



सत्यमेव जयते

Ministry of Ayush
Government of India

STANDARD TREATMENT GUIDELINES
ON
**MANAGEMENT OF
METABOLIC DISORDERS**
IN
SIDDHA SYSTEM OF MEDICINE

AYUSH VERTICAL
DIRECTORATE GENERAL OF HEALTH SERVICES
Government of India

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METABOLIC DISORDERS
IN
SIDDHA SYSTEM OF MEDICINE

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आयुष मंत्रालय और
राज्य मंत्री
स्वास्थ्य एवं परिवार कल्याण मंत्रालय
भारत सरकार



सत्यमेव जयते

प्रतापराव जाधव
PRATAPRAO JADHAV



MESSAGE

Minister of State
(Independent Charge) of
Ministry of Ayush and
Minister of State in
Ministry of Health and Family Welfare
Government of India



India has a rich legacy of traditional healthcare systems that offer time-tested approaches to health and well-being. In recent years, there has been a growing recognition of the role Ayush can play in addressing contemporary health challenges through holistic approach.

The release of the Standard Treatment Guidelines (STGs) for Metabolic Disorders in respective Ayurveda, Siddha, Unani, and Homoeopathy (ASU&H) systems, with the inclusion of Yoga, marks another significant milestone in our efforts to mainstream Ayush systems within India's healthcare landscape. Building on the success of STGs for musculoskeletal disorders, this initiative underscores our commitment to integrating traditional wisdom with modern scientific validation, enhancing healthcare quality and accessibility.

These guidelines offer evidence-based recommendations for the prevention and management of prevalent conditions such as Diabetes Mellitus, Dyslipidaemia, Obesity, Gout and Non-Alcoholic Fatty Liver Diseases (NAFLD), thereby equipping healthcare practitioners with structured, holistic approaches to patient care.

I am confident that these STGs will help to improve clinical outcomes, promote integrative healthcare models, and reinforce the relevance of Ayush systems in addressing the growing burden of lifestyle-related disorders in our nation.

I heartily appreciate the efforts and congratulate all the experts, institutions, and stakeholders who have contributed to the development of these comprehensive guidelines.


(Prataprao Jadhav)

25 April, 2025
New Delhi

वैद्य राजेश कोटेचा
सचिव
Vaidya Rajesh Kotecha
Secretary



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FOREWORD

Metabolic disorders represent a growing public health concern in India, contributing significantly to the national burden of non-communicable diseases. Addressing these conditions calls for a comprehensive, patient-centric approach—one that not only addresses symptoms but also fosters long-term health and well-being. Ayush systems hold immense potential in the prevention and management of lifestyle-related disorders, including Diabetes Mellitus, Dyslipidemia, Obesity, Gout and Non-Alcoholic Fatty Liver Disease (NAFLD).

Recognizing this potential, the Ayush vertical under the Directorate General of Health Services (DGHS) has undertaken a commendable step in formulating Standard Treatment Guidelines (STGs) for metabolic disorders across Ayurveda, Siddha, Unani, and Homeopathy systems. These guidelines have been developed through an extensive process of expert consultations, critical review of classical texts, and incorporation of contemporary clinical evidence. The STGs aim to support practitioners in delivering consistent, safe, and effective care through Ayush systems, promoting standardization and quality assurance in clinical practice.

I hope these guidelines will not only lead to improved clinical outcomes but also contribute meaningfully to realizing the vision of integrative healthcare in India. By establishing uniform standards of practice, they pave the way for generating high-quality evidence. This, in turn, can support the global pursuit of wellbeing by addressing one of today's most pressing healthcare challenges—non-communicable diseases—through the holistic and time-tested approaches of Ayush. As we move ahead, such initiatives will continue to affirm the evolving and vital role of Ayush in tackling lifestyle-related health issues and in shaping a more holistic, inclusive, and sustainable healthcare system.

I congratulate the teams of experts, institutions, and stakeholders whose dedication and collaborative efforts have made this initiative possible.

(Rajesh Kotecha)

New Delhi.
23.04.2025

प्रो.(डॉ.) अतुल गोयल

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स्वास्थ्य सेवा महानिदेशक

DIRECTOR GENERAL OF HEALTH SERVICES



सत्यमेव जयते

भारत सरकार
स्वास्थ्य एवं परिवार कल्याण मंत्रालय
स्वास्थ्य सेवा महानिदेशालय

Government of India
Ministry of Health & Family Welfare
Directorate General of Health Services



Foreword

In the past two decades, there has been a resurgence of traditional medicine globally, including the Ayush system in India. Advocates of the Ayush system of medicine, including practitioners and scientists, have consistently highlighted its personalized predictive approach and diversity of Ayush formulations and therapies. As we traverse the terrain of healthcare, necessity of a holistic treatment approach becomes increasingly important. Ayush system of medicine, with its centuries-old wisdom and emphasis on natural healing modalities, offers a distinct perspective on managing metabolic disorders. Its approach, centered on restoring an equilibrium of mind, body, and spirit, complements modern medicine, thereby widening the care available to patients

Publication of Standard Treatment Guidelines (STGs) on Metabolic Disorders by Ayush system of medicine represents a significant footstep towards our commitment to comprehensive healthcare for our citizens. These guidelines, curated by experts in the field, are a testament to efficacy and relevance of Ayush in addressing public health. In order to ensure clarity and accessibility for all stakeholders, conventional terminology has been seamlessly integrated throughout the document. Each disease condition is introduced alongside its corresponding ICD classification, providing a clear clinical narrative that enhances understanding for all stakeholders.

I appreciate the Ayush vertical of this directorate, as well as contributions of various experts from National Institutes and Research Councils under the Ministry of Ayush, in bringing forth this initiative. Additionally, my gratitude to experts from medicine department of LHMC for their invaluable support in incorporating modern perspective on metabolic disease conditions into the STGs. By bridging gaps between traditional and modern medicine, we attempt to foster inclusivity and collaboration between various systems of medicine for benefitting patients.

I sincerely hope that these guidelines will serve as a valuable resource for Ayush healthcare practitioners, empowering them to deliver optimal care to individuals afflicted with metabolic diseases.

(Atul Goel)

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TABLE OF CONTENTS

S.No.	Chapters	Page No.
I	Abbreviations	ii
II	Glossary	v
1.	Diabetes Mellitus	1
2.	Dyslipidemia	27
3.	Gout	53
4.	Non Alcoholic Fatty Liver Disease	73
5.	Obesity	97

ABBREVIATIONS

ACR	Albumin- to- Creatinine Ratio
ACR	American College of Rheumatology
ADA	Adenosine Deaminase Test
ALT	Alkaline Transaminase
Apo B	Apolipoprotein B
APRI	Aspartate Aminotransferase to Platelet Ratio Index
ASCVD	Atherosclerotic cardiovascular diseases
ASMD	Acid sphingomyelinase deficiency
AST	Aspartate Aminotransferase
BARD	Body Mass Index, Aspartate Aminotransferase/ Alkaline Transaminase(AST/ALT) ratio and Presence of Diabetes
BD	Twice a day
b-hCG	Beta-human chorionic gonadotropin
BMI	Body Mass Index
CAD	Coronary Artery Disease
CAP	Controlled Attenuation Parameter
CDT	Carbohydrate-deficient transferrin
CKD	Chronic Kidney Disease
CRP	C- Reactive Protein
CT scan	Computed Tomography
CVD	Cardiovascular disease
DALY	Disability-adjusted life year
DASH	Dietary Approaches to Stop Hypertension-style diet
DCS	Double contour sign
DECT	Dual-energy Computed Tomography
DIP	Distal Interphalangeal Joint
DXA	Dual Energy X-Ray absorptiometry
ECG	Electrocardiogram
ESR	Erythrocyte Sedimentation Rate
FAST	FibroScan- aspartate aminotransferase
FBS	Fasting blood glucose
FH	Follicle Stimulating Hormone
FPG	Fasting Plasma Glucose

FT4	Free Thyroxine
GFR	Glomerular Filtration Rate
HbA1c	Glycosylated Haemoglobin
HBsAg	Hepatitis B
HCC	Hepato cellular Carcinoma
HCG	Human Chorionic Gonadotropin
HDL	High Density Lipoprotein
HeFH	Heterozygous Familial Hypercholesterolemia
HELLP	Hemolysis, Elevated Liver enzymes and Low platelets
HLA-B27	Human Leucocyte Antigen B27
HOMA-IR	Homeostatic Model Assessment for Insulin Resistance
ICD	International Classification of Diseases
IFG	Impaired Fasting Glucose
IGT	Impaired Glucose Tolerance
kPa	Kilopascals
LAL	Lysosomal acid lipase
LDL	Low Density Lipoprotein
LDL-C	Low-density lipoprotein cholesterol
LFT	Liver Function Test
LH	Luteinizing Hormone
LSM	Liver Stiffness Measurement
MAFLD	Metabolic Dysfunction Associated Fatty Liver Disease
MEFIB	Magnetic Resonance Elastography plus Fibrosis - 4
MRCP	Magnetic Resonance Cholangiopancreatography
MRE	Magnetic Resonance Elastography
MRI	Magnetic Resonance Imaging
MS	Metabolic Syndrome
MSU	Monosodium Urate crystal
MTP	Metatarsophalangeal joint
MTTP	Microsomal Triglyceride Transfer Protein
MUFA	Monounsaturated Fatty Acid
NAFLD	Non-Alcoholic Fatty Liver Disease
NASH	Non-Alcoholic Steatohepatitis
NFHS	National Family Health Survey
NFS	BMI, diabetes status, AST/ALT ratio, platelet count, and albumin levels

Non-HDL-C	Non-High-Density Lipoprotein Cholesterol
OA	Osteoarthritis
OD	Once Daily
OGTT	Oral Glucose Tolerance Test
OHS	Obesity Hypoventilation Syndrome
OSA	Obstructive Sleep Apnea
PCOS	Polycystic Ovarian Syndrome
PUFA	Polyunsaturated Fatty Acid
RA factor	Rheumatoid Arthritis factor
RBSK	Rashtriya Bal Suraksha Karyakaram
RSSDI	Research Society for the Study of Diabetes in India
SF	Synovial Fluid
SM - S	Sphingomyelin
T2DM	Type 2 Diabetes Mellitus
TC	Total Cholesterol
TDS	To be taken three times a day
TG	Triglyceride
TSH	Thyroid Stimulating Hormone
USG	Ultrasonography
UTI	Urinary Tract Infection
VLDL	Very Low Density Lipoprotein
WAGR syndrome	Wilms tumor, Aniridia, Genitourinary malformations and a Range of developmental delays
WC	Waist Circumference
WHO	World Health Organisation
WHR	Waist-Hip Ratio
YLD	Years Lived with Disability
YLL	Years of Life Lost

GLOSSARY

1. Pūtam/ pañcapūtam /aimpūtam / añcupūtam - Five primordial elements

Earth, water, fire, air and space are the primordial elements in the formation of every single material (living and nonliving) in the world; the entire universe, including the creatures in it, is constituted and influenced by these five elements

2. Pirutivi / pirutivi pūtam (earth)

A primordial golden-coloured element formed from water element, with qualities such as heaviness, solidity, conglomeration, growth and development

3. Appu / calam / appu pūtam/ nīr (water)

A primordial colourless element formed from fire element, with qualities such as coldness, greasiness, lightning, soddening, spreading with ease, wetting and oozing, collecting scattered things and enriching the mind

4. Vaṅṅicam / tēyu pūtam / tī (fire)

A primordial red-coloured element formed from air element, with qualities like heat, sharpness, clarity, subtleness, burning, glowing, colouring, etc.; governs activities such as egoism, laziness, sexual intercourse, fear and sleep

5. Mārutam / vāyu / kārru /vaḷi / kāl (air)

A primordial black-coloured element formed from ether element, with qualities like dryness, weightlessness and roughness, governing motor activities, inhalation and exhalation

6. Ākāya pūtam / ākāyam /Vicumpu (ether)

The primordial element, whitish in colour, having qualities like subtleness, clarity, appeasing nature and occupying space and governing the activities of desire, vengeance, lust, etc.

7. Seevakini (Basal Metabolic heat)

Heat required for sustenance of life, (also referred to as *Kukkiyanal*, *Uyiranal* and *Uyirakkini*).

8. Samanikini (Optimal digestive fire)

It is constituted by *Samanan*, *Analam* and *Kilethakam*. It is the digestive fire which ensures proper and timely digestion of all the solid and liquid food materials taken by an individual. – Optimal digestive fire.

9. Vidamakini (Toxic digestive fire)

Delayed digestion due to deranged and displaced samanana leading to toxic digestion.

10. Deekashakini (Enhance fire of digestion/ Fiery digestion)

Due to increased digestive fire intake of even improperly cooked/under cooked food gets digested along with essence.

11. Manthakini (Sluggish/Delayed digestion)

Without digesting immediately, the food items taken eagerly, it produces rumbling noise (borborygmus) in the abdomen along with abdominal distension and heaviness of body

12. Vaḷi/ vātam / aṅḷam/ vāyu (bio-energy movement):

One of the three humours/ *mukkuṛam* / *muttōṭam* or principles of functional constitution of the body, condensed from the elements air and space. *Vaḷi* is responsible for all movements in the body and controls the functions of the nervous system, circulatory system and elimination of wastes etc. *Vatham* predominates in the region below umbilicus and based on its function it

is classified into ten types. They are *Pranan*, *Abanan*, *Viyanan*, *Samanan*, *Udhanan*, *Naagan*, *Koorman*, *Kirugaran*, *Devathathan* and *Thananjeyan*. Roughness, dryness, lightness and mobility are certain attributes of *Vatham*. It also strengthens the five sensory organs, regulates respiration, maintain the functions of *Udal thathukkal* (physical constituents) and 14 *Vegangal* (physiological reflexes)

13. *Uyirkkāl / pirāṇaṇ* (vāyu for respiration and digestion)

Responsible for respiratory functions and controls its organs; originates from the center of skull, also nourishes the life force

14. *Kīḷṇōkku kāl/apāṇavāyu /apāṇaṇ* (vāyu for downward biological movements)

*Responsible for absorption and assimilation of essence, excretion of urine and faeces, ejection of semen and expulsion of contents of the uterus, contracting and relaxing the sphincters; originates from coccygeal region *mūlātāram*/*

15. *Mēlnōkku kāl/ utāṇaṇ* (vāyu for upward biological movements)

Responsible for all upward movements; responsible for reflexes like cough, sneeze, hiccup and vomiting; also responsible for speech, stations the essence of foods at appropriate place (nutrition), thus helps in the digestion and assimilation of food; emanates from fire of stomach, resides in navel, neck, throat and nose

16. *Naṭukkāl/ camāṇaṇ /camāṇavāyu* (vāyu for homeostasis)

Balances the other components of vāyu and responsible for assimilation; balances the six tastes, water and foodstuffs during the process of digestion and gets them to their sites of action; originates from the navel region

17. *Paravukāl/viyāṇaṇ* (vāyu for circulation)

Disseminates throughout body via 72 000 vessels and nerves causing voluntary and involuntary functions; takes the essence of food to all parts of the body; responsible for touch sensation

18. *Nākaṇ* (vāyu for intellectual functions)

*Responsible for higher intellectual functions, hearing, thinking, singing, etc.; causes blinking of the eyes, opening of eyelids and goosebumps. *Nākaṇ* (vāyu for intellectual functions) Responsible for higher intellectual functions, hearing, thinking, singing, etc.; causes blinking of the eyes, opening of eyelids and goosebumps.*

19. *Kūrmaṇ* (vāyu for ophthalmic function)

Acts on the eyes, responsible for blinking, visual interpretation and lacrimation; responsible for the acts of yawning and closing of mouth

20. *Kirukaraṇ* (vāyu for secretion)

Responsible for oral and nasal secretion; causes thinking of one entity and produces much hunger, cough, sneeze, etc.

21. *Tēvatattaṇ vāyu* for fatigue

Responsible for laziness and tiredness on waking, causes movement of eyeball, causes one to be engaged in coaxing, fighting, verbal dispute and bouts of intense anger

22. *Taṇaṅceyaṇ* (vāyu for death)

During death, causes generalized swelling of the body and tinnitus; leaves the body through the head on the third day of death

23. *Azal/pittam* (bio-energy fire)

*One of the humours/ *mukkurram* or principles of constitution of the body, condensed from the elements water and earth; *azal* is responsible for normal metabolism and controls digestion,*

movement of limbs, function of eyes to enhance vision, complexion of skin, sharpness of mind, etc. Azal dominates the chest and abdominal area and exhibits itself in five forms. They are *Anarpitham*, *Ranjaga pitham*, *Saathag pitham*, *Aalosaga pitham* and *Prasaga pitham*. It is eliminated from the body through sweat.

24. Ākkaṇal / anarpittam (azal / pittam for digestion)

One of the five types of *azal*, exists in stomach and intestines; quality of increased fire, dries up water contents of foodstuffs, digests all ingested food

25. Vaṇṇa eri/ iraṅcaka pittam (azal / pittam for nourishment of blood)

One of the five types of *azal*, exists in stomach, responsible for nourishment of blood through conversion of chyle

26. Ārralaṅki/ cātaka pittam (azal / pittam for performing desired acts)

One of the five types of *azal*, exists in heart, performs desired acts with help of knowledge, intellect and affinity

27. Oḷḷoḷi tī / pirācakam (azal / pittam for Complexion)

One of the five types of *azal*, exists in skin and gives it lustre

28. Nōkkaṇal/ ālōcakam (azal pittam for vision)

One of the five types of *azal*, exists in eye and is responsible for vision

29. Aiyam/kapam (bio-energy water):

One of the three humors of body according to the humoral principles; is watery or frothy in general; a key influencer in all respiratory diseases. It dominates the head and neck region and exhibits itself into five forms. They are *Avalambagam*, *Kilaetham*, *Pothagam*

Tharpagam and *Santhigam*. It is eliminated from the body through the urine.

30. Aḷi aiyam / avalampakam (strengthening aiyam)

One of the five types of *aiyam*, exists in thoracic cavity, including heart; along with its innate potential and essence of food it strengthens the body

31. Nirppi aiyam / kilētakam (aiyam/ kapam for digestive functions)

One of the five types of *aiyam*, exists in stomach, breaks down ingested foodstuffs and promotes digestion

32. Cuvaikāṇ aiyam / pōtakam (aiyam/ kapam for taste)

One of the five types of *aiyam*, exists in tongue, helps to experience taste of food

33. Niraiyaiyam / tarpakam (aiyam/ kapam for strengthening sense organs)

One of the five types of *aiyam*, exists in head, strengthens sense organs, keeps the eyes cool

34. Onriyaiyam / cantikam (aiyam/ kapam for lubrication)

One of the five types of *aiyam*, exists in joints and lubricates them

35. Seven uṭartātu (physical constituents):

Seven *uṭartātu*, namely plasma (*cāram*), blood (*cennīr*), muscle (*ūṇ*), adipose tissue (*koḻuppu*), bone, (*eṇṇu*), bone marrow (*mūḷai*) or male or female hormones, reproductive tissue (*cukkilam curōṇitam*).

36. Eṇvakai tērvu / eṭṭuvakai pariṭcai (eight types of diagnosis)

Naadi (Unique Siddha pulse reading method), *Sparisam* (Examination of Touch / palpation), *Naa* (Examination of Tongue), *Niram* (Examination of Colour/ Complexion), *Mozhi* (Examination of Speech), *Vizhi* (Examination of Eye), *Malam* (Examination of Stool) *Neer*, *Neerkuri* (Urine examination), *Neikuri* (Urine Sign – Oil Drop Test).

37. Internal Medicines:

37.1 Cāru (juice): Extract of leaves, root, bark, flowers and unripe fruit, obtained by pounding and filtering or by adding astringent substances or by means of a heating process; juice should be taken within three hours after preparation

37.2 Kuṭinīr decoction

Aqueous extract prepared at a ratio of one part of medicine to four parts of water ($\frac{1}{4}$); decoctions are also prepared using other ratios, e.g., $\frac{1}{8}$ or $\frac{1}{16}$, as prescribed in Siddha texts; occasionally milk is also added; other methods of extraction are also described, including boiling and percolation. It is to be consumed within three hours of preparation

37.3 Kaṛkam (medicinal paste)

Paste of fresh or dried raw materials ground with water; should be consumed within three hours of preparation

37.4 Cūraṇam (medicinal powders)

Purified raw materials are pounded separately, sieved and mixed according to a given ratio; for certain preparations the purified raw materials are mixed as per the ratio prescribed, then powdered and sieved; shelf-life of three months

37.5 Vaṭakam (lozenges)

Medicinal powder mixed with sugar or jaggery is steam baked with vapours from a mixture of milk and water; the steamed flour is pounded when hot and rolled into pills of required size; shelf-life of three months

37.6 Maṇappāku (syrup)

Decoction or fruit juices boiled with sugar or jaggery until a sweet aromatic odour develops; powdered raw materials are sprinkled over this; shelf-life is six months

37.7 Ney (medicated ghee)

Ghee-based herbal preparation prepared by boiling a mixture of ghee with specified medicinal pastes, juices, decoctions and milk, according to composition of recipes; shelf life is six months

37.8 Iracāyaṇam semi-solid confection

Prepared by adding unrefined sugar and ghee to medicinal powders until a semi-solid consistency is attained; shelf-life is six months

37.9 Iḷakam (electuary)

This type of internal medicine is prepared by heating certain decoctions or juices or milk along with sugar or jaggery until sweet aromatic odour develops and volume is reduced; powdered raw drugs are added and mixed well; ghee and finally honey are added and mixed well; shelf-life is six months

37.10 Eṇṇey (medicinal oils)

Herbal juice, decoctions, powder or herbal paste is added with specific oils, boiled at specific heat level until definite consistency is reached and finally filtered and preserved; shelf-life is one year.

37.11 Māttirai/ kuḷikai /uruṇṭai (pills/tablets)

Certain drugs are ground well with herbal juices or decoctions, ginger juice, breast milk, etc. until the mixture becomes fine enough to be rolled to form pills and dried; size of pill depends on dose and method of preparation in classical texts; hours of grinding mixture vary with each medicine; usually round in appearance; shelf-life is one year.

37.12 *Tīnīr/ pukai nīr / Tirāvakam* (medicated liquid obtained by distillation)

Drugs are boiled with water in a distillation apparatus, vapour of medicated water is condensed and collected as a distillate. shelf-life is one year

37.13 *Mezuku* (medicated wax)

Prepared by grinding raw drugs to waxy consistency; There are two types: 1. obtained by grinding certain mercurial compounds separately or with other raw drugs, adding herbal juices or honey to a perfect stage of waxy consistency. 2. obtained by heating mercurial drugs or arsenical compounds with added oily substances or juices and grinding well; shelf-life is five years

37.14 *Kuzampu* (medicated viscous mixture)

Certain juices either mixed or separately taken in a pot along with jaggery, medicinal powders or fine powders of certain drugs, heated to semi-solid form. shelf-life is five years

37.15 *Pataṅkam* drugs obtained by sublimation

Heating of sublimating constituents either from organic or inorganic drugs. shelf-life is 10 years

37.16 *Centuram* (red calx)

Metallic/mineral substances are made into red microfine powder by burning or insolation or grinding with herbal juice or mineral distillates, or by incineration; shelf-life is 75 years

37.17 *Nīru / paṛpam* (white calx)

Metallic/mineral substances made into white powder by burning or frying or by grinding with juices or by incineration; an ancient method of calcination; different processes are employed with variation in duration of incineration, hours of grinding and/or hours of burning; shelf-life is 100 years

37.18 *Cuṇṇam* (calcine)

Metallic/mineral substances, ground well with leaf juices, pungent liquids, mineral distillates; dried, kept in crucible, incinerated to obtain calcine; become red when turmeric is added due to lime content; shelf-life is 100 years

37.19 *Kaṛpam* rejuvenating drugs

Certain leaves, roots, salts and mineral compounds are consumed in a specific dose for a specific period of time while following a prescribed dietary regimen

38. External Medicines

38.1 *Kaṭṭu* (compress or bandage)

External application in which raw drugs and medicines like leaves, bark, etc. are either ground or cooked; then tied or bandaged over affected part

38.2 *Parru* (poultice)

External application in which medicines in the form of pastes or juices are applied over inflammation, wounds and skin lesions, some time after spreading them on a piece of cloth

38.3 *Pūccu* (liniment / semi-solid application)

External application in which boiled leaf juices or medicated oil are applied on affected part

38.4 *Pacai* (medicated cream)

Raw materials are added to melted wax or castor oil and applied over affected areas

38.5 *Poṭi* dusting powder

Powdered and purified herbs/inorganic substances are applied over wounds and ulcers; usually, astringent substances are used

38.6 Kaḷimpu (ointment)

Used externally; certain mineral compounds / astringent materials are powdered, ground with butter and applied over wounds and ulcers

38.7 Nīr (medicated liquid)

Medicated water for washing wounds and ulcers; antiseptic solutions prepared by either soaking raw drugs in water and making a decoction or diluting caustic substances

38.8 Naciyam (nasal instillation)

Instilling nasal drops

38.9 Poṭi timirtal (powder massage)

Massage with herbal powders containing turmeric and horse gram, occasionally mixed with camphor

38.10 Tokkaṇam (manipulation techniques)

There are nine types. 1. pressing, 2. holding or grasping, 3. tight-hug manoeuvre, 4. pulling, 5. moving, 6. gripping, 7. twisting, 8. laying or supinating, 9. striking with fist with or without applying oil

38.11 Orraṭam (fomentation)

Application of hot topically; substances such as lime powder, bran, brick powder, eggshell, leaves of medicinal plants such as Vitex negundo, Calotropis gigantea, Ricinus communis, Abutilon indicum, etc. are tied in a cloth as a bundle, which is heated and applied over affected area

38.12 Vētu (steam inhalation / steam exposure therapy)

Steam inhalation and steam application either to localized regions or the whole body; materials used are medicinal herbs such as Vitex negundo and Leucas aspera, turmeric powder, salt water, red brick and medicinal aromatic gums

38.13 Poṭṭaṇam/ Kizl (medicated pouch)

Raw drugs that are pounded or fried leaves of medicinal plants are tied in a piece of cloth as a bundle; this is dipped in a particular medicated oil and applied over an affected area.

38.14 Pukai (fumigation)

Using medicated fumes or smoke from any herbal/animal/ aromatic substance, such as cumin seeds, dried ginger, turmeric and flower of Datura metel.

38.15 Cuṭṭikai (cautery)

Application of heat using needle, broken earthen pots, piece of wood, heated air and insolation; usually on vertex, forehead, chest, back, hands and legs

38.16 Kāram (caustic ablation)

Application of drugs to parts to be excised or chronic ulcers, to remove unwanted growth, slough and debris and after healing process initiated

38.17 Pīccu (enema)

Medicated liquid substances injected into rectum to expel its contents; laxative solutions are administered through anal canal, leading to purgation

38.18 Aṭṭai viṭal (leech)

Medicinal leech therapy or hydrotherapy, a technique used for blood-letting to extract poisonous substances from affected areas and to purify blood

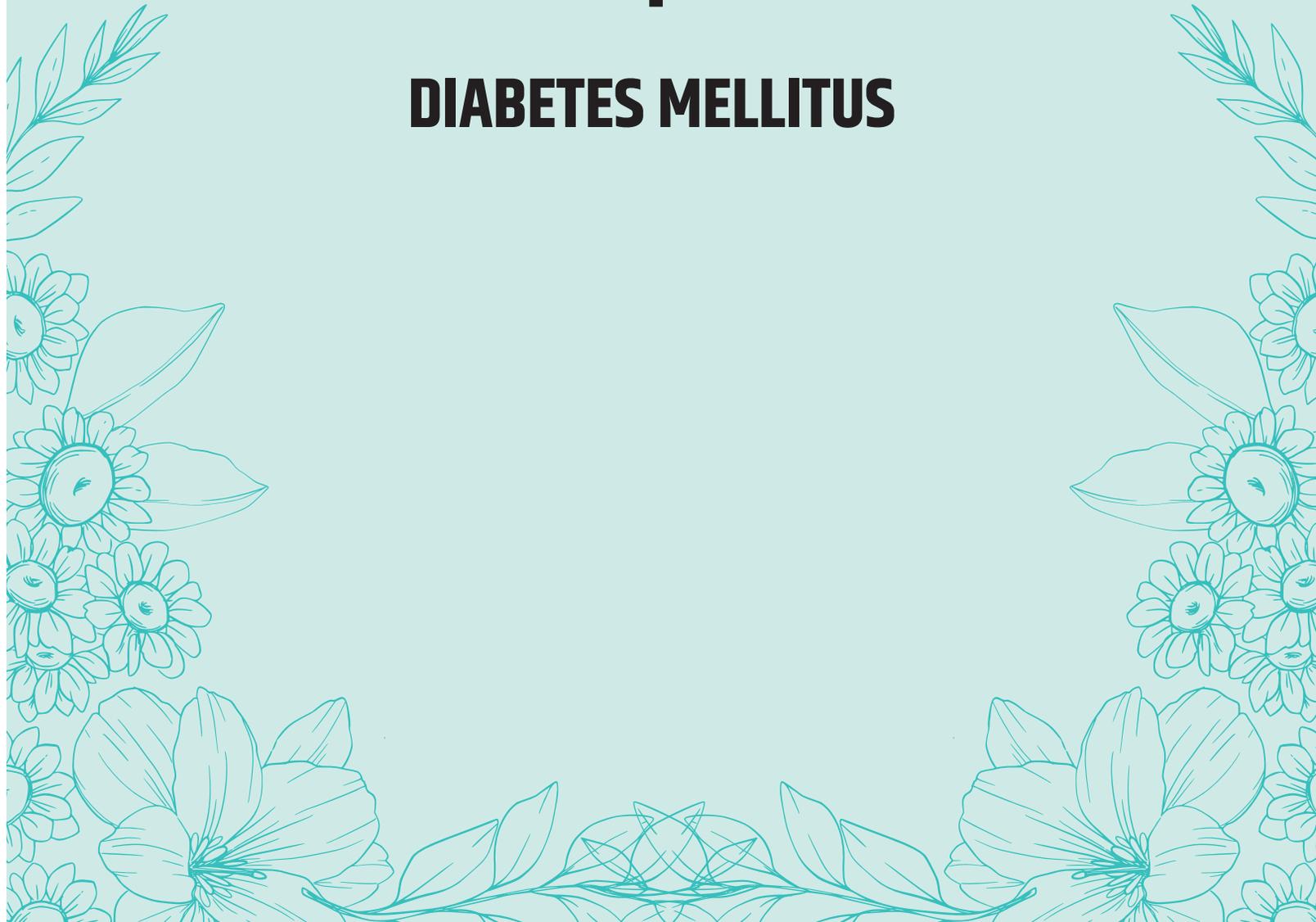
38.19 Kīral (incision)

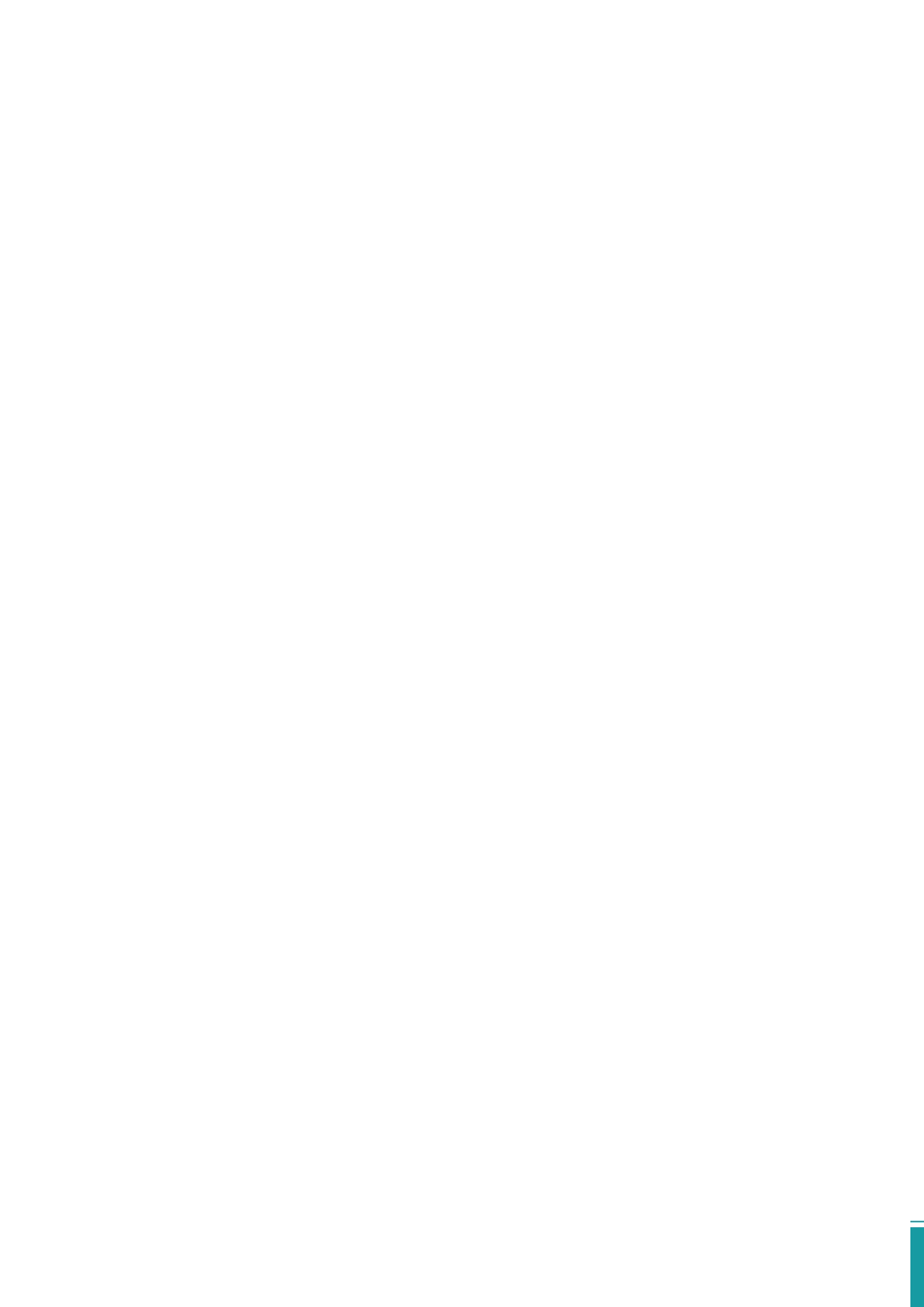
A surgical procedure to remove accumulated pus, blood etc.

CHAPTER

1

DIABETES MELLITUS





DIABETES MELLITUS

ICD 11 - 5A11

ICD 10 – E11.0 TO E11.9

மதுமேகம் – *Matumēkam* (TYPE 2 DIABETES MELLITUS)

Name of the disease:

மதுமேகம்

- NSMC – XGB1.4
- WHO Code - ISMT-4.11.40¹

TYPE 2 DIABETES MELLITUS²

CASE DEFINITION

Matumēkam is a disease characterized by frequency of urination, gradual deterioration of seven body constituents (*Uṭaltātu*) followed by emaciation.³ The primary vitiation of Azhal humour slowly affects the other humours and eventually leads to the deterioration of seven body constituents one by one. The salient clinical features of Matumēkam are correlated with Type 2 Diabetes Mellitus (T2DM) in conventional biomedical system.

