimatization to the laboratory conditions

Test Animals and Test Conditions:

Sexually mature Female Wistar albino rats (150-200gm) were obtained fromKings institute,Guindy, Chennai.All the animals were kept under standard environmental condition (22±3°C). The animals had free access to water and standard pellet diet (Saimeera foods, Bangalore).

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 7 days prior to dosing to allow for acclimatization to the laboratory conditions

Preparation for Acute Toxicity Studies

Rats were deprived of food overnight (but not water 16-18 h) prior to administration of the, ***PERUNGAYA CHOORANAM***.

The principles of laboratory animal care were followed and the Institutional Animal Ethical Committee approved the use of the animals and the study design.

IAEC No: LI/10/CLBMCP/2017

Test Substance	: **PERUNGAYA CHOORNAM**
Animal Source	: Kings institute,Guindy, Chennai.
Animals	: Wister Albino Rats (Female-3+3)
Age	**:** 6-8 weeks
Body Weight on Day 0	**:**150-200gm.
Acclimatization	**:** Seven days prior to dosing.
Veterinary examination	**:** Prior and at the end of the acclimatization period.
Identification of animals	**:** By cage number, animal number and individual marking by using Picric acid.
Numberofanimals	: 3 Female/group,
Routeofadministration	: Oral
Diet	**:** Pellet feed supplied by Saimeera foods Pvt Ltd,Bangalore
Water	**:** Aqua guard portable water in polypropylene bottles.
Housing & Environment	**:** The animals were housed in Polypropylene cages provided with bedding of husk.
Housing temperature	**:** between 22ºC $\pm$3ºC.
Relative humidity	**:** between 30% and 70%,
Air changes	**:** 10 to 15 per hour and

Dark and light cycle **:** 12**:**12 hours.

Duration of the study **:** 14 Days

Administration of Doses:

PERUNGAYA CHOORANAM was suspended in water and administered to the groups of wistar albino rats in a single oral dose by gavage using a feeding needle. The control group received an equal volume of the vehicle. Animals were fasted 12 hours prior to dosing. Following the period of fasting, the animals were weighed and then the test substance was administered. Three Female animals are used for each group. The dose level of 5, 50, 300and 2000 mg/kg body weight was administered stepwise. After the substance has been administered, food was withheld for a further 3-4 hours. The principle of laboratory animal care was followed. Observations were made and recorded systematically and continuously as per the guideline after substance administration. The visual observations included skin changes, mobility, aggressively, sensitivity to sound and pain, as well as respiratory movements. Finally, the number of survivors was noted after 24 hrs and these animals were then monitered for a further 14 days and observations made daily. The toxicological effect was assessed on the basis of mortality.

Observations:

Animals are observed individually after dosing at least once during the first 30 minutes, periodically during the first 24 hours, with special attention given during the first 4 hours, and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead. It should be determined by the toxic reactions, time of onset and length of recovery period, and may thus be extended when considered necessary. The times at which signs of toxicity appear and disappear are important, especially if there is a tendency for toxic signs to be delayed. All observations are systematically recorded with individual records being maintained for each animal.

Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somato motor activity and behavior pattern. Attention was directed to observations of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma. The principles and criteria summarized in the Humane Endpoints Guidance Document taken into consideration. Animals found in a moribund condition and animals showing severe

pain or enduring signs of severe distress was humanly killed. When animals are killed for human reasons or found dead, the time of death was recorded.

Acute oral toxicity study of PERUNGAYA CHOORANAM

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

Observation done:

SL	Group CONTROL	Observation	SL	Group TEST GROUP	Observation
1	Body weight	Normal	1	Body weight	Normally increased
2	Assessments of posture	Normal	2	Assessments of posture	Normal
3	Signs of Convulsion Limb paralysis	Normal	3	Signs of Convulsion Limb paralysis	Absence of sign (-)
4	Body tone	Normal	4	Body tone	Normal
5	Lacrimation	Normal	5	Lacrimation	Absence
6	Salivation	Normal	6	Salivation	Absence
7	Change in skin color	No significant color change	7	Change in skin color	No significant color change
8	Piloerection	Normal	8	Piloerection	Normal
9	Defecation	Normal	9	Defecation	Normal
10	Sensitivity response	Normal	10	Sensitivity response	Normal
11	Locomotion	Normal	11	Locomotion	Normal
12	Muscle gripness	Normal	12	Muscle gripness	Normal

13	Rearing	Mild	13	Rearing	Mild
14	Urination	Normal	14	Urination	Normal

Behaviour:

The animals will be observed closely for behaviour in the first four hours which includes abnormal gait, aggressiveness, exophthalmos, ptosis, akinesia, catalepsy, convolusion, excitation, head twitches, lacrimation, loss of corneal reflex, loss of traction, piloerection reactivity of touch, salivation, scratching, sedation, chewing, head movements, sniffing, straub, tremor and writhes, diarrhea, leathery, sleep and coma.

Body Weight:

Individual weight of animals was determined before the test substance was administered and weights will be recorded at day 1, 7, and 14 of the study. Weight changes were calculated and recorded. At the end of the test, surviving animals were weighed and humanly killed.

Food and water Consumption:

Food and water consumed per animal was calculated for control and the treated dose groups.

Mortality:

Animals were observed for mortality throughout the entire period.

Results:

All data were summarized in tabular form, (Table-1-4) showing for each test group the number of animals used, the number of animals displaying signs of toxicity, the number of animals found dead during the test,description of toxic symptoms, weight changes, food and water intake

No of animals in each group:3

Table 2 (Observational study Results)

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	Control	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2.	2000mg	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-

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

Table 3(Body weight Observation)

DOSE	DAYS		
	1	7	14
CONTROL	320.2±42.30	322.4 ± 60.10	323.6 ±52.10
HIGH DOSE	302.4± 1.21	302 ± 2.04	304.2 ± 2.10
P value (p)*	NS	NS	NS

Table 3Water intake (ml/day) of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM:*

DOSE	DAYS		
	1	6	14
CONTROL	58 ± 1.02	58±9.20	59.4±1.04
HIGH DOSE	59.4±2.20	59.8±3.40	59.9±6.24
P value (p)*	NS	NS	NS

N.S- Not Significant,**(p > 0.01), *(p >0.05), n = 10 values are mean ± S.D (One-wayANOVA followed by Dunnett's test)

Table 4:Food intake (gm/day) of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM*