Diabetes Mellitus is a chronic disorder resulting from aberrations in insulin secretion, insulin action, or both. Long term damage, dysfunction and failure of different organs resulting in this condition is attributed to the persistent hyperglycaemia state.⁴ T2DM previously referred as non-insulin-dependent diabetes accounts for approximately 90 – 95% of all diabetes cases. The condition also known as adult-onset diabetes is due to insulin resistance and relative insulin deficiency.^{4,5}

INTRODUCTION

- Matumēkam is a progressive metabolic disorder which is due to deranged three humors and deteriorating seven fundamental tissues. Siddhar *Yūkimuṇi* classified this disease under *Aḷal* type of *Nīriṇai Perukkal Nōykaḷ* (Clinical conditions with polyuria) in the classical text of *Yūki Vaittiya Cintāmaṇi*. This is synonymously known as *Iṇippu Nīr* and *Nīriḷivu*.^{3,6}
- Diabetes is the eighth-leading cause of mortality and has a prevalence of 529 million cases worldwide in 2021 with a global age standardised prevalence of 6.1%. International Diabetes Federation report indicated an expenditure of US\$ 996 billion globally due to the disease.^{7,8}
- Diabetes is also contributing to two-fold excess risk for ischemic heart disease and stroke, which attributes to the first and second leading cause of death worldwide.⁷
- A report published by the Lancet commission in 2020 highlights that the majority of disease burden (80%) is from Low- and Middle-income countries (LMICs).⁹
- Globally, the disease attributed to 37.8 million Years of Life Lost (YLL), 41.4 million Years of healthy life lost due to disability (YLD) and 79.2 million Disability-adjusted life year (DALY) in 2021.
- Between 2021-2050, the global age-standardised total diabetes prevalence is expected to increase by 59.7% resulting in 1.31 billion cases in 2050.⁷

- The NFHS-5 survey reported prevalence of diabetes of 4.90% among Indian individuals aged 15-49 years with 24.82% of individuals with undiagnosed diabetes.¹⁰
- The ICMR-INDIAB survey conducted reported 26.6% of Indians above 20 years having dysglycaemia with 11.4% suffering from diabetes and 15.3% suffering from a pre-diabetic state.¹¹
- Several non-modifiable risk factors like age, ethnicity, genetic predisposition, family history of diabetes and modifiable factors like sedentary lifestyle, obesity, unhealthy diet, stress, intrauterine environment, environmental pollutants, etc. are associated with the incidence of the disease.
- The COVID-19 pandemic has resulted in a significant rise of new-onset of T2DM in all age groups especially during the post-acute phase of the disease.¹² The pandemic shows an increase of 14.4% of new onset of diabetes mellitus including T2DM among the hospitalized patients.¹³

CLINICAL PRESENTATION AND EXAMINATION

According to the Siddha system of medicine, the common symptoms are frequent and excessive passage of brownish yellow coloured urine producing white sediments, which invite ants and flies; emaciation, polydipsia, xerostomia, polyphagia, general debility even after sufficient food intake. The uncontrolled disease condition over some time gradually deteriorates the seven body constituents causing *Matumēka Avataikaḷ* (Sequelae of T2DM) which is described as complications of T2DM. Ten stages of *Avataikaḷ* describe the complications of T2DM are as under:³

- Obesity and Bladder dysfunction
- Polyuria, Spermatorrhoea and UTI
- Xerostomia and Gastroparesis
- Polydipsia and Delirium due to Diabetic ketoacidosis
- Debility due to deterioration of seven body constituents
- Orthopnea
- Syncope
- Glandular swelling and Carbuncle
- Diarrhoea due to autonomic neuropathy, Parasitic infestations (maggots)
- Cachexia and death

The presentation of T2DM to the clinician is quite varied. A majority is discovered incidentally during regular blood testing for routine, pre-surgery, dental care, or any medical procedure. The classical presentation of T2DM like polyuria, polydipsia and fatigue is observed mainly in older individuals. Often recurring bacterial and fungal infections, blurred vision and delayed wound healing are classically observed in patients, especially older individuals.

With a majority of the cases being asymptomatic, the patient may present to the clinician with a macrovascular complication of coronary heart disease, peripheral vascular disease, cerebrovascular disease or a microvascular one of diabetic nephropathy, retinopathy, Neuropathy, or diabetic foot ulcer. In the recent years cancers (hepatocellular, pancreatic, colorectal, etc.), infections, Non-Alcoholic Fatty Liver Disease including steatohepatitis and cirrhosis, obstructive sleep apnoea, affective disorders, dementia, erectile dysfunction and functional disability at workplace is also considered as emerging complications of T2DM. In severe cases especially in older individuals, hyperosmolar coma is observed especially during medications for major events like myocardial infraction and stroke.¹⁴

The assessment of a patient with T2DM shall first involve the diagnosis and confirmation of the type of diabetes by blood glucose and HbA1c evaluation. Additional evaluation includes the assessment of the diabetes complications, presence of co-morbidities and overall health status. The clinician must explore behavioural factors (eating patterns, calorie counting, physical activities, sleep behaviour, addictions), medications and vaccinations, technology use and social life assessment. A comprehensive physical examination of the patient must be conducted with special emphasis on fundoscopic examination, skin examination, foot examination, cognitive function, mental state examination and bone health assessment.¹⁵

DIFFERENTIAL DIAGNOSIS

Table 1

Condition	Differential features
Type 1 Diabetes Mellitus ¹⁶	<ul style="list-style-type: none"> • Associated with autoimmune β cell destruction of the pancreas • Onset in a younger age group • Family history of auto-immunogenicity • Serum insulin levels are diminished • C-peptide levels are diminished <200 pmol/L • Detection of antibodies in serum
Maturity onset of diabetes in Young/ Monogenic diabetes ¹⁶	<ul style="list-style-type: none"> • Onset at an age before 25 years of age • Impaired serum insulin levels • Usually, obesity is not co-existent
Diseases of the exocrine pancreas ¹⁶	<ul style="list-style-type: none"> • Associated with conditions like pancreatitis (acute or chronic), trauma/pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, etc. • Demonstration of pancreatic injury by blood parameters like amylase, lipase, faecal elastase and imaging studies.
Stress induced hyperglycaemia ¹⁵	<ul style="list-style-type: none"> • Usually noted in persons within 48 hours of hospital admission • Blood levels 180 mg/dl and above • Increased levels of cytokines, cortisol, glucagon, catecholamines in blood.
Medications like steroids ¹⁷	<ul style="list-style-type: none"> • Develops due to side effects of glucocorticoids used as anti-inflammatory or immunosuppressive purposes • Mostly observed with oral and injected glucocorticoids
Acromegaly ¹⁸	<ul style="list-style-type: none"> • Increased secretion of Growth Hormone and Insulin like Growth Factor-1 results in gluconeogenesis, impairs insulin sensitivity • Characteristic physical appearance • Often surgery for pituitary tumour causing reversal of diabetes
Cushing's Disease	<ul style="list-style-type: none"> • Circulating glucocorticoids results in increased glucose levels in the blood. • Cortisol levels after dexamethasone suppression test aids in the diagnosis.

SUPPORTIVE INVESTIGATIONS

i Essential Investigations:

- Blood Sugar Profile:
- Fasting Blood sugar (FBS) \geq 126 mg/dL
- Post-prandial Blood sugar (PPBS) \geq 200 mg/dL

- Glycated Haemoglobin HbA1c \geq 6.5%
- Complete haemogram.
- Urine examination for glucose, proteins, ketone bodies and microscopic examination for pus cells.

ii Advanced Investigations:

- Blood for serum creatinine, lipid profile and liver function tests
- Serum electrolytes, Blood urea, Urine microalbumin
- Creatinine clearance and ACR

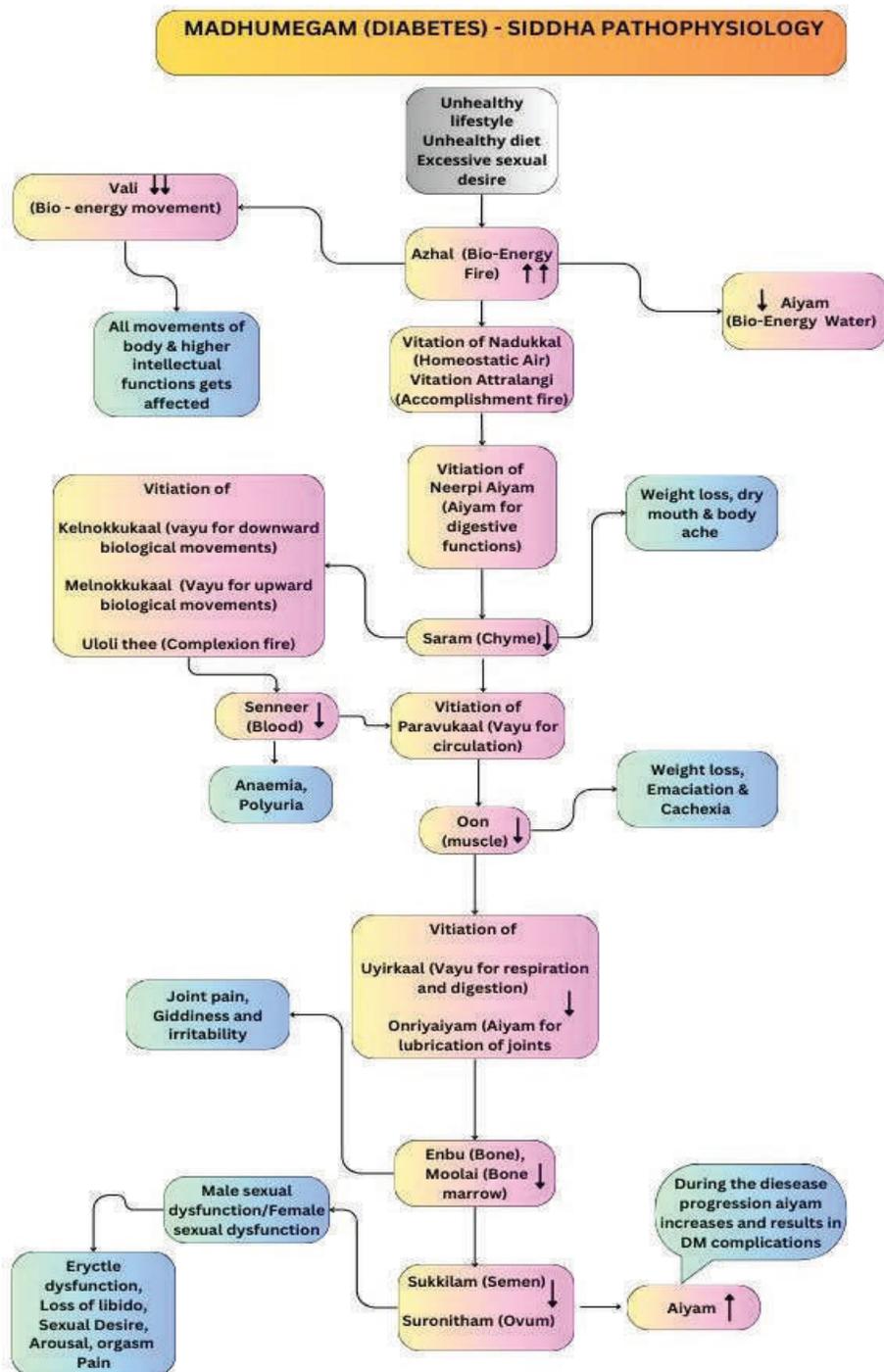


Fig. 1 Diabetes Pathophysiology Siddha

DIAGNOSTIC CRITERIA

The diagnosis of Diabetes Mellitus among non-pregnant individuals has been defined by the American Diabetes Association (ADA) and Research Society for the Study of Diabetes In India (RSSDI) as per the following criteria:¹⁶

Table 2

Criteria of diagnosis of Diabetes among non-pregnant individuals
HbA1c \geq 6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay*
Or
FPG \geq 126 mg/dL. Fasting is defined as no caloric intake for at least 8h*
Or
In an individual with classic symptoms of hyperglycaemia or hyperglycaemic crisis, a random plasma glucose \geq 200 mg/dL. Random is any time of the day without regard to time since previous meal.

*In the absence of unequivocal hyperglycaemia, diagnosis requires two abnormal test results obtained at the same time (e.g., HbA1c and FPG) or at two different time points.

The criterion for specific detection of T2DM is difficult and diagnosis is often mistaken especially in ~40% of adults with new onset of Type 1 diabetes mellitus and maturity-onset diabetes in young.

Pre-diabetes:

Pre-diabetes is defined as a clinical condition where the levels of glucose and HbA1c do not meet the criteria for diabetes, but yet the individual suffers from abnormal carbohydrate metabolism. The condition poses significant risk for the progression to overt Diabetes, cardiovascular diseases and several other cardio-metabolic outcomes.

The criteria for diagnosis of prediabetes have been defined by the American Diabetes Association and RSSDI as follows:

Table 3

Impaired fasting glucose (IFG): FPG 110 mg/dL to 125 mg/dL
Or
HbA1c \geq 5.7%-6.4%

Siddha Assessment

Eṇvakai Tērvu (Eightfold examination)^{3,19}

- *Nāṭi* (Pulse) – *Vali aḷal/Ayya vali/Vali Aiyam/Rapidly pulsating aḷal naadi/Aḷal naadi* like worm
- *Sparisam* (Touch) – Warmth/ dryness/ light brown or red scaly patches
- *Nā* (Tongue) – Pallor/dryness/ fissured/ sweet taste
- *Nīram* (Colour) – Pallor/dark
- *Moḷi* (Speech) – Normal/low pitched
- *Viḷi* (Eye) – Red/pallor, dryness/reduced touch sensation, visual impairment/ distorting vision/floaters

- *Malam* (Stool) – Normal / constipated, Yellowish
- *Mūttiram* (Urine)
- a) *Nīrkuṛi* (Uro-macroscopy)
 - *Nīṛam* (Colour) – Ghee/Cow's urine/ meat/ toddy colour urine represent
 - *Aṭartti* (Specific gravity) – *Vitiated vali*, Dark yellow colour- *Vitiated aḷal*
 - *Maṇam* (Odour) – Crystal clear urine –*Vitiated aiyam*, Dense
 - *Nuṛai* (Froth) – Smells like honey/ghee/cow's urine/ toddy/meat Increased in later stages
 - *Eñcal* (Deposit) – Small deposit in urine
 -
- b) *Neykuṛi* (Oleo uro-macroscopy) – Oil may spread in the form of sieve / ring/ fast dispersal / irregular margin. If the oil is immersed in the urine it denotes *Aiya neer*.

PRINCIPLES OF MANAGEMENT

Red Flag signs:

These conditions are addressed by modern medicine, hence they should be assessed before initiating the Siddha treatment.

- Hypoglycaemia
- Hyponatremia
- Severe cardiovascular disease including valvular and ischemic heart disease
- Cerebro vascular accident
- Severe associated infective morbidity like pneumonia, tuberculosis, sepsis, etc
- Advanced stages of malignancy
- Visual loss due to diabetic retinopathy
- Severe motor or autonomic dysfunction
- Severe renal dysfunction with severely reduced GFR
- Diabetic ketoacidosis
- Hyperosmolar nonketotic coma

A) Preventive management²⁰

Prevention of diabetes includes primary, secondary and tertiary management of the condition. The primary measures shall target persons with obesity/increased BMI. A targeted 7% weight loss and moderate physical exercise may be useful for prevention or reversal of the disease. Trials also suggest that individualized low- calorie diet plans and lifestyle/ behavioural therapy result in the prevention or delay of T2DM and related cardiovascular morbidity. Opportunistic screening must be conducted for the following criteria.

Table 4

Persons with age of 18 years and above
Persons with a high BMI (≥ 25 kg/m ²)
Women with a history of gestational diabetes
First- or second-degree relative with diabetes

Hypertensive individuals
Sedentary lifestyle
Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovarian syndrome, small-for-gestational age birth weight)
If results are normal, testing should be repeated at a minimum of 3-year intervals, with consideration of more frequent testing depending on initial results and risk status.

Yogam and Pranayamam Adherence to practices of yoga and physical exercises on a regular basis will help regulate the eating patterns and aid physical fitness thereby facilitating good glycaemic control.

The general guidelines of **Yogasanam** recommended for T2DM patients

Table 5

Criteria	Yoga Techniques	Approximate duration	Effects
Asanas (Yoga postures)	<i>Trikonasana</i> (Triangle pose)	Recommended to hold the final pose for 15 seconds, gradually increasing the duration up to 1 minute	Enhances insulin receptor expression in the muscles, causing increased glucose uptake by muscles. Have positive effects on glucose utilization and fat redistribution in T2DM
	<i>Tadasana</i> (Palm tree pose)		
	<i>Vakrasana</i> (Spinal twist)		
	<i>Paschimottasana</i> (Seated forward bend)		
	<i>Bhujangasana</i> (Cobra pose)		
	<i>Naukasana</i> (Boat pose)		
	<i>Pavanamuktasana</i> (Wind releasing pose),		
	<i>Setubandhasana</i> (Bridge pose)		
	<i>Sarvangasana</i> (Shoulder stand)		
	<i>Surya namaskara</i>	Slow speed, 3–7 rounds according to an individual's capacity	Stimulates insulin production through brain signaling Significantly decreases hip circumference, exerting beneficial effects on glycaemic outcomes
Pranayama (Yogic breathing)	<i>Anuloma viloma</i> (Alternate nostril breathing)	5–10 minutes	Improves components of health- related fitness, i.e., cardiorespiratory endurance, flexibility, and body fat percentage
	<i>Chandra bhedana</i> (Left nostril breathing)	5 minutes	Parasympathetic stimulation

Criteria	Yoga Techniques	Approximate duration	Effects
	<i>Surya bhedana</i> (Right nostril breathing)	5 minutes	Sympathetic stimulating effect; may be recommended in people with diabetes.
	<i>Bhastrika</i> (Bellows breath)	3–5 minute	Regulation of pineal, pituitary, and adrenaline glands, important role in the regulation of metabolism
	<i>Bhramari</i> (Humming bee breath)	3–5 minutes	Soothing and calming effect on the mind, improves mental and physical health
	<i>Sheetali/Sitkari</i> (Cooling breath)	5 rounds	Lowers blood pressure, cooling effect
<i>Bandha</i> (Lock)	<i>Uddiyan bandha</i> (Abdominal lock)	5 rounds	Negative pressure created in the abdominal cavity may improve pancreatic function
<i>Mudras</i> (Hand gestures)	<i>Linga mudra, surya mudra, prana mudra, apana mudra, gyana mudra</i>	15–45 minutes	Promote deep relaxation and eliminate stress. Boost metabolic rates, promote weight loss, and reduce sugar levels.
<i>Shuddhi kriya</i> (Cleansing processes)	<i>Kapalbhati</i> (Frontal brain purification)	5 rounds, 120 strokes	Abdominal pressure created during exhalation improves the efficiency of β -cells of the pancreas Helps in the production of insulin and controlling glucose levels in the blood
	<i>Agnisara kriya</i> (Stimulating the digestive fire)	5 rounds	The 'vacuum' effect of this action massages the internal organs and increase blood flow to the area Boosts metabolism and facilitates proper functioning of the abdominal organs
	<i>Vaman dhauti</i> (Stomach cleansing)	Once a week	Increases glucose uptake, minimizes insulin resistance, and promotes the function of insulin by reducing levels of circulating free fatty acids in the body
	Full <i>shankhprakashalana</i> (Intestine cleansing)	Once a year	Significantly reduces blood glucose levels, Increases insulin production
	<i>Laghu shankhprakashalana</i> (Short cleansing)	Every 40 day	
<i>Dhyana</i> (Meditation)	Meditation	10 minutes or more	Beneficial psychological effects, such as faster reactions to stimuli and being less prone to various forms of stress

*Yoga and exercise should be performed as per the advice of qualified yoga instructor or physiotherapist

ICMR guidelines explain four stages of opportunities for the prevention of diabetes.

a) Primordial prevention attempts to reduce the risk factors for diabetes, e.g., reducing or preventing obesity to reduce the future risk of diabetes.

b) Primary prevention targets people who are in the stage of pre-diabetes to prevent the onset of diabetes.

c) Secondary prevention is to prevent the onset of complications in those who are already diagnosed with diabetes.

d) Tertiary prevention of diabetes is aimed at limiting physical disability and rehabilitation measures in those who have already developed diabetic complications and preventing them from going into end-stage complications of diabetes.

Siddha System of Medicine emphasis adhering to *Tēraiyaṟ piṇi aṇukā viti* for prevention of disease and lead to healthy life. Thus, personalized daily and seasonal regimens of food and lifestyle are the key advantages of the Siddha medical system to prevent T2DM.

Table 6

Dietary Habits (Uṇavu Muṛaikaḷ)	
Do's - Pattiyam	Don'ts - Apattiyam
<ul style="list-style-type: none"> • Eat consistently at an interval of 3 - 5 hrs. daily • Include traditional rice varieties like Pūṅkār, kāṭṭu yāṇam karuppu kavuṇi, māppiḷai campā, iluppai campā, kuḷḷakkār • Millet diet advised 3 days/week • Include mostly whole grains, legumes, greens and mostly vegetables • Prefer sea fish varieties • Increase fiber rich food • Include non-starchy vegetables • Include lean proteins and low fat dairy in diet 	<ul style="list-style-type: none"> • Avoid skipping meals • Food should never be consumed during excessive hunger, anger or grief • Avoid root tubers except yam – <i>Typhonium trilobatum (L.) Schott</i> • Avoid highly processed refined carbohydrate diet and advised to take complex carbohydrates • Limit added sugars and refined grains • Strictly avoid sweets, carbonated drinks • Avoid saturated food and trans fats • Avoid heavy meals late at night • Avoid Cold, Stale, and Heavy Foods
Lifestyle Practices (Vāḷviyal Muṛaikaḷ)	
Do's	Don'ts
<ul style="list-style-type: none"> • Practice post meal walk • Practice at least 30 minutes of moderate activity (eg: walking) 5 days a week • Prefer left lateral position for sleeping • Better balance of mood and sleep • Practice regular meditation • Consume food to the level of hunger • Undergo therapeutic purgation once in four months 	<ul style="list-style-type: none"> • Avoid daytime sleep • Avoid excessive sexual indulgence • Don't suppress any natural urge • Avoid sedentary life style • Avoid stress • Don't Self-Medicate

CURATIVE INTERVENTIONS

At every level of care, if the patient is already under standard care, the physician may advise continuing the same along with add-on Siddha and can be assessed for the same in the follow-ups for tapering or discontinuing the treatment in consultation with a conventional physician.

- **Clinical Diagnosis**

Type 2 Diabetes mellitus presents at the clinic in an adult with either the classical presentation of polydipsia, polyuria, fatigue, or often as an incidental discovery of raised blood glucose levels during a routine health check-up. There may be an increase in occurrences of bacterial and fungal infections and pruritus vulva in women. In many cases, any disease complication may be the initial presenting symptom of the disease. Patients may also present with levels of prediabetes on incidental discovery. The diagnosis will be made by using the following investigations:

- Blood Sugar Profile: Fasting Blood sugar (FBS) \geq 126 mg/dL, Post-prandial Blood sugar (PPBS) \geq 200 mg/dL, Glycated Haemoglobin HbA1C \geq 6.5%.
- Urine examination for glucose, proteins, ketone bodies, and microscopic examination for pus cells.
- Serum creatinine, lipid profile and liver function tests.

Management:

Patients may seek Siddha management for different stages of T2DM i.e., pre-diabetic/ newly detected/diabetic with various complications and the line of treatment may vary accordingly. The first line of treatment is to normalize the altered or deranged humours and revitalization of seven fundamental tissues through detoxification methods followed by internal medications.

The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

Day 1

i. *Eṇṇey kuḷiyal* (Therapeutic oilbath):¹⁹

Eṇṇey kuḷiyal is a preparatory procedure in which medicated oil massage with a bath of lukewarm water. It will strengthen the five sensory organs. According to disease severity, *eṇṇey kuḷiyal* can be advised for one day to three days.

- *Keezhanelli Thylam* – Quantity sufficient (External use)
- *Arakku Thylam* – Quantity sufficient (External use)

Note: Anyone of the *Thylam* may be used

Rules to be followed during *Eṇṇey kuḷiyal*:

Apply oil before 7 am. Instil one drop in each eye, two drops in each nostril and three drops in each ear. Spread over the medicated oil from head to foot and give a gentle massage. After application, leave it for 45 minutes and bathe with lukewarm water using herbal hair wash powder.

Take tender vegetables and easily digestible food. Avoid daytime sleep, intercourse and exposure to sunlight and cold items on the day of the oil bath.

Day 2

ii. *Kaḷiccal maruttuvam* (Therapeutic Purgation):

- *Agathiyar Kuzhambu*-100-130 mg with *Chukku Karkam* (*Zingiber officinalis*), OD, in the early morning on an empty stomach.¹⁹

Rules to be followed during Therapeutic Purgation:

- The medication should be taken in the early morning 5 to 6 AM
- After the average number (5-6 times) of bowel evacuations, watery diarrhoea commences. In this stage, the patient is advised to take buttermilk/ lemon juice/fried cumin seeds decoction/Ash of sweet flag (*Vacampū*).
- After purgation, the patient may have symptoms like tiredness, slimness, lightness of the body and tiredness of sense organs which is a good sign.
- Dietary regimen during purgation:
 - Mōr (Butter milk)
 - Kañci (Rice porridge)
 - Irumuṛaivaṭṭitta kañci (Double boiled porridge)
 - Kāyntāriya vennīr (Lukewarm water)
- Precautions:
 - Avoid daytime sleep during purgation therapy
 - Should not take heavy meals before or during the procedure
 - Avoid intercourse

Day 3 onwards

A. Internal medicine

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 7: Single herbs

S. No.	Single herbs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ <i>Aṇupāṇam</i>
1.	<i>Aavarai (Cassia auriculata)</i> flower decoction ²¹	Decoction	30-60ml	BD	40 days	Nil
2.	<i>Kovai (Coccinia grandis)</i> tuber juice ²¹	Juice	10-15ml	BD after food with diuretic drugs	--	Nil
3.	<i>Nilappanai Ver Chooranam</i> ²¹	Root powder	8g	BD after food	--	Water
4.	<i>Kalyaṇa murukku (Erythrina variegata)</i> root decoction ²¹	Decoction	80-90ml	BD after food	--	<i>Vasantha kusumakara Maathirai</i>
5.	<i>Thottarsurungi (Mimosa pudica)</i> Chooranam ²¹	Leaf and root Powder	4-8g	BD after food	--	Milk
6.	<i>Peercku (Luffa Acutangula)</i> ²¹	Leaf juice	5ml	BD after food	--	Nil

Table 8: Compound formulations

S.No.	Compound formulations	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Anupaanam
Kudineer / Decoction						
1.	<i>Aavarai Kudineer</i> ²²	Decoction	60- 80ml	BD before food	40 days	--
2.	<i>Vilva ilai Kudineer</i> ¹⁹	Decoction	30- 60ml	BD before food	-	--
3.	<i>Vilvathy Kudineer</i> ²³	Decoction	40- 80ml	BD before food	-	-
4.	<i>Seenthil kodi Kudineer</i> ²⁴	Decoction	30- 60ml	BD before food	--	--
Chooranam / Medicinal Powder						
1	<i>Aavaraiyathi pattai Chooranam</i> ¹⁹	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
2	<i>D5 Chooranam</i> ²⁵	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
3.	<i>Veppampisin Chooranam</i> ²³	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
4.	<i>Kadal Azhingil Chooranam</i> ¹⁹	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
5.	<i>Mathumega Chooranam</i> ²⁶	Medicinal Powder	1-2 g	TDS before food	--	Lukewarm water
6.	<i>Seenthil Chooranam</i> ³	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
7.	<i>Naaval Chooranam</i> ³	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
8.	<i>Santhana Chooranam</i> ³	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
9.	<i>Thiripala Chooranam</i> ²⁷	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
10.	<i>Sanjeevi Chooranam</i> ²⁸	Medicinal Powder	1-2 g	BD after food	--	Lukewarm water
Maathirai / Tablet						
1.	<i>Naaval kottai Maathirai</i> ²⁹	Tablet	1	BD after food	-	Water
Ilakam / Electuary						
1.	<i>Lavanga Ilakam</i> ³	Electuary	2-3 g	BD after food	-	-

Recommended diet and lifestyle

Diet

The diet is responsible for promoting weight loss, improving glycaemic control and reducing of cardiovascular complications.³⁰ Carbohydrates in the diet (50-60% of total caloric intake) should include grains with low glycaemic index and low glycaemic load. Complex

carbohydrates must be preferred over refined products. Total fiber consumption should be 25-40 g/day. Protein intake must be 15% of the total caloric intake depending on the age, sarcopenia, and renal function. Oils rich in MUFA and PUFA must be advised.

Dietary habits

- A diet rich in fruits, nuts, leafy vegetables, fiber, whole grains and unsaturated fat is preferred. The plate must also include pulses, legumes, unprocessed vegetables and low-fat dairy.
- Change in eating patterns like early dinner must be advised.
- Extreme diets like low-carbohydrate ketogenic diet must be planned and executed in consultation with a physician and trained nutritionist, and for a short period.
- Intermittent fasting reduces body weight and reduces diabetes parameters such as fasting glucose, fasting insulin, insulin resistance (HOMA-IR) index, and glycated hemoglobin (HbA1c).³¹

Siddha Culinary Medicine ^{3,19,24}

- Advice to take food after sunrise and before sunset.
- Millet diet may be advised 3 days/ week.

Table 9: Preferred food according to Siddha

Rice	<ul style="list-style-type: none"> • Hand pounded boiled rice • <i>Maṇicampā</i> rice (<i>Oryza sativa</i>) • <i>Kēḷvaraku/Raagi</i> (<i>Eleusine coracana</i>) • <i>Kambu/Pearl Millet</i> (<i>Pennisetum typhoides</i>) • <i>Tiṇṇai/Foxtail millet</i> (<i>Setaria italica</i>) • <i>Saamai</i> (<i>Panicum sumatrense</i>) • <i>Mūṅkil</i> rice (<i>Bambusa arundinaceae</i>),
	<ul style="list-style-type: none"> • Any one of the above items can be consumed per day. • They can be taken in various forms like <i>Kichadi</i>, <i>Upma</i>, <i>Pongal</i>, and <i>Poha</i> except as porridge • Avoid dishes made from batter
Tender vegetables	<ul style="list-style-type: none"> • <i>Pākal</i> / Bitter guard (<i>Momordica charantia</i>) • <i>Sūrai</i>/ Bottle guard (<i>Lagenaria siceraria</i>) • <i>Veṇṭai/Ladies' finger</i> (<i>Abelmoschus esculentus</i>) • <i>Avarai/Broad beans</i> (<i>Lablab purpureus</i>) • <i>Muruṅkai /Drumstick</i> (<i>Moringa oleifera</i>) • <i>Ciṇṇa veṅkāyam/Shallots</i> (<i>Allium cepa</i>) • <i>Suṇṭai/Turkey berry</i> (<i>Solanum torvum</i>) • <i>Kōvai/Coccinia</i> (<i>Coccinia grandis</i>) • <i>Vāḷai piṅcu</i> (<i>Musa paradisiaca</i>), • <i>Pālā piṅcu</i> (<i>Artocarpus heterophyllus</i>) <p>Any one or more vegetables can be included in the daily diet in the forms of soup, salad, veg curry, etc.</p>
Greens	<ul style="list-style-type: none"> • <i>Muruṅkai</i> (<i>Moringa oleifera</i>) • <i>Maṇattakkāḷi</i> (<i>Solanum nigrum</i>) • <i>Kīrai taṇṭu</i> (<i>Amaranthus gangeticus</i>) • <i>Kottumalli/Coriander</i> (<i>Coriandrum sativum</i>)

	<ul style="list-style-type: none"> • <i>Putiṇā</i> /Mentha (<i>Mentha arvensis</i>) • <i>Karuvēppiḷai</i>/Curry leaves (<i>Murraya koenigii</i>) • <i>Puliyārai</i>/Creeping wood sorrel (<i>Oxalis corniculata</i>) • <i>Vacalai</i>/Chickenweed (<i>Portulaca quadrifida</i>) • <i>Arukīrai</i>/Amaranthus (<i>Amaranthus tristis</i>) • <i>Tāḷi kīrai</i> (<i>Ipomoea marginata</i>) • <i>Vēntaya kīrai</i> /Methi leaves (<i>Trigonella foenumgraecum</i>) • <i>Koṭivācalai</i>/Climbing spinach(<i>Basella alba</i>) • <i>Puḷiyarai</i>/Creeping wood sorrel(<i>Oxalis corniculata</i>) • <i>Tūttuvaḷai</i> (<i>Solanum trilobatum</i>) • <i>Vallaik koṭi</i> (<i>Convolvulus repens</i>) <p>Traditional recipes like <i>kūṭṭu</i>, <i>poriyal</i>, and <i>racam</i> of madeup of the greens can be included in the daily diet</p>
Fruits	<ul style="list-style-type: none"> • <i>Atti</i>/Fig (<i>Ficus racemosa</i>) • <i>Koyyā</i>/Guava (<i>Psidium guajava</i>) • <i>Mātuḷai</i>/Pomegranate (<i>Punica granatum</i>) • <i>Pappāḷi</i>/Papaya (<i>Carica papaya</i>) • <i>Nāval</i>/Jamun fruit (<i>Syzygium cumini</i>) • <i>Nelli</i>/Indian gooseberry (<i>Phyllanthus emblica</i>) • <i>Vāḷai</i>/ <i>Musa textilis</i> (Banana – peyan type) <p>A small bowel of fruit salad of the above fruits can be included daily</p>
Pulses	<ul style="list-style-type: none"> • <i>Uḷuntu</i>/Urad dal (<i>Vigna mungo</i>) • <i>Pācipparuppu</i>/Moong dal (<i>Vigna radiata</i>) • <i>Koṇṭai kaṭalai</i>/Channa dal (<i>Cicer arietinum</i>) • <i>Thuvarai</i>/Toor dal (<i>Cajanus cajan</i>) • <i>Kollu</i>/Horse gram (<i>V.unguiculeta</i>) <p>These pulses can be used as sprouts and in the form of various culinary preparations like <i>Sāmpār</i>, <i>Vaṭai</i>, <i>Dōcai</i>, <i>Aṭai</i>, Variety rice etc.</p>
Nuts	<ul style="list-style-type: none"> • <i>Vātumai</i> (<i>Prunus dulcis</i>) • <i>Muntiri</i> (Cashew)
Dairy products	<ul style="list-style-type: none"> • <i>Mōr</i>/Buttermilk <p>1-2 glass of buttermilk can be consumed in the afternoon to enhance gut microbiota</p>
Non-vegetarian diet	<ul style="list-style-type: none"> • <i>Ayirai mīṇ</i>/Spined Loaches (<i>Cobitis taenia</i>) • <i>Kāṭai</i>/Quail (<i>Coturnix coturnix</i>) • <i>Kautāri</i>/ Grey francolin (<i>Francolinus pondicerianus</i>) • <i>Veḷḷāṭu</i>/Goat (<i>Capra aegagrus hircus</i>) <p>Any of the above non-vegetarian items can be consumed once a week</p>

Physical exercise

- Physical activity must be included on the basis of patient's willingness and ability.
- ≥ 30 minutes of moderate-intensity aerobic exercise each day including, swimming, walking, cycling, running, jogging, and rowing.
- 15-30 minutes of work-related activity.
- 15 minutes of muscle-strengthening exercise (at least 3 times a week), which includes

lifting weights, working with resistance bands, hill climbing/ inclined walking, sit-ups, and squats.

- At least 5000 steps per day
- A minimum of 150 minutes/week of exercise is recommended for healthy Indian individual considering the high risk of T2DM and CVD.
- Use of smart watch or fitness bands for monitoring of physical activity must be encouraged.

Varma Maruttuvam³²

- *Koṇṭai kolli varmam*
- *Vāyu kalam*
- *Nāṅkāṇa poṭṭu*
- *Aāmaikālam*
- *Urumikālam*
- *Tummi kālam*
- *Aṭappā kālam*
- *Muṇṭellu varmam*
- *Aṇṇa varmam*

Precautions and contraindications for Siddhar Varmam³²

- *Varmam* should be done by Siddha physician
- The better posture for *Varmam* is sitting and analyse *Nāṭi* before performing *Varmam*
- Beginners should avoid extreme practices
- Monitor carefully for any reactions to any new fitness activity

1) Restricted diet (Pathiyam) and lifestyle

- Consumption of processed grains should be avoided.
- Intake of red meat must be limited. Fats should be < 30% of the total caloric intake especially from nuts and seeds.
- Saturated fats like butter, ghee, margarine, coconut oil must be limited to <10% of caloric intake. Use of hydrogenated vegetable oils and re-cooking or re-frying of oil must be avoided.
- Sugar intake must be reduced to 6 teaspoons (25g) daily. Salt intake must be restricted to <5 g/day. Artificial sweeteners must be avoided as it alters the gut microbiota and increases insulin resistance.
- Sweetened beverages must be avoided.
- Avoid smoking and alcohol.

2) Follow-up: At an interval of 7 days or as per the need.

3) Reviews should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.
- Management of T2DM in terms of diet, exercise, and other interventions.

- Discussing the person's knowledge of the condition, any concerns they have, their personal preferences, and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.
- Self-management support.
- Monitoring the long-term course of the condition with periodic review.