DOSE	DAYS		
	1	7	14
CONTROL	61.04±2.62	62.2±4.76	64.3±6.26
High DOSE	69.4±4.23	70.4±6.22	71.6±4.18

REPEATED DOSE 28-DAY ORAL TOXICITY (407) STUDY OF *PERUNGAYA CHOORANAM*

Test Substance : **PERUNGAYA CHOORANAM**
Animal Source : Kings institute,Guindy, Chennai
Animals : Wister Albino Rats (Male -24, and Female-24)
Age **:** 6-8 weeks
Body Weight **:**150-200gm.
Acclimatization **:** Seven days prior to dose.
Veterinary examination **:** Prior and at the end of the acclimatization period.
Identification of animals **:**By cage number, animal number and individual marking by using Picric acid
Diet **:** Pellet feed supplied by Sai Meera Foods Pvt Ltd, Bangalore
Water **:** Aqua guard portable water in polypropylene bottles.
Housing & Environment **:** The animals were housed in Polypropylene cages provided with bedding of husk.
Housing temperature **:** between 22ºC±3ºC.
Relative humidity **:** between 30% and 70%,
Air changes **:** 10 to 15 per hour
Dark and light cycle **:** 12**:**12 hours.
Duration of the study **: 28 Days.**

Table 5

Groups	No of Rats
Group I Vehicle control (Water)	12(6male,6 female)
Group II low dose X (20mg)	12 (6male,6 female)
Group III Mid dose 10X (200mg)	12 (6male,6female)
Group IV High dose 20X(400mg)	12(6male,6female)

PERUNGAYA CHOORANAM

Methodology

Randomization, Numbering and Grouping of Animals:

48 Wistar Albino Rats (24M + 24F) were selected and divided into 4 groups. Each group consist of 12 animals (Male -6, and Female-6). First group treated as a control and other three group were treated with test drug (low, mid, high) for 28 days. Animals were allowed acclimatization period of 7 days to laboratory conditions prior to the initiation of treatment. Each animal was marked with picric acid. The females were nulliparous and non-pregnant.

Justification for Dose Selection:

As per OECD guideline three dose levels were selected for the study. They are low dose (X), mid dose (10X), high dose (20X). X is calculated by multiplying the acute toxicity dose (2000mg) i.e X dose is (20mg/kg), 10X dose is (200mg/kg), 20X dose is (400mg/kg).

Preparation and Administration of Dose:

PERUNGAYA CHOORANAM suspended in with water, It was administered to animals at the dose levels of X, 10X, 20X. The test substance suspensions were freshly prepared every two days once for 28 days. The control animals were administered vehicle only. The drug was administered orally by using oral gavage once daily for 28 consecutive days.

Observations:

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

Body Weight:

Weight of each rat was recorded on day 0, at weekly intervals throughout the course of study.

Food and water Consumption:

Food and water consumed per animal was calculated for control and the treated dose groups.

Clinical signs:

All animals were observed daily for clinical signs. The time of onset, intensity and duration of these symptoms, if any, were recorded.

Mortality:

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

Necropsy:

All the animals were sacrificed by excessive anesthesia on day 29. Necropsy of all animals was carried out.

Laboratory Investigations:

Following laboratory investigations were carried out on day 29 in animals fasted over-night. Blood samples were collected from orbital sinus using sodium heparin (200IU/ml) for Bio chemistry and potassium EDTA (1.5 mg/ml) for Hematology as anticoagulant. Blood samples were centrifuged at 3000 r.p.m. for 10 minutes.

Haematological Investigations:

Haematological parameters were determined using Haematologyanalyzer.

Biochemical Investigations:

Biochemical parameters were determined using auto-analyzer.

Histopathology:

Control and highest dose group animals will be initially subjected to histo pathological investigations. If any abnormality found in the highest dose group than the low, then the mid dose group will also be examined. Organs will be collected from all animals and preserved in 10% buffered neutral formalin for 24 h and washed in

running water for 24 h. The organ sliced 5 or 6µm sections and were dehydrated in an auto technicon and then cleared in benzene to remove absolute alcohol. Embedding was done by passing the cleared samples through three cups containing molten paraffin at 50°C and then in a cubical block of paraffin made by the "L" moulds. It was followed by microtome and the slides were stained with Haematoxylin-eosin red.

Statistical analysis:

Findings such as body weight changes, water and food consumption,hematology and blood chemistry were subjected to One-way ANOVA followed by dunnett test using a computer software programme – Graph pad version7. All data were summarized in tabular form, (Table-6 to 12)

RESULTS:

Repeated Dose 28-day oral toxic study of PERUNGAYA CHOORANAM

Table 6: Body weight of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM*

DOSE	DAYS				
	1	7	14	21	28
CONTROL	235.2±18.46	236.5 ± 35.10	236.6 ± 45.60	238.7± 56.16	238.4 ± 66.15
LOW DOSE	248.2 ± 65.24	250.7 ± 66.28	254.6± 55.34	256 ±56.34	256.8± 35.36
MID DOSE	252.4± 18.34	253.3 ± 16.24	253.4± 14.12	255.2 ± 15.20	256.4 ± 54.10
HIGH DOSE	261.6± 62.24	261.4±42.22	262.4 ± 52.24	263 ± 54.28	264 ± 74.60
P value (p)*	NS	NS	NS	NS	NS

NS- Not Significant, **(p > 0.01),*(p >0.05), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test)

Table 7: Water intake (ml/day) of Wistar albino rats group exposed to *PERUNGAYA CHOORNAM*

DOSE	DAYS				
	1	6	14	21	28
CONTROL	60.1 ± 8.72	60±1.52	60.2±1.40	61±1.32	61.4±1.62
LOW DOSE	65.1±1.21	65.6±4.22	66.6±1.02	65.2±2.06	66.4±1.20
MID DOSE	62.1±1.02	62.3±1.21	62.1±2.62	63.4±4.32	63.4±1.64
HIGH DOSE	64.1±1.81	64.2±1.32	64.4±1.14	64.6±1.62	65.8±2.02
P value (p)*	NS	NS	NS	NS	NS

N.S- Not Significant, **(p > 0.01), *(p >0.05), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test)

Table 8:Food intake (gm/day) of Wistar albino rats group exposed to *PERUNGAYA CHOORNAM*

DOSE	DAYS				
	2	7	23	22	28
CONTROL	34±4.14	34.2±6.12	34.3±2.18	34.2±1.14	34±5.62
LOW DOSE	36.3±1.64	36.3±1.51	36.2±1.51	36.5±1.62	36.5±1.22
MID DOSE	34.1±2.12	34.2±3.50	34.2±2.14	34.2±2.16	35.2±1.64
HIGH DOSE	32.4±1.62	32.1±1.64	32.6±2.36	32.6±1.20	36.4±2.32
P value (p)*	NS	NS	NS	NS	NS

N.S- Not Significant, **(p > 0.01), *(p >0.05), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett'stest.

Table 9: Haematological parameters of Wistaralbino rats group exposed to *PERUNGAYA CHOORNAM*

Category	Control	Low dose	Mid dose	High dose	P value (p)*
Haemoglobin(g/ dl)	13.4±0.71	13.30±0.14	13.4±0.13	13.72±0.13	N.S
Total WBC ($\times 10^3$ l)	09.41±0.22	09.32±0.22	09.34±0.22	09.30±1.10	N.S
Neutrophils (%)	21.13±0.60	21.02±0.52	22.11±1.42	22.02±2.71	N.S
lymphocyte (%)	82.10±1.26	82.12±1.42	83.10±2.44	83.20±2.54	N.S
Monocyte (%)	1.1±0.03	1.1±0.01	1.2±0.04	1.1±0.03	N.S
Eosinophil (%)	0.8±0.03	0.8±0.04	0.9±0.05	0.9±0.08	N.S
Platelets cells10^3/µl	900.17±3.18	902.11±4.62	902.11±2.20	902.22±2.64	N.S
Total RBC 10^6/µl	9.32±0.11	9.47±0.33	9.50±0.64	9.60±0.46	N.S
PCV%	48.10±0.2	48.62±5.30	48.8±4.70	48.4±.71	N.S
MCHC g/dL	36.5±1.61	36.2±1.51	36.8±1.30	36.13±1.60	N.S
MCV fL(µm^3)	58.2±2.02	58.2±1.80	58.7±1.10	59.7±1.30	N.S

N.S- Not Significant, **($p > 0.01$), *($p > 0.05$), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test)

Table 10 :Bio chemical Parameters of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM*

BIOCHEMICAL PARAMETERS	CONTROL	LOW DOSE	MID DOSE	HIGH DOSE	P Value (p)*
GLUCOSE (R) (mg/dl)	138.10±2.02	138.12±2.10	138.9±12.06	138.12±5.25	N.S
T.CHOLESTEROL(mg/dl)	140.14±5.10	140.15±5.20	142.40±1.68	143.21±1.10	N.S
TRIGLY(mg/dl)	74.15±1.82	74.11±1.32	74.15±1.22	76.16±1.21	N.S
LDL	78.6±2.13	78.7±2.05	78.10±1.03	78.40±01.32	NS
VLDL	14.2±1.52	14.20±2.41	14.02±1.32	14.04±12.15	NS
HDL	28.12±4.32	28.32±2.50	28.46±1.20	28.51±1.23	NS
Ratio 1(T.CHO/HDL)	3.73±1.16	3.72±1.80	3.73±1.32	3.74±2.33	NS
Ratio 2(LDL/HDL)	1.92±1.22	1.92±1.20	1.93±2.20	1.94±06.02	NS
Albumin(g/dL)	6.21±0.22	6.22±0.52	6.4±7.20	6.55±6.48	NS

NS- Not Significant,**(p > 0.01), * (p >0.05), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test)

Table 11: Renal function test of of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM*

PARAMETERS	CONTROL	LOW DOSE	MID DOSE	HIGH DOSE	P Value (p)*
UREA (mg/dl)	14.50±0.29	14.50±0.29	14.46±1.18	14.42±1.22	N.S
CREATININE (mg/dl)	0.42±0.02	0.41±0.04	0.43±0.03	0.44±0.09	N.S
BUN(mg/dL)	19.1±0.02	19.10±0.34	19.6±0.42	19.26±1.02	NS
URIC ACID(mg/dl)	4.02±0.04	4.06±0.21	4.4±0.12	4.20±0.10	N.S

NS- Not Significant, **(p > 0.01), * (p >0.05) , n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test)

Table 12: Liver Function Test of of Wistar albino rats group exposed to *PERUNGAYA CHOORANAM*

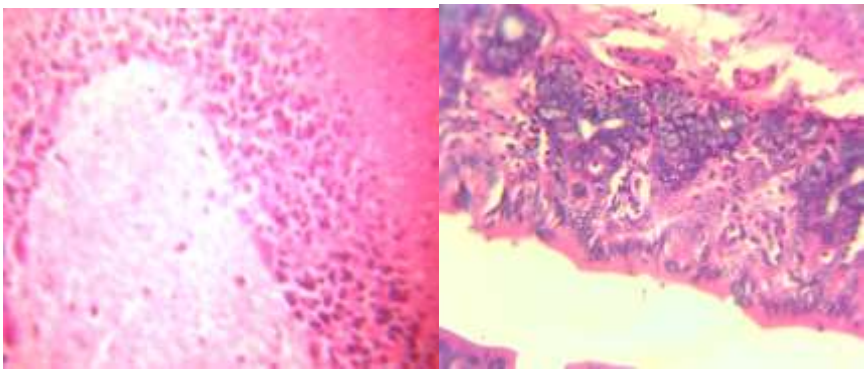
PARAMETERS	CONTROL	LOW DOSE	MID DOSE	HIGH DOSE	P Value (p)*
T BILIRUBIN(mg/dl).	0.08±0.01	0.08±0.03	0.08±0.03	0.08±0.01	N.S
SGOT/AST(U/L)	64.11±1.53	64.12±0.22	64.24±1.54	65.74±1.53	N.S
SGPT/ALT(U/L)	79.21±1.02	79.34±1.04	79.44±1.16	79.38±0.21	N.S
ALP(U/L)	137.11±2.21	137±2.20	139±1.24	140.03±6.02	N.S
T.PROTEIN(g/dL)	7.2.40±0.14	7.2±0.41	7.2±0.60	7.3±0.61	N.S

NS- Not Significant, **(p > 0.01), * (p >0.05), n = 10 values are mean ± S.D (One way ANOVA followed by Dunnett's test.

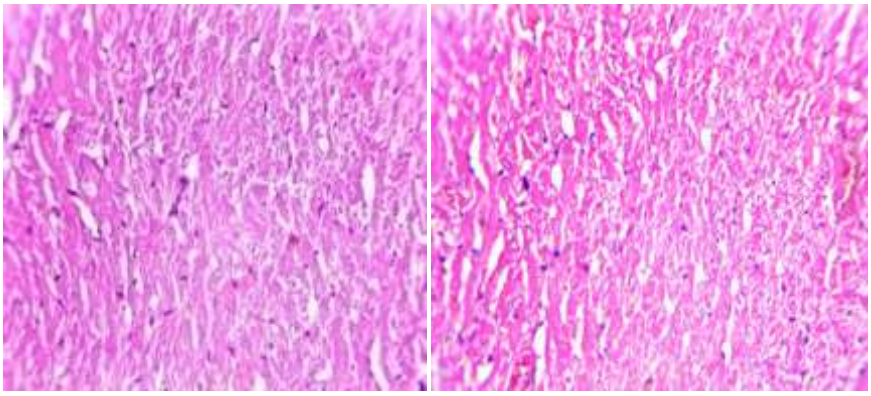
HISTOPATHOLOGY OF VITAL ORGANS:

Low dose **High dose**

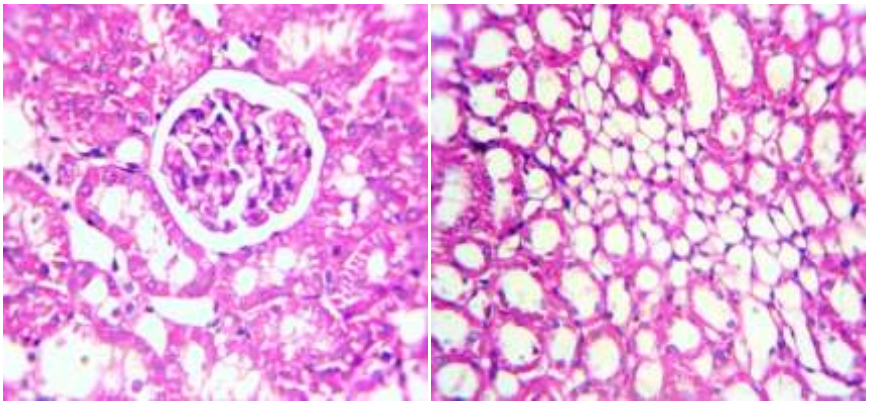
BRAIN



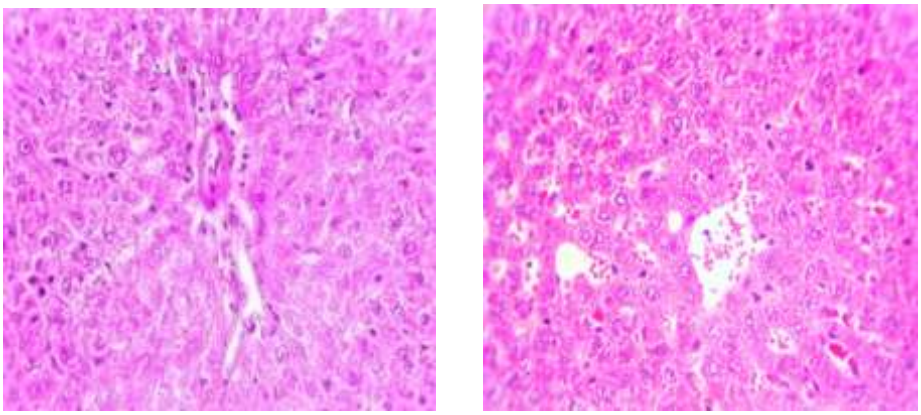
HEART



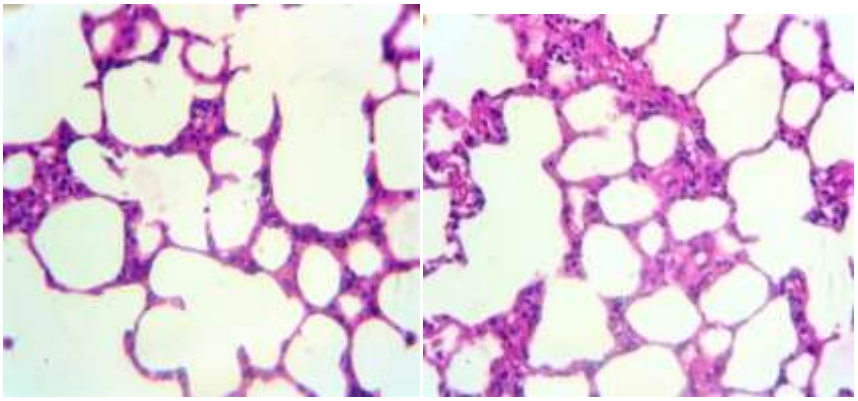
KIDNEY



LIVER



LUNG



BRAIN

Regular marginal alignment on the neurons was observed. No signs of oedema or degeneration were observed.

HEART

No evidence of pyknotic nucleus was observed.

LUNG

No signs of airway secretion and bronchial secretion. Bronchial blood vessels and connective tissue appears normal with no signs of pulmonary oedema.

LIVER

Periportal zone appears normal. No evidence of phagocytosis in intra cytoplasmic region were observed.

KIDNEY

Appearance of proximal and distal convolutes tubules was normal with no evidence of atrophy. Lumen of distal convolutes tubule and collecting duct was normal.

AN EVALUATION OF THE SIDDHA DRUG PERUNGAYA CHOORANAM FOR ITS ANTI-DIABETIC ACTIVITY IN WISTAR ALBINO RATS

ANIMAL PROCUREMENT AND MAINTENANCE:

Wistar Albino rats of either sex, weighing 150g to 200g were purchased from Kings Institute of Preventive Medicine Animal House, Chennai, India. Animal ethical guidelines of CPCSEA, Ministry of Animal Husbandry and Welfare, Govt. of India were strictly followed for the care and maintenance of procured animals. The animals were fed on standard rodent pellet and RO water was provided *ad libitum*. The animals were kept for overnight fasting before experimentation.

Diabetes was induced in male Wistar Albino rats aged 2–3 months (180–200 g body weight) by intraperitoneal administration of STZ (single dose of 55 mg/kg b.w) dissolved in freshly prepared 0.01 M citrate buffer, pH 4.5.

After injection the animals had food and water *ad libitum* and were given 5% glucose in their drinking water for the first 24 hours to counter any initial hypoglycemia. The development of diabetes was confirmed after 72 hours of the Streptozotocin injection. After 72 hrs of STZ injection under mild anesthesia the blood was withdrawn from the tip of the tail of each rat and the blood glucose level was analyzed. Animals with more than 250 mg/dl was considered as diabetic.

The animals were divided into 5 groups each constituting 6 rats. Group I were normal rats, Group II were STZ (55 mg/kg b.w., i.p) induced diabetic rats. Group III STZ (55 mg/kg b.w., i.p) induced diabetic rats were treated with Glibenclamide 5mg/kg b.w/p.o Group IV STZ (55 mg/kg b.w., i.p) induced diabetic rats were treated with *Perungaya Chooranam* 200mg/kg b.w/ p.o Group V STZ (55 mg/kg b.w., i.p) induced diabetic rats were treated with *Perungaya Chooranam* 400mg/kg b w/p.o for 28 days.