4) Referral criteria

- Nonresponse to treatment
- Target organ involvement and investigations
- Complications of diabetes mellitus including all macrovascular, microvascular, and emerging complications
- Complications related to glycaemic control including uncontrolled hyperglycaemia and frequent hypoglycaemic episode.
- Substantial impact on their quality of life and activities of daily living
- Diagnostic uncertainty

At Level 2 (CHC/Small hospitals (10-20 bedded hospitals with basic facilities such as routine, investigation, X-ray)

Clinical Diagnosis: Same as Level 1. Any fresh case or referred case from Level 1 shall be evaluated thoroughly for confirmation of diagnosis and complications.

Investigations: Same as Level 1.

Supportive investigations to assess organ involvement include:

1. Serum electrolytes
2. Blood urea
3. Urine microalbumin, creatinine clearance, ACR
4. Electro-cardiography
5. Chest skiagram- Postero-anterior view
6. Ophthalmoscopic examination

Management: Same as Level 1. For the patients referred from Level-1, treatment given in Level-1 may be continued if appropriate for the presenting condition or the case may be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the medications mentioned for Level-1 may also be considered; however, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient. Complications of the disease are the important clinical presentations at this stage of care especially the early signs and symptoms of such complications. Conditions like diabetic foot ulcers may require surgical debridement of the lesion and antiseptic dressing along with integrative management for glycaemic control. Hypoglycaemia state requires acute management by fast-acting glucose and long-term management with constitutional treatment. In case of hypoglycaemia, patients on oral hypoglycaemic agents and/or insulin therapy may require a review of the dosage of conventional medications.^{33,34} Other complaints of neurological, ophthalmological, hepatic, cardiovascular, and nephrological involvement may be managed by integrative management of Siddha and Modern Medicine.

Management: (Along with level 1 medications)

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 10: Compound formulations

S.No	Compound formulations	Dosage form	Dose	Time	Duration and Frequency	Adjuvant (Anubanam)
Chooranam / Medicinal Powder						
1.	<i>Pungampoo Chooranam</i> ²⁸	Medicinal Powder	1-2 g	OD	45 days	Lukewarm water
2.	<i>Keezhaneli Chooranam</i> ¹⁹	Medicinal Powder	1-2 g	BD, after food	--	Lukewarm water
3.	<i>Aavaraiyathi pattai Chooranam</i> ¹⁹	Medicinal Powder	1-2 g	BD, after food	--	Lukewarm water
4.	<i>Kadalazhinjil Chooranam</i> ¹⁹	Medicinal Powder	1-2 g	BD, after food	--	Lukewarm water
Nei / Medicated Ghee						
1.	<i>Naval pattai Nei</i> ⁽²⁸⁾	Medicated Ghee	2-5ml	BD, after food	---	--
2.	<i>Perichangai Nei</i> ⁽²⁸⁾	Medicated Ghee	5-10ml	BD, after food	-	--
Iracāyaṇam / Semi-solid confection						
1.	<i>Kathali poo rasayanam</i> ⁽²⁸⁾	Semi-solid confection	5-10 g	BD, after food	--	--
Eṇṇey / Medicinal oils						
1.	<i>Kannaththennai</i> ⁽²⁸⁾	Medicinal oils	1-5 ml	OD	--	Till the disease gets cured
Parpam / White Calx						
1.	<i>Velvanga Parpam</i> (27)	White Calx	65 -130 mg	BD, after food	--	Ghee
2.	<i>Aya Parpam</i> ¹⁹	White Calx	30-65mg	BD, after food	--	water/ milk
3.	<i>Velli Parpam</i> ¹⁹	White Calx	65-130 mg	BD, after food	--	<i>Nīrmuḷḷi flower (Hygrophila auriculata) juice</i>
Chenduram / Red Calx						
1.	<i>Aya Chenduram</i> ¹⁹	Red Calx	65-130 mg	BD, after food	--	<i>Aracampiṅcu powder (Ficus religiosa) / Athimathura Chooranam/ honey / ghee / Ālam piṅcu powder (Ficus benghalensis),</i>
2.	<i>Kaantha Chenduram</i> ¹⁹	Red Calx	100 - 130 mg	BD, after food	--	Honey

Recommended Diet & Lifestyle

Restricted Diet & Lifestyle

same as in level 1

Follow-up: At an interval of 7 days or as per the need.

Referral criteria:

Same as level 1 and nonresponsive to treatment

At Level 3 (Ayush hospitals attached with teaching institution, District Level/Integrated/ State Ayush Hospitals, Tertiary care allopathic hospitals having Ayush facilities), multiple departments/facilities for diagnosis and interventions.

- **Clinical Diagnosis:** Same as Level 1 and 2. Confirmatory diagnosis with advanced biochemistry and serological tests. Evaluation and assessment of complications.

Investigations: Same as Levels 1 and 2. Additional Investigations may be done as follows:

- Ultrasonography with colour doppler for upper and lower extremity arteries
- Nerve conduction velocity tests
- Electroencephalogram
- Serum C-peptide, Insulin autoantibodies, and Fasting insulin levels
- Genetic testing (INSR Single Gene Test)
- Psychological assessment with a trained psychiatrist

Management: Same as Levels 1& 2. For the patients referred from Level-1 or 2, treatment given in Level-1 &/or 2 may be continued if appropriate for the presenting condition or the case may be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient.

The treatment strategy includes single herbs with Herbo-mineral formulations and other supportive medicines to prevent T2DM complications. Along with stage I& II medicines, the following medicines can be advised according to the discretion of the physician.

(**Note:** Administration of medicine, dosage and treatment duration may vary according to the condition of patient and disease severity. Administration of mineralo-metallic medicines shall be prescribed with a drug holiday as ascertained by the treating Siddha Physician).

Table 11

S.No.	Compound formulations	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ <i>Aṇupāṇam</i>
Maathirai/ Tablet						
1	<i>Maha elathy Maathirai</i> ⁽²⁷⁾	Tablet	(50 mg) -1-2 pills	BD	--	Lukewarm water
Parpam / White calx						
1.	<i>Abraka Parpam</i> ⁽²⁷⁾	White calx	30-60 mg	BD	45 days	<i>Ney/Ghee, Vetrilai charu/</i> betel leaves juice

S.No.	Compound formulations	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ <i>Aṇupāṇam</i>
2.	<i>Gandaga Parpam</i> ¹⁹	White calx	25-50 mg	BD	-	<i>Ghee/ butter</i>
Chenduram/Red calx						
1.	<i>Abraka Chenduram</i> 28	Red calx	130 mg	BD after food	16 days /10 days drug free then 16 days	<i>Ghee for Vatha thaegi Honey for Pittha thaegi</i>
2.	<i>Abraka Chenduram</i> 19	Red calx	100-150mg	BD after food	45 days	<i>Ghee or betel leaves juice</i>
3.	<i>Gowri chinthamani Chenduram</i> ²⁷	Red calx	60-130 mg	BD after food	40 days	<i>Avarai Kuli Thylam</i>
4.	<i>Naga Chenduram</i> ¹⁹	Red calx	100 - 200 mg	BD, after food	--	<i>Thirikadugu Chenduram along with ghee/ honey/ milk,</i>
5.	<i>Sornabiraka Chenduram</i> ¹⁹	Red calx	30 - 60 mg	BD, after food	--	<i>Seenthilathi Ilakam</i>
6.	<i>Poorna Chandrothayam</i> ¹⁹	Red calx	30-65 mg	BD, after food	--	<i>Karpoorathi Chooranam and leaf juice of <i>verrilai</i> (Piper betel),</i>
Mezhugu / Medicated wax						
1.	<i>Van Mezhugu or Indu varna Mezhugu</i>	Medicated wax	50-100 mg	BD	3 or 5 days	<i>Panai vellam/Palm jaggery</i>

Recommended diet and lifestyle: Same as Levels 1 and 2

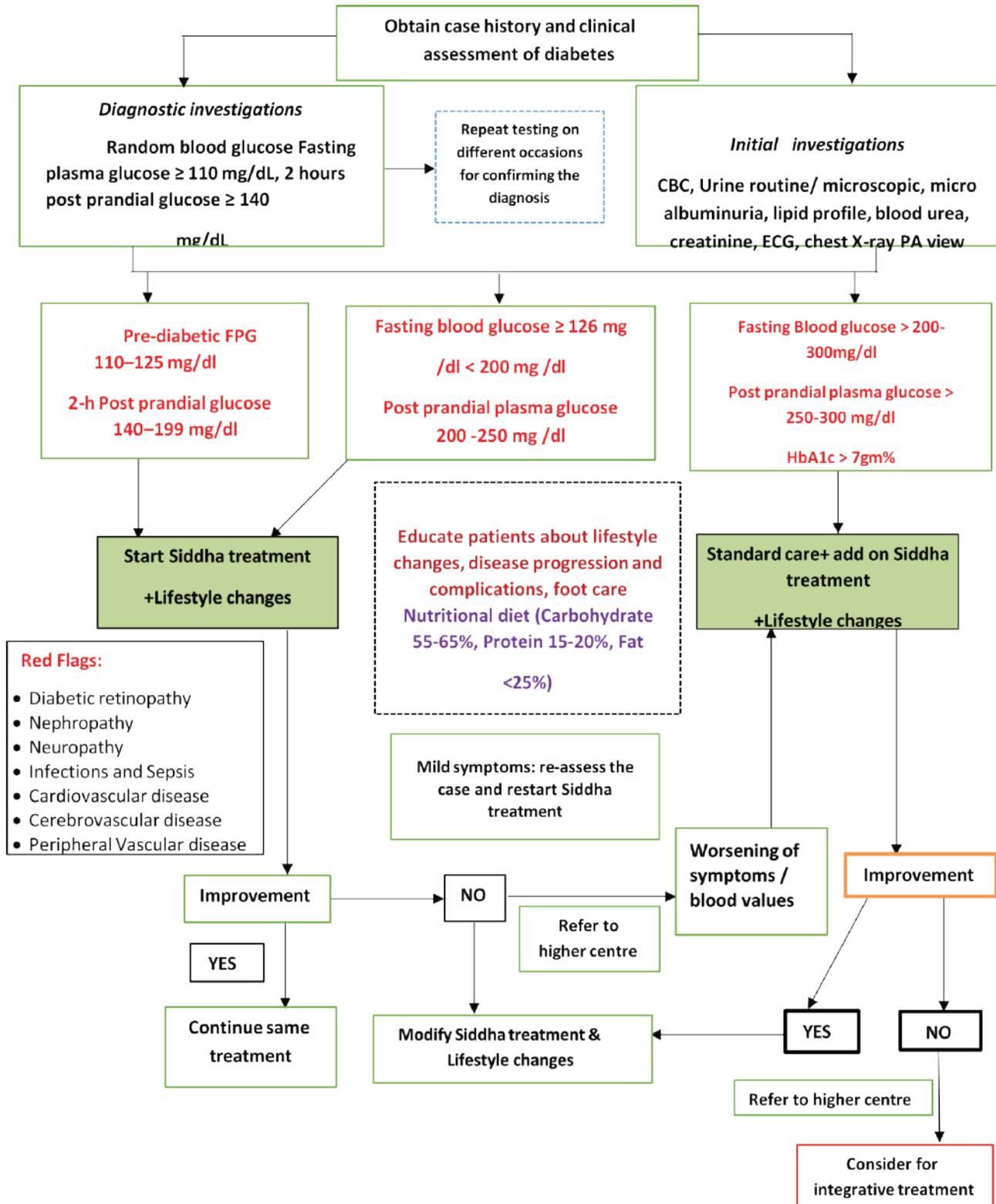
Restricted diet and lifestyle: Same as Levels 1 and 2

Follow-up: At an interval of 7 days or as per the need

Referral criteria

Same as Level 1, 2 and any condition or serious complication not responding to treatment

ALGORITHM OF TREATMENT PROCESS FOR TYPE 2 DIABETES MELLITUS



REFERENCES

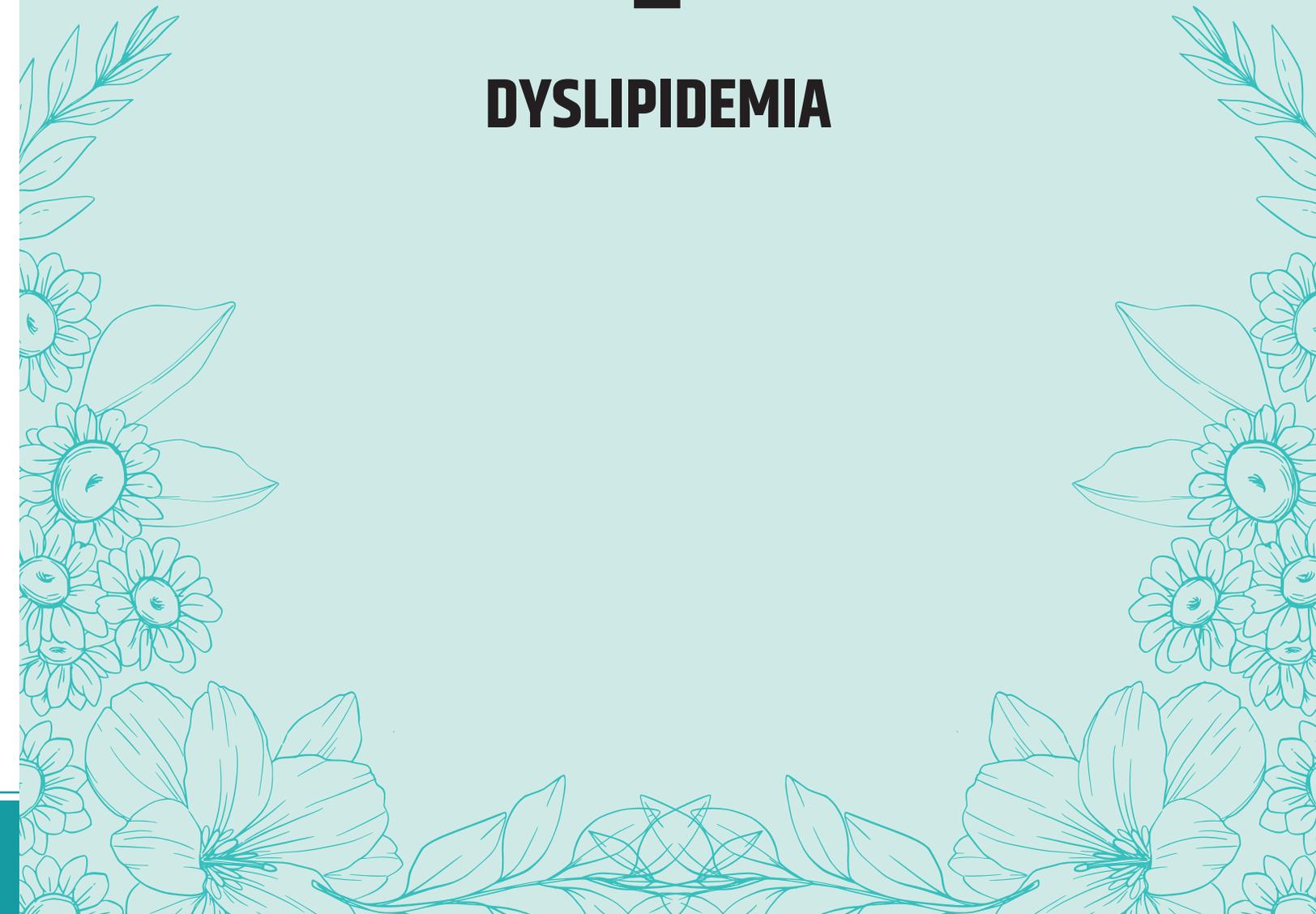
1. <https://www.who.int/publications/i/item/9789240064973>
2. <https://www.icd10data.com/ICD10CM/Codes/E00-E89/E08-E13/E11->
3. Kuppusamy Mudaliar K.N, Siddha Maruthuvam (Pothu), Published by Directorate of Indian Medicine and Homoeopathy, Chennai-106, 6th edition, 2004.Pg no 501-524.
4. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care [Internet] 2014 [cited 2024 Jan 17];37(Supplement_1):S81–90. Available from: <https://dx.doi.org/10.2337/dc14-S081>
5. Petersmann A, Müller-Wieland D, Müller UA, Landgraf R, Nauck M, Freckmann G, et al. Definition, Classification and Diagnosis of Diabetes Mellitus. Exp Clin Endocrinol Diabetes [Internet] 2019 [cited 2024 Jan 17];127(S 01):S1–7. Available from: <https://pubmed.ncbi.nlm.nih.gov/31860923/>
6. Anonymous, Yugi vaithya chinthamani Perunool 800.Pub: Directorate of Indian Medicine, Chennai. 1976.
7. Ong KL, Stafford LK, McLaughlin SA, Boyko EJ, Vollset SE, Smith AE, et al. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. The Lancet [Internet] 2023 [cited 2024 Jan 17];402(10397):203–34. Available from: <http://www.thelancet.com/article/S0140673623013016/fulltext>
8. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels, Belgium: 2021. Available at: <https://www.diabetesatlas.org>5. Sarwar N, Gao P, Kondapally Seshasai SR, Gobin R, Kaptoge S, Di Angelantonio E, et al. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta- analysis of 102 prospective studies. Lancet [Internet] 2010 [cited 2024 Jan 17];375(9733):2215–22. Available from: <https://pubmed.ncbi.nlm.nih.gov/20609967/>
9. Chan JCN, Lim LL, Wareham NJ, Shaw JE, Orchard TJ, Zhang P, et al. The Lancet Commission on diabetes: using data to transform diabetes care and patient lives. Lancet [Internet] 2021 [cited 2024 Jan 17];396(10267):2019–82. Available from: <https://pubmed.ncbi.nlm.nih.gov/33189186/>
10. Sahadevan P, Kamal VK, Sasidharan A, Bagepally BS, Kumari D, Pal A. Prevalence and risk factors associated with undiagnosed diabetes in India: Insights from NFHS-5 national survey. J Glob Health [Internet] 2023 [cited 2024 Jan 19];13:04135. Available from: <https://pubmed.ncbi.nlm.nih.gov/38063336/>
11. Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). Lancet Diabetes Endocrinol [Internet] 2023 [cited 2024 Jan 19];11(7):474–89. Available from: <http://www.thelancet.com/article/S2213858723001195/fulltext>
12. Pal R, Bhadada SK, Misra A. Resurgence of COVID-19 and diabetes in India. Diabetes Metab Syndr [Internet] 2021 [cited 2024 Apr 25];15(3):1037. Available from: </pmc/articles/PMC8102081>
13. Pantea Stoian A, Bica IC, Salmen T, Al Mahmeed W, Al-Rasadi K, Al-Alawi K, et al. New-Onset Diabetes Mellitus in COVID-19: A Scoping Review. Diabetes Therapy [Internet] 2024 [cited 2024 Apr 25];15(1):33–60. Available from: <https://link.springer.com/article/10.1007/s13300-023-01465-7>
14. .Campbell I, Edinburgh F. Epidemiology and Clinical Presentation of Type 2 Diabetes. Value in Health [Internet] 2000 [cited 2024 Jan 19];3(SUPPL. 1):3–6. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1046/j.1524-4733.2000.36014.x>
15. Vedantam D, Poman DS, Motwani L, Asif N, Patel A, Anne KK. Stress-Induced Hyperglycemia: Consequences and Management. Cureus [Internet] 2022 [cited 2024 Jan 23];14(7). Available from: </pmc/articles/PMC9360912/>
16. American Diabetes Association. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2024. Diabetes Care [Internet] 2024 [cited 2024 Jan 19];47(Supplement_1):S20–42. Available from: <https://dx.doi.org/10.2337/dc24-S002>

17. Tamez-Pérez HE, Quintanilla-Flores DL, Rodríguez-Gutiérrez R, González-González JG, Tamez-Peña AL. Steroid hyperglycemia: Prevalence, early detection and therapeutic recommendations: A narrative review. *World J Diabetes* [Internet] 2015 [cited 2024 Jan 23];6(8):1073. Available from: /pmc/articles/PMC4515447/
18. Ferraù F, Albani A, Ciresi A, Giordano C, Cannavò S. Diabetes Secondary to Acromegaly: Physiopathology, Clinical Features and Effects of Treatment. *Front Endocrinol (Lausanne)* [Internet] 2018 [cited 2024 Jan 23];9(JUL):358. Available from: /pmc/articles/PMC6043782/
19. Standard Treatment Guidelines. (2019) published by National Institute of Siddha, Tambaram sanatorium, Chennai 47. Pg no 290-299.
20. American Diabetes Association. 3. Prevention or Delay of Type 2 Diabetes and Associated Comorbidities: Standards of Medical Care in Diabetes—2022. *Diabetes Care* [Internet] 2022 [cited 2024 Feb 29];45(Supplement_1):S39–45. Available from: <https://dx.doi.org/10.2337/dc22-S003>
21. Murugesu Mudhaliyar K. S. Gunapadam Mooligai Vaguppu, Directorate of Indian medicine and Homeopathy, Chennai-106, 5th reprint, 1998. Pg no 84,248,414,552, 576,685
22. Anonymous. *Theran Kudineer-100* (74th stanza). 2nd ed. New Delhi: Central Council for Research in Ayurveda and Siddha. Pg no:40,41.
23. Hakkim Pa. Mohammed Abdhula Sayubu, *Mega nivarana bogini ennum Neerizhivu Maruthuvam*, Thamarai Library, Chennai-26, -1 st Edition 1998.
24. .Kannusamy Pillai S. *Pathartha Guna Velakkam (Vegetable Kingdom)* by re-edition, Chennai: B. Rathnayakar and Sons; 2017.
25. A Monograph on D5 Chooranam. Pub: Central Council for Research in Siddha. Chennai 2017.
26. Narayanaswami V, *Pharmacopoeia of Hospital of Indian Medicine Part I & II*, Tamilnadu Siddha Medical Board, Chennai-106, 2nd Edition 1995
27. *The Siddha Formulary of India, Part I*, Govt. of India, Ministry of health and family welfare, Department of AYUSH, 1992. Pg no 30, 90,91,156
28. *The Siddha Formulary of India, Part II*, Govt. of India, Ministry of health and family welfare, Ministry of AYUSH, 2011. Pg no 23,31,50-52,57-59,105,106.
29. Thiyagarajan R. *Text Book of Materia Medica (Gunapadam)*. Department of Indian Medicine and Homoeopathy; Chennai. 2008. Pg. 12.
30. RSSDI Clinical Practice Recommendations for the Management of Type 2 Diabetes Mellitus 2022. *Int J Diabetes Dev Ctries* 2022;42(S1):1–143.
31. Nowosad K, Sujka M. Effect of various types of intermittent fasting (IF) on weight loss and improvement of diabetic parameters in human. *Current nutrition reports*. 2021 Jun; 10:146-54.
32. Kannan Rajaram.T, *Varma pulligalin irupidam*, ATVS Siddha medical college, Kanniyakumari, 1st edition, 2007.
33. Nakhleh A, Shehadeh N. Hypoglycemia in diabetes: An update on pathophysiology, treatment, and prevention. *World J Diabetes* [Internet] 2021 [cited 2024 Jan 29];12(12):2036. Available from: /pmc/articles/PMC8696639
34. /Lowe RN, Williams B, Claus LW. Diabetes: how to manage patients experiencing hypoglycaemia. *Drugs Context* [Internet] 2022 [cited 2024 Feb 29];11. Available from: /pmc/articles/PMC9205569/

CHAPTER

2

DYSLIPIDEMIA





DYSLIPIDEMIA

(ICD) 11-5C8Z¹

International Classification of Diseases

Unspecified disorders of lipoprotein metabolism or lipidemias 5C8Z¹

Koḷuppu Piṛaḷvu Nōy

CASE DEFINITION

- Dyslipidemia are the disorders of lipoprotein metabolism resulting in High total cholesterol (TC), High low-density lipoprotein cholesterol (LDL-C), High non-high-density lipoprotein cholesterol (non-HDL-C), High triglycerides.²
- The clinical features seen in Dyslipidaemia are varyingly learnt from the signs and symptoms as described in *Koḷuppu Miku kuṇam* (a condition of excess fat) such as increased fatty tissue resulting in symptoms similar to that of muscle excess, along with fatigue, dyspnoea on exertion, associated with excess muscle formation in buttocks, genitals, chest, abdomen and thighs³.

INTRODUCTION

- The global prevalence of hypercholesterolemia among adults was 39% (males 37% & females 40%) as per the WHO 2008 report. Further WHO estimates showed that the prevalence of hypercholesterolemia in adults was (53.7%) in Europe, (47.7%) in America, (30.3%) in Southeast Asia and (23.1%) in Africa.⁴ In India specific, the prevalence of hypercholesterolemia varies from 10 to 15 % in rural to 25–30 % in urban populations.⁵
- Dyslipidemia is one of the established risk factors for cardiovascular disease. In-depth reviews concluded that elevated LDL-c is a significant contributor to atherosclerotic cardiovascular disease (CVD)⁶⁻⁹ while some studies had shown that non-HDL-c predicts CV risk better than LDL-C.¹⁰
- Epidemiological studies have reported variable prevalence rates of important dyslipidemias in India. The prevalence of total cholesterol 200 mg/dl ranges from 25 to 30 %, non-HDL cholesterol 160 mg/dl 25-30 %, LDL cholesterol 130 mg/dl: 25-30 %, non-HDL cholesterol 130 mg/dl: 50-55 %, LDL cholesterol >100 mg/dl: 50-55 %, triglycerides >150 mg/dl: 30-40 % and low HDL cholesterol: 60-70 %. Most national studies have reported higher prevalence of hypercholesterolemia in most Southern and a few North Indian states, more in urban than rural areas, whereas the prevalence of high triglycerides and low HDL cholesterol is similar throughout the country.¹¹

DYSLIPIDEMIA - SIDHA PATHOPHYSIOLOGY

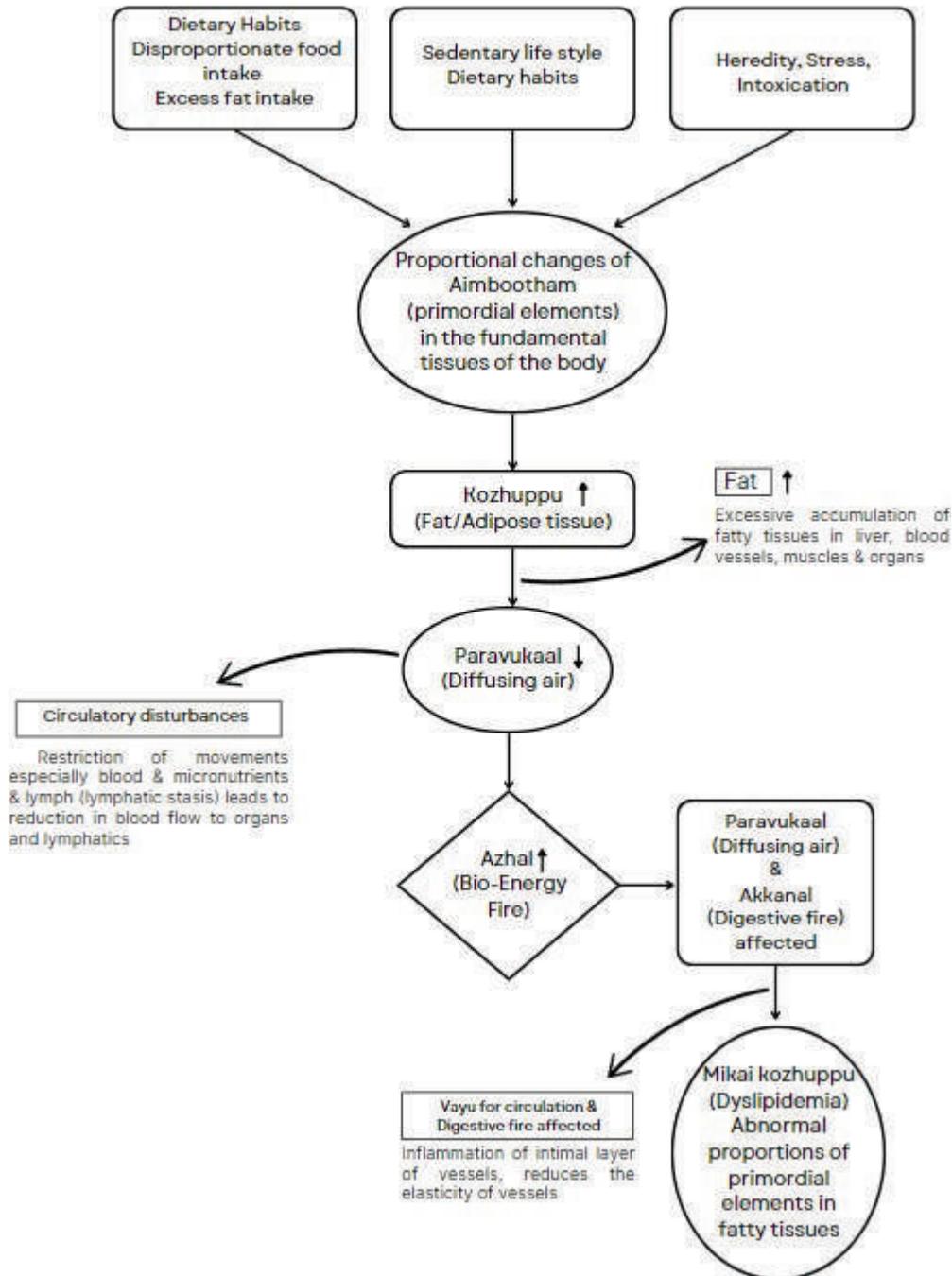


Figure 1. Dyslipidemia Siddha Pathophysiology

CLINICAL PRESENTATION AND EXAMINATION^{12,13}

Dyslipidemia majority of the times are asymptomatic and are accidentally diagnosed on routine blood tests. Few patients with severe or untreated dyslipidemia may present with signs and symptoms related to the complications of dyslipidemia, such as coronary artery disease, peripheral arterial disease, stroke, atherosclerosis and heart failure. Some of the possible presentations (signs & symptoms) of dyslipidemia are as below:

1. Xanthomas (yellowish fat deposits visible on the skin).



2. Arcus senilis (Gray or white ring around the eye's cornea that is caused by cholesterol depositing in the corneal margin).
3. Lipemia retinal is (milky appearance in the retinal vessels due to high blood triglyceride levels with blurred vision).
4. Lower limb ischemia (common symptom of peripheral artery disease, caused by the narrowing or blockage of the arteries that supply blood to the legs due to atherosclerosis; this condition is usually characterized by pain or cramping during physical activity and improves with rest).
5. Angina (caused by the narrowing or blockage of the arteries that supply blood to the heart due to atherosclerosis. The uncomfortable pressure, fullness, squeezing or pain in the centre of the chest usually occurs when the heart needs more oxygen, such as during physical or emotional stress and may radiate to the neck, jaw, shoulders, left arm or back).
6. Transient ischemic attacks and strokes (atherosclerosis in cerebral arteries, contributing to sudden interruption of blood flow to the brain due to a clot or a bleed in weakened blood vessel walls. Symptoms may include sudden weakness, slurred speech, transient loss of consciousness or visual disturbances).
7. Non- Alcoholic Fatty liver disease.

DIFFERENTIAL DIAGNOSIS¹⁴⁻¹⁶

Several disease conditions remain as secondary causes for dyslipidemia. They are as follows:

Table 1

Sl. No.	Disease condition	Findings
1.	Hypothyroidism	Fatigue, increased sensitivity to cold, dryness of skin, constipation, hair loss, dyspnea, hoarse voice, irregular menses, paresthesia, peripheral edema and elevated TSH levels.
2.	Nephrotic syndrome	Swelling in legs, feet, ankles, face and hands. Weight gain, fatigue, foamy or bubbly urine, anorexia, high protein levels in urine, low levels of protein in blood and kidney biopsy to confirm exact cause.
3.	Biliary obstruction, Hepatoma	Right upper quadrant abdominal pain, fever, nausea, vomiting and weight loss. Jaundice with clay colored or acholic stools, dark urine and pruritis, elevated bilirubin levels, Endoscopic ultrasound (EUS), Magnetic Resonance Cholangiopancreatography (MRCP), or direct cholangiography.

Sl. No.	Disease condition	Findings
4.	Pregnancy	Elevated HCG levels, USG abdomen.
5.	Drugs (oral estrogens, glucocorticoids, tamoxifen, thiazides)	Past history of drugs intake, elevated levels of estrogen, cortisol etc., in Blood tests.
6.	Alcohol abuse	Past history of excess alcohol intake.
7.	Obesity	Weight gain, breathlessness, swellings, joint pains and skin changes.
8.	Niemann Pick Disease Type C	Lipidosis due to intracellular cholesterol transport defect (Acid Sphingomyelinase Deficiency) (ASMD), that catalyzes the hydrolysis of sphingomyelin (SM) to ceramide and phosphocholine. Due to this, SM and its precursor lipids begin to accumulate in lysosomes, mainly in macrophages.
9.	Wolman's Disease	It is an autosomal recessive storage condition characterized by extremely low (or nonexistent) lysosomal acid lipase (LAL) activity. This enzyme deficiency results in significant intracellular buildup of cholesteryl esters and triglycerides.
10.	Cerebrotendinous xanthomatosis	A rare autosomal recessive genetic condition caused by a mutation in the CYP27A1 gene, resulting in a lack of the mitochondrial enzyme sterol 27-hydroxylase. This enzyme is required to convert cholesterol into chenodeoxycholic acid, a bile acid.

In Siddha medicine:

- *Atitūlam*
- *Aiyam miku kuṇam*³
- *Valarccitai mārra nōykal*¹⁷

1) Supportive Investigations^{18 - 20}

i. Essential investigations:

- **Fasting lipid profile:** The National Cholesterol Education Program provides the Adult Treatment Panel III—widely acknowledged guidelines for dyslipidemia screening. Guidelines recommend a fasting lipid panel every 5 years for adults 20 years and older.
- **Body Mass Index:** Measuring Body Mass Index as follows:

Table 2: WHO's Classification of Adults according to BMI

Classification	BMI	Risk of comorbidities
Underweight	<18.50	Low (but risk of other clinical problems increased)
Normal range	18.50-24.99	Average
Overweight: Preobese	≥25.00	
Obese class I	25.00-29.99	Increased
Obese class II	30.00-34.99	Moderate
Obese class III	35.00-39.99	Severe
	≥40.00	Very severe

Table 3: Classification of weight by BMI in adult Asians:²¹

Classification	BMI (kg/m ²)	Risk of co- morbidities
Underweight	<18.5	Low (but increased risk of other clinical problems)
Normal range	18.5-22.9	Average
Overweight	23-24.9	Increased
Obese I	25-29.9	Moderate
Obese II	<30	Severe

ii. Advanced Investigations:

As per the need and symptomatology, the following may be done:

- Apolipoprotein B (ApoB), apolipoprotein A1
- Lipoprotein (a)
- Treadmill Test
- High sensitivity C-reactive protein
- Glycosylated haemoglobin (HbA1c)
- Fasting blood glucose (FBS)
- Thyroid stimulating hormone level (TSH)
- Liver function tests
- Serum creatinine
- Creatine kinase
- Urine analysis
- Homocysteine levels
- Fundoscopy
- Waist hip ratio, waist circumference, skin fold thickness
- Plasma leptin
- Upper Abdominal Ultrasound

DIAGNOSTIC CRITERIA:

Dyslipidemia is often diagnosed with routine screening tests. Dyslipidemia is diagnosed by measuring serum lipids. Routine measurements (lipid profile) include total cholesterol (TC), TGs, HDL-C and LDL-C; these results are used to calculate LDL-C and VLDL-C. A modern updated clinical algorithm for the diagnosis of dyslipidemia is as below:

Table 4: Diagnostic biochemical parameters for dyslipidemia in adults^{18,22,24}

	TC	LDL-C	TG	HDL-C
Mild-to-moderate risk				
Levels	200-239 mg/dL	130-194 mg/dL	175-499 mg/dL	25-35 mg/dL
Severe risk				
Levels	≥ 240 mg/dL	≥ 194 mg/dL	≥ 449 mg/dL	< 25 mg/dL

Abbreviations: TC, Total Cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglyceride.

Classification ^{12,18,22,23}

Dyslipidemia are mainly classified into two types:

Primary:

Primary dyslipidemia is caused by genetic mutations and can be inherited as an autosomal dominant, autosomal recessive, or X-linked.

Secondary:

Secondary dyslipidemia is caused by improper lifestyle such as lack of physical activity, unhealthy food habits, alcohol intake, smoking etc., and by some health conditions such as obesity, hypothyroidism. Diabetes, CKD, liver disease etc.

International Classification of dyslipidemia gives 5 categories, according to Frederickson phenotype (World Health Organization):¹²

- Phenotype I is an abnormality of chylomicrons and will result in triglycerides greater than 99 percentiles.
- Phenotype IIa consists mainly of LDL cholesterol abnormality and will have a total cholesterol concentration greater than 90 percentile and possibly apolipoprotein B greater than 90 percentile.
- Phenotype IIb consists of abnormality in LDL and VLDL cholesterol. This type will result in total cholesterol or triglycerides greater than the 90 percentile and apolipoprotein greater than the 90 percentile.
- Phenotype III is an abnormality in VLDL remnants and chylomicrons, which results in elevated total cholesterol and triglycerides greater than 90 percentile.
- Phenotype IV is mainly when VLDL is abnormal and results in total cholesterol greater than 90 percentile. This type can also present with triglycerides greater than 90 percentile and low HDL.
- Phenotype V is when chylomicrons and VLDL are abnormal and triglycerides are greater than 99 percentiles.

Eṅvakai Tērvu (Eightfold examination) ^{24,25}

- *Nāṭi* (Pulse) – *Azhal aiyam/ Aliya azhal*
- *Sparicam* (Touch)– *Cool*
- *Nā* (Tongue) –*Pallor and Shiny / coated*
- *Niram* (Colour) – *Pallor*
- *Moḷi* (Speech) – *Low pitched Voice*
- *Viḷi* (Eye) – *Pallor*
- *Malam* (Stool) - *Colour-White Consistency – Ilakal or Malakkaṭṭu*

- *Mūttiram (Urine)*-Oil stands like a Pearl and disappears as a ring

PRINCIPLES OF MANAGEMENT:

The principles of management include assessment of signs and symptoms before initiating treatment and the need for management through conventional treatment for associated co-morbidities. If the patient is already under standard care, the physician may advise to continue the same along with add-on homoeopathy and can be assessed for the same in the follow ups for tapering or discontinue the treatment in consultation with the conventional physician.

Red Flag Signs²⁶⁻²⁷

- Early age of onset for coronary artery disease in self or in family (includes heart attack, stent, bypass)
- Recurrent vascular events and Atherosclerotic cardiovascular diseases (ASCVD) with genetic dyslipidemia (FH& High Lp (a))
- Clinical evidence of atherosclerotic CAD
- Atherosclerotic disease in other vascular beds
- Heterozygous Familial Hypercholesterolemia (HeFH) with ASCVD, or coronary imaging showing >50 % lesion in 2 coronary vessels
- Total cholesterol \geq 220 mg/dL or LDL cholesterol \geq 190 mg/dL in individual
- Tendon Xanthomas
- Uncontrolled co-morbidities

(A) Prevention Management²⁴

Preventing dyslipidemia is essential to reduce the risk of cardiovascular complications and improve the quality of life. The prevention strategies include:

- Screening for dyslipidemia regularly, especially for people with a family history or other risk factors. The frequency and type of screening depend on the individual's age, sex and health status, but generally, a lipid profile test is recommended every 4 to 6 years for adults and every 2 years for children and adolescents.
- Adopting a healthy lifestyle by eating a balanced diet with plenty of fruits, vegetables, whole grains, lean proteins and healthy fats, such as omega-3 fatty acids from fish, nuts and seeds. Avoid foods high in cholesterol, saturated fats, trans fats, added sugars and salt. If possible, engage in physical activity for at least 150 minutes weekly.
- Maintaining a healthy weight and body mass index, quitting smoking and limiting alcohol intake are all recommended.
- Comorbidities such as diabetes, hypertension, hypothyroidism, chronic kidney disease, or liver disease can affect lipid levels or increase the risk of cardiovascular disease; therefore, it is important to remain compliant with any medications.

Table 5: Common Yoga Protocol

S. No.	Name of Posture/Procedure	
Invocation/Prayer		
Chalana Kriyas (Loosening Practices/Warmups)		
1.	Neck Movements	Forward/Backward Bending
		Right/Left Bending
		Right/Left Twisting
		CW/ACW Rotation
2.	Shoulder Movements	Stretching
		CW/ACW Rotation
3.	Trunk Movements	Right/Left Twisting
4.	Knee Movements	Squats
Standing Yoga Positions		
5.	<i>Samasthiti</i>	Standing Alert Posture
6.	<i>Tadasana</i>	Palm Tree Posture
7.	<i>Vrksasana</i>	Tree Posture
8.	<i>Uttanasanan</i>	Standing Forward Bend
9.	<i>Pada-Hastasana</i>	Hand to Feet Posture
10.	<i>Ardha Chakrasana</i>	Half Wheel Pose
11.	<i>Trikonasana</i>	Triangle Pose
Sitting Yoga Positions		
12.	<i>Visramasana</i>	Long Sitting Posture
13.	<i>Sukhasana</i>	Easy Pose
14.	<i>Padmasana</i>	Lotus Pose
15.	<i>Dandasana</i>	Stick/Staff Pose
16.	<i>Bhadrasan</i>	Gracious Pose or Butterfly Pose
17.	<i>Vajrasana</i>	Thunderbolt Pose
18.	<i>Ushtrasana</i>	Camel Pose
19.	<i>Ardha-Ushtrasana</i>	Half Camel Pose
20.	<i>Sasankasana</i>	Hare Posture
21.	<i>Balasana</i>	Child Pose
22.	<i>Uttana Mandukasana</i>	Stretched Up Frog Posture
23.	<i>Vakrasana</i>	Spinal Twist Posture
24.	<i>Paschimottanasana</i>	Seated Forward Bend
25.	<i>Simhasana</i>	Lion Pose

S. No.	Name of Posture/Procedure		
26.	<i>Marjarasana</i>	Cat Pose	
Prone Positions			
27.	<i>Makarasana</i>	Crocodile Posture	
28.	<i>Bhujangasana</i>	Cobra Pose	
29.	<i>Salabhasana</i>	Locust Posture	
30.	<i>Dhanurasana</i>	Bow Pose	
Supine Positions			
31.	<i>Chatuspadasana</i> <i>Setubandhaasana</i>	Bridge Posture	
32.	<i>Uttanapadasana</i>	Raised Leg Posture	
33.	<i>Matsyasana</i>	Fish Pose	
34.	<i>Ardhahalasana</i>	Half Plough Pose	
35.	<i>Pavanmuktasana</i>	Wind Releasing Posture	
36.	<i>Markatasana</i>	Monkey Pose	
37.	<i>Shavasana</i>	Corpse Body Posture	
38.	<i>Kapalbhati</i>	Forceful Rapid Exhalations	Sukhasana/Padmasana/V ajrasana 1 inhalation :20-30 exhalation
Breathing Exercises			
39.	Anuloma-Viloma/ Nadishodhana Pranayam/ <i>Suryabhedan</i>	Alternate Nostril Breathing	Left Palm on Left Knee (Jnana Mudra) Right palm in Nasagra Mudra Without Kumbhaka With Kumbhaka (Kumbhaka means retention of breath)
40.	<i>Shitali Pranayam</i>	Cooling breath	<i>Jnana Mudra or Dhyana Mudra or Anjali Mudra (Namaste Pose)</i> Inhale through Tongue Tube and exhale through nostrils
41.	<i>Bhramari Pranayam</i>	Humming bee breath	<i>Sanmukhi Mudra</i> <i>IMRL Thumb-Eye Nose Mouth Ear</i>
42.	<i>Dhyana</i>	Meditation	<i>Jnana Mudra or Dhyana Mudra or Anjali Mudra</i> <i>Tip of thumb to Tip of index finger</i> <i>Other fingers straight/relaxed</i>

Primordial Prevention

Table 6: Vegetables to be Added:

Tamil Name	Common English Name	Botanical Name	Part Used	Consumption Advice
<i>Veḷḷari</i> ²⁸	Cucumber	<i>Cucumis sativus</i>	Fruit	Consume raw as salads or juice
<i>Pācipparuppu</i> ²⁸	Pumpkin	<i>Cucurbita pepo</i>	Fruit	Consume as curry or soups

Tamil Name	Common English Name	Botanical Name	Part Used	Consumption Advice
<i>Curai</i> ²⁸	Bottle gourd	<i>Lagenaria siceraria</i>	Fruit	Consume as curry, soup, or stir-fry
<i>Vālai</i> ²⁸	Banana stem	<i>Musa paradisiaca</i>	Stem	Consume as curry/salad
<i>Murunkai</i> ²⁸	Drumstick	<i>Moringa oleifera</i>	Pods	Consume as curry/salad/stir fry
<i>Pīṇs</i> ²⁸	Beans	<i>Phaseolus vulgaris</i>	Pods	Consume as curry, soup, or stir-fry
<i>Veṇṭakkāy</i> ²⁸	Ladies finger	<i>Abelmoschus esculentus</i>	Fruit	Consume as curry, soup, or stir-fry
<i>Kīraikāl</i> ²⁸	Green leafy vegetables	<i>Various species</i>	Leaves	Include one variety in the salads, soups and curries.
<i>Inji</i> ²⁸	Ginger	<i>Zingiber officinale</i>	Rhizome	Consume as tea or include it in cooking as one of the ingredients for digestive benefits
<i>Pūṇṭu</i> ²⁸	Garlic	<i>Allium sativum</i>	Bulb	Can be used in cooking
<i>Čiṇṇa vēṅkayam</i> ²⁸	Small onion	<i>Allium parvum</i>	Bulb	Use for making curries
<i>Elumiccai</i> ²⁸	Lemon	<i>Citrus limon</i>	Fruit	Use for salads, juice, or cooking for flavouring dishes
<i>Kōvai</i> ²⁸	Ivy gourd	<i>Coccinia grandis</i>	Fruit	Consume as stir-fries or curries
<i>Kudampuli</i>	Malabar tamarind	<i>Garcinia cambogia</i>	Fruit Pulp	For cooking purpose instead of tamarind

Others:²⁹

- Whole grains (brown rice, Millets etc.)
- Plant oils (vegetable oils)
- Regular exercises for at least 30 minutes.
- Brisk walking for 30-45 minutes
- Oil bath - weekly twice
- Steam bath - weekly once

To be avoided:

- Oily foods, fried items
- Tubers like potato (*Solanum tuberosum*), Tapioca (*Manihot esculenta*), etc.
- Excessive intake of coconut (*Cocos nucifera*)
- Ground nut (*Arachis hypogaea*)
- Sesame seeds (*Sesamum indicum*)
- Milk and milk products
- High glycaemic index foods (rice, corn, sugar, white bread, white pasta).

Levels of Prevention for Dyslipidemia

- Primordial Prevention: Prevent the development of risk factors that lead to dyslipidemia (such as unhealthy diet, physical inactivity, obesity, and smoking).
- Primary Prevention: Target individuals with borderline or elevated lipid levels who have not yet developed cardiovascular disease.
- Secondary Prevention: Manage dyslipidemia in individuals with established cardiovascular disease (CVD) (e.g., past heart attack, stroke, angina).
- Tertiary Prevention: Prevent or limit disability and complications in individuals with advanced cardiovascular consequences of long-standing dyslipidemia.

Siddha System of Medicine emphasis adhering to *Tēraiṃar piṇi aṇukā viti* for prevention of disease and lead to healthy life.

Table 7:

Dietary Habits (<i>Uṇavu Muṛaika!</i>)	
Do's - <i>Pattiyam</i>	Don'ts - <i>Apattiyam</i>
<ul style="list-style-type: none"> • Drink warm water • Add <i>Trithoda sama porutgal</i> inclusive of turmeric, pepper, cumin seeds, asafoetida, dry ginger, cardamom, fenugreek and garlic in diet • Consume low fat, low-calorie & high fiber diet, fresh vegetables, whole grains, legumes, greens & citrus fruits • Easily digestible foods should be taken such as rice gruel /double boiled rice gruel, buttermilk, Tender coconut water • Include moderate intake of nuts • Include lean proteins and low fat dairy in diet 	<ul style="list-style-type: none"> • Always avoid fatty meals and late-night snacking • Avoid highly processed refined carbohydrate diet and advised to take complex carbohydrates • Limit added sugars, trans fat and refined grains • Avoid deep fried food and junk foods • Avoid Overeating or Skipping Meals
Lifestyle Practices (<i>Vālvīyal Muṛaika!</i>)	
Do's	Don'ts
<ul style="list-style-type: none"> • Practice Siddha <i>kāyakaṛpam</i> – take ginger, dried ginger and chebulic myrobalan in the morning, afternoon and evening respectively • Follow intermittent fasting (<i>Oru poḷutu, Iru poḷutu</i>) • Practice at least 45 minutes of moderate physical activity (like walking) 5 days a week and <i>Cilampāṭṭam, Uppukuṇṭam, Mālyutam</i> • Consume food to the level of hunger • Consume food only half of stomach, liquid quarter of stomach and always leave quarter stomach empty • Better balance of mood and sleep • Powder massage (<i>Poṭi Timirtal</i>) with <i>Panja Karpa Kuliya Podi</i> or <i>Kollu Chooranam</i> or <i>Thiripala Chooranam</i> 	<ul style="list-style-type: none"> • Avoid daytime sleep or oversleeping • Avoid sedentary life style • Avoid stress • Avoid nap after food • Avoid high sodium diet • Avoid alcohol and smoking

(B) Curative Interventions

At Level 1:

(Solo Siddha Physician Clinic/Health & Health Clinic/PHC (Optimal Standard of treatment in a situation where technology and resources are limited)

Clinical diagnosis:

Understanding the signs and symptoms of dyslipidemia is crucial for timely intervention and preventing associated complications. Clinicians should consider the broader clinical context, including family history and risk factors, to guide appropriate interventions and reduce the burden of cardiovascular diseases associated with dyslipidemia. Pertinent social history would include tobacco use or specific details about diet. Diagnosis of dyslipidemia is primarily arrived at with the help of investigations as fasting lipid profile. However other investigations may be advised based on the presentation.

Management

The treatment plan (*Marutuvā vaḷimurāi*) for managing *Koḷuppu piṛaḷvu nōy* in Siddha focuses on addressing the root causes of the imbalance.

The first line of treatment is to normalize the altered or deranged humours and revitalization of seven fundamental tissues through detoxification methods followed by internal medications. The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

Fasting and dietary modifications are also advised, including practices like *Pattini* (complete fasting), *Viratam* (intermittent fasting) and *Oru poḷutu* (restricting meals to one specific time a day). *Kāyakaṛpam* drugs are prescribed to enhance immunity and vitality. Additionally, medicines with pungent and bitter tastes are recommended to increase *Aḷal*, improve digestion, reduce excess *Aiyam* and balance metabolism. This comprehensive approach targets both internal and external factors to restore balance and promote overall health^{2,11}.

Day 1

Eṇṇey muḷukku (Therapeutic oil bath): ⁽³⁾

Eṇṇey muḷukku is a preparatory procedure in which medicated oil massage with a bath of lukewarm water. It will strengthen the five sensory organs. According to disease severity, *eṇṇey kuḷiyal* can be advised for one day to three days.

- *Arakku Thylam* – Quantity sufficient (External use)³⁰

Rules to be followed during Therapeutic oil bath:

Apply oil before 7 am. Instil 2 drops of medicated oil in each nostril, ear and eye. Spread over the medicated oil from head to foot and give a gentle massage. After application, leave it for 15 to 45 minutes and bathe with lukewarm water.

Take tender vegetables and easily digestible food. Avoid daytime sleep, intercourse and exposure to sunlight and cold items on the day of the oil bath.

Day 2

Therapeutic Purgation (*Kalīccal maruttuvam*):

- *Akathiyar Kuzhambu* -100-130 mg with *Ginger Juice (Zingiber officinalis)*, weekly once for 2 weeks on an empty stomach.⁽³⁰⁾

Rules to be followed during Therapeutic Purgation

- The medication should be taken in the early morning 5 to 6 AM
- After the average number (5-6 times) of bowel evacuations, watery diarrhoea commences. In this stage, the patient is advised to take buttermilk/ lemon juice/tea decoction/ fried cumin seeds decoction.
- After purgation, the patient may have symptoms like tiredness, slimness, lightness of the body and tiredness of sense organs which is a good sign.
- Dietary regimen during purgation:
 - Buttermilk
 - Rice porridge
 - Double-boiled porridge
 - Luke-warm water
- Precautions
 - Avoid daytime sleep on the day of purgation therapy
 - Should not take heavy meals before or during the procedure

Day 3 onwards

Treatment:

- Selection of drug, dosage form dose, time, choice of adjuvant and its dose duration will depend upon the disease severity, chronicity, body constitution and age. These criteria solely depend upon the discretion of the treating physician.

Table 8: Single Herbs

Sl. No	Herbs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Aṇupāṇam
<i>Kudineer – Decoction / Powder/ juice</i>						
1.	<i>Neermuli (Hygrophila auriculata)</i> ³¹	Decoction or powder	30ml/1-2g	BD	30 days	Water
2.	<i>Nerunjil (Tribulus terrestris)</i> ³¹	Decoction or powder	30ml/1-2g	BD	30 days	Water
3.	<i>Sirukanpeelai (Aerva Lanata)</i> ³¹	Decoction or powder	30ml/1-2g	BD	30 days	Water
4.	<i>Brahmi (Bacopa monnieri)</i> ³¹	Decoction or powder	30ml/1-2g	BD	30 days	Water

Sl. No	Herbs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Anupānam
5.	Seenthil (<i>Tinospora cordifolia</i>) ^{31, 32}	Decoction or powder	1-2 g	BD	30 days	Water
6.	Kattrazhai (<i>Aloe vera</i>) ³¹	Juice; avoid excessive use.	30ml	BD	30 days	--
7.	Korai kizhangu (<i>Cyperus rotundus.</i>) ³¹	Decoction or powder	30ml/1-2g	BD	30 days	Water
8.	Kungilyam (<i>Shorea Robusta</i>) ³¹	Powder	1-2g	BD	30 days	Water
9.	Mantharai (<i>Bauhinia purpurea</i>) ³¹	Powder	1-2 g	BD	30 days	Water
10.	Ashoka (<i>Saraca asoca</i>) ³⁰	Decoction or powder	30ml/1-2g	BD	30 days	Water
11.	Kazharchi (<i>Caesalpinia crista</i>) ³¹	Powder	1-2 g	BD	30 days	Water
12.	Kodam puli (<i>Garcinia cambogia</i>) ³¹	Dried fruit juice	30ml	BD	30 days	--
13.	Nathaisoori (<i>Spermacoce hispida</i>) ³¹	Decoction or powder	1-2 g	BD	30 days	Water
14.	Vellulli (<i>Allium sativum</i>) ³³	Decoction	30ml	BD	30 days	--
15.	Karunjeergam (<i>Nigella sativa</i>) ³⁴	Powder	1-2 g	BD	30 days	Water
16.	Kadukkai (<i>Terminalia chebula</i>) ³⁵	Powder	1-2 g	BD	30 days	Water
17.	Chukku (<i>Zingiber officinale</i>) ³⁶	Decoction or powder	1-2 g	BD	30 days	Water
18.	Alisividhai (<i>Linum usitatissimum</i>) ³⁷	Powder	1-2 g	BD	30 days	Water
19.	Kothamalli (<i>Coriandrum sativum</i>) ³⁸	Decoction or powder	130ml/1-2 g	BD	30 days	Water
20.	Nelli (<i>Phyllanthus emblica</i>) ³⁹	Juice or Powder	30ml/1-2 g	BD	30 days	Water
21.	Manjal (<i>Curcuma longa</i>) ⁴⁰	Powder	1-2 g	BD	30 days	Water

Table 9: Compound formulations

Sl. No	Drugs	Dosage Form	Dose	Time	Duration and Frequency	Adjuvants/ Anupānam
Kudineer - Decoction						
1.	Neermulli Kudineer ³⁰	Decoction	30-60 mls	Twice a day, Before food	60 days	-
2.	Mandurathi Kudineer ³⁰	Decoction	30-60 ml	Twice a day, Before food	21 days	-

Sl. No	Drugs	Dosage Form	Dose	Time	Duration and Frequency	Adjuvants/ Aṅupānam
3.	Venthamarai Kudineer ³⁰	Decoction	60 ml	Twice a day, Before food	60 days	-
Chooranam - Medicinal powder						
4.	Karisalankanni Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, Before food	60 days	-
5.	Seeraga Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Honey or Milk
6.	Thiripala Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Honey or Milk
7.	Thirikadugu Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Honey or Milk
8.	Nilavagai Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Honey or Milk
9.	Elathi Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Honey or Milk
10.	Venthamarai Chooranam ³⁰	Medicinal powder	1-2 g	Twice a day, After food	60 days	Warm water
Maathirai- Tablet						
11.	Veppampoo Maathirai ⁴¹	Tablet	1-2 Nos	Twice a day, After food	60 days	Honey
12.	Kasthuri Maathirai ⁴²	Tablet	1-2 Nos	Twice a day, After food	60 days	Honey

Varma maruttuvam ^{43,44}

- Tivalai kalam
- Puruva varmam
- Kaikāvuḷi varmam
- Mūttira kalam
- Uḷkuttu varmam
- Taṭciṇai kalam

1. Recommended Diet & Lifestyle:

- Healthy Diet regimens - Mediterranean diet, Dietary Approaches to Stop Hypertension [DASH].⁴⁵
- Systematic physical activity such as aerobics enhances cardiorespiratory fitness and ameliorates dyslipidemia. High-intensity intermittent aerobic training can reduce myocardial oxygen demand and help control exercise intensity and increase HDL-C levels vs. moderate- intensity continuous aerobic training. Aerobic training can bring about an approximate 30– 40% reduction in TG and 20% increase in HDL-C levels in patients with moderate hypertriglyceridemia.⁴⁶

Integrative treatment approach:

If a case of dyslipidemia is associated with other co-morbid conditions (diabetes, hypothyroidism, etc.), a multidisciplinary integrative approach with other medical experts such as diabetologists, endocrinologists and registered nutritionists is essential to achieve a sustained improvement and benefit to the patient.⁴⁷

2. Restricted Diet and Lifestyle ⁴⁸⁻⁵¹

- Avoid high carbohydrate diet.
- Avoid consumption of red and processed meat.
- Avoid consumption of alcohol and smoking.
- Avoid strenuous physical exercises which may trigger cardiac events.
- Avoid diet rich in trans fats such as fried food.

3. Follow Up: Every 14 days or as per need⁵²

Reviews should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.
- Monitoring the long-term course of the condition.
- Management of dyslipidemia in terms of lifestyle modifications.
- Discussing the person's knowledge of the condition, concerns, personal preferences and ability to access services.
- Review the effectiveness and tolerability of ongoing treatment. If the patient is improving, continue treatment and if not, review the totality for further prescription.
- Self-management support.

4. Referral criteria

- Non-response to treatment.
- Evidence of an increase in severity/complications
- Substantial impact on their quality of life and activities of daily living
- Diagnostic uncertainty
- Uncontrolled co-morbidities, such as diabetes, hypertension or associated cardiac disease.

At Level 2:

(CHC/Small hospitals (10-20 bedded hospitals with basic facilities such as routine, investigation, X-ray).

Clinical Diagnosis: Same as level 1. The case referred from Level 1, or a fresh case, must be evaluated thoroughly for any complications.

Investigations:

The diagnosis would be primarily clinical. However, investigations may be necessary to investigate complications or exclude other differential diagnoses as follows:

- High sensitivity C-reactive protein.
- Apolipoprotein B (ApoB), apolipoprotein A1.
- Lipoprotein(a).
- Glycosylated hemoglobin (HbA1c).
- Fasting blood glucose (FBS).
- Thyroid stimulating hormone level (TSH).
- Transaminase (ALT).
- Serum creatinine.
- Creatine kinase.
- Urine analysis.
- Homocysteine levels.
- Fundoscopy.

Management

Along with level 1 medications including detoxification treatment any of the following medicines can be used.

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 10

Sl. No	Drugs	Dosage Form	Dose	Time	Duration and Frequency	Adjuvants/ Anupānam
Chooranam - Medicinal powder						
1.	<i>Thiratchathi Chooranam</i> ⁴²	Medicinal powder	1-2 g	Twice a day, After meals	90 days	Honey or Milk
Parpam - White calx						
2.	<i>Nandukal Parpam</i> ³⁰	White Calx	200- 400 mg	Twice a day	90 days	<i>Neermulli Kudineer</i>
3.	<i>Silasathu Parpam</i> ³⁰	White Calx	100- 300 mg	Twice a day	90 days	Ghee
4.	<i>Kungiliya Parpam</i> ³⁰	White Calx	100- 300 mg	Twice a day	90 days	Ghee
5.	<i>Vengara Parpam</i> ³⁰	White Calx	65- 125 mg	Twice a day	90 days	Ghee

Varma maruttuvam ^{42,43}

- *Tivalai kalam*
- *Puruva varmam*
- *Kaikāvuḷi varmam*
- *Mūttira kalam*
- *Uḷkuttu varmam*
- *Taṭciṇai kalam*

1. Recommended Diet & Lifestyle As per level 1

2. Restricted Diet & Lifestyle

3. **Follow Up** every 14 days or as per the need

4. Referral Criteria

Same as mentioned in Level 1 and any of these

- Psychological imbalance
- Any red flag signs.
- Signs of CVD as stroke, transient ischaemic attack and angina.

At Level 3:

(Ayush hospitals attached with teaching Institution, District Level/Integrated/State Ayush Hospitals, Allopathic hospitals also having tertiary care facilities either standalone or integrative management facilities.

- Multiple departments/facilities for diagnosis and interventions
- Must provide additional facilities like dieticians, counselling, Physiotherapy unit and sophisticated procedures. (as applicable)

Clinical Diagnosis: Same as levels 1 & 2. Confirm diagnosis and severity with the help of the following investigations:

- Plasma Leptin
- Treadmill Test or Exercise stress Test to evaluate the efficacy of functioning of heart during exercises

Management

Along with level 1 medications including detoxification treatment any of the following medicines can be used.

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 11

Sl. No	Drugs	Dosage Form	Dose	Time	Duration and Frequency	Adjuvants/ Aṇupāṇam
Maathirai - Tablet						
1.	<i>Maha vasantha Kusumagaram</i> ³⁰	Tablet	65-130 mg	Twice a day	90 days	Honey
Parpam - White calx						
2.	<i>Thanga Parpam</i> ³⁰	White calx	30-65 mg	Twice a day	90 days	Honey
Chenduram - Red calx						
3.	<i>Annabedhi Chenduram</i> ³⁰	Red calx	500-200 mg	Twice a day	90 days	Honey

Sl. No	Drugs	Dosage Form	Dose	Time	Duration and Frequency	Adjuvants/ Anupānam
4.	<i>Ayakantham Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
5.	<i>Aya Chenduram</i> ³⁰	Red calx	60-130 mg	Twice a day	90 days	Honey
6.	<i>Vedi Annabedhi Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
7.	<i>Vediyuppu Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
8.	<i>Rasa Chenduram</i> ³⁰	Red calx	130 mg	Twice a day	90 days	Honey
9.	<i>Mandura Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
10.	<i>Gowri Chinthamani Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
11.	<i>Padikara Chenduram</i> ³⁰	Red calx	65-130 mg	Twice a day	90 days	Honey
12.	<i>Purna Chandrothayam</i> ³⁰	Red calx	30-65 mg	Twice a day	90 days	Honey

Varma maruthuvam^{43,44}

- *Tivalai kalam*
- *Puruva varmam*
- *Kaikāvuḷi varmam*
- *Mūttira kalam*
- *Uḷkuttu varmam*
- *Taṭciṇai kalam*

1. Recommended Diet & Lifestyle As per level 1

2. Restricted Diet & Lifestyle

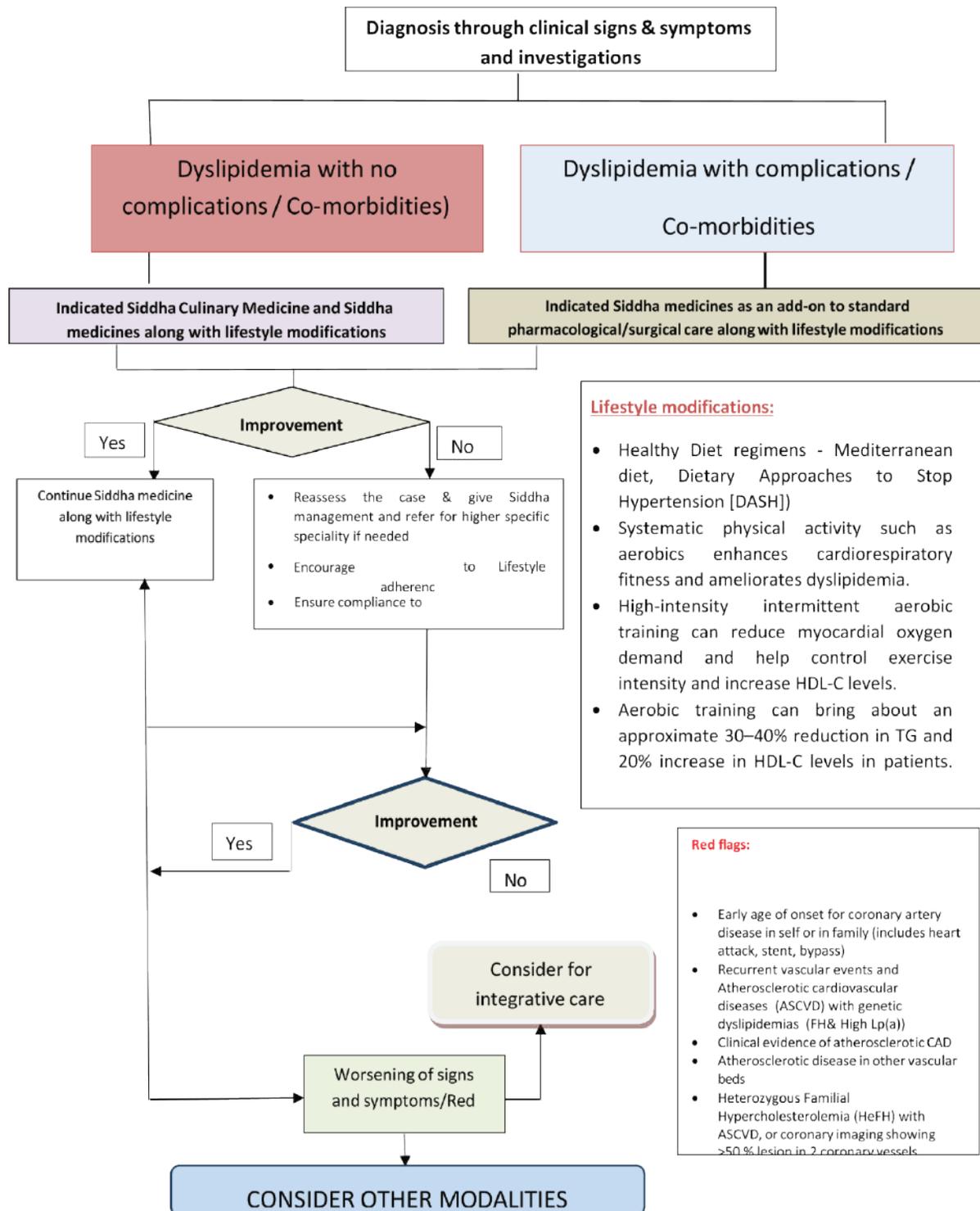
3. Follow Up every 14 days or as per need

4. Referral Criteria⁵³

Same as mentioned in Level 1 and any of these

- Morbid obesity not responding to treatment
- Uncontrolled hypertension
- Worsening Hypertriglyceridemia
- Worsening insulin resistance and hyperglycaemia
- Suspected Cardiac arrhythmias
- Recurrent vascular events and ASCVD with genetic dyslipidemia (FH& High Lp(a))
- Suspected Polycythaemia
- Other modalities can be considered depending on the case and to rehabilitate properly.

ALGORITHM OF TREATMENT PROCESS FOR DYSLIPIDEMIA



REFERENCES

1. ICD-11 for Mortality and Morbidity Statistics [Internet]. Who.int. 2025 [cited 2025 Feb 14]. Available from: <https://icd.who.int/browse/202501/mms/en#1191191920%2Fun specified>.
2. De Ferranti SD, Newburger JW. Dyslipidemia in children and adolescents: Definition, screening, and diagnosis. UpToDate, Waltham, MA, USA. 2020.
3. Uthamarayan KS. Siddha Maruthuvanga Surukkam. 3rd ed. Vol. 1. Chennai: Directorate of Indian Medicine and Homeopathy, Govt. of Tamil Nadu; 2006; 180-189.
4. World Health Organization. Global health observatory data repository Geneva: World Health Organization;2018
5. Gupta R, Rao RS, Misra A, Sharma SK. Recent trends in epidemiology of dyslipidemias in India. *Indian Heart J.* 2017;69(3):382-392. doi: 10.1016/j.ihj.2017.02.020.
6. Mohamed-Yassin MS, Baharudin N, Abdul-Razak S, Ramli AS, Lai NM. Global prevalence of dyslipidaemia in adult populations: a systematic review protocol. *BMJ Open.* 2021;11(12): e049662. Published 2021 Dec 3. doi:10.1136/bmjopen-2021-049662
7. Collins R, Reith C, Emberson J, et al. Interpretation of the evidence for the efficacy and safety of statin therapy. *Lancet* 2016; 388:2532–61.
8. Ference BA, Ginsberg HN, Graham I, et al. Low-Density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement.
9. Silverman MG, Ference BA, Im K, et al. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis. *JAMA* 2016; 316:1289–97.
10. Ramjee V, Sperling LS, Jacobson TA. Non-high-density lipoprotein cholesterol versus apolipoprotein B in cardiovascular risk stratification: do the math. *J Am Coll Cardiol* 2011; 58:457–63.
11. Sawhney JP, Ramakrishnan S, Madan K, Ray S, Jayagopal PB, Prabhakaran D, et al. CSI clinical practice guidelines for dyslipidemia management: executive summary. *Indian Heart Journal.* 2024 Mar 1;76:S6-19.
12. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. *Ann Intern Med.* 1971 Sep;75(3):471-2.
13. Karantas ID, Okur ME, Okur NÜ, Siafaka PI. Dyslipidemia Management in 2020: An Update on Diagnosis and Therapeutic Perspectives. *Endocr Metab Immune Disord Drug Targets.* 2021;21(5):815-834.
14. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, Goldberg AC, Gordon D, Levy D, Lloyd-Jones DM, McBride P, Schwartz JS, Shero ST, Smith SC, Watson K, Wilson PW., American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014 Jul 01;63(25 Pt B):2889-934.
15. Pappan N, Awosika AO, Rehman A. Dyslipidemia. In: *StatPearls.* Treasure Island (FL): StatPearls Publishing; March 4, 2024.
16. Loscalzo J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. *Harrison's principles of internal medicine.* 2022 May.
17. Ministry of Ayush. NAMASTE - Portal [Internet]. Ayush.gov.in. MoA, Govt. of India; 2020 [cited 2024 Sep 12]. Available from: <https://namstp.ayush.gov.in/#/Siddha>
18. Berberich AJ, Hegele RA. A modern approach to dyslipidemia. *Endocrine reviews.* 2022 Aug 1;43(4):611-53.
19. Nikolaus Marx, Massimo Federici, Katharina Schütt, Dirk Müller-Wieland, Ramzi A Ajjan, Manuel J Antunes, Ruxandra M Christodorescu, Carolyn Crawford, Emanuele Di Angelantonio, Björn Eliasson, Christine Espinola-Klein, Laurent Fauchier, Martin Halle, William G Herrington, Alexandra Kautzky-Willer, Ekaterini

- Lambrinou, Maciej Lesiak, Maddalena Lettino, Darren K McGuire, Wilfried Mullens, Bianca Rocca, Naveed Sattar, ESC Scientific Document Group , 2023 ESC Guidelines for the management of cardiovascular disease in patients with diabetes: Developed by the task force on the management of cardiovascular disease in patients with diabetes of the European Society of Cardiology (ESC), European Heart Journal, Volume 44, Issue 39, 14 October 2023, Pages 4043–4140, <https://doi.org/10.1093/eurheartj/ehad192>
20. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American association of clinical endocrinologists and american college of endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease. *Endocr pract.* 2017;23(suppl 2):1-87. Doi: 10.4158/ep171764.appgl.
 21. Pacific WHO RO for the W. The Asia-Pacific perspective : redefining obesity and its treatment [Internet]. iris.who.int. Sydney : Health Communications Australia; 2000. Available from: <https://iris.who.int/handle/10665/206936>.
 22. Garg A, Garg V, Hegele RA, Lewis GF. Practical definitions of severe versus familial hypercholesterolaemia and hypertriglyceridaemia for adult clinical practice. *The lancet Diabetes & endocrinology.* 2019 Nov 1;7(11):880-6.
 23. Carmena R. Primary Mixed Dyslipidemias, Editor(s): Ilpo Huhtaniemi, Luciano Martini, Encyclopedia of Endocrine Diseases (Second Edition), Academic Press, 2019, Pages 314-319, ISBN 97801281222006, <https://doi.org/10.1016/B978-0-12-801238-3.65333-3>
 24. R.S.Ramaswamy, K.Kanakavalli, P.Sathyarajeswaran, et.al, Siddha Treatment Guidelines for selected Non-Communicable Disease Conditions, Siddha Central Research Institute, CCRS, Chennai, 1st edition 2019; pp.195-210.
 25. Shanmughavelu HP. Noi naadal Noi mudhal naadal thirattu Part 1. Department of Indian Medicine and Homoeopathy, Chennai. 2007.
 26. Goldberg AC, Hopkins PN, Toth PP, et al. Familial Hypercholesterolemia: Screening, diagnosis and management of pediatric and adult patients. *J Clin Lipidol.* 2011; 5:133-140.
 27. Marks D, Thorogood M, Neil HA, Humphries SE. A review on the diagnosis, natural history, and treatment of familial hypercholesterolaemia. *Atherosclerosis* 2003; 168(1):1-14.
 28. Kannusamy Pillai S. Pathartha Guna Vilakkam (Vegetable Kingdom) by re-edition, Chennai: B. Rathnayakar and Sons; 2017.
 29. Standard Siddha Treatment Guidelines [Internet]. National Institute of Siddha; 2019 [cited 2024 Sep11]. Available from: https://namayush.gov.in/sites/all/themes/webcms/images/org_str/SiddhaStandardTreatmentGuidelines.pdf
 30. The Siddha Formulary of India, Part-I. 1st ed. New Delhi: Government of India, Ministry of Health and family Welfare, Department of Health; 1992.
 31. K.S.Murugesu mudaliyar, Gunapadam mooligai vaguppu part 1, 9th edition, Dept of Indian medicine and homeopathy.Chennai, 2013.
 32. Sparshadeep EM, Nayak RP, Kavana GV, Rai M. Evaluation of hypolipidemic effect of *Tinospora cordifolia* in cholesterol diet induced hyperlipidemia in rats. *International Journal of Basic & Clinical Pharmacology.* 2016 Jul;5(4):1286.
 33. Choudhary R. Beneficial effect of *Allium sativum* and *Allium tuberosum* on experimental hyperlipidemia and atherosclerosis. *Pakistan Journal of Physiology.* 2008 Dec 31;4(2):7-10.
 34. Pourghassem-Gargari B, Ebrahimzadeh-Attary V, Rafraf M, Gorbani A. Effect of dietary supplementation with *Nigella sativa* L. on serum lipid profile, lipid peroxidation and antioxidant defense system in hyperlipidemic rabbits. *J Med Plants Res.* 2009 Oct 1;3(10):815-21.
 35. Maruthappan V, Shree KS. Hypolipidemic activity of *Haritaki* (*Terminalia chebula*) in atherogenic diet induced hyperlipidemic rats. *Journal of advanced pharmaceutical technology & research.* 2010 Apr 1;1(2):229-35.
 36. Jafarnejad S, Keshavarz SA, Mahbubi S, Saremi S, Arab A, Abbasi S, Djafarian K. Effect of ginger (*Zingiber officinale*) on blood glucose and lipid concentrations in diabetic and hyperlipidemic subjects: A meta-analysis of randomized controlled trials. *Journal of functional foods.* 2017 Feb 1;29:127-34.