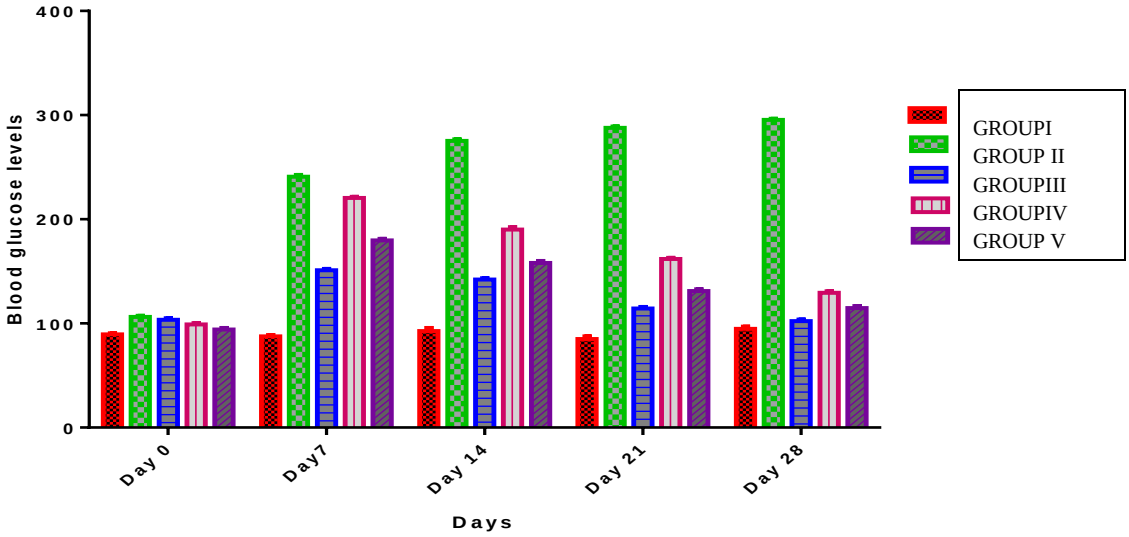
Fasting blood glucose levels was measured before the administration of extracts. The blood glucose levels were checked on 0th, 7th, 14th, and 21st day of the treatment period. Blood was collected from snipping of the rat tail. Blood glucose levels were measured.

Groups	Treatment
Group I	Normal Control
Group II	Diabetic control- STZ (55 mg/kg)
Group III	Diabetic control- Glibenclamide (5 mg/kg)
Group IV	Diabetic control- *Perungaya Chooranam* 200mg/kg
Group V	Diabetic control- *Perungaya Chooranam* 400mg/kg

Effect of *Perungaya Chooranam* in blood glucose level:

Group	Blood glucose (mg/dl)				
	Day – 0	Day – 7	Day – 14	Day – 21	Day – 28
I	90.40±1.13	88.23±1.328	92.45±2.78	87.13±2.43	93.60±2.18
II	104.22±1.14	244.9±1.66	276.3±1.46	290.8±1.33	295.4±1.22
III	103.5±1.46	152.0±1.32	142.05±1.30	116.3±1.48	104.3±1.46
IV	99.13±1.22	221.5±1.24	191.2±2.27	161.8±1.26	128.5±1.44
V	94.23±1.38	179.8±1.34	158.04±1.98	131.03±1.76	114.8±1.84

Effect of PERUNGAYA CHOORANAM in blood glucose level



CONCLUSION:

By the observed result, the values of trial drug Perungaya chooranam treated with animals were compared with the positive control drug STZ 55mg/kg b.w/i.p, single dose. The results (mean value) are assured as a Anti- Diabetic activity of trial drug,

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PHYSIOCHEMICAL ANALYSIS OF –PERUNGAYA CHOORANAM

1. **Loss On Drying:**

An accurately weighed 2g of ***Perungaya Chooranam*** formulation was taken in a tarred glass bottle. The crude drug was heated at 105^{0}C for 6 hours in an oven till a constant weight. The Percentage moisture content of the sample was calculated with reference to the shade dried material.

2. **Determination of total ash:**

Weighed accurately 2g of ***Perungaya Chooranam*** formulation was added in crucible at a temperature 600^{0}C in a muffle furnace till carbon free ash was obtained. It was calculated with reference to the air dried drug.

3. **Determination of acid insoluble ash:**

Ash above obtained, was boiled for 5min with 25ml of 1M Hydrochloric acid and filtered using an ash less filter paper. Insoluble matter retained on filter paper was washed with hot water and filter paper was burnt to a constant weight in a muffle furnace. The percentage of acid insoluble as was calculated with reference to the air dried drug.

4. **Determination of water soluble ash:**

Total ash 1g was boiled for 5min with 25ml water and insoluble matter collected on an ash less filter paper was washed with hot water and ignited for 15 min at a temperature not exceeding 450^{0}C in a muffle furnace. The amount of soluble ash is determined by drying the filtrate.

5. **Determination of water soluble Extractive:**

5gm of air dried drug, coarsely powered ***Perungaya Chooranam*** was macerated with 100ml of distilled water in a closed flask for twenty-four hours, shaking frequently.The Solution was filtered and 25 ml of filtrated was evaporated in a tarred flat bottom shallow dish, further dried at 100^0 C and weighted. The percentage of water soluble extractive was calculated with reference to the air dried drugs.

6. **Determination of alcohol soluble extractive:**

2.5gm. of air dried drugs, coarsely powdered ***Perungaya Chooranam*** was macerated with 50 ml. alcohol in closed flask for 24 hrs. With frequent shaking, it was filtered rapidly taking precaution against loss of alcohol. 10ml of filtrate was then evaporated in a tarred flat bottom shallow dish, dried at 100^0C and weighted. The percentage of alcohol soluble extractive was calculated with reference to air dried drug.

S.no	Parameters	Percentage
1	Loss on drying	3.68%
2	Total ash value	38.37%
3	Acid insoluble ash	Less than 1%
4	Water soluble ash	33.88%
5	Water soluble extraction	47.53%
6	Alcohol soluble extraction	13.92%

The above stated physiochemical properties of the given sample certified to be present.

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PRELIMINARY PHYTOCHEMICAL SCREENING - PERUNGAYA CHOORANAM

The preliminary phytochemical screening test was carried out for each extracts of ***Perungaya Chooranam*** as per the standard procedure.

1. Detection of alkaloids:

Extracts were dissolved individually in dilute Hydrochloric acid and filtered.

a) Mayer's Test: Filtrates were treated with Mayer's reagent (Potassium Mercuric Iodide). Formation of a yellow colored precipitate indicates the presence of alkaloids.

b) Wagner's Test: Filtrates were treated with Wagner's reagent (Iodine in Potassium Iodide). Formation of brown/reddish precipitate indicates the presence of alkaloids.

c) Dragendroff's Test: Filtrates were treated with Dragendroff's reagent (solution of Potassium Bismuth Iodide). Formation of red precipitate indicates the presence of alkaloids.

d) Hager's Test: Filtrates were treated with Hager's reagent (saturated picric acid solution). Presence of alkaloids confirmed by the formation of yellow colored precipitate.