37. Kanikowska D, Korybalska K, Mickiewicz A, Rutkowski R, Kuchta A, Sato M, Kreft E, Fijałkowski M, Gruchala M, Jankowski M, Bręborowicz A. Flaxseed (*Linum usitatissimum* L.) supplementation in patients undergoing lipoprotein apheresis for severe hyperlipidemia—A pilot study. *Nutrients*. 2020 Apr 18;12(4):1137.
38. Chika CU, Onuoha N, Ajah O, Nnaoma IE. Effect of Flavonoid Rich Fraction of *Coriandrum sativum* Leaf on Lipid Profile, Nitric Oxide, Ang II and Cardio Histopathology in L-NAME Intoxicated Experimental Rats. *Sch Bull*. 2022;8(6):201-11.
39. Jeevangi S, Manjunath S, Sakhare PM. A study of anti-hyperlipidemia, hypolipidemic and anti-atherogenic activity of fruit of *Embllica officinalis* (amla) in high fat fed albino rats. *International Journal of Medical Research & Health Sciences*. 2013;2(1):70-7.
40. Babu PS, Srinivasan K. Hypolipidemic action of curcumin, the active principle of turmeric (*Curcuma longa*) in streptozotocin induced diabetic rats. *Molecular and cellular biochemistry*. 1997 Jan;166:169-75.
41. Chitra, S. M.. (2023). In silico Computational Analysis of Siddha Formulation Veppampoo Maathirai against Hypertension. *Current Overview on Disease and Health Research Vol. 11*, 46–60. <https://doi.org/10.9734/bpi/codhr/v11/17815D>
42. Anonymous. *Siddha Vaidhya Thirattu*. 11nd ed. Department of Indian Medicine and Homeopathy; 2006.
43. Kannan Rajaram, T., *Varma Points and Relieving Methods on the basis of Finger Measurement Technique in Varmam Therapy*. Centre for Varma Medicine & Research, Rajaram Hospital: 2010.
44. Prof Ramasamy, R.S et al., *Guidelines for Practice of Siddha Varmam Therapy*. Central Council for Research in Siddha; 2017.
45. Tyson CC, Nwankwo C, Lin PH, Svetkey LP. The Dietary Approaches to Stop Hypertension (DASH) eating pattern in special populations. *Curr Hypertens Rep*. 2012;14(5):388-396. doi:10.1007/s11906-012-0296-1.
46. Vanhees L., Geladas N., Hansen D., Koudi E., Niebauer J., Reiner Z., Cornelissen V., Adamopoulos S., Prescott E., Börjesson M. Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular risk factors: recommendations from the EACPR. Part II. *Eur J Prev Cardiol*. 2012; 19:1005–1033. doi: [10.1177/1741826711430926](https://doi.org/10.1177/1741826711430926)
47. Kirkpatrick CF, Sikand G, Petersen KS, Anderson CA, Aspary KE, Bolick JP, Kris-Etherton PM, Maki KC. Nutrition interventions for adults with dyslipidemia: A Clinical Perspective from the National Lipid Association. *Journal of clinical lipidology*. 2023 Jul 1;17(4):428-51.
48. Hickey JT, Hickey L, Yancy Jr WS, Hepburn J, Westman EC. Clinical use of a carbohydrate- restricted diet to treat the dyslipidemia of the metabolic syndrome. *Metabolic syndrome and related disorders*. 2003 Sep 1;1(3):227-32.
49. Kim SA, Shin S. Red meat and processed meat consumption and the risk of dyslipidemia in Korean adults: A prospective cohort study based on the Health Examinees (HEXA) study. *Nutrition, Metabolism and Cardiovascular Diseases*. 2021 Jun 7;31(6):1714-27.
50. Thongtang N, Sukmawan R, Llanes EJ, Lee ZV. Dyslipidemia management for primary prevention of cardiovascular events: Best in-clinic practices. *Preventive Medicine Reports*. 2022 Jun 1; 27:101819.
51. Franklin BA. Preventing exercise-related cardiovascular events: is a medical examination more urgent for physical activity or inactivity? *Circulation*. 2014 Mar 11;129(10):1081-4.
52. Hoover L. Cholesterol management: ACC/AHA updates guideline. *American Family Physician*. 2019 May 1;99(9):589-91.
53. Olefsky JM. Obesity. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, editors. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw-Hill Education; 1994. p. 446-452.



CHAPTER

3

GOUT





GOUT

(ICD) 10- M10.9 ¹

(ICD) 11- FA25.2Z

Peruviral vātam (பெருவிரல் வாதம்)

Viral vātam (விரல் வாதம்)²

WHO International Standard Terminologies on Siddha medicine: ISMT-4.24.218

Amuri amila Paṭital (அமுரி அமில படிதல்)

CASE DEFINITION

Gout is a chronic disease of deposition of monosodium urate crystals (crystal-induced arthritis), which form in the presence of increased urate concentrations. It is characterized by severe pain, redness, tenderness in joints which occur due to too much uric acid crystal deposits in the joints. ³⁻⁵

INTRODUCTION

- It is the most common inflammatory arthritis in men and in older women.
- Globally, the Gout is prevalent in a range of <1% to 6.8% and an incidence of 0.58-2.89 per 1,000 person-years. Gout is more prevalent in men than in women with increasing age and in some ethnic groups.
- In India, approximately 0.12-0.19% population is affected by gout with male preponderance. The reported male to female ratio is approximately 7:1 to 9:1 but in people over the age of 65 this ratio is reduced to 3:1. Polyarticular gout is more frequent in the elderly and females.
- Initial presentation is predominantly monoarticular with the ankle joint being the commonest to be involved. But overall, the first metatarsophalangeal (MTP) joint is the commonest joint affected with > 90% having this joint involvement at some point of the disease. ⁶⁻⁸
- Risk factors include hyperuricemia, genetic factors, dietary factors like intake of meat, seafood, sugar-sweetened soft drinks and foods high in fructose, alcohol consumption, especially beer and hard liquor, obesity, hypertriglyceridemia, metabolic syndrome, increased diuretic use, chronic renal disease and recent surgery or trauma, hypertension, diabetes and menopause. ⁹⁻¹²

CLINICAL EXAMINATION

The signs and symptoms of gout almost always occur suddenly and often at night. They include:

- **Intense joint pain:** Gout usually affects the large joint of your big toe, but it can occur in any joint. Other commonly affected joints include the ankles, knees, elbows, wrists and fingers. The pain is likely to be most severe within the first four to 12 hours after it begins.

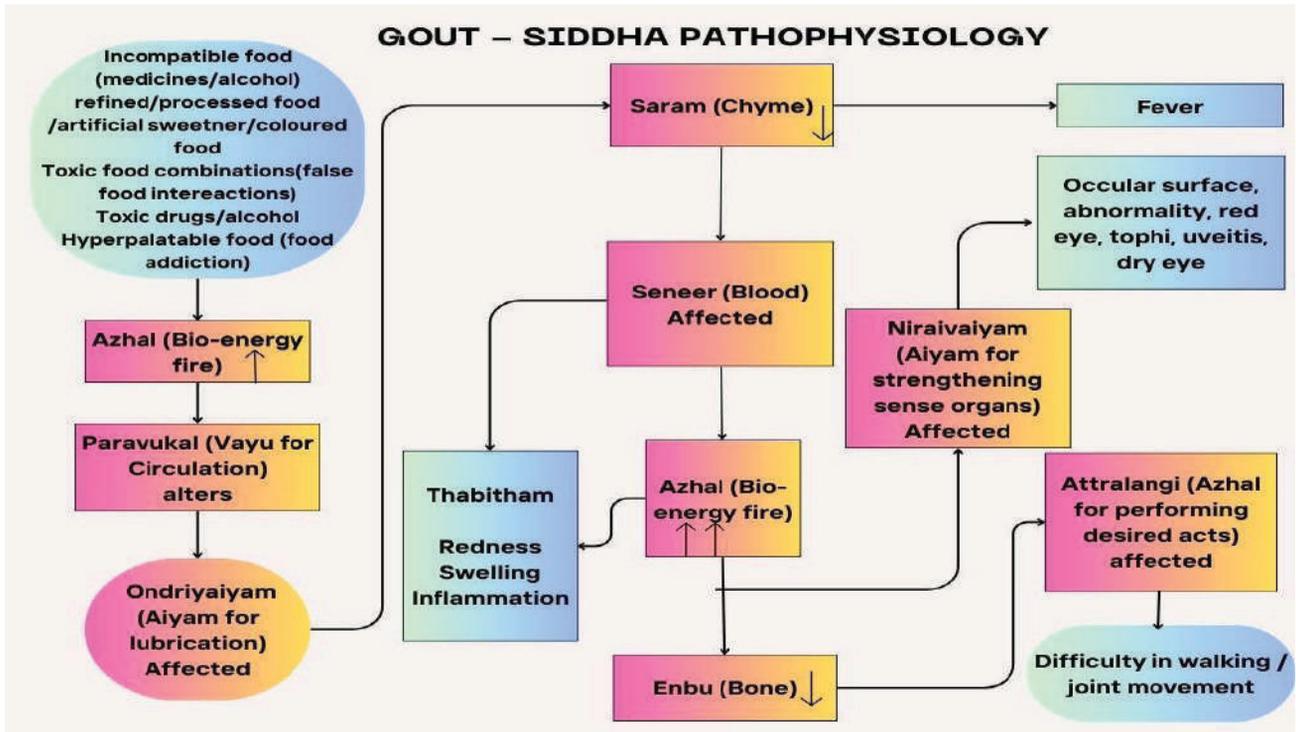


Fig. 1 Gout Siddha Pathophysiology

- **Lingering discomfort:** After the most severe pain subsides, some joint discomfort may last from a few days to a few weeks. Later attacks are likely to last longer and affect more joints.
- **Inflammation and redness:** The affected joint or joints become swollen, tender, warm and red.
- **Limited range of motion:** As gout progresses, patients may not be able to move joints normally.



Fig. 2: (a) Acute gout. Note the swelling and erythema of the first metatarsal phalangeal joint. (b) Diffuse swelling of the dorsum of the left hand is evident in this patient with acute gouty arthritis (left panel).¹³



Fig. 3: Generalized chronic tophaceous Gout (a) Nodules located in the hands, elbows, legs, buttocks and abdominal wall (arrows) (b) Nodules in periarticular structures and arthritis only in few joints.¹⁴

DIFFERENTIAL DIAGNOSIS

The following diseases must be considered in differential diagnosis of acute gout:

Table 1

Condition	Differential Features
Septic arthritis	<ul style="list-style-type: none"> ● Knee is most commonly involved (may be any joint distribution) ● Synovial fluid findings: <ul style="list-style-type: none"> ○ WBC Count > 50,000 per mm³ ○ Culture positive ○ Synovial fluid crystals absent ○ Radiography findings- Joint effusion; radiography results otherwise normal early in the disease
Trauma	History of injury will be present.
Pseudogout	<ul style="list-style-type: none"> ● Knee, wrist, or first metatarsophalangeal joints are commonly involved. ● Synovial fluid findings: <ul style="list-style-type: none"> ○ WBC Count 2,000 to 50,000 per mm³ ○ Culture negative ○ Synovial fluid crystals-Rhomboid shaped, weak positive birefringence ○ Radiography findings-soft tissue swelling, chondrocalcinosis (calcification of cartilage)
Rheumatoid arthritis	<ul style="list-style-type: none"> ● Arthritis of three or more joint areas ● Symmetrical arthritis ● Morning stiffness (> 1 hour) ● Positive rheumatoid factor ● Positive anti-CCP antibody ● Elevated ESR and CRP
Psoriatic arthritis	<ul style="list-style-type: none"> ● Onset usually between 25 and 40 years of age ● Most commonly in patients with current or previous skin psoriasis (70%) ● Affection of the DIP joints of the hands. However, unlike hand OA, psoriatic arthritis may target just one finger, often as dactylitis and characteristic nail changes are usually present. ● HLA-B27 Positive.

Condition	Differential Features
Reactive arthritis	<ul style="list-style-type: none"> • Monoarthritis or oligoarthritis following a recent infection (e.g., urethritis, enteric). • Asymmetric pattern of joint involvement • Symptoms or signs of enthesopathy, Keratoderma blennorrhagica or circinate balanitis • Radiologic evidence of sacroiliitis and/or spondylitis • The presence of human leukocyte antigen (HLA) B27
Monoarthritis	<ul style="list-style-type: none"> • Inflammation of single joint. Laboratory tests (blood chemistries, urinalysis) and diagnostic modalities (X-rays, CT scans, MRI) should be considered to confirm clinical impression.
Acute bursitis	<ul style="list-style-type: none"> • Gout can mimic bursitis as well, especially at the olecranon, prepatellar and infrapatellar bursa, as these joints are common locations for the formation of gouty tophi or pain from pseudogout. • Imaging can be helpful to narrow down the differential diagnosis. MRI can be used to evaluate the deeper bursa. Aspiration of the inflamed bursa can be helpful when there is a question of septic bursitis.
Tenosynovitis	<ul style="list-style-type: none"> • Centesis of the tenosynovial sheath and microscopic examination should be encouraged in acute tenosynovitis as gout flares may mimic infectious tenosynovitis.

- In Siddha Medicine: ^{15,16}
- *Vātacurōṇitam*
- *Narittalai Vātam*
- *Karastampam*
- *Vātakarṣaṇam*
- *Pēy Vātam*

SUPPORTIVE INVESTIGATIONS ¹⁷⁻¹⁹

Identification of urate crystals in fluid from an affected joint is the definitive diagnostic test for the diagnosis of gout. In practice, this test is applied to only a minority of patients. Guidelines exist for clinical diagnosis without joint aspiration. Other tests which may be considered are:

Table 2

Test	Comment
Essential investigations	
Serum urate concentration	Level may go down in few cases during an acute attack (serum uric acid levels ≤ 6 mg/dL)
Advanced investigations	
X ray	X-ray has low sensitivity for diagnosis of Gout. In the initial presentation only an increased soft tissue volume and density can be seen. In chronic tophaceous gout, radiographic signs include visualizing tophi as soft tissue or intraosseous masses, whether or not containing calcifications; and the presence of a non- demineralizing arthropathy accompanied by erosions presenting margins which may be sclerotic or protruding. The Martel's sign (Fig. 3) consists in the presence of a protruding, salient bone edge separated from a tophus and leaning on it.

Test	Comment
	 <p data-bbox="943 720 1013 753">Fig. 4</p>
Ultrasonography (USG)	Characteristic for the diagnosis of gout is the “double contour signal”, which is characterized by an irregular linear hyper echoic layer on the superficial margin of the anechoic hyaline cartilage and parallel to the bone cortex, without a posterior acoustic shade.
Dual Energy Computed tomography (DECT)	CT allows the visualization of tophi in both the subcutaneous tissue and in intra-articular areas. This method also helps to identify bone erosion.
Complete blood count /ESR	To exclude myeloproliferative disorders; raised white cell count may indicate septic arthritis
Renal function	Hyperuricemia can occur in renal failure
Fasting lipids, glucose and thyroid functions	Hyperlipidaemia, diabetes mellitus, hypothyroidism and possibly hyperthyroidism are associated with gout
Urinary urate excretion	Some authorities advise measuring this if the serum urate concentration is >0.8 mmol/l because of risk of renal stone formation
CRP	High levels of CRP are expected in patients experiencing acute gout attacks.
RA factor	To rule out Rheumatoid arthritis.

DIAGNOSTIC CRITERIA^{7,20}

The diagnosis of Gout is primarily clinical and made after a complete medical history and physical examination. Gout undergoes four phases during its course, which are stated below:

- Asymptomatic hyperuricaemia:** In this stage, patients have no symptoms or signs and are usually accidentally discovered when measuring serum uric acid (serum level greater than 7 mg/dL). This condition should not be treated with any medication.
- Acute gouty attack:** Classically, there is no hyper uricemia. It produces an acute monoarthritis of rapid onset, often waking patients from sleep, reaching a peak within 24 to 48 hours. The pain is intense and patients often cannot wear socks or touch bed sheets during flare-ups with marked exacerbation of pain even at the simple touch. The affected joints become red, shiny and tender in a few hours. The most affected joints are big toe also

known as podagra (50% of initial attacks), foot, ankle, mid tarsal, knee, wrist, finger and elbow. Acute flares also occur in periarticular structures, including bursae and tendons.

- **Inter-critical period:** During the period between acute attacks the patient is asymptomatic even if monosodium Urate (MSU) deposition may continue to increase silently.
- **Chronic tophaceous gout:** It is characterized by the deposition of solid MSU crystal aggregates in various locations including joints, bursae and tendons as tophi. Tophaceous gout may lead to significant morbidity and, if untreated, can cause prominent joint damage and marked functional impairment.

The ACR/EULAR gout classification criteria 2015 ²¹

STEP 1: Entry Criterion: If yes, Classification criteria required for positive diagnosis ε 1 episode of swelling, pain or tenderness in a peripheral joint/ bursa

STEP 2: Sufficient Criterion: If yes, diagnosis is positive Presence of Monosodium Urate (MSU) crystals in a symptomatic joint, bursa or tophus

STEP 3: Classification Criteria:

Table 3

Criteria	Categories	Score	
Clinical	Pattern of joint/bursa involvement	Ankle or midfoot (mono-/oligo-)	1
	Characteristics of the episode(s) ever	MTP1 (mono-/oligo-)	2
	(erythema overlying joint, cannot bear touch/pressure to the affected joint, walking difficulty)	One characteristic	1
		Two characteristics	2
		Three characteristics	3
	Time-course of episode(s) ever	One typical episode	1
Clinical evidence of tophus	Recurrent typical episode	2	
	Present	4	
Laboratory	Serum uric acid level (SU)	6 to <8 mg/dL	2
		8 to <10 mg/dL	3
		>10 mg/dL	4
Imaging	Imaging evidence of urate deposition	Present (US: DCS or DECT)	4
	Imaging evidence of gout-related joint damage	Present (X-ray gouty erosion)	4
		Maximum score	23
If SU <4 mg/dL: take -4 points; if MSU is negative take -2 points			

* MTP1: the first metacarpophalangeal joint, US: ultrasonography, DCS: double contour sign DECT: dual-energy computed tomography, MSU: monosodium urate.

According to the diagnostic criteria, gout is considered when the sum of scores from domains such as the presence of clinical symptoms, level of serum urate and radiographic imaging (plain X-ray and ultrasound) is **greater than 8 points**.

PRINCIPLES OF MANAGEMENT

Red Flag signs:

These signs should be assessed before initiating treatment for need for management consultation through modern medicine.

- Uncontrollable pain
- Joint destruction
- Constitutional features such as fever, weight loss and malaise
- Renal failure

Patients should be educated on their diagnosis. They should be educated about the natural history of disease with possible complications. Therapeutic options need to be discussed along with dietary restrictions and lifestyle changes such as exercise and weight control that might be helpful.

A. Prevention Management

Levels of Prevention for GOUT

- Primordial Prevention: Prevent development of risk factors like high uric acid, poor diet, or metabolic syndrome.
- Primary Prevention: Prevent the onset of gout in high-risk individuals (e.g., family history, hyperuricemia).
- Secondary Prevention: Prevent recurrence of gout attacks and development of chronic gout.
- Tertiary Prevention: Manage chronic tophaceous gout and prevent joint damage or disability.

Siddha System of Medicine emphasis adhering to *Tēraiyaṛ piṇi aṇukā viti* for prevention of disease and lead to healthy life.

Table 4

Dietary Habits (<i>Uṇavu Muṛaika!</i>)	
Do's - <i>Pattiyam</i>	Don'ts - <i>Apattiyam</i>
<ul style="list-style-type: none"> • Drink warm water • Add <i>Trithoda sama porutgal</i> inclusive of turmeric, pepper, cumin seeds, asafoetida, dry ginger, cardamom, fenugreek and garlic in diet • Include moderate intake of nuts • Include lean proteins and low fat dairy in diet • Follow Dietary Approaches to Stop Hypertension (DASH)-style diet and to avoid use of diuretics²² 	<ul style="list-style-type: none"> • Avoid deep fried food and junk foods • Avoid overeating or skipping meals • Avoid of triggering foods such as red meats, seafood and fermented items • Avoid carbonated drinks, ice creams and chocolate

Lifestyle Practices (Vālvīyal Muraika!)	
Do's	Don'ts
<ul style="list-style-type: none"> • Meditation and Physical activity • Better balance of mood and sleep • Advised therapeutic purgation once in every four month • Regular stress management • Maintain ideal weight²² 	<ul style="list-style-type: none"> • Avoid daytime sleep or oversleeping • Avoid sedentary life style • Avoid stress • Avoid nap or sleep after food • Avoid purine rich diet like sea food, red meat, lentils, chickpeas, kidney beans etc., • Avoid alcohol and smoking • Avoid extreme weather exposures • Avoid vigorous exercise

Yoga: Various Yoga practices are helpful for the management of Gout. These include Pranayama like Bhastrika, Kapalabhati and Anuloma-Viloma; various relaxation techniques viz. twisting movement of the body; yogasanas like Vajrasana, Trikonasana, Dhanurasana, Naukasana, Ardha Matsyendrasana, Pavana Muktasana and Surya namaskara.

B. Curative Interventions

At Level 1: (Solo Siddha Physician Clinic/Health & Health Clinic/PHC (Optimal Standard of treatment in a situation where technology and resources are limited)

- **Clinical Diagnosis:** The diagnosis of Gout is primarily clinical and made after a complete medical history and physical examination. However, some investigations, like a complete hemogram, urine routine/microscopic and serum uric acid level, RA factor, CRP may be done.

Management

The first line of treatment is to normalize the altered or deranged humours and revitalization of seven fundamental tissues through detoxification methods followed by internal medications. The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

Enṇey kuḷiyal (Therapeutic oil bath):²³

Enṇey kuḷiyal is a preparatory procedure in which consists medicated oil massage with a bath of lukewarm water. It will strengthen the five sensory organs. According to disease severity, *enṇey kuḷiyal* can be advised. The days of *ennai kuḷiyal* are decided according to the discretion of the physician.

- *Arakku Thylam* – Quantity sufficient (External use)
- *Santhanathi Thylam* – Quantity sufficient

Rules to be followed during *Enṇey kuḷiyal*

Apply oil before 7 am. Instil 1 drop of medicated oil in each eye, 2 drops in each nostril and 3 drops in each ear. Spread over the medicated oil from head to foot and give a gentle massage. After application, allow it for 45 minutes and bathe with lukewarm water with herbal hair wash powder.

Take tender vegetables and easily digestible food. Avoid daytime sleep, intercourse and exposure to sunlight and cold items on the day of oil bath.

Kalical maruttuvam (Therapeutic purgation)²⁴

Table 5

S. No.	Drugs	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ Aṅupāṇam
1.	<i>Kumari Ennai</i> ²⁵	Internal oil	30 ml	Once in a Day Morning in an empty stomach	Once a week for 2 weeks	Milk
2.	<i>Siddhathi Ennai</i> ²⁵	Internal oil	5-10 ml	Once in a Day (OD) - Morning in an empty stomach	Once a week for 2 weeks	Milk

*Any of the medicine can be used

Rules to be followed during Kalical Maruttuvam

- The medication should be taken in the early morning 5 to 6 AM
- After the average number (5-6 times) of bowel evacuations, the patient is advised to take buttermilk/ lemon juice/fried cumin seeds decoction/Ash of sweet flag (*Vacampū*).
- At the end of proper purgation, watery diarrhoea commences. This indicates that the purgation therapy has been completed properly.
- After purgation, the patient may have symptoms like tiredness, slimness, lightness of the body and tiredness of sense organs which is a good sign.
- Dietary regimen during purgation:
 - Buttermilk
 - Rice porridge
 - Double-boiled porridge
 - Luke warm water
- Precautions:
 - Avoid daytime sleep on the day of purgation therapy
 - Should not take heavy meals before or during the procedure
 - Avoid intercourse

Compound formulation

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 6

S. No	Drugs	Dosage form	Dose	Time	Duration & Frequency	Adjuvants/ Aṇupāṇam
Kudineer /Decoction						
1.	<i>Neermulli Kudineer</i> ²⁴	Decoction	60 ml	BD Before food	15 days	-
2.	<i>Vatha Sura Kudineer</i> ²⁵ (if fever persists)	Decoction	60 ml	BD Before food	15 days	-
3.	<i>Kurunthotti Kudineer</i> ²⁶	Decoction	60 ml	BD Before food	15 days	-
Chooranam/medicinal powder						
1.	<i>Parangipattai Chooranam</i> ²⁵	Medicinal powder	1-2 g	BD After food	15 days	Ghee/Palm Jaggery
2.	<i>Seenthil Chooranam</i> ²⁵	Medicinal powder	1-2 g	BD After food	15 days	Ghee/Palm Jaggery
3.	<i>Thirikaduku Chooranam</i> ²⁵	Medicinal powder	1-2 g	BD After food	15 days	Honey
Maathirai /Tablet						
1.	<i>Vatha Ratchasan Maathirai</i> ²³	Tablet (100 mg)	1-2 Nos	BD After food	15 days	Honey/Hot Water/ suitable herbal decoction
2.	<i>Karuppu Vishnu chakkaram</i> ²³	Tablet (100 mg)	1-2 Nos	OD After food	7-14 days	Honey/Hot Water/ suitable herbal decoction
External application						
1.	<i>Sivappu kukkil Thylam</i> ²⁷	External Oil	q.s	BD	15 days	-
2.	<i>Vathakesari Thylam</i> ²³	External Oil	q,s	BD	15 days	-

Varmam Maruttuvam^{28,29}

- Muṭiccu varmam
- Moḷi piṛalkai
- Cavvukālam
- Kavulikālam
- Kaimūṭṭu varmam

1) Recommended Diet & Lifestyle³⁰⁻³⁴

Lifestyle and dietary recommendations for gout patients should consider overall health benefits and risk since gout is often associated with metabolic syndrome and an increased future risk of cardiovascular disease (CVD) and mortality. Some of the measures are:

- Exercise: Apparently in healthy, vigorously active men, the prevention of weight gain through the promotion of vigorous physical activity may help to prevent gout.
- Overweight patients should aim for a normal weight but should not crash-diet or follow protein rich diet.

- Patients known to suffer from gout and kidney stones should be instructed to consume sufficient fluids (>2 L /day).
- Adherence to Dietary Approaches to Stop Hypertension (DASH)-style diet.
- Encourage low fat or non-dairy products, yellow lentil (moong dal).

2) **Restricted Diet & Lifestyle** ³⁵⁻³⁷

- Reduced-fat foods and vegetarian sources of protein should be integrated into the diet.
- Avoid or reduce purine (protein) rich foods such as meat and yeast, sweet breads, liver, kidney, consumption of alcohol, particularly beer and spirits. Patients should be encouraged to refrain from consuming alcohol on at least 3 days per week.
- Avoid sugar-sweetened beverages, fruit juices and sweetened soda as fructose inhibits uric acid excretion by the kidneys.
- Avoid sea foods, juicy fruits, oats and germinated gram.

3) **Follow Up:** Every 7 days or as per the need.

Reviews should include:³⁸

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.
- Monitoring of serum uric acid levels.
- Monitoring the long-term course of the condition.
- Discussing the person's knowledge of the condition, any concerns they have, their personal preferences and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.
- Reviewing the co-morbidities associated with gout.

Referral Criteria

- Uncontrollable pain and no response to treatment
- Joint destruction
- High fever, weight loss and malaise
- Rise in serum creatinine and serum urea above normal limits
- Suspected cardiovascular complications due to Gout
- Patients taking chemotherapy for neoplastic diseases
- Uncontrolled comorbidities
- Evidence of an increase in severity/complications
- Diagnostic uncertainty
- Substantial impact on their quality of life and activities of daily living.

II. **At Level 2:** (CHC/Small hospitals (10-20 bedded hospitals with basic facilities such as routine, investigation, X-ray))

- **Clinical Diagnosis:** Same as level 1. The case referred from Level 1, or a fresh case must be evaluated thoroughly for any complications.

- **Investigations:** The diagnosis would be primarily clinical along with some investigations which will be necessary to investigate complications or exclude other differential diagnoses as follows:
 - Serum urate concentration
 - Complete blood count/ESR
 - Renal function Test
 - Fasting lipids, glucose and thyroid functions
 - Urinary urate excretion

Management

Same as level 1. For the patients referred from Level-1, treatment given in Level-1 may be continued if appropriate for the presenting condition or the case may be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the medications mentioned for Level-1 may also be considered, however, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient. Along with level 1 medications including detoxification treatment any of the following medicines can be used.

The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

a. Internal medication

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 7

S. No	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Añupāñam
Mezhugu / Medicinal Wax						
1.	<i>Rasagandhi Mezhugu</i> ²³	Medicinal Wax	250-500 mg	BD, After food	1 month	Palm jaggery
2.	<i>Idivallathi Mezhugu</i> ²³	Medicinal Wax	250-500 mg	BD, After food	2-3 weeks	Palm jaggery
Any one of the drugs can be used.						
Chenduram / Red calx						
1.	<i>Arumuga Chenduram</i> ²³	Red calx	50-100mg	BD, After food	2-3 weeks	Honey / Palm jaggery / betel leaf
2.	<i>Kalamega Narayana Chenduram</i> ²⁷	Red calx	50-100mg	BD, After food	2-3 weeks	Honey / Palm jaggery / betel leaf
3.	<i>Poorna Chandrothyam</i> ²³	Red calx	50-100mg	BD, After food	2-3 weeks	Honey / Palm jaggery / betel leaf
4.	<i>Linga Chenduram</i> ²⁵	Red calx	50-100mg	BD, After food	2-3 weeks	Honey / Palm jaggery / betel leaf
5.	<i>Ayaveera Chenduram</i> ²⁷	Red calx	50-100mg	BD, After food	1 week	Honey / Palm jaggery / betel leaf

S. No	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Aṅṅupāṅṅam
Parpam / White calx						
1.	<i>Sangu Parpam</i> ²³	White calx	100-200mg	BD, After food	1 month	Ghee / Butter
2.	<i>Muthuchippi Parpam</i> ²³	White calx	100-200mg	BD, After food	1 month	Ghee / Butter
3.	<i>Pavala Parpam</i> ²³	White calx	100-200mg	BD, After food	2-3 weeks	Ghee / Butter
4.	<i>Thanga Parpam</i> ²³	White calx	100-200mg	BD, After meal	1 week	Ghee
5.	<i>Muthu Parpam</i> ²³	White calx	100-200mg	BD, After meal	2-3 weeks	Ghee
Maathirai / Tablet						
1.	<i>Soolai Kudaaram</i> ²³	Tablet (100 mg)	1-2 Nos	BD, After meal	7 days	Honey / Suitable herbal decoction

External Therapies / other procedures

- *Attai Vidal* (Leech Therapy) twice a week on the swollen areas
- *Patru* (poultice)
 - *Mosambara Patru* (Resin derived from *Aloe Vera*)
 - *Kazharchi Patru* (*Caesalpinia bonducella*)
 - *Aavarai Ulunthu Patru* – (*Cassia auriculata* & *Vigna mungo*)
 - *Kaavikal Patru* (Red Ochre)
 - *Amukkura Kizhangu podi Patru* - (Root Tuber of *Withania somnifera*)
- *Kattu* (Compress /Bandage)
 - Fry the dry stems of *Piraṅṅai* (*Cissus quadrangularis*) with the juice of *Erukku* (*Calotropis gigantea*), crush well and apply as a compress
- *Pochu* (Liquid/ Oil Poultice)
 - *Ulundhu Thylam*
 - *Mezhugu Thylam*
 - *Karpoorathi Thylam*
 - *Vathakesari Thylam*
- *Kalimbu* (Ointment Application)
 - *Kungiliya Vennai*
- *Thylam*
 - *Pinda Thylam*
 - *Mayana Thylam*
- b. Siddhar Varmam Maruttuvam** ^(28,29)
 - *Muṅṅiccu varmam*
 - *Moli piraṅṅkai*

- *Cavvu kālam*
- *Kavuḷi kālam*
- *Kaimūṭu varmam*
- *Cuṇṭikai kālam*
- *Kālnerukku varmam*
- *Virutti kālam*
- *Uḷḷaṅkāl veḷḷai varmam*
- *Kompēri kālam*
- *Uppukkuṟi varmam*

1) Recommended Diet & Lifestyle

As per level 1

2) Restricted Diet & Lifestyle

3) Follow Up: Every 7 days or as per the need

4) Referral Criteria

Same as mentioned earlier in level 1 and any of these

- Failure of acute exacerbation to respond to initial medical management.
- Cases with prominent joint damage and marked functional impairment.
- Extra-articular tophi
- Uncontrolled complications such as acute uric acid nephropathy
- Any other complications that threaten the life of the patient.