2. Detection of carbohydrates:

Extracts were dissolved individually in 5 ml distilled water and filtered. The filtrates were used to test for the presence of carbohydrates.

a) Molisch's Test:

To 2 ml of plant sample extract, two drops of alcoholic solution of α- naphthol are added. The mixture is shaken well and few drops of concentrated sulphuric acid is added slowly along the sides of test tube. A violet ring indicates the presence of carbohydrates.

b) Benedict's Test:

Filtrates were treated with Benedict's reagent and heated gently. Orange red precipitate indicates the presence of reducing sugars.

3. Detection of glycosides:

Extracts were hydrolyzed with dil. HCl, and then subjected to test for glycosides.

a) Modified Borntrager's Test: Extracts were treated with Ferric Chloride solution and immersed in boiling water for about 5 minutes. The mixture was cooled and extracted with equal volumes of benzene. The benzene layer was separated and treated with ammonia solution. Formation of rose-pink color in the ammonical layer indicates the presence of anthranol glycosides.

b) Cardiac glycoside (Keller-Killiani test): Extract was shaken with distilled water (5 mL). To this, glacial acetic acid (2 mL) containing a few drops of ferric chloride was added, followed byH2SO4 (1 mL) along the side of the test tube. The formation of brown ring at the interface gives positive indication for cardiac glycoside and a violet ring may appear below the brown ring

4. Detection of saponins

a) Froth Test: Extracts were diluted with distilled water to 20ml and this was shaken in a graduated cylinder for 15 minutes. Formation of 1 cm layer of foam indicates the presence of saponins.

b) Foam Test: 0.5 gm of extract was shaken with 2 ml of water. If foam produced persists for ten minutes it indicates the presence of saponins.

5. Detection of phytosterols

a) Salkowski's Test: Extracts were treated with chloroform and filtered. The filtrates were treated with few drops of Conc. Sulphuric acid, shaken and allowed to stand. Appearance of golden yellow color indicates the presence of triterpenes.

6. Detection of phenols Ferric Chloride Test:

Extracts were treated with 3-4 drops of ferric chloride solution. Formation of bluish black color indicates the presence of phenols.

7. Detection of tannins Gelatin Test:

The extract is dissolved in 5 ml of distilled water and 2 ml of 1% solution of Gelatin containing 10% NaCl is added to it. White precipitate indicates the presence of phenolic compounds.

8. Detection of Flavonoids

a) Alkaline Reagent Test: Extracts were treated with few drops of sodium hydroxide solution. Formation of intense yellow color, which becomes colorless on addition of dilute acid, indicates the presence of flavonoids.

b) Lead acetate Test: Extracts were treated with few drops of lead acetate solution. Formation of yellow color precipitate indicates the presence of flavonoids.

9. Detection of proteins and aminoacids

a) Xanthoproteic Test: The extracts were treated with few drops of conc. Nitric acid. Formation of yellow color indicates the presence of proteins.

b) Ninhydrin Test: To the extract, 0.25% w/v ninhydrin reagent was added and boiled for few minutes. Formation of blue color indicates the presence of amino acid.

10. Detection of diterpenes Copper Acetate Test:

Extracts were dissolved in water and treated with 3-4 drops of copper acetate solution. Formation of emerald green color indicates the presence of diterpenes

11. Gum and Mucilage:

To 1ml of extract add 2.5ml of absolute alcohol and stirring constantly. Then the precipitate was dried in air and examine for its swelling properties. Swelling was observed that will indicate presence of gum and mucilage.

12. Test for Fixed oils and Fats

a. Spot test : A small quantity of extract is pressed between two filter papers. Oil stain on the paper indicates the presence of fixed oils.

13. Test for Quinones

Extract was treated with sodium hydroxide blue or red precipitate indicates the presence of Quinones.

The Preliminary phytochemical studies of aqueous extract of ***Perungaya Chooranam*** were done using standard procedures. The results were presented in tables. The present study reveals that the bioactive compounds were present in all the extracts of ***Perungaya Chooranam***.

S.no	Phytochemicals	Test Name	H2O Extract
1.	Alkaloids	Mayer's Test	-ve
		Wagner's Test	-ve
		Dragendroff's Test	-ve
		Hager's Test	-ve
2.	Carbohydrates	Molisch's Test:	+ve

		Benedict's Test	+ve
3.	Glycoside	Modified Borntrager's Test	-ve
		Keller Killiani	-ve
4.	Saponin	Froth Test	+ve
		Foam Test	-ve
5.	Phytosterol	Salkowski's Test	-ve
6.	Phenols	Ferric Chloride Test	+ve
7.	Tannins	Gelatin Test	-ve
8.	Flavonoids	Alkaline Reagent Test	+ve
		Lead acetate Test	+ve
9.	Proteins and amino acids	Xanthoproteic Test	+ve
10.	Diterpenes	Copper Acetate Test	+ve
11.	Gum & Mucilage	Extract + Alcohol	-ve
12.	Fat & Fixed Oil	Spot Test	-ve
13.	Quinones	NAOH + Extract	+ve

+ve/-ve present or absent if component tested

The above stated phytochemical properties of the given sample certified to be present.

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BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug 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	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b	2ml of extract is added with 2mlof dilute hydrochloric acid until the effervescence ceases off. Then2ml 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 nitrates solution is added.	Presence of white precipitate obtained	Presence of Chloride.
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 obtained	Presence of Phosphate.
4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate	Absence of white precipitate	Absent
5	**Test for Sulphide:** 1 gm of the substance is treated	Rotten egg smelling	Absent

	with 2ml of concentrated Hydrochloric acid.		
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
7a	**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.	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.	KMNO4 solution Decolourization obtained	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 nonluminous part of the flame.	Absence of Bluish green coloured flame.	Absent
b	2ml of the extract is added with excess of Ammonia solution.	Presence of deep blue	Presence of Copper.
12	**Test for Aluminium** To the 2ml 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 HNO_3 is added.	Blood red colour obtained.	Presence of Iron.
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Presence of White precipitate.	Presence of Zinc.
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 Benedicts qualitative solution is taken in a test tube and	Absence of Green colour	Absent

	allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.		
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium Iodide solution.	Absence of Red colour	Absent
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH, mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

The given sample (Perungaya Chooranam) contains,

1. Chloride
2. Phosphate
3. Copper
4. Iron
5. Zinc.

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethics Committee (IEC)