III. At Level 3:

(Ayush hospitals attached with teaching Institution, District Level/Integrated/State Ayush Hospitals, Allopathic hospitals also having tertiary care facilities either standalone or integrative management facilities.

In this facility, all four stages of Gout can be managed through a combination of Siddha internal medication, external medicine and personalized dietary advice.

- **Clinical Diagnosis:** Same as levels 1 & 2.

Confirm diagnosis and severity with the help of investigations. MRI, CT scan, DECT, Cystatin C, IVP, chemical analysis of uric acid renal stones if present.

Management

Along with level 1 & 2 medications including detoxification treatment any of the following medicines can be used.

a. Internal medication

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 8

S. No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Aṅupāṇam
Chenduram / Red calx						
1.	<i>Kalamega Narayana Chenduram</i> ²⁷	Red calx	50-100 mg	BD After meal	2 weeks	Honey / Palm jaggery/ betel leaf
2.	<i>Poorna Chandrothyam</i> ²³	Red calx	50-100 mg	BD After meal	2-3 weeks	Honey / Palm jaggery/ betel leaf
Parpam /White calx						
1.	<i>Thanga Parpam</i> ²³	White calx	100-200 mg	BD After meal	2 weeks	Ghee
2.	<i>Muthu Parpam</i> ²³	White calx	100-200 mg	BD After meal	2-3 weeks	Ghee

External Therapies / other procedures – As per level 2**Varmam Maruttuvam**

- *Naṅkaṅapūtu*
- *kaṅṅupukai varmam*
- *Veḷḷai varmam*
- *kāri varmam*
- *Kaṅṅāṭi kālam*
- *Kāl caṅṅi aṅaṅkal*
- *Viḷaṅku varmam*
- *Kutikāl varmam*
- *Maṅṅai aṅaṅkal*
- *Pāta varmam*

Recommended Diet & Lifestyle

- 1) **Restricted Diet & Lifestyle** As per level 1
- 2) **Follow Up:** Every 7 days or as per the need
- 3) **Referral Criteria**
 - Same as mentioned earlier at level 2 along with
 - Any condition or serious complication not responding to treatment.

REFERENCES

1. 2024 ICD-10-CM Diagnosis Code M10.9: Gout, unspecified (icd10data.com) [Internet]. 2024 ICD-10-CM Diagnosis Code [updated 2024; cited 2024 May 23]. Available from: <https://www.icd10data.com/ICD10CM/Codes>
2. World Health Organization. WHO international standard terminologies on siddha medicine [Internet]. www.who.int. World Health Organization ; 2023 [cited 2024 May 20]. Available from: <https://www.who.int/publications/i/item/9789240064973>
3. Neogi T. Clinical practice. Gout. *N Engl J Med*. 2011;364(5):443-452
4. Davidson S, Bouchier I, Edwards C. Davidson's principles and practice of medicine. 21st ed. London: E.L.B.S. and Churchill Livingstone;1991
5. Dalbeth N, Merriman TR, Stamp LK. Gout. *Lancet*. 2016;388(10055):2039-2052.
6. Dehlin M, Jacobsson L, Roddy E. Global epidemiology of gout: prevalence, incidence, treatment patterns and risk factors. *Nat Rev Rheumatol*. 2020 Jul;16(7):380-390. doi: 10.1038/s41584-020-0441-1. Epub 2020 Jun 15. PMID: 32541923.
7. Kumar S, Gupta R, Suppiah R. Gout in women: differences in risk factors in young and older women. *NZMJ*.2012;125(1363):39-45.
8. Paul BJ, James R. Gout: an Asia-Pacific update. *Int J Rheum Dis*. 2017; 20(4): 407- 416.
9. Singh JA, Reddy SG, Kundukulam J. Risk factors for gout and prevention: a systematic review of the literature. *Curr Opin Rheumatol*. 2011;23(2):192-202.
10. Roddy E, Doherty M. Gout. *Epidemiology of gout. Arthritis Research & Therapy*. 2010; 12(6):223
11. Saag KG, Choi H. Epidemiology, risk factors and lifestyle modifications for gout. *Arthritis Res Ther*.2006;8(Suppl 1):2
12. Walker S.W. Laboratory reference ranges. In: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston, editors. Davidson's principles practice of medicine. 21st ed. Edinburgh; New York: Churchill Livingstone/Elsevier; 2010: p.1296.
13. Jelley MJ, Wortmann R. Practical Steps in the Diagnosis and Management of Gout. *BioDrugs*. 2000; 14 (2): 99-107.
14. Tristano AG. Generalised chronic tophaceous gout. *BMJ Case Rep*. 2009;2009: bcr03.2009.1668. doi: 10.1136/bcr.03.2009.1668. Epub 2009 Jun 3. PMID: 21686975; PMCID: PMC3027919.
15. CCRS. National AYUSH Morbidities and Standard Terminology Portal (NAMSTP)- Siddha. New Delhi: CCRAS, Ministry of AYUSH, Govt. of India; Available from: www.namstp.ayush.gov.in.
16. Anonymous Yugimuni vaithiya chinthamani - 800. Chennai: B.Rathina naiyakar and sons,Thirumagal vilasa achuu Nilayam (1969):54-67
17. Underwood M. Diagnosis and management of gout. *BMJ*. 2006;332(7553):1315- 1319.
18. Schlesinger N, Norquist JM, Watson DJ. Serum urate during acute gout. *J Rheumatol*.2009; 36(6):1287-89
19. Fernandes EDA, Bergamaschi SB, Rodrigues TC, Dias GC, Malmann R, Ramos GM, Monteiro SS. Relevant aspects of imaging in the diagnosis and management of gout. *Rev Bras Reumatol Engl Ed*. 2017 Jan-Feb;57(1):64-72. English, Portuguese. doi: 10.1016/j.rbre.2016.05.001. Epub 2016 Jun 24. PMID: 28137404
20. Grassi W, Angelis RD. Clinical features of gout. *Reumatismo*.2011; 63(4):238-245.
21. Neogi T, Jansen TLTA, Dalbeth N, et al. 2015 Gout classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Annals of the Rheumatic Diseases* 2015; 74:1789-1798.
22. McCormick N, Rai SK, Lu N, Yokose C, Curhan GC, Choi HK. Estimation of Primary Prevention of Gout in Men

- Through Modification of Obesity and Other Key Lifestyle Factors. *JAMA Netw Open*. 2020 Nov 2;3(11): e2027421. doi: 10.1001/jamanetworkopen.2020.27421. PMID: 33231639; PMCID: PMC7686865.
23. Anonymous. *Siddha Vaidhya Thirattu*. 11nd ed. Department of Indian Medicine and Homeopathy; 2006.
 24. Kuppusamy Mudaliyar, K.S., *Siddha Maruthuvam Pothu*. Chennai: Directorate of Indian Medicine & Homeopathy; 1936
 25. *The Siddha Formulary of India, Part-I*. 1st ed. New Delhi: Government of India, Ministry of Health and family Welfare, Department of Health; 1992.
 26. Kannusamy Pillai S. *Pathartha Guna Velakkam (Vegetable Kingdom)* by re-edition, Chennai: B. Rathnayakar and Sons; 2017.
 27. Thiyagarajan R. *Siddha Materia Medica (Mineral & Animal section)* in Tamil. 1st ed. Chennai: Department of Indian Medicine and Homeopathy; 2008.
 28. Kannan Rajaram, T., *Varma Points and Relieving Methods on the basis of Finger Measurement Technique in Varmam Therapy*. Centre for Varma Medicine & Research, Rajaram Hospital: 2010.
 29. Ramasamy, R.S et al., *Guidelines for Practice of Siddha Varmam Therapy*. Central Council for Research in Siddha; 2017.
 30. McCormick N, Rai SK, Lu N, Yokose C, Curhan GC, Choi HK. Estimation of Primary Prevention of Gout in Men Through Modification of Obesity and Other Key Lifestyle Factors. *JAMA Netw Open*. 2020 Nov 2;3(11): e2027421. doi: 10.1001/jamanetworkopen.2020.27421. PMID: 33231639; PMCID: PMC7686865.
 31. Janssen CA, Voshaar MAHO, Klooster PMT, Vonkeman HE, Laar MAFJVD. Development and validation of a patient-reported gout attack intensity score for use in gout clinical studies. *Rheumatology*. 2019; 58(11):1928-1934.
 32. Choi HK. A prescription for lifestyle changes in patients with hyperuricemia and gout. *Curr Opin Rheumatol*. 2010 Mar;22(2):165-72.
 33. Williams PT. Effects of diet, physical activity and performance, and body weight on incident gout in ostensibly healthy, vigorously active men. *Am J Clin Nutr*. 2008 May;87(5):1480-7. doi: 10.1093/ajcn/87.5.1480. PMID: 18469274; PMCID: PMC4090353.
 34. Kusmayanti GAD, Dewantari NM. The influence of low purine diet and physical activity on changing uric acid levels in hyperuricemia. *International Journal of Health Sciences*. 2017;1(3):1-9.
 35. Eggebeen AT. Gout: An Update. *American Family Physician*. 2007;76(6):801-808.
 36. Doherty M, Abhishek A. Clinical manifestations and diagnosis of osteoarthritis. Characteristics of specific joint involvement. In up to date. Post TW (Ed), UpToDate, Waltham, MA. (Accessed on August 11, 2022.) Available from: https://wolterkluwer.ccrhlibrary.in/contents/clinical-manifestations-and-diagnosis-ofosteoarthritis?Search=osteoarthritis&source=search_result&selectedtitle=2~150&us_age_type=default&display_rank=2
 37. Choi HK, Atkinson K, Karlson EW, et al. Purine-rich foods, dairy and protein intake, and the risk of gout in men. *N Engl J Med* 2004; 350:1093–103
 38. Ministry of Health & Family Welfare, Government of India. *Standard Treatment Guidelines. Management of Osteoarthritis Knee*. Macro Graphics Pvt. Ltd. August 2017.



CHAPTER

4

NON ALCOHOLIC FATTY LIVER DISEASE



NON ALCOHOLIC FATTY LIVER DISEASE

Siddha terminology - Aiyā kallīral noy¹

ICD 10 CODE: K76.0 Fatty (change of) liver, not elsewhere classified (non-alcoholic fatty liver disease) ²

CASE DEFINITION

Non-alcoholic fatty liver disease (NAFLD) is a spectrum of chronic liver disease characterized by accumulation of fat in the liver, Non-alcoholic steatohepatitis (NASH), and liver fibrosis unrelated to recent or ongoing significant amount of alcohol intake and due to over-nutrition and its associated metabolic syndrome.³ An international group of expert consensus statement suggested to change the name to Metabolic-associated Fatty Liver Disease (MAFLD).⁴ But due to the unavailability of an acceptable definition of metabolic dysfunction, currently the nomenclature of the condition is still to be accepted as NAFLD.⁵

INTRODUCTION (incidence/prevalence, mortality/morbidity)

- NAFLD is a spectrum of disorder ranging from Non-alcoholic Fatty liver to Non- Alcoholic steatohepatitis (NASH), NASH with fibrosis, NASH- cirrhosis and NASH associated with hepatocellular carcinoma (HCC).^{6,7}
- The prevalence of NAFLD in India varies from 9-35% as per the accordance to ultrasonography data.^{8,9} Studies demonstrated area-wise prevalence data of NAFLD with 16.6 % in Western India, 24.5 % in Eastern India, and 32 % in South India.⁸
- A certain proportion of patients suffering from NAFLD may have normal body mass index and such cases are known as 'Lean NAFLD'. A pooled proportion of studies show that Lean NAFLD consists of 16.97% of all persons suffering from NAFLD.⁵
- Metabolic syndrome (MS) or 'Syndrome X' characterized by a constellation of various components namely, obesity, type 2 diabetes, dyslipidemia, and hypertension. NAFLD and MS share the same associations and risk factors, and often NAFLD is considered as the hepatic manifestation of MS.⁹
- NAFLD is consistently associated with type 2 diabetes mellitus (28-55%) and dyslipidemia (27-92%). Two other factors namely hypertriglyceridemia (62%) and low HDL-cholesterol (54%) are found in NAFLD patients.⁹
- NAFLD is known to be associated with several extrahepatic conditions like chronic kidney disease (CKD),¹⁰ cardiovascular diseases,¹¹⁻¹³ osteopenia, osteoarthritis,¹⁴ obstructive sleep apnoea,¹⁵ hypothyroidism,¹⁶ and polycystic ovarian syndrome.^{17,18} NAFLD has also been shown to increase the risk of extrahepatic malignancies like carcinoma colon, gastric cancer, carcinoma pancreas, uterine, and breast conditions.¹⁹

- The most common cause of mortality in patients with NAFLD is cardiovascular diseases. Cancer related mortality is among the top three causes of death in patients with NAFLD. Patients with NASH have a higher liver-related mortality rate.²⁰
- Diseases of the liver is mentioned under *Kallīral nōykal* in *Siddha* literatures with synonyms such as *Valappērral noy*, *mānta kaṭṭi*, *kal māntam*, *yākkutam*. There are three types of *Kallīral nōykal* caused by vitiation of humors. *Ayyā kallēral nōy* can be interpreted as Non-alcoholic fatty liver disease. *Ayyā kallēral nōy* results by aggravation *Aḷal kurram* along with *Ayyā kurram*. It is usually characterised by enlargement of liver, Fever, Vomiting, Headache, Dysuria, Dark coloured urine, Jaundice, Swelling of the body, Pallor followed by ascites in the later stages.¹

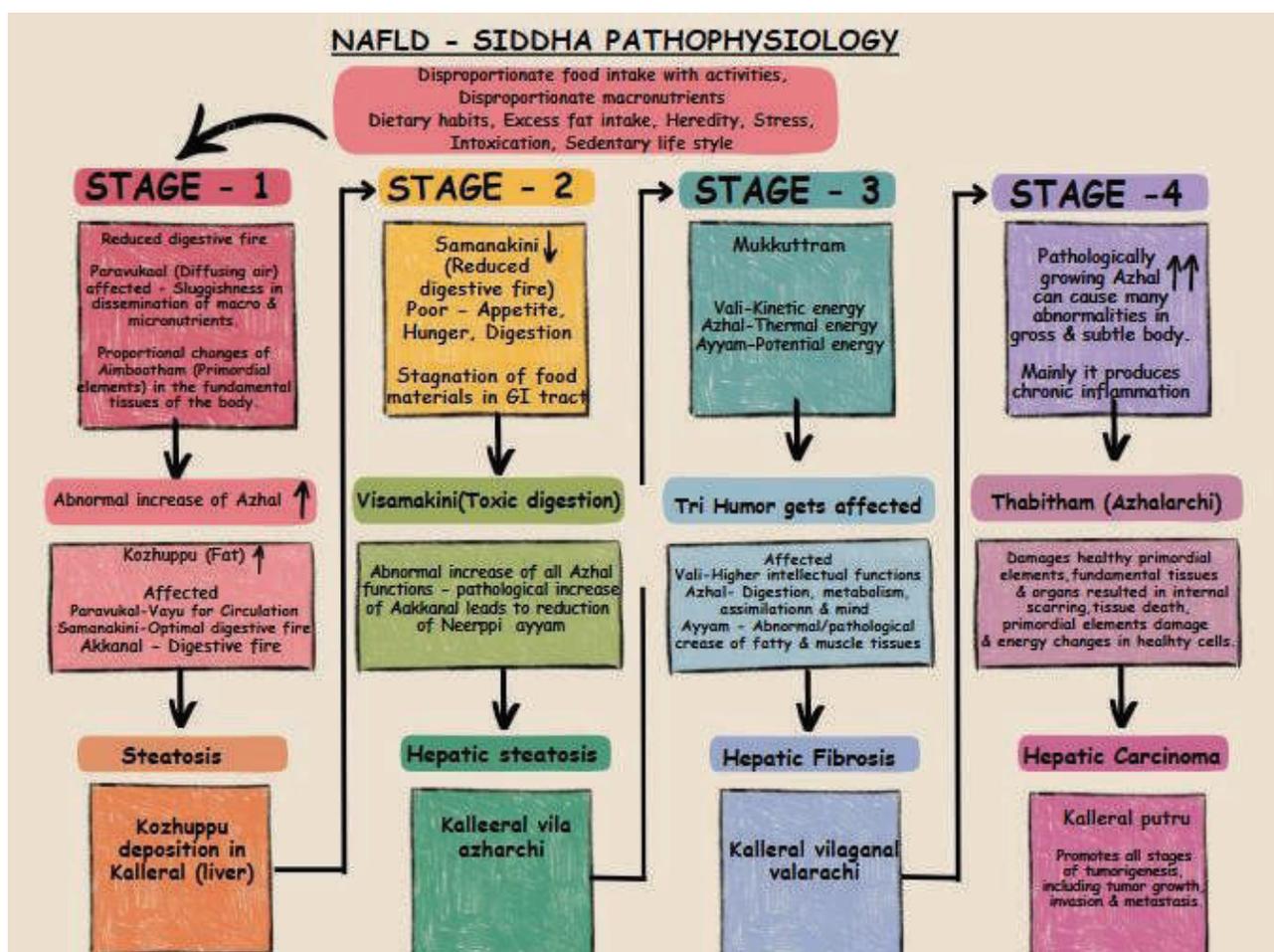


Fig 1. NAFLD- Siddha Pathophysiology

CLINICAL PRESENTATION AND EXAMINATIONS

The majority of patients with NAFLD are asymptomatic and do not experience any specific symptoms related to the disease. Few individuals complain of symptoms like fatigue, nausea, vomiting, pruritis, ascites, memory impairment, right upper quadrant discomfort, hepatomegaly,

acanthosis nigricans and lipomatosis.²¹ A certain proportion of patients with NASH-cirrhosis may present with signs of end stage liver disease such as spider angiomas, erythema, caput medusae, gynecomastia, petechiae, dupuytren scontracture. On clinical examination, mild to moderate hepatomegaly may be the most common finding. Patients of NAFLD may often present with obesity and hypertension.²² The National cholesterol Education Program – Adult treatment Panel III (NCEP ATP III) criteria modified for Indians has been developed for determining certain risk factors associated with metabolic syndrome.²³ Patients with such risk factors must be screened as it has been observed that Metabolic syndrome is closely associated with NAFLD.²⁴

Table 1

Abdominal obesity	Waist circumference > 90 cms in males and > 80 cms in female
Impaired fasting glucose	Fasting glucose ≥ 110 mg/dl or on pharmacological treatment
Hypertension	Blood pressure ≥ 130/85 mm of Hg or on antihypertensives
Hypertriglyceridemia	Serum triglycerides ≥ 150 mg/dl or on pharmacological treatment that lowers triglycerides
Decreased HDL	Serum HDL < 40 mg/dl in males and < 50 mg/dl in females

DIFFERENTIAL DIAGNOSIS

As the diagnosis of NAFLD is mainly driven by exclusion of the alternate causes of hepatic steatosis. The alternate causes of hepatic steatosis are as follows:

Table 2

Macro-vesicular steatosis	Micro-vesicular steatosis
Excessive alcohol consumption	Reye's syndrome
Hepatitis C (genotype 3)	Medications like valproate and antiretroviral drugs
Wilson's disease	Acute fatty liver of pregnancy
Lipodystrophy	HELLP syndrome
Starvation	Inborn errors of metabolism
Parenteral nutrition	
Abetalipoproteinemia	
Medications like methotrexate and steroids	
Kwashiorkor	
Anorexia nervosa	
Personality Disorders	

SUPPORTIVE INVESTIGATION

With a paucity of specific symptoms for the diagnosis of NAFLD, imaging and other investigations remain the main diagnostic indicator for the condition. Though hepatic histology is considered as the gold standard for the diagnosis of the condition, the complexity, complications associated with the procedure, and lack of preference among the patients

prevents this method of investigation as a popular modality for diagnosis.⁵ Non- invasive tests remain the investigation of choice among the physicians and patients alike.

Table 3

Investigations	Findings	
Essential Investigations		
Liver function tests	Mild to moderately elevated serum transaminases (AST and ALT), ALT elevation more common than AST, raised alkaline phosphatase levels, albumin and bilirubin levels raised. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are often somewhat raised, ranging from two to five times the upper limit of normal, with ALT being larger in a 2:1 ratio to AST. Since the AST and ALT in alcoholic hepatitis typically differ by a ratio of more than 2:1, this pattern of elevated serum aminotransferase aids in the differentiation of NAFLD from alcoholic hepatitis.	
Other blood investigations	Serum ferritin and transferrin saturation levels, abnormal clotting time, HbA1c, Fasting Blood glucose, Celiac disease screening test, Lipid Profile, HBsAg, Hepatitis C	
Ultrasonography	The grading of hepatic steatosis in ultrasonography are done as per the following criteria:	
	Grade of fatty liver	USG findings
	Grade 1 (Mild)	Increased echogenicity of the liver in comparison to spleen and right kidney
	Grade 2 (Moderate)	Blurring of intravascular structures in addition to Grade 1 findings
	Grade 3 (Severe)	Deep attenuation of ultrasound signal; diaphragm cannot be readily discerned from posterior surface of live in addition to Grade1/2 findings
Advanced Investigations		
Non contrast CT scan	Hepatic steatosis can be inferred by comparing the attenuation of liver in comparison to the spleen. Liver attenuation index (LAI) < - 10 HU is suggestive of moderate to severe macrovesicular steatosis, while LAI > + 5 HU suggests absence of significant steatosis ²⁷	
Magnetic resonance – proton density fat fraction (MR-PDFF)	Higher sensitivity compared to all imaging procedures but not recommended for routine detection of hepatic steatosis.	

Assessment of hepatic fibrosis

Hepatic fibrosis is the most important parameter for the prognosis, treatment, and outcome in patients with NAFLD. Non-invasive scoring methods of assessing hepatic inflammation and fibrosis are performed using certain scores by combining results of elastography and blood parameters.

Table 4

Name of score	Measuring components	Utility
FAST score ²⁸	Median liver stiffness by TE, CAP and blood AST	Hepatic inflammation. FAST score varied on a scale from 0 to 1, with the patients being classified as having low (<0.35), intermediate (0.35–0.67), or high (>0.67) probability of having SH with significant inflammatory activity and fibrosis.
AST to Platelet Ratio Index (APRI) score ²⁹	AST and platelet levels	Hepatic fibrosis.
Fibrosis-4 score (Fib-4) ³⁰	AST, ALT, age, and platelets	Hepatic fibrosis
NAFLD fibrosis scores (NFS) ^{31,32}	BMI, Age, AST/ALT ratio, Albumin, and presence of insulin resistance and diabetes	Hepatic fibrosis
BARD score ³²	BMI, Age, AST/ALT ratio, and presence of diabetes	Hepatic fibrosis
Magnetic resonance elastography (MRE) and Fibrosis-4 score (MEFIB) ³³	Magnetic resonance elastography and Fibrosis-4 scores	NASH

A score of greater than 1 with APRI less than 0.676 with NFS and greater than 2.67 with Fib-4 predicts the presence of advanced fibrosis, while NFS less than -1.455 and Fib-4 score less than 1.3 suggests a low risk for advanced fibrosis.²⁶

DIAGNOSTIC CRITERIA

Most of the diagnosis of NAFLD takes place incidentally on ultrasonographic (USG) examination of the abdomen done for dyspepsia or asymptomatic rise of blood transaminases. There are also recommendations for screening of NAFLD in patients with type 2 diabetes mellitus, obesity and metabolic syndrome^{5,20,25}. The diagnosis of NAFLD includes documentation of hepatic steatosis of variable severity on imaging and exclusion of secondary causes of hepatic steatosis. Investigations for alcoholic hepatic steatosis especially with an history of significant alcohol intake, hepatitis B and C, and autoimmune hepatitis must be done to rule out alternate causes of hepatic steatosis.

Siddha Diagnostic Criteria

Eṇvakai tērvu (Eight types of diagnosis) ²⁶

- Nāṭi - Aḷal vali/ vali aiyam nāṭi
- Sparicam - Warmth
- Nā - Yellowish Coated
- Niram - Pallor yellow
- Moḷi - Low pitched
- Viḷi - Yellowish discolouration
- Malam - Yellow/ Pale, constipation altered with diarrhoea
- Mūttiram
 - a) Nīrkuṛi (Uro-macroscopy) - Yellow / red, decreased output
 - A. b) Neykūri - Oil may spread in the form of ring / pearl.
 - B. (Oleo Uro-macroscopy)

PRINCIPLES OF MANAGEMENT

The principles of management include assessment of signs and symptoms before initiating treatment and the need for management through conventional treatment for associated co-morbidities. If the patient is already under standard care, the physician may advice to continue the same along with add-on homoeopathy and can be assessed for the same in the follow ups for tapering or discontinue the treatment in consultation with the conventional physician.

Red Flag Signs

- NASH-associated cirrhosis
- End-stage liver disease
- Hepatocellular carcinoma (HCC)
- Uncontrolled co-morbidities
- LSM \geq 20
- Platelet count $<$ 150 \times 10⁶ / L
- Portal hypertension
- Hepatic encephalopathy
- Weight loss or anorexia

The major challenge in the management of the condition is that there are no specific symptoms for the disease and the majority of the patients are asymptomatic. Such circumstances become difficult to the physicians to encourage the patients to undergo treatment or lifestyle modification. The first step for initiation of treatment includes appropriate counselling of the patients and educating them about the disease condition. The patient must be educated that NAFLD is not a mere gastrointestinal disorder, but a metabolic disorder and dietary modification alone may not be helpful for resolving the condition. Adequately guided individualized therapy and overall lifestyle modification is essential for the treatment of the condition.

A) Prevention management

Lifestyle interventions including dietary calorie management and exercise constitute the main pillars of NAFLD management. Studies have demonstrated that there is a dose-response relationship between the magnitude of weight loss and the degree of histological improvement of NAFLD. 3-5%, $\geq 7\%$, and $\geq 10\%$ of weight loss has been associated with regression in steatosis, steatohepatitis, and fibrosis respectively.³⁴ Daily caloric restriction by 30% with cutting down of both carbohydrates and fat in the staple diet. Intermittent fasting (e.g. alternate day fasting, 5:2 fasting with 2 days of severely reduced caloric intake and 5 days of normal consumption) may be a promising approach but sufficient evidence is still not available to routinely recommend such practice.³⁵ Exercise shall consist of moderate-intensity aerobic exercises such as brisk walking, jogging, running, swimming, etc. supplemented by resistance exercises.^{36,37}

Levels of Prevention for NAFLD

- Primordial Prevention: Prevent risk factors like obesity, insulin resistance, and poor diet.
- Primary Prevention: Prevent NAFLD in high-risk groups (obese, prediabetic individuals).
- Secondary Prevention: Detect NAFLD early and prevent progression to NASH (Non-Alcoholic Steatohepatitis) or cirrhosis.
- Tertiary Prevention: Manage complications like fibrosis, cirrhosis, or hepatocellular carcinoma.

Siddha System of Medicine emphasis adhering to *Tēraiyaṛ piṇi aṇukā viti* for prevention of disease and lead to healthy life.

Table 5

Dietary Habits (<i>Uṇavu Muṛaikaḷ</i>)	
Do's - <i>Pattiyam</i>	Don'ts - <i>Apattiyam</i>
<ul style="list-style-type: none"> • Drink warm water • Add <i>Triphoda sama porutgal</i> inclusive of turmeric, pepper, cumin seeds, asafoetida, dry ginger, cardamom, fenugreek and garlic in food preparations • Consume low fat, low-calorie & high fiber diet, fresh vegetables, whole grains, legumes, greens & citrus fruits • Easily digestible foods should be taken such as rice gruel/double boiled rice gruel, buttermilk, tender coconut water • Include moderate intake of nuts • Include lean proteins and low fat dairy in diet 	<ul style="list-style-type: none"> • Always avoid fatty meals especially at night • Avoid highly processed refined carbohydrate diet and advised to take complex carbohydrates • Avoid foods containing added sugars, trans fat and refined grains • Avoid deep fried food and junk foods • Avoid night and late-night snacking
Lifestyle Practices (<i>Vāḷviyaḷ Muṛaikaḷ</i>)	
Do's	Don'ts
<ul style="list-style-type: none"> • Practice at least 45 minutes of moderate physical activity (like walking) 5 days a week • Consume food to the level of hunger • Consume food only half of stomach, liquid quarter of stomach and always leave quarter stomach empty • Prefer left lateral position for sleeping • Better balance of mood and sleep 	<ul style="list-style-type: none"> • Avoid daytime sleep or oversleeping • Avoid sedentary life style • Avoid stress • Avoid nap or sleep after food • Avoid salty foods • Avoid alcohol and smoking • Don't self-medicate

<ul style="list-style-type: none"> • Undergo therapeutic emesis once in six months • Undergo therapeutic purgation once in four months • Practice siddha <i>kāyakaṛpam</i> – take ginger, dried ginger and chebulic myrobalan in the morning, afternoon and evening respectively • Routine liver screening test 	
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B) Interventions

At level 1- Solo Physician Clinic / Health Clinic / PHC (Optimal Standard of treatment where technology and resources are limited).

Clinical diagnosis

The diagnosis of NAFLD shall be done in level 1 especially in cases who have incidental discovery of fatty liver disease. Depending on the infrastructural setup of the clinic/health center an ultrasonography examination may be conducted. To confirm the diagnosis the alternate causes of hepatic steatosis must be ruled out by clinical history and available investigations.

Investigations

1. Liver function tests (Bilirubin, transaminases, total protein), Lipid profile (Total cholesterol, HDL, LDL, VLDL, Triglycerides), Fasting and post-prandial blood sugar, Urea, Creatinine, Complete haemogram, HBsAg, Celiac disease screening.
2. Assessment scores like APRI, Fib-4, and BARD.
3. Ultrasonography of upper abdomen (if available)

Management

Patients may seek *Siddha* management at different stages of NAFLD and the line of treatment may vary accordingly.

The first line of treatment is to normalize the altered or deranged humours and revitalization of seven fundamental tissues through detoxification methods followed by internal medications.

The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

a. *Vāmaṇa maruttuvam* (Therapeutic emesis)

- *Marukarai Kudineer* - 15- 30 ml, OD, in the early morning on an empty stomach
- *Marukarai nei* - 10-15 ml, OD, at the early morning in an empty stomach (Either one medicine can be administered for inducing emesis)

b. *Kalical maruttuvam* (Therapeutic purgation)

- *Kakkarattan ver Kudineer*¹ - 30ml
- *Kattamanakku ver Kudineer*¹ - 30ml

To eliminate the vitiated humour, either one *Kudineer* can be advised to induce purgation. After *purgation*, the patient will experience lightness in the body and improvement in appetite.

Rules to be followed during purgation:

- It is advised to take purgative medicine early morning at 5-6 am on an empty stomach.
- If bouts of purgation do not commence, the patient may be advised to drink hot water.

- Some patients may have nausea, profuse sweating, and vomiting symptoms during this treatment.
- After the average number (5-6 times) of bowel evacuations, watery diarrhoea commences. This indicates that the purgation therapy has been completed. Then, the patient is advised to take buttermilk/ lemon juice/tea decoction/ fried cumin seeds decoction.
- After purgation, the patient may have symptoms like tiredness, slimness, and lightness of the body which is a good sign.

Dietary regimen during purgation:

- Easily digestible foods should be taken
- Luke warm water
- Butter milk
- Rice porridge
- Double boiled porridge
- Tender coconut water
- Non spicy Moong dal curry

Precautions:

- Avoid sleeping during daytime of purgation therapy
- Should not take heavy meals before or during the procedure
- Avoid high oil/ spicy masala diet.

Treatment:

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

c. Single herbs:

Table 6

Sl. No	Herb	Dosage form	Dose	Time	Frequency and Duration	Adjuvants /Anupanam
1.	<i>Avuri ver Kudineer</i> / <i>Indigofera tinctoria</i> 39,40	Decoction	30 ml	BD after food	48 days	--
2.	<i>Sombu vitai / Pimpinella anisum</i> 39,41	Seed Powder	½2- g	BD after food	48 days	Plain or with sugar
3.	<i>Adathodai/ Justicia beddomei</i> 39,42,	Leaf Juice	10 to 20 drops	BD after food	48 days	Honey
4.	<i>Amanakku and Keezhanelli / Ricinus communis and Phyllanthus amarus</i> 39,43,44	Leaf Paste	5–10 g	Morning empty stomach	3 days	On 4 th day <i>Sivathai potṭi /Ipomea turpethum</i> – for Purgation ⁴
5.	<i>Karisalai / Eclipta alba</i> 39,45	Leaf powder	2 g	BD after food	48 days	--

Table 7: Compound Formulations:

Sl. No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Aṅupāṇam
Chooranam / Medicinal Powder						
1.	Mandura Chooranam -1 ^{46,47}	Medicinal powder	5 g	BD	48 days	Butter milk
2.	Mandura Chooranam-2 ^{46,48}	Medicinal powder	2 g	BD	48 days	Hot water
3.	Paavettai Chooranam ^{39,49}	Medicinal powder	40-80 ml	BD	48 days	Rice Water
Maathirai / Tablet						
1.	Chithiramoola Maathirai ^{46,50}	Tablet	1 - 2	BD	48 days	Water

Targeting a weight loss of 7-10% is recommended in overweight and obese patients with NAFLD.⁵

Table 8: Recommended diet and lifestyle modifications

Tender vegetables ⁵¹	<ul style="list-style-type: none"> • Drumstick/ <i>Muruṅkai</i> (<i>Moringa oleifera</i>) • Broad beans/ <i>Āvarai</i> (<i>Lablab purpureus</i>) • Brinjal/ <i>Kattiri</i> (<i>Solanum melongena</i>) • Fig/ <i>Atti</i> (<i>Ficus racemosa</i>) • Lady finger/ <i>Veṅṅai</i> (<i>Hibiscus esculentus</i>) • Unripe Papaya/ <i>Pappāli piṅcu</i> (<i>Carica papaya</i>) • Cucumber/ <i>Veḷḷari</i> (<i>Cucumis sativus</i>) • Bottle guard/ <i>Curaikkāy</i> (<i>Lagenaria siceraria</i>) • Ivy gourd/ <i>Kōvai</i> /(<i>Coccinia grandis</i>)
Any of the vegetables can be prepared as curry, Kūttu, Poriyal etc.	
Green leafy vegetables	<ul style="list-style-type: none"> • Black night shade/ <i>Maṅattakkāḷi</i> /(<i>Solanum nigrum</i>) • Dwarf copper leaf spinach/ <i>Poṅṅāṅkaṇi</i> (<i>Alternanthera sessilis</i>) • False daisy/ <i>Karicālai</i> / (<i>Eclipta prostrata</i>) • Creeping woodsorrel / <i>Puḷiyarai</i> /(<i>Oxalis corniculata</i>) • Kidney leaved moon-seed/ <i>Poṅmucuṭṭai</i> /(<i>Rivea ornata</i>) • Amaranthus green/ <i>Kīrai taṅṅu</i> /(<i>Amaranthus gangeticus</i>) • Purslane seeds/ <i>Paruppukīrai</i> /(<i>Portulaca oleracea</i>) • Curry leaves / <i>Karuvēppilai</i> /(<i>Murraya koenigii</i>)-
Soup, saute and curry-like preparations can be made from these vegetables for consumption	
Pulses	<ul style="list-style-type: none"> • Black Gram / <i>Uḷuntu</i> (<i>Vigna munga</i>) • Green gram / <i>Pāci payaru</i> (<i>Vigna radiata</i>)
Pulses can be used to make Rice, Porridge, Salad, Chutney, Pongal, Idly, Dosa and Curry	
Fruits	<ul style="list-style-type: none"> • Indian gooseberry/ <i>Nelli</i> (<i>Phyllanthus emblicus</i>) • Lemon/ <i>Eḷumiccai</i> (<i>Citrus lemon</i>) • Pomegranate/ <i>Mātulai</i> (<i>Punica granatum</i>) • Guava/ <i>Koyyā</i> (<i>Psidium guava</i>)
Fruits can be taken as salad, juice (without sugar), or cut fruit	

Exercise recommendations⁵

- Moderate intensity aerobic or resistance exercises for 30-45 min/day at least 5 days in a week in all patients of NAFLD irrespective of body weight.

- Moderate intensity aerobic exercise includes brisk walking, jogging, running, swimming, cycling, etc.
- Resistance exercises may supplement aerobic exercises and may be particularly useful for patients with who cannot partake in aerobic exercises like patients with arthritis, morbid obesity, poor cardiorespiratory fitness, etc.
- Asanas as in yoga that involve physical exertion and the maintenance of certain body postures like isometric resistance exercises may be beneficial.
- **Yoga:** Various Yoga practices are helpful for the management of NAFLD. These include Pranayama like Bhastrika, Kapalabhati and Anuloma-Viloma; various relaxation techniques viz. twisting movement of the body; yogasanas like Vajrasana, Trikonasana, Dhanurasana, Naukasana, Ardha Matsyendrasana, Pavana Muktasana and Surya namaskara.

Restricted diet and lifestyle⁵

- In obese and overweight individuals, the dietary calorie intake should be restricted by 30% or 500-1000 kcal by cutting down carbohydrates and fats in staple diet. In lean individuals, energy intake should be balanced with energy expenditure.
- Total fat consumption should not exceed 30% of total energy intake with saturated fats being <10% and trans-fat <1% of total energy intake.
- Free sugar intake must be limited to < 10% of total energy intake and further 5% reduction may have additional benefits. Fructose and sweetened beverages should be curtailed.
- Protein restriction is not required in patients with NAFLD, although meat proteins may be replaced with plant, dairy and fish proteins.
- Evidence shows benefit of > 2 cups of caffeinated coffee per day in NAFLD. But the standard habit of sweetening and use of milk/cream should be avoided.

Follow-up: (at an interval of 14 days or as required)

Reviews should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.
- Management of NAFLD in terms of diet, exercise, and other interventions.
- Discussing the person's knowledge of the condition, any concerns they have, their personal preferences, and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.
- Self-management support.
- Monitoring the long-term course of the condition with periodic review.

Referral criteria

- Non-response to treatment
- Progression of the disease to NASH, NASH- associated Cirrhosis, or NASH associated Hepatocellular Carcinoma

- Any other hepatic or extra-hepatic complications such as Gallstone disease commonly seen in older age and higher grade of NAFLD.
- Evidence of an increase in severity/complications
- Co-morbidities, such as cardiac disease.
- Substantial impact on their quality of life and activities of daily living
- Diagnostic uncertainty

At level 2- (CHC/Small hospitals (10-20 bedded hospitals with basic facilities such as routine investigations and imaging facilities)

Clinical diagnosis

Same as Level 1. Any fresh case, cases on incidental discovery, or referred case from Level 1 shall be evaluated thoroughly for confirmation of diagnosis and complications.

Investigations:

Same as Level 1. Ultrasonography examination must be conducted compulsorily with proper grading of the hepatic steatosis.

Management:

Same as Level 1. For the patients referred from Level-1, treatment given in Level-1 may be continued if appropriate for the presenting condition or the case may be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the medications mentioned for Level-1 may also be considered, however, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient. Complications of the disease is an important clinical presentation at this stage of care especially the early signs and symptoms of such complications. Conditions progressing to steatohepatitis and fibrosis may be treated according to the presenting complications. Accessory management of co-morbidities like diabetes mellitus, dyslipidemia, and hypertension must be accordingly managed.

d. *Ennai mulukku* (Therapeutic oil bath)

Ennai mulukku is a preparatory procedure in which medicated oil massage with a bath of lukewarm water. It will strengthen the five sensory organs. According to disease severity oil bath can be advised.

- *Keezhanelli Thylam* - Quantity Sufficient (For ext. use only)

Rules to be followed during *Ennai mulukku*

Apply oil before 7 am. Instil 2 drops of medicated oil in each nostril, ear, and eye. Spread the oil from head to foot and give a gentle massage. After application, leave it for 15 to 45 minutes and bathe with lukewarm water

Take tender vegetables and easily digestible food. Avoid daytime sleep, intercourse, and exposure to sunlight and cold items on the day of the oil bath

e. **Kaḷiccal maruttuvam (Therapeutic purgation):**

Table 9

Sl.No.	Drugs	Dose form	Dose	Time	Adjuvant/ Aṇupāṇam
1.	Agathiyar Kuzhambu ⁵¹	Kuzhambu (Medicated viscous Mixture)	100-200 mg	At early morning on an empty stomach.	Lemon (<i>Citrus limon</i>) juice / white onion (<i>Allium cepa</i>) juice
2.	Meganatha Kuligai ⁵¹	Tablet	1-2 pills	-do-	Ginger juice (<i>Zingiber officinalis</i>)
3.	Sivathaiver Kudineer ⁵¹	Decoction	15-30 ml	at early morning on an empty stomach	--

Along with Siddha formulations mentioned in Level 1, the following formulations are also recommended:

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

a. Single herbs:

Table 10

Sl. No.	Herb	Dosage form	Dose	Time	Frequency and Duration	Adjuvant / Aṇupāṇam
Karkam - Medicinal paste						
1.	Adathodai Karkam (<i>Justicia beddomei</i>) ⁵¹	Medicinal paste	10-20 drops	BD after food.	30 days	Honey
2.	Serangkottai (<i>Cassia fistula Linn</i>) ⁵¹	Medicinal paste	5- 10 g	OD	if there is chronic constipation	Lukewarm water
3.	Sivanar Vembu Karkam (<i>Indigofera aspalathoides</i>) ⁵¹	Medicinal paste	5- 10 g	OD	30 days	Lukewarm water
4.	Muthirukkanevi Karkam (<i>Elytraria acaulis</i>) ⁵¹	Medicinal paste	5- 10 g	OD	30 days	Lukewarm water
5.	Kadukkai Karkam (<i>Terminalia chebula</i>) ⁵¹	Medicinal paste	5 – 10 g	OD before food	30 days	Lukewarm water
6.	Amukkura Ilai Karkam (<i>Withaniya Somnifera</i>) ⁵¹	Medicinal paste	5-10 g	OD before food	30 days	--
Any one of the herb may be used.						

b. Compound Formulations:

Table 11

Sl. No	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants / <i>Aṅupāṇam</i>
Kudineer - Decoction						
1.	<i>Sarakondrai Kudineer</i> ⁵¹	Decoction	30 – 60 ml	early morning on an empty stomach	2 – 3 days	--
2.	<i>Pidangunaari Kudineer</i> ⁵¹	Decoction	30 – 60 ml	BD after food	48 days	--
3.	<i>Mookirattai ver Kudineer</i> ⁵¹	Decoction	40-80 ml	BD after food	48 days	--
4.	<i>Sodakku takkali Kudineer</i> ⁵¹	Decoction	30-60 ml	BD after food	48 days	--
5.	<i>Nerunjil Kudineer</i> ⁵¹	Decoction	60-80 ml	BD after food	48 days	--
6.	<i>Kadukkai Kudineer</i> ⁵¹	Decoction	60 -80 ml	BD after food	48 days	--
7.	<i>Paavettai ver Kudineer</i> ⁵¹	Decoction	40-80 ml	BD after food	48 days	--
8.	<i>Mandurathi Adai Kudineer</i> ⁵¹	Decoction	60-80 ml	BD after food	48 days	--
Any one of the Kudineer can be prescribed						
Maathirai –Tablet						
9.	<i>Keezhanelli Maathirai</i> ⁵¹	Tablet	1 - 2	BD/TDS, after food	48 days	Buttermilk
10.	<i>Santha chandrothaya Maathirai</i> ⁵¹	Tablet	1 -2	BD after food	48 days	Honey
11.	<i>Bavana Kadukka</i> ⁵¹	Tablet	1 -2	BD, after food	48 days	(to be chewed)
12.	<i>Amirthathi Maathirai</i> ⁵¹	Tablet	1 -2	BD after food	If there is Jaundice	Honey
13.	<i>Nannari Maathirai</i> ⁵¹	Tablet	1 -2	BD after food	45 days	Honey
14.	<i>Maha elathi Maathirai</i> ⁵¹	Tablet	1 -2	BD after food	45 days	Honey/ milk
15.	<i>Maha vasantha kusumakara Maathirai</i> ⁵¹	Tablet	1 -2	BD after food	21 days	Honey
Any one of the tablets can be advised if jaundice is present						
Chooranam – Medicinal Powder						
16.	<i>Nilavgaai Chooranam</i> ⁵¹	Medicinal powder	1 - 2 g	BD/TDS after food	if constipation is present	Lukewarm water

Sl. No	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants /Aṇupāṇam
17.	<i>Thiriphala Chooranam</i> ⁵¹	Medicinal powder	1 - 2 g	BD/TDS, after food	90 days	Water
18.	<i>Pancha lavana Chooranam</i> ⁵¹	Medicinal powder	1 - 2 g	BD/TDS after food	to neutralize electrolyte imbalance	Lukewarm water
19.	<i>Elathi Chooranam</i> ⁵¹	Medicinal powder	1 - 2 g	BD/TDS after food	if there is gastric irritation	Lukewarm water
20.	<i>Dhratchathi Chooranam</i> ⁵¹	Medicinal powder	1 - 2 g	BD/TDS after food	48 days	Lukewarm water
21.	<i>Kadukkai Chooranam</i> ⁵¹	Medicinal powder	1-2 g	BD after food	48 days	Lukewarm water
Manapagu - Syrup						
22.	<i>Turunji Manapagu</i> ⁵¹	Syrup	15 – 30 ml	BD after food	if vomiting is present	Water
23.	<i>Nannari Manapagu</i> ⁵¹	Syrup	15 -30 ml	BD after food	if there is fatigue	Water
24.	<i>Mathulai Manapagu</i> ⁵¹	Syrup	15 - 30 ml	BD after food	if there is nausea and vomiting	Water
25.	<i>Naarathai Manapagu</i> ⁵¹	Syrup	15 - 30 ml	BD after food	if there is nausea and vomiting	Water
Karpam - Rejuvenating drug						
26.	<i>Ayabringaraja Karpam</i> ⁵¹	Rejuvenating drug	100-200 mg	BD, after food	40 days	Honey/ tender coconut water/ palm jaggery
27.	<i>Ayasambeera Karpam</i> ⁵¹	Rejuvenating drug	one piece (1/4 of lemon)	BD after food	48 days	--
28.	<i>Ponnankanni Karpam</i> ⁵¹	Rejuvenating drug	2 - 3g	BD, after food	48 days	

a. **Puramaruttuvam** (External Medicines)/ other procedures

Patru (Semi Solid Poultice): *Carakkonrai puli Patru* - Quantity sufficient

b. **Varma maruttuvam**

- *Aṭappā kālam*
- *Kāraraḷ varmam*

2. **Recommended diet and lifestyle:** Same as Level 1

3. **Restricted diet and lifestyle:** Same as Level 1

4. **Follow-up:** At an interval of 15 days or as per the need

5. **Referral criteria:**

Same as level 1 and

- Failure of acute exacerbation to respond to initial medical management.

At level 3-

Ayush hospitals attached with teaching institution, District Level/Integrated/State Ayush Hospitals, Tertiary care allopathic hospitals having Ayush facilities, multiple departments/facilities for diagnosis and interventions.

Clinical diagnosis

Same as Level 2. The diagnosis must be confirmed using advanced biochemistry, serology and imaging studies.

Investigations: Same as Level 1

Supportive investigations:

1. Non-contrast CT scan
2. MRI based Elastography
3. Blood levels for carbohydrate-deficient transferrin (CDT), Gamma glutamyl transferase for determination of chronic alcoholism.
4. Hepatitis C antigen
5. Serum copper levels and ceruloplasmin to rule out Wilson's disease (only if needed)
6. Metabolic profile for ruling out lipodystrophy, and starvation
7. Genetic testing for apo B and MTP to rule out abetalipoproteinemia (only if needed)

Management

Same as Levels 1& 2. For the patients referred from Level-1 or 2, treatment given in Level-1 &/or 2 may be continued if appropriate for the presenting condition or the case may be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient. Along with Siddha formulations mentioned in Level 1 and Level 2 Management, the following Siddha formulations are also recommended:

Day 1 - Vāmaṇa maruttuvam (Therapeutic emesis):⁵¹

- *Marukkarai vidhai Chooranam* (2-5 g) with unripe papaya (*Carica papaya*) juice (5-10 ml) in the early morning on an empty stomach.

Day 2 – Rest

Day 3 – First Line of Treatment:³⁰

- *Sara kondrai Kudineer* - 30 – 60 ml in the early morning on an empty stomach for 2 – 3 days.
- *Keezhanelli Maathirai* (500 mg) - 1 - 2 tabs with butter milk BD/TDS, after food.
- *Santha chandrodaya Maathirai* (100 mg) - 1 -2 pills with honey, BD after food.

a. Compound Formulations:

(Note: Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 12

Sl. No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants /Anupānam
Kudineer - Decoction						
1.	<i>Mandurathi adai Kudineer</i> ⁵¹	Decoction	60-80 ml	BD after food	48 days	--
Ney- Medicated Ghee						
2.	<i>Vallarai nei</i> ⁵¹	Medicated Ghee	5 – 10 ml	BD after food	45 days	Lukewarm water
Maathirai - Tablet						
3.	<i>Elathi Maathirai</i> ⁵¹	Tablet	1 – 2 tabs	BD after food	45 days	Butter milk
4.	<i>Karisalai Maathirai</i> ⁵¹	Tablet	1 – 2 tabs	BD after food	45 days	Butter milk
5.	<i>Panchadeepakini Maathirai</i> ⁵¹	Tablet	1 – 2 tabs	BD after food	45 days	Butter milk
Kuzhambu- Medicated viscous mixture						
6.	<i>Lavaṇa Kuzhambu</i> ⁵¹	Medicated viscous mixture	100 – 200 mg	BD after food.	Discretion of the Physician.	Palm jaggery
7.	<i>Narathangai Kuzhambu</i> ⁵¹	Medicated viscous mixture	100 – 200 mg	BD after food	if there is vomiting	Palm jaggery
8.	<i>Vilvathi Kuzhambu</i> ⁵¹	Medicated viscous mixture	100 – 200 mg	BD after food	Discretion of the Physician.	Palm jaggery
Chenduram – Red calx						
9.	<i>Vedi Annabehi Chenduram</i> ⁵¹	Red calx	100-200 mg	BD after food	40 days	Honey
Parpam – White calx						
10.	<i>Kariyuppu Parpam</i> ⁵¹	White calx	65-130 mg	BD after food.	40 days	<i>Oma ilagam</i>
11.	<i>Silasathu Parpam</i> ⁵¹	White calx	100 – 200 mg	BD after food.	40 days	Ghee / butter
Karpam - Rejuvenating drug						
12.	<i>Karisalai karpam</i> ⁵¹	Rejuvenating drug	1 – 2 tabs	BD after food	40 days	Lukewarm water

Recommended diet and lifestyle: Same as Levels 1 & 2

1. **Restricted diet and lifestyle:** Same as Levels 1 & 2

2. **Follow-up (at an interval of 15 days or as per the need)**

3. **Referral criteria:**

- Same as mentioned in Level 1 & 2 and any of these
- Hepatic encephalopathy
- Portal hypertension
- Hematemesis or melaena or any condition requiring blood transfusion or critical care management
- Any condition or serious complication beyond the scope of homoeopathic treatment
- Other modalities can be considered depending on the case and to rehabilitate properly.

REFERENCES

1. K.N.Kuppusamy Mudaliar, Siddha Maruthuvam (Pothu), Department of Indian Medicine and Homeopathy publication, 7th Edition 2007 pg no.:339,344
2. Hayward KL, Johnson AL, Horsfall LU, et al Detecting non-alcoholic fatty liver disease and risk factors in health databases: accuracy and limitations of the ICD-10- AMBMJ Open Gastroenterology 2021;8:e000572. doi: 10.1136/bmjgast-2020- 000572.
3. Liu SYW, Wong VWS, Wong SKH, Wong GLH, Lai CMS, Lam CCH, et al. A prospective 5-year study on the use of transient elastography to monitor the improvement of non-alcoholic fatty liver disease following bariatric surgery. *Sci Rep* 2021;11(1):5416
4. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *J Hepatol* [Internet] 2020 [cited 2024 Aug 29];73(1):202–9. Available from: <http://www.journal-of-hepatology.eu/article/S0168827820302014/fulltext>.
5. Duseja A, Singh SP, De A, Madan K, Rao PN, Shukla A, et al. Indian National Association for Study of the Liver (INASL) Guidance Paper on Nomenclature, Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease (NAFLD). *J Clin Exp Hepatol* 2023.
6. Duseja A, Singh SP, Mehta M, Shalimar, Venkataraman J, Mehta V, et al. Clinicopathological Profile and Outcome of a Large Cohort of Patients with Nonalcoholic Fatty Liver Disease from South Asia: Interim Results of the Indian Consortium on Nonalcoholic Fatty Liver Disease. *Metab Syndr Relat Disord* 2022;20(3):166–73.
7. De A, Duseja A. Natural History of Simple Steatosis or Nonalcoholic Fatty Liver. *J Clin Exp Hepatol* [Internet] 2020 [cited 2024 Aug 29];10(3):255–62. Available from: <http://www.jcehepatology.com/article/S0973688319302385/fulltext>
8. Duseja A, Chalasani N. Epidemiology and risk factors of nonalcoholic fatty liver disease (NAFLD). *Hepatol Int* [Internet] 2013 [cited 2021 Nov 24];7 Suppl 2:S755– 64. Available from: <https://pubmed.ncbi.nlm.nih.gov/26202291/>
9. Duseja Ajay, Singh Shivaram P, Saraswat Vivek A, Acharya Subrat K, Chawla Yogesh K, Chowdhury Subhankar, et al. Non-alcoholic Fatty Liver Disease and Metabolic Syndrome - Position Paper of the Indian National Association for the Study of the Liver, Endocrine Society of India, Indian College of Cardiology and Indian Society of Gastroenterology. *J Clin Exp Hepatol* 2015;5(1):51–68
10. Byrne CD, Targher G. NAFLD as a driver of chronic kidney disease. *J Hepatol* [Internet] 2020 [cited 2024 Aug 29];72(4):785–801. Available from: <http://www.journal-of-hepatology.eu/article/S0168827820300301/fulltext>
11. Oni ET, Agatston AS, Blaha MJ, Fialkow J, Cury R, Sposito A, et al. A systematic review: Burden and severity of subclinical cardiovascular disease among those with nonalcoholic fatty liver; Should we care? *Atherosclerosis* [Internet] 2013 [cited 2024 Aug 29];230(2):258–67. Available from: <http://www.atherosclerosis-journal.com/article/S0021915013004577/fulltext>
12. Targher G, Day CP, Bonora E. Risk of Cardiovascular Disease in Patients with Nonalcoholic Fatty Liver Disease. *New England Journal of Medicine* [Internet] 2010 [cited 2024 Aug 29];363(14):1341–50. Available from: <https://www.nejm.org/doi/abs/10.1056/NEJMra0912063>
13. Guleria A, Duseja A, Kalra N, Das A, Dhiman R, Chawla Y, Bhansali A. Patients with non-alcoholic fatty liver disease (NAFLD) have an increased risk of atherosclerosis and cardiovascular disease. *Tropical Gastroenterology*. 2013 Sep 26;34(2):74-82.
14. De A, Antony J, Bhagat N, Charak S, mehta M, Singh P, et al. Higher prevalence of metabolic bone disease (MBD) but similar fracture risk in non-alcoholic fatty liver disease (NAFLD) compared to chronic viral hepatitis. *J Clin Exp Hepatol* [Internet] 2022 [cited 2024 Aug 29];12:S70–1. Available from: <http://www.jcehepatology.com/article/S0973688322003395/fulltext>

15. Bhatt SP, Guleria R, Vikram NK, Gupta AK. Non-alcoholic fatty liver disease is an independent risk factor for inflammation in obstructive sleep apnea syndrome in obese Asian Indians. *Sleep and Breathing* [Internet] 2019 [cited 2024 Aug 29];23(1):171–8. Available from: <https://link.springer.com/article/10.1007/s11325-018-1678-7>
16. Harsha Varma S, Tirupati S, Pradeep TVS, Sarathi V, Kumar D. Insulin resistance and hyperandrogenemia independently predict nonalcoholic fatty liver disease in women with polycystic ovary syndrome. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews* 2019;13(2):1065–9.
17. Harsha Varma S, Tirupati S, Pradeep TVS, Sarathi V, Kumar D. Insulin resistance and hyperandrogenemia independently predict nonalcoholic fatty liver disease in women with polycystic ovary syndrome. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews* 2019;13(2):1065–9.
18. Chakraborty S, Ganie MA, Masoodi I, Jana M, Shalimar, Gupta N, et al. s as a non- invasive predictor of hepatic steatosis in women with polycystic ovary syndrome. *Indian Journal of Medical Research, Supplement* [Internet] 2020 [cited 2024 Aug 29];151(4):333–41. Available from: https://journals.lww.com/ijmr/fulltext/2020/51040/fibroscan_as_a_non_invasive_predictor_of_hepatic.12.aspx
19. Mantovani A, Petracca G, Beatrice G, Csermely A, Tilg H, Byrne CD, et al. Non- alcoholic fatty liver disease and increased risk of incident extrahepatic cancers: a meta-analysis of observational cohort studies. *Gut* [Internet] 2022 [cited 2024 Aug 29];71(4):778–88. Available from: <https://gut.bmj.com/content/71/4/778>
20. Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology* [Internet] 2018 [cited 2024 Aug 30];67(1):328–57. Available from: https://journals.lww.com/hep/fulltext/2018/01000/the_diagnosis_and_management_of_nonalcoholic_fatty.31.aspx
21. Pouwels S, Sakran N, Graham Y, Leal A, Pintar T, Yang W, et al. Non-alcoholic fatty liver disease (NAFLD): a review of pathophysiology, clinical management and effects of weight loss. *BMC Endocr Disord* [Internet] 2022 [cited 2024 Aug 30];22(1):1–9. Available from: <https://bmcendocrdisord.biomedcentral.com/articles/10.1186/s12902-022-00980-1>
22. Basaranoglu M, Neuschwander-Tetri BA. Nonalcoholic Fatty Liver Disease: Clinical Features and Pathogenesis. *Gastroenterol Hepatol (N Y)* [Internet] 2006 [cited 2024 Aug 30];2(4):282. Available from: </pmc/articles/PMC5335683/>
23. Rezaianzadeh A, Namayandeh SM, Sadr SM. National Cholesterol Education Program Adult Treatment Panel III Versus International Diabetic Federation Definition of Metabolic Syndrome, Which One is Associated with Diabetes Mellitus and Coronary Artery Disease? *Int J Prev Med* [Internet] 2012 [cited 2024 Sep 2];3(8):552. Available from: </pmc/articles/PMC3429802/>
24. Sharma B, John S. Nonalcoholic Steatohepatitis (NASH) [Updated 2023 Apr 7]. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK470243/>
25. Berzigotti A, Tsochatzis E, Boursier J, Castera L, Cazzagon N, Friedrich-Rust M, et al. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis – 2021 update. *J Hepatol* [Internet] 2021 [cited 2024 Aug 30];75(3):659–89. Available from: <http://www.journal-of-hepatology.eu/article/S0168827821003986/fulltext>
26. Dr.M.Shanmugavelu H.I.M., Siddha Maruthuva Noi Nadal, Noi Mudhal Nadal thirattu Part-I Department of Indian Medicine and Homeopathy publication, 3rd Edition 2003 pg no:187-347
27. Limanond P, Raman SS, Lassman C, Sayre J, Ghobrial RM, Busuttil RW, et al. Macrovesicular hepatic steatosis in living related liver donors: correlation between CT and histologic findings. *Radiology* [Internet] 2004 [cited 2024 Aug 30];230(1):276–
80. Available from: <https://pubmed.ncbi.nlm.nih.gov/14695401/>

28. .De A, Keisham A, Mishra S, Mehta M, Verma N, Premkumar M, et al. FibroScan- AST (FAST) Score for Nonalcoholic Steatohepatitis – Validation in an Indian Cohort. *J Clin Exp Hepatol* [Internet] 2022 [cited 2024 Sep 2];12(2):440–7. Available from: <http://www.jcehepatology.com/article/S097368832100150X/fulltext>
29. Loeza-del-Castillo A, Paz-Pineda F, Oviedo-Cárdenas E, Sánchez-Ávila F, Vargas- Vorácková F. AST to platelet ratio index (APRI) for the noninvasive evaluation of liver fibrosis: Original Article. *Ann Hepatol* 2008;7(4):350–7.
30. Xu X lan, Jiang L shun, Wu C si, Pan L ya, Lou Z qi, Peng C ting, et al. The role of fibrosis index FIB-4 in predicting liver fibrosis stage and clinical prognosis: A diagnostic or screening tool? *Journal of the Formosan Medical Association* 2022;121(2):454–66
31. Mathew JF, Panackel C, Jacob M, Ramesh G, John N. A Validation Study of Non- invasive Scoring Systems for Assessing Severity of Hepatic Fibrosis in a Cohort of South Indian Patients With Non-alcoholic Fatty Liver Disease. *J Clin Exp Hepatol* [Internet] 2024 [cited 2024 Sep 2];14(5). Available from: <http://www.jcehepatology.com/article/S0973688324000641/fulltext>
32. Cichoz-Lach H, Celiński K, Prozorow-Król B, Swatek J, Słomka M, Lach T. The BARD score and the NAFLD fibrosis score in the assessment of advanced liver fibrosis in nonalcoholic fatty liver disease. *Med Sci Monit* [Internet] 2012 [cited 2024 Sep 2];18(12):CR735. Available from: [/pmc/articles/PMC3560810/](https://pubmed.ncbi.nlm.nih.gov/23081010/)
33. Jung J, Loomba RR, Imajo K, Madamba E, Gandhi S, Bettencourt R, et al. MRE combined with FIB-4 (MEFIB) index in detection of candidates for pharmacological treatment of NASH-related fibrosis. *Gut* [Internet] 2021 [cited 2024 Sep 2];70(10):1946–53. Available from: <https://gut.bmj.com/content/70/10/1946>
34. Vilar-Gomez E, Martinez-Perez Y, Calzadilla-Bertot L, Torres-Gonzalez A, Gra- Oramas B, Gonzalez-Fabian L, et al. Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. *Gastroenterology* [Internet] 2015 [cited 2024 Sep 2];149(2):367–378.e5. Available from: <http://www.gastrojournal.org/article/S0016508515004965/fulltext>
35. Memel ZN, Wang J, Corey KE. Intermittent Fasting as a Treatment for Nonalcoholic Fatty Liver Disease: What Is the Evidence? *Clin Liver Dis (Hoboken)* [Internet] 2022 [cited 2024 Sep 2];19(3):101–5. Available from: https://journals.lww.com/cld/fulltext/2022/03000/intermittent_fasting_as_a_treatment_for.5.aspx
36. Younossi ZM, Corey KE, Lim JK. AGA Clinical Practice Update on Lifestyle Modification Using Diet and Exercise to Achieve Weight Loss in the Management of Nonalcoholic Fatty Liver Disease: Expert Review. *Gastroenterology* [Internet] 2021 [cited 2024 Sep 2];160(3):912–8. Available from: <http://www.gastrojournal.org/article/S0016508520355384/fulltext>
37. Sung KC, Ryu S, Lee JY, Kim JY, Wild SH, Byrne CD. Effect of exercise on the development of new fatty liver and the resolution of existing fatty liver. *J Hepatol* [Internet] 2016 [cited 2024 Sep 2];65(4):791–7. Available from: <http://www.journal-of-hepatology.eu/article/S0168827816302124/fulltext>
38. Kwak MS, Kim D. Non-alcoholic fatty liver disease and lifestyle modifications, focusing on physical activity. *Korean J Intern Med*. 2018 Jan;33(1):64-74. doi: 10.3904/kjim.2017.343. Epub 2017 Dec 6. PMID: 29202557; PMCID: PMC5768549
39. K.S.Murugesu Mudaliar, Gunapadam (Porut Panpu Nool) First Part Siddha Materia Medica, Department of Indian Medicine and Homeopathy publication, 3rd Reedition 1988 pg no:36,376,47,55,183
40. Chitra M, Muthusudha N, Sasikala R. Bioefficiency of indigogera tinctoria linn. On isoniazid induced hepatotoxicity in albinorats. *Anc Sci Life*. 2003 Oct;23(2):79-89. PMID: 22557116; PMCID: PMC3330958
41. Asadollahpoor A, Abdollahi M, Rahimi R. Pimpinella anisum L. fruit: Chemical composition and effect on rat model of nonalcoholic fatty liver disease. *J Res Med Sci*. 2017 Mar 15;22:37. doi: 10.4103/1735-1995.202147. PMID: 28465696; PMCID: PMC5393100
42. Afzal, Umara & Gulfracz, Muhammad & Hussain, Shahzad & Malik, Farnaz & Maqsood, Sadaf & Syed, Imamshah & Mahmood, Sidra. (2013). Hepatoprotective effects of Justicia adhatoda L. against carbon tetrachloride (CCl₄) induced liver injury in Swiss albino mice. *African journal of pharmacy and pharmacology*. 7. 8-14. 10.5897/AJPP12.501.

43. Evan Sabina Princea, Poorna Parameswarib, Rasool Mahaboob Khanc,* Protective Effect of Ricinus communis Leaves Extract on Carbon Tetrachloride Induced Hepatotoxicity in Albino Rats Iranian Journal of Pharmaceutical Sciences,Autumn 2011: 7(4): 269-278.
44. Al Zarzour RH, Ahmad M, Asmawi MZ, Kaur G, Saeed MAA, Al-Mansoub MA, Saghir SAM, Usman NS, Al-Dulaimi DW, Yam MF. Phyllanthus Niruri Standardized Extract Alleviates the Progression of Non-Alcoholic Fatty Liver Disease and Decreases Atherosclerotic Risk in Sprague-Dawley Rats. Nutrients. 2017 Jul 18;9(7):766. doi: 10.3390/nu9070766. PMID: 28718838; PMCID: PMC5537880.
45. Satheesh Naik K, Gurushanthaiah M, Kavimani M, Prabhu K, Lokanadham S. Hepatoprotective Role of Eclipta alba against High Fatty Diet Treated Experimental Models - A Histopathological Study. Maedica (Bucur). 2018 Sep;13(3):217-222. doi: 10.26574/maedica.2018.13.3.217. PMID: 31490461; PMCID: PMC6290179.
46. K.Vasudeva Sasthri, Dr.S.Venkataraman Sarabendra Vaithya Muraigal Pandu Kamalai Chikichai Muraigal Saraswathi Magal Library, Thanjavur 1992 4th Edition Pg no 40,77,80-82,93-94
47. Pratibha devarshi et al., Effect of mandur bhasma on lipolytic activities of liver, kidney and adipose tissue of albino rat during CCl₄ induced hepatic injury , J. Biosci., Vol. 10, Number 2, June 1986, pp. 227-234.
48. Hanqing Chen Iron metabolism in non-alcoholic fatty liver disease: A promising therapeutic target* Liver Research,Volume 6, Issue 4, December 2022, Pages 203-213
49. Globale, Pharmacie & Singh, N & Saravanan, Nandanam. (2012). The effect of Pavetta indica in CCl₄ induced hepatotoxicity in rats. International Journal of Comprehensive Pharmacy. 6. 1-4.
50. Silva RL, Melo GB, Melo VA, Antonioli AR, Michellone PR, Zucoloto S, Picinato MA, Franco CF, Mota Gde A, Silva Ode C. Effect of the aqueous extract of Sida cordifolia on liver regeneration after partial hepatectomy. Acta Cir Bras. 2006;21 Suppl 1:37-9. doi: 10.1590/s0102-86502006000700009. PMID: 17013511
51. Siddha standard treatment guidelines, National Institute of Siddha, 2019, namastp.ayush.gov.in. Available at: http://namayush.gov.in/sites/all/themes/webcms/images/org_str/SiddhaStandardTreatmentGuidelines.pdf Pg no:75-79.

CHAPTER

5

OBESITY



OBESITY

ICD 10 code: E 66.0-E 66.9²
ICD 11 code: 5B81.0-5B81.Z

அதிதூலம்¹

WHO Code -ISMT-4.4.20

CASE DEFINITION

Obesity is a chronic complex disease defined by excessive fat deposits that can impair health. Obesity in ICD- 10 (and in ICD- 11) is defined as a body mass index (BMI) of 30 kg/m² or higher and BMI between 25 and 30 kg/m² is defined as overweight. The WHO Asia-Pacific region defined BMI ≥ 23kg/m² as overweight and ≥ 25kg/m² as Obesity. Obesity is defined as a body mass index (BMI) equal to or greater than the 95th percentile for age and sex.³

INTRODUCTION

- In 2022, 1 in 8 people in the world were living with obesity. 2.5 billion Adults (18 years and older) were overweight. Of these, 890 million were living with obesity.⁴
- As per National Family Health Survey-5 (NFHS-5), one in every four Indians is now having obesity. There are 135 million obese individuals in India. The prevalence of abdominal obesity in the country was found to be 40% in women and 12% in men.⁵
- In 2022, overweight affected around 37 million children under 5 globally and over 390 million children and adolescents aged 5–19 years were overweight, including 160 million who were living with obesity – 75% of whom live in low- and middle-income countries.⁶
- Obesity and overweight are a major risk factor for non-communicable diseases such as heart disease, stroke, type 2 diabetes, PCOS and certain cancers (endometrial, breast, ovarian, prostate, liver, gallbladder, kidney and colon).⁷ Therefore, Obesity is more effectively defined by assessing its linkage to morbidity and mortality.⁸ The current guidelines deal with management of both overweight and obesity.
- Obesity is synonymously known as *Tūlaṅ nōy*, *Uṭal parumaṅ* in Siddha. According to Siddha system of medicine, Obesity is explained by increased *Aiyam* which influences *Vāli* (*viyāṅṅ maṛṛum camaṅṅ*) *aḷal* (*aṅarpitam*, *catakāpitam*). This alteration influences the seven body constituents.

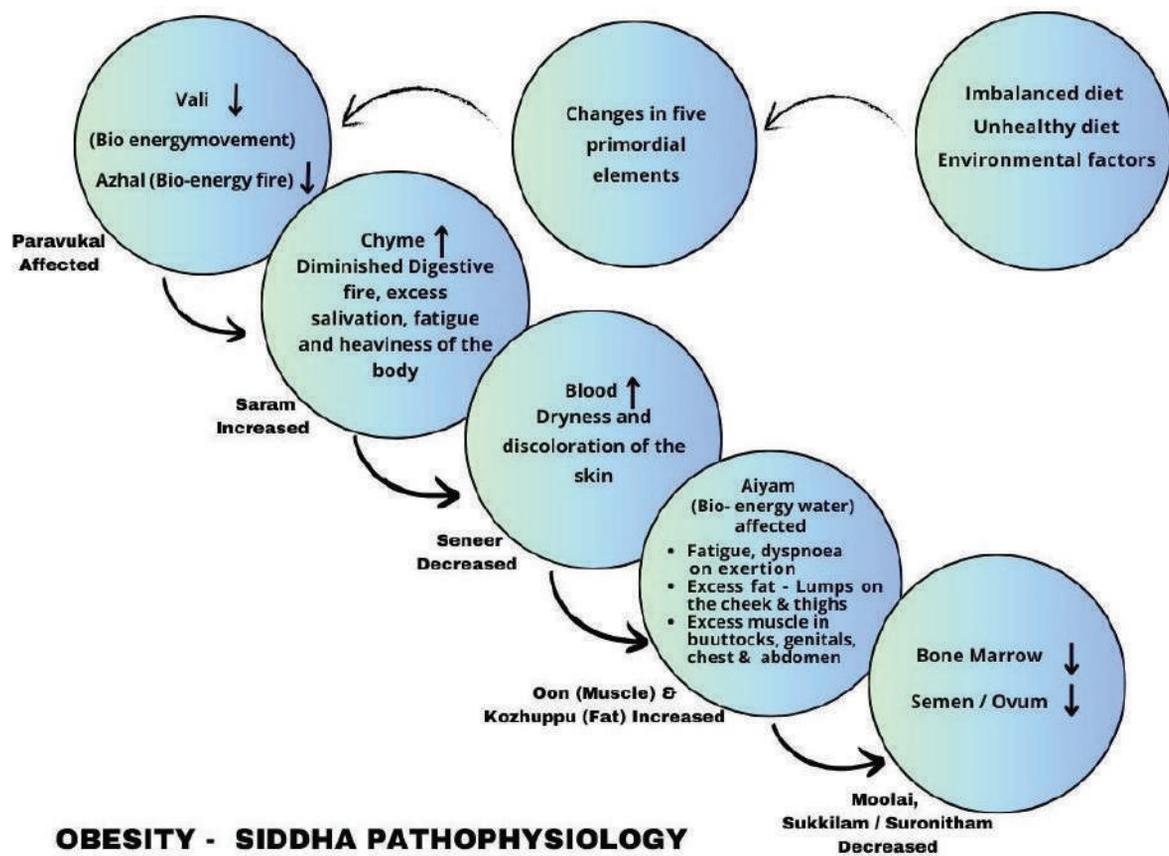


Fig 1: Pathophysiology of Obesity

CLINICAL EXAMINATION⁹

Persons presenting with overweight or obesity must have a detailed history taken, a clinical examination performed and appropriate investigations done (Figure – 2). This is done to identify the environmental, genetic and lifestyle factors responsible for obesity and at the same time identify impact of overweight and obesity on the individual, physically, mentally and socially.