IEC No: GSMC-CH-ME-5/007/2016

Protocol title: AN OPEN NON-RANDOMIZED CLINICAL TRIAL OF PERUNGAYA CHOORANAM IN MADHUMEGAM (DIABETES MELLITUS - TYPE II).
Principal Investigator: Dr. K. NITHYA
Name & Address of Institution: Government Siddha Medical College, Arumbakkam, Chennai-106
☑ New Review ☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY): 05-04-2016
Date of Previous Review, If Revised Application:
Decision of the IEC ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions / Reasons / Remarks: 1) Duration should be changed to 90 days. 2) Lipid profile and C-Peptide assay should be included in lab investigation.
Recommended for a period of 1 year from date of completion of preclinical studies :

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

Dr.P.Jeyaprakashnarayanan, M.D(s)
Chairman

5/4/16

Dr.K.Kanakavalli, M.D(s)
Member Secretary

INSTITUTIONAL ETHICS COMMITTEE

Date : 05/04/2016.
Sub : IEC review of research proposals.
Ref : Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
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Dr.K.KANAKAVALLI, M.D(S)., Member secretary	✓	
Dr.P.SATHYA RAJESWARAN, M.D(S)., Clinician – Siddha	✓	
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Dr. P. Jeyaprakashnarayanan, M.D(s)
Chairman

Dr.K. Kanakavalli, M.D(s)
Member secretary

BIO STATISTICAL ANALYSIS

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

FASTING BLOOD SUGAR :

S.no	Fasting blood sugar level in mg	
	Before treatment	After treatment
1	127	106
2	133	119
3	139	126
4	132	127
5	134	106
6	135	109
7	140	130
8	128	98
9	133	111
10	131	105
11	126	95
12	129	107
13	128	100
14	127	94
15	134	112
16	138	113
17	129	100
18	133	108
19	140	122
20	133	99
21	127	114
22	135	123
23	132	113
24	129	93
25	130	107
26	128	102
27	126	97
28	133	119
29	128	107
30	137	113
31	128	110
32	128	93
33	132	117
34	134	111
35	138	106
36	126	98
37	137	107
38	136	129
39	136	100
40	132	95

Fasting blood sugar :

	ft_bt	ft_at
N	40	40
Mean	132.0250	108.5250
Minimum	126.00	93.00
Maximum	140.00	130.00

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ft_bt	132.0250	40	4.16633	.65876
	ft_at	108.5250	40	10.29311	1.62748

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	ft_bt - ft_at	23.50000	8.30199	1.31266	20.84489	26.15511	17.903	39	0.000

Ft_bt= fasting before treatment

Ft_at=fasting after treatment

Inference:

Since the p value is highly significant (p<0.000). The hypothesis is not accepted. So there is significant reducing of Fasting blood sugar level (mg) among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

Effect of Perungaya chooranam on Postprandial blood Sugar level in Madhumegam cases.

S.no	Post prandial blood sugar level in mg	
	Before treatment	After treatment
1	192	162
2	186	141
3	240	169
4	214	180
5	184	152
6	245	205
7	258	208
8	244	192
9	220	181
10	200	166
11	229	180
12	242	196
13	219	179
14	208	177
15	239	189
16	257	221
17	218	165
18	230	195
19	268	211
20	222	181
21	203	179
22	275	242
23	229	197
24	228	177
25	247	198
26	191	180
27	219	178
28	182	163
29	260	216
30	219	182
31	214	175
32	186	159
33	213	183
34	233	190
35	251	212
36	183	170
37	268	199
38	225	176
39	188	163
40	184	153

Post prandial blood sugar:

	pp_bt	pp_at
N	40	40
Minimum	182.00	141.00
Maximum	275.00	242.00

Paired Samples Statistics

	Mean	N	Std. Deviation	Std. Error Mean
pp_bt	222.8250	40	26.65726	4.21488
pp_at	183.5500	40	20.62479	3.26107

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	pp_bt - pp_at	39.27500	12.77013	2.01914	35.19091	43.35909	19.451	39	0.000

Inference:

Since the p value is highly significant (p<0.000). The hypothesis is not accepted. So there is significant reducing of postprandial blood sugar level (mg) among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

Effect of Perungaya chooranam on HbA1C level in Madhumegam cases.

S.no	HbA1C	
	Before treatment	After treatment
1	7.7	7.1
2	8	7.2
3	7.8	7.0
4	7.3	6.8
5	7.6	7.1
6	7.4	6.3
7	7.9	7.6
8	6.6	6.1
9	6.7	5.8
10	7.1	6.5
11	6.8	6.0
12	6.7	6.5
13	7.3	6.9
14	7.0	6.5
15	7.5	6.5
16	8	7.4
17	6.8	5.9
18	6.7	6.2
19	7.8	6.8
20	7.4	7.2
21	6.9	6.5
22	8.0	7.9
23	7.1	6.8
24	7.0	6.5
25	7.4	6.8
26	7.5	6.9
27	7.8	6.6
28	6.8	6.0
29	7.9	7.2
30	7.2	6.8
31	6.9	6.2
32	8	6.8
33	6.6	5.8
34	7.7	6.7
35	7.5	6.6
36	7.6	6.6
37	7.2	6.7
38	8.0	7.9
39	7.8	7.2
40	7.0	6.5

HbA1C:

	hba_bt	hba_at
N	40	40
Minimum	6.60	5.70
Maximum	8.00	7.80

Paired Samples Statistics

	Mean	N	Std. Deviation	Std. Error Mean
hba_bt	7.3350	40	.45548	.07202
hba_at	6.7700	40	.51201	.08096

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Pair 1 hba_bt - hba_at	.56500	.29919	.04731	.46932	.66068	11.944	39	0.000

Inference:

Since the p value is highly significant ($p<0.000$). The hypothesis is not accepted. So there is significant reducing of HbA1C level among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

GOVERNMENT SIDDHA MEDICAL COLLEGE

ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE

CHENNAI – 600 106

CLINICAL STUDY ON **"PERUNGAYA CHOORANAM"** IN THE TREATMENT OF **"MADHUMEGAM" (DIABETES MELLITUS TYPE 2)**

INFORMED CONSENT FORM

"I have read the foregoing information. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.

I consent voluntarily to participate in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care".

"I have received a copy of the information sheet/consent form".

Date:

Station:

Signature of participant:

Signature of the Guide: Signature of theInvestigator:

அரசினர் சித்த மருத்துவக் கல்லூரி சென்னை 106
அறிஞர் அண்ணா மருத்துவமனை சென்னை

மதுமேக நோய்க்கான சித்த மருந்தின் (பெருங்காய சூரணம்)
பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்
ஒப்புதல் படிவம்

ஆய்வாளரால் சான்றளிக்கப்பட்டது

நான் இந்த ஆய்வை குறித்த அனைத்து விபரங்களையும் நோயாளிக்கு புரியும் வகையில் எடுத்துரைத்தேன் என உறுதியளிக்கிறேன்

தேதி : கையொப்பம் :
இடம் : பெயர் :

நோயாளியின் ஒப்புதல்

என்னிடம் இந்த மருத்துவ ஆய்வின் காரணத்தையும், மருந்தின் தன்மை மற்றும் மருத்துவ வழிமுறை பற்றியும், தொடர்ந்து எனது உடல் இயக்கத்தை கண்காணிக்கவும் ,அதனை பாதுகாக்கவும் பயன்படும் மருத்துவ ஆய்வுக்கூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் மருத்துவ ஆய்வின் போது ,காரணம் எதுவும் கூறாமல் ,எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்து கொள்ளும் உரிமையை தெரிந்திருக்கின்றேன். நான் என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு மதுமேக நோய்க்கான பெருங்காய சூரணம் மருந்தின் பரகரிப்புத் திறனைக் கண்டறியம் மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

தேதி : கையொப்பம் :
இடம் : பெயர் :
உறவுமுறை:சாட்சிக்காரர் கையொப்பம் பெயர்:
தேதி :
இடம் :

CASE SHEET PROFORMA
GOVERNMENT SIDDHA MEDICAL COLLEGE
POST GRADUATE DEPARTMENT – MARUTHUVAM BRANCH
CHENNAI – 600 106.

CASE SHEET PROFORMA FOR MADHUMEGAM
(NON INSULIN DEPENDENT DIABETES MELLITUS – NIDDM)

OP No / IP No	:	Nationality	: Indian
Ward No	:	Religion	:
Bed No	:	D.O.A	:
Name (In Block Letters)	:	D.O.D	:
Age	:	No of Days Treated	:
Sex	: Male/ Female		
Occupation	:	Diagnosis	:
Income	: /Month	Result	:
Permanent Address	:		

Temporary Address : Govt. Siddha Medical College,
Chennai – 600 106.

1. Complaint and duration :

2. History of present illness :

3. History of previous illness :

4. Personal history

Marital History :

Occupation :

Environment :

Social History :

Habits :

5. Family history

SIDDHA ASPECT

GENERAL CONDITION ON ADMISSION

1. NILAM: 5

Kurinji

Mullai

Marutham

Neithal

Paalai

2. PARUVA KAALAM: 6

Kaar Kaalam : (Aavani, Purattasi)

Koothir Kaalam : (Ayppasi, Karthigai)

Munpani Kaalam : (Maarkazhi, Thai)

Pinpani Kaalam : (Maasi, Panguni)

Elavenil Kaalam : (Chittirai, Vaikasi)

Mudhuvenil Kaalam : (Aani, Aadi)

3. UDAL: 4

Vali Udal

Azhal Udal

Iya Udal

Kalappu Udal

4. KANMENTHIRIYANGAL: 5

Vaai

Kaal

Kai

Eruvai

Karuvai

5. PORI / PULANGAL: 5

Mei - Ooru

Vaai - Suvai

Kann - Oli

Mookku - Nattram

Sevi - Osai

6. GUNAM: 3

Sathuva Gunam

Rajo Gunam

Thamo Gunam

7. UDAL KATTUGAL: 7

Saaram

Senneer

Oon

Kozhuppu

Enbu

Moolai

Sukkilam / Suronitham

8. MALAM: 3

Malam

Moothiram

Viyarvai

9. MUKKUTRANGAL

VALI

Piraanan

Abaanan

Uthaanan

Viyaanan

Samaanan

AZHAL

Anala Pitham

Ranjaga Pitham

Aalosaga Pitham

Praasaga Pitham

Saathaga Pitham

IYAM

Avalambakam

Kilethagam

Pothagam

Tharpagam

Santhigam

10. ENVAGAI THERVU

Naadi:

Sparisam:

Naa:

Niram:

Mozhi:

Vizhi:

Malam:

Niram

Irugal

Ilagal

Moothiram:

Neerkuri:

Niram

Edai

Manam

Nurai

Enjal

Neikuri:

MODERN METHODS:

GENERAL EXAMINATION:

Consciousness and Intelligence

Voice and Speech

General appearance

Height and Weight

Anaemia

Cyanosis

JVP

Jaundice

Clubbing

Ascites

Oedema

Lymphadenopathy

Temperature

Respiration

Pulse

Blood Pressure

INVESTIGATION

A)BLOOD INVESTIGATIONS:

BLOOD INVESTIGATIONS		**BEFORE TREATMENT**	**AFTER TREATMENT**
Hb (gms/dl)			
T.RBC (millions cells/cu.mm)			
ESR (mm)	½ hr		
	1 hr		

T.WBC (cells/cu.mm)			
Differential Count (%)	Polymorphs		
	Lymphocytes		
	Monocytes		
	Eosinophils		
	Basophils		

BLOOD INVESTIGATIONS		**BEFORE TREATMENT**	**AFTER TREATMENT**
Blood glucose (mg/dl)	F		
	PP		
	R		
Renal Function Test	Blood Urea		
	Serum creatinine		

B) URINE INVESTIGATIONS:

URINE INVESTIGATIONS	**BEFORE TREATMENT**	**AFTER TREATMENT**
Albumin		
Sugar		
Deposits		
Ketone Bodies		

C) SPECIFIC INVESTIGATIONS:

HbA1C:

C- peptide assay:

D) BODYMASS INDEX (BMI)

SIGN AND SYMPTOMS:

S.No	CLINICAL FEATURES	BEFORE TREATMENT	DURATION TREATMENT						AFTER TREATMENT
			14th Day	21st Day	28th Day	35th Day	42nd Day	49th Day	
1	POLYURIA								
2	POLYPHAGA								
3	POLYDIPSIA								
4	PRURITIS VULVA								
5	ITCHING ALL OVER THE BODY								
6	DRYNESS OF THE MOUTH AND THROAT								
7	CONSTIPATION								
8	DISTURBED SLEEP								
9	PAIN ALL OVER THE BODY								
10	SKIN INFECTION								
11	EMACIATION								
12	ABDOMINAL PAIN								
13	IMPOTENCE								
14	GLYCOSURA								

Others specify, if any

DIAGNOSIS

MADHUMEGAM (TYPE II DIABETES MELLITUS)

TRIAL MEDICINE: PERUNGAYA CHOORANAM
DOSE : 2 Gram/ bd
Anubanam: LUKE WARM WATER
Duration of Treatment: 90 days

DAILY REPORT	MEDICINE

ADVICE:

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