Clinical History

- **Body weight history** in persons who are overweight or present with pre- obesity/obesity may begin with an assessment of body weight increases or reductions over the individual's lifetime (e.g., slow and gradual, rapid and sudden or a combination) and factors influencing weight change. Short sleep duration and poor sleep quality may increase the risk of obesity, making it important to record sleep patterns in patients.¹⁰
- **A detailed family history** is important and often suggests a genetic predisposition.
- **Drug history** should be taken to identify possible drugs that may be contributing to weight gain, such as steroid hormones, antidepressants (tricyclics), antipsychotics (phenothiazines and butyrophenones), anticonvulsants (valproate and carbamazepine), lithium and antihyperglycemics (insulin, sulfonylurea and thiazolidinediones).

- The psychological aspects of eating behaviour should be explored, such as loneliness, boredom or stress. Often obese persons express feelings of low self-esteem and depression. Eating disorders should be particularly sought.
- A thorough review of systems must be taken to assess any co-morbidities that are directly or indirectly related to obesity to identify any evidence of endocrine disease as an occult aetiology of obesity.
- A thorough examination of the patient's present dietary habits is essential. This evaluation can be conducted by a dietitian. It should involve assessing the total daily calorie intake and determining the percentage of calories derived from fat. Individuals with obesity often show abnormal eating patterns. The eating disorders that have been most frequently studied in individuals with obesity are binge eating disorder and bulimia nervosa.
- History pertaining to physical activity Physically active and fit individuals are considerably less likely to be obese than physically inactive and unfit individuals. Therefore, it's essential to gather comprehensive information to understand their current activity level any past injuries or limitations, their exercise preference and Lifestyle Factors.

Clinical and imaging indicators of obesity

Apart from BMI, waist circumference, waist-hip ratio and skin-fold thickness, the variations in lean muscle mass and body fat percentage are also assessed utilizing the body composition analyzer.¹¹

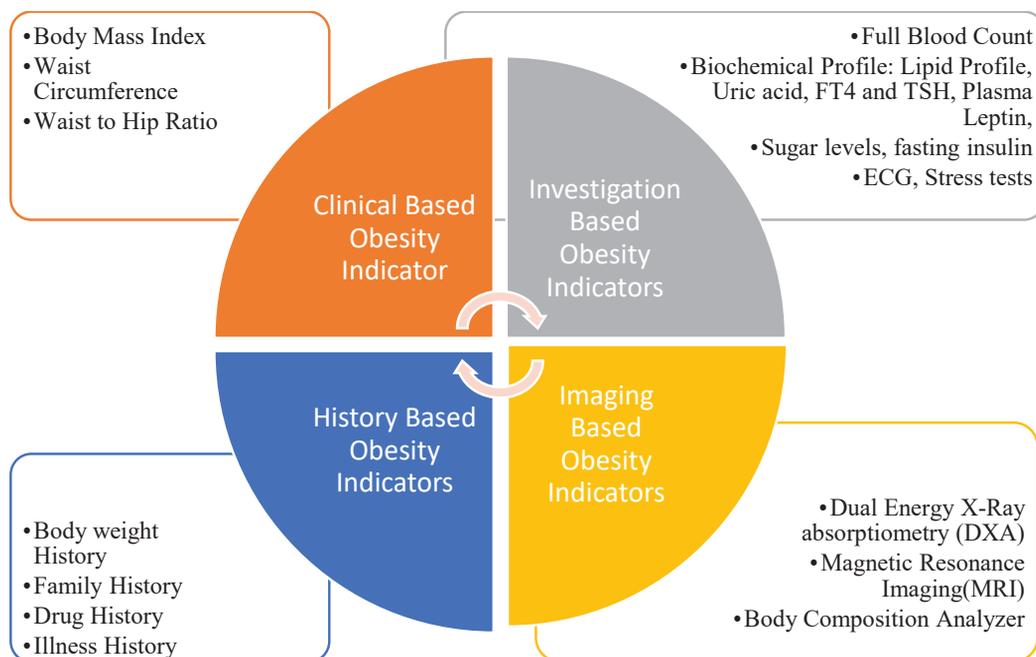


Figure 2 Assessments in overweight and obese persons Physical Examination¹²

- Height
- Weight
- BMI
- Waist Circumference, Hip circumference, neck circumference, wrist circumference
- Waist to Hip Ratio (WHR)
- Blood Pressure
- Pulse
- Percentage of body fat determined by skinfold thickness measurements¹³
- Tongue examination (Size, Colour and Texture)

Markers of insulin resistance- Skin tags and acanthosis nigricans

DIFFERENTIAL DIAGNOSIS

Obesity is known to be multifactorial, occurring due to complex interactions occurring between genetics and environmental factors. Where genetic factors per se can affect lipid metabolism and adiposity, the endocrinal factors affecting metabolism may also have genetic and environmental causations.

Identification of underlying cause of overweight and obesity are the mainstay of its management and treatment.

Table 1: Differential diagnosis

Sl. No.	Condition	Features
1.	Obesity due to Lifestyle Factors	<ul style="list-style-type: none"> • Imbalanced diets and sedentary lifestyles are linked to weight gain and adiposity. Physical inactivity is a hallmark of sedentary living and is often associated with increased body weight • Unhealthy eating patterns, including frequent consumption of fast food and sugary beverages, along with a low intake of fruits and vegetables, eating much more rapidly than usual, eating until uncomfortably full and consuming large amounts of food when not physically hungry are symptoms of Binge Eating and may contribute to the rising rates of obesity • Snacking and reliance on fast food are recognized as significant contributors to childhood overweight and obesity¹⁴
2.	Obesity due to Endocrinal conditions¹⁵	<p>The mechanisms underlying the development of obesity vary according to the abnormalities of endocrine function, whilst at the same time, increase in body fats also tends to lead to abnormalities in endocrinal functions.</p> <p>Some endocrinal disorders associated with obesity are:</p> <ul style="list-style-type: none"> • Hypothyroidism • Cushing's Syndrome • Insulinoma • Ovarian disorders and hyper ovarian syndrome • Hypogonadism in men • Hypothalamic tumours or damage to this part of the brain as a consequence of irradiation, infection or trauma

Sl. No.	Condition	Features
3.	Obesity with Genetic conditions¹⁶	Genetic and epigenetic variations contribute to obesity by influencing the function of metabolic pathways in the body and regulating neural pathways and appetite centres. Subsequently, these variations influence insulin resistance, dyslipidaemia, inflammation, hypertension and ectopic fat deposition-especially in the liver, which are the markers of obesity.
		Obesity can be syndromic due to <ul style="list-style-type: none"> • Chromosomal rearrangements, monogenic due to mutations in leptin signalling pathways or polygenic i.e. multiple mutations coding for proteins in skeletal and adipose tissues • Down's syndrome • Prader-Willi syndrome • WAGR syndrome • SIM1 syndrome • Bardet-Biedl syndrome • Fragile X syndrome • Cohen syndrome • Albright hereditary Osteodystrophy/PHP Type 1 a • Alstrom syndrome • Carpenter syndrome • Chudley-Lowry syndrome, etc.
4.	Drugs-Induced obesity^{17,18}	Weight gain or body fat redistribution are common side effects of many widely used drugs, some of which are given below: <ul style="list-style-type: none"> • Anticonvulsants: Sodium Valproate, Phenytoin • Hypoglycaemics: Insulin, Sulfonylurea (SU), Thiazolidinediones • Beta-Blockers: Atenolol, Metoprolol, Propranolol • Antidepressants: Amitriptyline, Nortriptyline, Imipramine, Desipramine, Dosulepin, Doxepin, Clomipramine • Antipsychotics: Haloperidol, Perphenazine

SUPPORTIVE INVESTIGATIONS¹⁹

The role of laboratory and other investigations is to exclude possible underlying causes of overweight/ obesity and its complications. Some key investigations that can be conducted for identifying causes / complications of overweight and obesity are as follows:

i. Essential Investigations

- Complete Blood Count/ESR
- Fasting lipid profile
- Fasting plasma glucose
- Fasting Insulin levels
- Serum uric acid
- Serum FT4 and TSH
- HbA1c

ii. Advanced Investigations

- 24-hour urine free cortisol
- Electrolyte Panel test
- ECG and chest x-ray
- Respiratory function tests
- Liver function test
- USG whole abdomen and pelvis
- Plasma Leptin
- Test For Insulin Resistance (OGTT, Insulin Sensitivity Test and Insulin Tolerance Test)
- Hormonal Assay (FH, LH, Prolactin, Androstenedione, Progesterone and Testosterone) in cases of Females

DIAGNOSTIC CRITERIA

Diagnosis of overweight and obesity is made by measuring people's weight and height and by calculating the body mass index (BMI). BMI equals the ratio of weight in kilograms divided by height in meters squared (kg/m^2): $\text{weight (kg)}/\text{height (m}^2\text{)}$.

The BMI categories for defining obesity vary by age and gender in infants, children and adolescents.

- Obesity in adults is defined as a BMI greater than or equal to 30; overweight is defined as a BMI greater than or equal to 25
- In children aged below 5 years, overweight is 2 standard deviations and obesity is greater than 3 standard deviations above the WHO Growth Reference median.²⁰
- In children aged between 5–19 years, overweight is 1 standard deviation and obesity is greater than 2 standard deviations above the WHO Growth Reference median²¹

The classification of body weight as per BMI in adults and children is given in Tables 1 & 2 respectively.

Table 2: Classification of obesity by BMI in adults²²

CLASSIFICATION	OBESITY CLASS	BMI
Obesity	I	30.0-34.9
Severe Obesity	II	35.0-39.9
Morbid Obesity	III	40.0-49.9
Severe Morbid Obesity	IV	>50

Table 3: Classification of weight by BMI in adult Asians

Classification	BMI (kg/m^2)
Underweight	<18.5
Normal range	18.5-22.9

Classification	BMI (kg/m ²)
Overweight	23-24.9
Obese I	25-29.9
Obese II	≥ 30

Table 4: Classification of BMI in children²²

CLASSIFICATION	BMI
Overweight	85 th percentile to less than the 95 th percentile
Obesity	95 th percentile or greater
Severe Obesity	120% of the 95 th percentile or greater 35 kg/m ²

The BMI percentile chart for children aged 6 to 18, as provided by RBSK, is given in Annexure - I

The body mass index is a surrogate marker of fatness and additional measurements, such as the waist circumference, are also used to diagnose obesity.²³ Measures of overweight and obesity and their cut-off for the Indian population are given in Table 3.

Table 5: Indian cut-offs for Indicators²⁴

PARAMETER	INDIAN CUT-OFF MALE	INDIAN CUT-OFF FEMALE
Waist Circumference (WC)(cm)	>90	>80
Waist-Hip Ratio (WHR)	>0.9	>0.85
Wrist circumference (cm)	>16.5	>15.7
Neck circumference (NC) (cm)	>35.25	>34.25
Body Fat Percentage	>25%	>30%
Body Mass Index (kg/m ²)	>23 Overweight, >25 – Obesity	

The 5th National Family Health Survey (NFHS) conducted in India (2019–21) assessed abdominal obesity through waist circumference for the first time. The survey identified that the prevalence of abdominal obesity was high in India. Overall, 40% of women and 12% of men were abdominally obese in the country, but 49.3% of women in the age group of 30–39 and 56.7% of women in the age group of 40–49 crossed the cut-off mark. Measured on BMI, only 23% of the women crossed the cut-off mark for obesity. Thus, some women who have healthy BMI also happened to have abdominal obesity.²⁵

Types of Body Fat Distribution^{26,27}

The distribution of accumulating adipose tissue varies among individuals but can generally be classified as lower body, abdominal subcutaneous (underneath the skin), overall coverage or visceral fat (Figure 3)

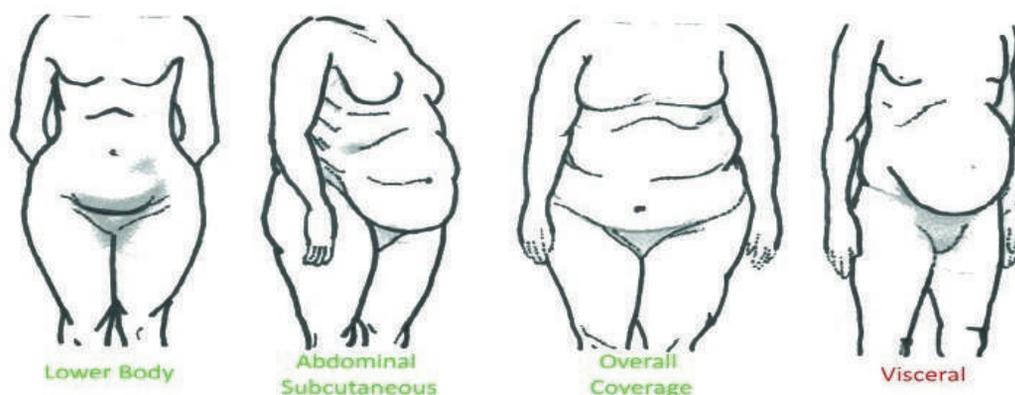


Figure – 3 Body fat distribution is characterized as **Lower body:** fat storage around the buttocks, hips and thighs; **Abdominal subcutaneous:** subcutaneous fat storage around the stomach and chest; **Overall coverage:** fat accumulation in the arms, breast, thighs, buttocks, lower back and breast, **Visceral:** Intra-abdominal fat deposition among organs such as the intestines, stomach, liver and pancreas. Fat distributed within the visceral cavity is highly associated with obesity-related health consequences whereas other fat distribution is not.

Siddha Diagnostic Criteria

Envagai Thervu (Eight-Fold System of clinical Assessment)^{28,29}

- *Nāṭi* (Pulse) – *Aiyam/ Aiyā aḷal / Aḷal aiyam*
- *Sparisam* (Touch)– Cool
- *Nā* (Tongue) -Thick/ coated
- *Niram* (Colour) – Normal/Fair
- *Moḷi* (Speech) – Normal/Rough/Hoarseness
- *Viḷi* (Eye) – Normal
- *Malam* (Stool) - Normal / constipation
- *Mūttiram* (Urine) -
 - *Nīrkkuri* (Uro-macroscopy) Yellowish in colour, tamarind odour
 - *Neykūri*(Oleo Uro-macroscopy) - Oil may spreads in the form of pearl

A(a). Comorbidities and Complications³⁰

Obesity and Overweight are associated with raised risk of disabilities and a number of comorbidities and complications²⁸ as listed in Table 4, which must be diagnosed timely.

Table 6: Complications and Comorbidities

SYSTEM	DISEASES
Respiratory	<ul style="list-style-type: none"> • Obstructive sleep apnoea (OSA) • Obesity Hypoventilation Syndrome (OHS)
Cardiovascular	<ul style="list-style-type: none"> • Coronary Heart Disease • Congestive Cardiac Failure • Hypertension
Cerebrovascular	<ul style="list-style-type: none"> • Stroke

SYSTEM	DISEASES
Gastrointestinal	<ul style="list-style-type: none"> • Gastroesophageal Reflux Disease • Barrett's Oesophagus • Erosive Oesophagitis • Diverticular Disease • Oesophageal Cancer • Colon Cancer • Abdominal Hernia
Metabolic	<ul style="list-style-type: none"> • Dyslipidemia • Type 2 Diabetes Mellitus • Hyperinsulinemia • Metabolic Syndrome • Gout • Gestational Diabetes
Hepato-biliary	<ul style="list-style-type: none"> • NASH (Non-alcoholic steatohepatitis) • Liver Cirrhosis • Hepatocellular Carcinoma • Gallstone • Gall Bladder Cancer
Musculoskeletal	<ul style="list-style-type: none"> • Osteoarthritis
Cutaneous	<ul style="list-style-type: none"> • Acanthosis nigricans • Cutaneous fungal and yeast infections • Venous stasis
Reproductive disorders	<ul style="list-style-type: none"> • Male: gynaecomastia • Female: Menstrual Irregularities, PCOS, Infertility
Cancer	<ul style="list-style-type: none"> • Male: Liver cancer, Pancreas cancer, Rectum cancer, Prostate • Female: Gall bladder, Bile duct, Breast, Ovary, Uterine, Cervix

PRINCIPLES OF MANAGEMENT

The principles of management involve evaluating signs and symptoms before beginning treatment and addressing any co-morbidities with appropriate conventional therapies. If the patient is already receiving standard of care, the physician may recommend continuing the current regimen along with Siddha medications. Follow-up assessments can then help to determine whether to taper or discontinue the additional treatment, in consultation with the conventional healthcare provider.

Red Flag Signs

- Breathlessness
- Sleep apnoea syndrome
- Unintentional weight gain
- Rapid Onset of weight gain
- Body Mass Index (BMI) greater than 40 kg/m² – Morbid obesity
- Weight gain associated with other systemic complications
- Cardiac arrhythmia and unstable cardiac conditions

PREVENTIVE MANAGEMENT

- Measures that focus on dietary intake, the home nutrition environment, nutrition knowledge, physical self-concept, body perception and overcoming barriers to exercise are effective in preventing obesity, especially in younger individuals.^{31,32}
- The primary goals of treatment are to improve obesity-related comorbid conditions, improve quality of life and reduce the risk of developing future obesity-related complications.
- Obesity in children and adolescents also requires an interprofessional team approach. Failure to adequately diagnose and treat overweight/obesity results in comorbid medical conditions and the likelihood that a child will become an obese adult.³³

Patients who present with obesity-related comorbidities and who would benefit from weight-loss intervention should be managed proactively.

a) Levels of Prevention for obesity

- Primordial Prevention: Prevent the development of obesity risk factors (sedentary behavior, poor diet).
- Primary Prevention: Prevent onset of obesity in at-risk individuals (e.g., children of obese parents, sedentary adults).
- Secondary Prevention: Early detection of overweight and obesity to prevent complications (diabetes, hypertension).

Tertiary Prevention: Manage established obesity and prevent further health deterioration or disability. Siddha System of Medicine emphasis adhering to *Tēraiyaṛ piṇi aṇukā viti* for prevention of disease and lead to healthy life.

Table 7

Dietary Habits (<i>Uṇavu Muṛaika!</i>)	
Do's - <i>Pattiyam</i>	Don'ts - <i>Apattiyam</i>
<ul style="list-style-type: none"> • Drink warm water • Add <i>Trithoda sama porutgal</i> inclusive of turmeric, pepper, cumin seeds, asafoetida, dry ginger, cardamom, fenugreek and garlic in food preparations • Consume low fat, low-calorie & high fiber diet, fresh vegetables, whole grains, legumes, greens & citrus fruits • Nuts, calcium rich foods – <i>Rāki aṭai, Pēriṅgam, Muruṅkai kīrai cūp, Tūtuvēḷai tuvaiyal</i> • Include traditional rice varieties like <i>Pūṅkār, kāṭṭu yāṇam karuppu kavuṇi, māppiḷai campā, iluppai campā, kuḷḷakkār</i> • Advised millet diet 3 days/week • Include lean proteins and low fat dairy in diet • <i>Thiriphala chooranam</i>³⁴ 2g BD before food 	<ul style="list-style-type: none"> • Avoid skipping meals • Always avoid heavy meals especially at night • Avoid untimely food, overcooked food, poorly cooked food • Food should never be consumed during excessive hunger, anger or grief • Food should never be taken full stomach • Avoid highly processed refined carbohydrate diet and advised to take complex carbohydrates • Limit added sugars, trans fat and refined grains • Avoid root tubers except yam – <i>Typhonium trilobatum (L.) Schott</i>

Table 8

Lifestyle Practices (<i>Vālvīyal Muraika!</i>)	
Do's	Don'ts
<ul style="list-style-type: none"> • Practice Siddha <i>Pancha Karpa</i> bath and sunbath • Practice at least 40 minutes of moderate physical activity (like walking) 5 days a week • Practice regular meditation • Consume food to the level of hunger • Consume food only half of stomach, liquid quarter of stomach and always leave quarter stomach empty • Always practice post meal walk • Sleep in left lateral position • Maintain balanced mood • Undergo therapeutic purgation once in four months • Practice Siddha <i>kāyakaṛpam</i> – take ginger, dried ginger and chebulic myrobalan in the morning, afternoon and evening respectively 	<ul style="list-style-type: none"> • Avoid daytime sleep or oversleeping • Avoid sedentary life style • Avoid stress • Avoid nap or sleep after food • Avoid alcohol and smoking

i. Siddha culinary medicine for prevention

1. Pre-meal herbal water infusion include

- Cumin seeds
 - Tulsi leaves
 - Mint + lemon
 - Cucumber
- Anyone of the water can be used

2. Take high fiber food like *Kīrai maciyal* (Spinach), Black gram and Sprouted fenugreek

3. Following medicinal seeds may be included in diet

- *Muruṅkai vitai* (Moringa seed)–Powder mixed with honey
- *Alici vitāy* (Flax seed) – with palm jaggery
- *Curai* (Bottle guard) and *Pūcāṇi* (Pumpkin) – roast in ghee and add pepper and salt

4. Unique Siddha foods for preventions

a. Marutam Tea - The following ingredients can be used for preparing one serving and can be taken.

- *Marutampaṭṭai* (*Terminalia Arjuna*) bark 1 part
- *Chukku* (Dry ginger) 1 part
- Rose petals 1 part
- *Milāku* (Pepper) ½ part
- *Karuñcīrakam* (Blackcumin seeds) 1/2 part
- *Ēlakkāy* (Cardamom) -1 No

b. Annapodi - Dry roast the ingredients and grind them as a coarse powder. Take 3tsp/day with buttermilk /hot water

- *Chukku* (Dry ginger)- 1 part
- *Milāku* (Pepper) -1 part
- *Cīrakam* (Cumin seeds) - 1 part
- *Karuñcīrakam* (Black cumin) 1 part
- *Peruñkāyam* (Asafoetida) -1 part
- *Karuvēppilai* (Curry leaves) -10 part

c. Idhaya Avizhtham - Add equal quantity of *Veṅtāmarai* (White lotus), *Cemparutti* (Hibiscus) and Rose petals with Palm Jaggery and make into balls.

- Herbal tea prepared with *Koḷḷu* (Horse gram), *Karuvēppilai* (curry leaves), *Intuppu* (Rock salt), *Milāku* (Pepper) and *Kōṭampuli* (Malabar tamarind) juice can be used.
- *Moringa olifera* (*Muruñkai kīrai*)³⁵ can be taken in the form of rasam
- One teaspoon of *Cissus quadrangularis*, rock salt powder and lemon juice (5 lemons) and dry it. Take ½ teaspoon of this mixture with food or water after food to reduce body weight. It also cures low back pain especially for ladies³⁵

5. Kōllu puṭtu - Horse gram rice cake³⁶

- Raw rice powder – 1 cup
- Horse gram –½ Cup
- Grated coconut – ¼ cup
- Ghee – 2 table spoon
- Cashew nut – 5
- Almond - 4
- Lemon Juice – 1 table spoon
- Turmeric powder –1 teaspoon
- Mustard – little
- Salt – required quantity

Clean the horse gram and roast until it turns aromatic. Once cooled, grind it coarsely. Mix it with pre heated raw rice powder, salt to taste and add sufficient quantity of water and make a moist flour with crumbly texture. Then, keep the flour in a closed container for ten minutes, add a pinch of turmeric powder and steam the mixture. Temper it with ghee, mustard seeds and curry leaves. Add grated coconut, lemon juice to the mixture. Top it with fried almond and cashew before serving.

Quantity to be taken – 100 g / Serving

6. Koḷḷu Aṭai - Sprouted Horse gram pan cake³⁶

- Par boiled rice - 1
- Sprouted horse gram – 1 cup

- Ginger –1 piece
- Pepper – 5
- Black sesame seed –2 table spoon
- Curry leaves –small quantity
- Oil, Salt –Required quantity.

Preparation

Soak parboiled rice for one hour. Grind it coarsely with sprouted horse gram, ginger and pepper. Add sesame seeds, curry leaves and salt to the batter. Make it as *Aṭai* (pan cake).

Quantity to be taken – 2 Nos - 100 g / 1 Serving

7. *Koḷḷū* soup - Horse gram soup³⁶

- Horse gram - ½ Cup
- Tomato - 3 Nos
- Lemon Juice - ½ table spoon
- Coriander leaves - Q.S
- Salt - Required quantity
- Ghee- 2 teaspoon
- Black pepper - 1 teaspoon
- Cumin Seeds- 1 teaspoon
- Garlic - 2 pearls
- Curry leaves - Small

Preparation

Soak horse gram overnight and pressure cook it for 4-5 whistles. Filter the water and keep it aside. Pound the garlic and curry leaves. Saute the tomato in ghee, add the pounded mixture, powdered cumin seeds and pepper for 3 minutes. Now, add the horse gram water, salt and boil it for 5 minutes. Then add lemon juice and garnish it with coriander leaves.

Quantity to be taken – 100 ml / Serving

8. *Sāmai kañji*- Little millet Porridge³⁶

- Well fried Little millet (*Panicum sumatrense*) -50 g
- Groundnut oil cake flour -25 g
- Urad dhal flour-25 g
- Jaggery-20 g

Preparation

Dissolve jaggery in water, filter and boil it. Mix all the flour in water. Pour this mixture slowly into the boiling jaggery. Stir it continuously for 10 to 15 minutes until the porridge is ready.

Quantity to be taken – 100 ml / Serving

9. **Vāragu kañji- Kodo millet rice porridge**³⁷

- Kodo millet rice
- Buttermilk
- Onion
- Salt

Mix Kodo millet with water and boil it for 10 Minutes. Once it is ready add buttermilk and salt to it. Take it along with onion.

Quantity to be taken – 100 ml / Serving

- Fasting

'Pāṭiṇi perumaruntu' is one of the Siddha schools of thought for well-being. Hence, fasting can be followed with the proper guidance of a Siddha physician. Following fasting methods can be adopted.

- Once a day fasting - one-time meal may be skipped
- Moon cycle fasting - 15 days once, 11th lunar day (Ēkātaci)
- Monthly once any specific day
- Honey fasting - Take 1 tsp of honey and keep it in the oral cavity at least for 3 min. After mixing it with saliva swallow, it slowly. Repeat the same procedure whenever feel stressed, tired and hungry. This can be done 15-20 times /day for a maximum of 3 days/week.
- Eat ½ hr after sunrise and ½ hr prior to sunset

ii. **Behavioural therapy:**

- Self-monitoring techniques- (e.g., journaling, weighing and measuring food and activity)
Stress management
- Stimulus control-(e.g., using smaller plates, not eating in front of the television or in the car)
- Follow sleep hygiene
- Avoid nap after food
- Have dinner 3 ½ hrs before sleep

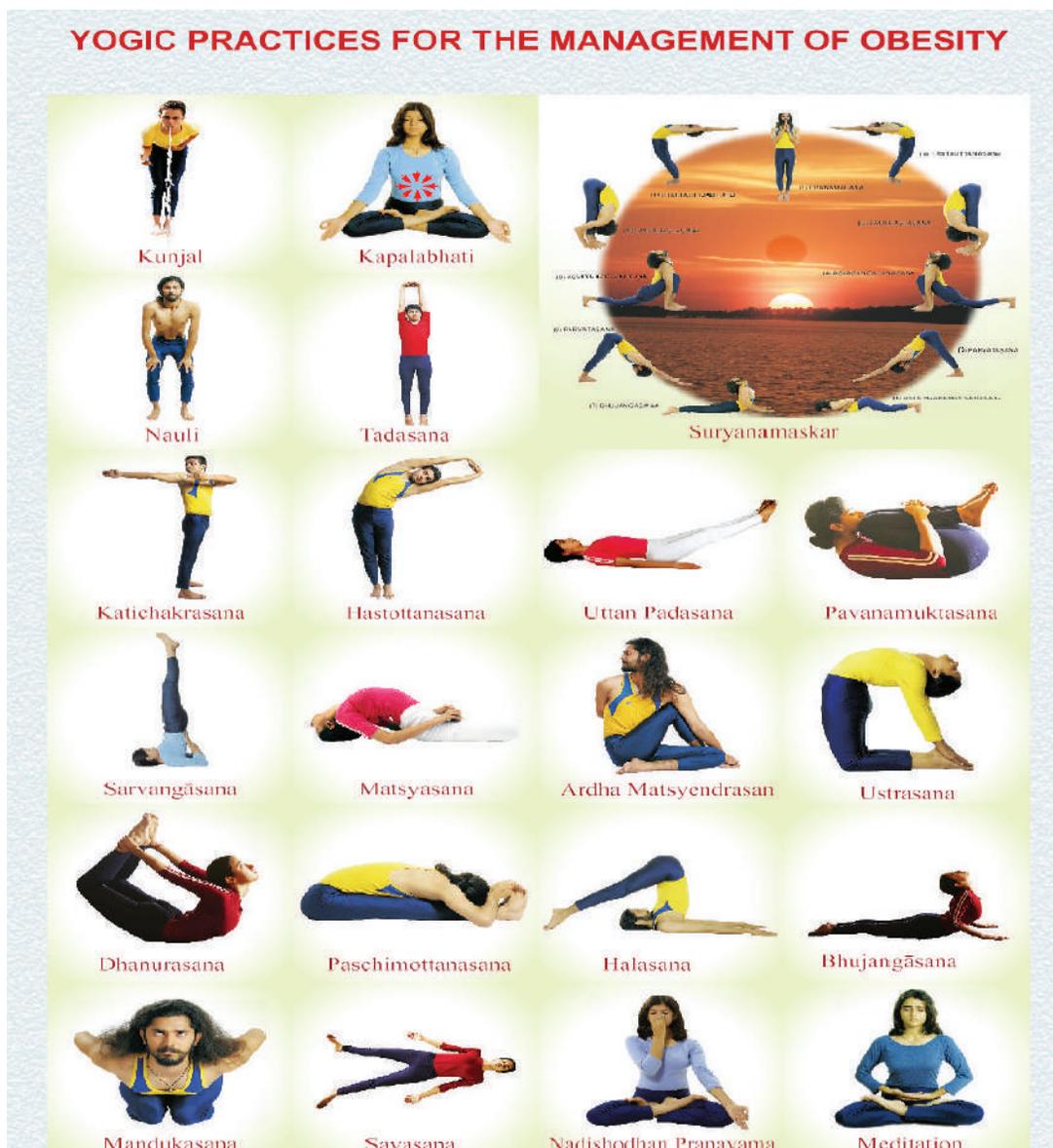
iii. **Physical activity**³⁸

The combination of dietary modification and exercise is the most effective behavioural approach for the treatment of obesity. Walking is one of the easiest and healthiest ways to exercise. At least a minimum period of 150 min of moderate-intensity or 75 min a week of vigorous-intensity aerobic physical activity per week is necessary. Examples include brisk walking, using the stairs, doing housework and yard work and engaging in sports. A high level of physical activity (>300 min of moderate-intensity activity per week) is often needed to lose weight and sustain weight loss.

• **Yogic practices include:**

- Om chanting and Prayer
- Shodhana Kriyas: Kapalabhati, Kunjal, Agnisara, Nauli

- Suryanamaskar
- Sukshma Vyayama
- Yogasanas: Tadasana, Katichakrasana, UrdhwaHastottanasana, Pawanamuktasana, Sarvangasana, Matsyasana, Halasana, Bhujangasana, Dhanurasana, UttanPadasana, Paschimottanasana, Ardha Matsyendrasana, Ushtrasana, Mandukasana, Shavasana
- Pranayama: Nadishodhana, Suryabhedhi Pranayama, Bhramari, Sitali, Bhastrika
- Special Practice: Yoga Nidra
- Dhyana (Meditation): Om Chanting, Om Meditation, and Anapana Meditation
- Yama and Niyama: This will help to have a controlled behaviour and would help to pacify the wandering mind and in turn help to have control over the eating and other habits of a person.
- Physical activity can be in the form of moderate to vigorous intensity aerobic activity, resistance training and muscle strengthening exercises.



Dance with *Mudhra*, *Baratham* with *mudhra*, *Parai isai*, *Kummi*, *Kavadi attam* may be practiced. Do any traditional sports like *Silambam*, *Kayittrattam* (skipping), *Pandi/Hopscotch*, etc.

8 (b) Curative Interventions

1) At Level 1:

- a. (Solo Physician Clinic/Health & Health Clinic/PHC (Optimal Standard of treatment in a situation where technology and resources are limited)

Clinical symptoms

There are no specific symptoms of overweight and obesity. Overweight and obesity are diagnosed based on clinical history and high body mass index (BMI).

Clinical Diagnosis

Based on anthropometry, clinical assessment of risk of co-morbidities and complications, the following investigations may be conducted:

- Complete Blood Count / ESR
- Fasting lipid profile
- Fasting plasma glucose
- HbA1c
- Serum uric acid
- Serum FT4 and TSH

Management

Siddha line of management:

Patients may seek Siddha management for overweight/ different stages of Obesity i.e., level I, level II and level III with or without comorbidities. Hence, the line of treatment may vary accordingly. The treatment algorithm is attached as Annexure – II.

The first line of treatment is to normalize the altered or deranged humours and revitalization of seven fundamental tissues through detoxification methods followed by internal medications.

The application of detoxification methods like therapeutic oilbath and purgation therapies may be decided by the Siddha physician.

Day 1

***Eṇṇey muḷukku* (Therapeutic oilbath):**

Eṇṇey muḷukku is a preparatory procedure, in which medicated oil massage with a lukewarm water bath. It will strengthen the five sensory organs. According to disease severity, *eṇṇey muḷukku* can be advised for one day to three days.

- *Arakku Thylam* (medicinal oil) – Quantity sufficient (External use)
- *Citramutti Thylam* - Quantity sufficient

Rules to be followed during *Eṇṇey muḷukku*

Apply oil before 7 am. Instil one drop in each eye, two drops in each nostril and three drops in each ear. Spread over the medicated oil from head to foot and give a gentle massage. After

application, leave it for 45 minutes and bathe with lukewarm water using herbal hair wash powder.

Take tender vegetables and easily digestible food. Avoid daytime sleep, intercourse and exposure to sunlight and cold items on the day of the oil bath.

Day 2

Therapeutic purgation (*Kaḷiccal maruttuvam*)³⁰

Table 9

Sl. No.	Drugs	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ <i>Anupaana</i>
1.	<i>Agathiyar Kuzhambu</i>	<i>Kuzhambu</i> (medicated viscous mixture)	100-200 mg	early morning on an empty stomach	1 st day of Treatment	Ginger juice (<i>Zingiber officinalis</i>)
2.	<i>Meganatha</i>	<i>Maathirai</i> (pills/tablets)	1-2 pills	early morning on an empty stomach	1 st day of Treatment	<i>Kāyntāriya vennīr</i> (lukewarm water)
3.	<i>Sanjeevi Maathirai</i>	<i>Maathirai</i> (pills/tablets)	1-2 pills	early morning on an empty stomach	1 st day of Treatment	<i>Kāyntāriya vennīr</i> (lukewarm water)

Rules to be followed during *Kaḷiccal Maruttuvam*

- The medication should be taken in the early morning 5 to 6 AM
- After the average number (5-6 times) of bowel evacuations, watery diarrhoea commences. In this stage, the patient is advised to take buttermilk/ lemon juice/fried cumin seeds decoction/Ash of sweet flag (*Vacampū*).
- After purgation, the patient may have symptoms like tiredness, slimness, lightness of the body and tiredness of sense organs which is a good sign.
- Dietary regimen during purgation:
 - *Mōr* (Butter milk)
 - *Kaṅci* (Rice porridge)
 - *Irumūraivaṭṭitta kaṅci* (Double boiled porridge)
 - *Kāyntāriya vennīr* (Luke warm water)
- Precautions
 - Avoid daytime sleep during purgation therapy
 - Should not take heavy meals before or during the procedure

Day 3 onwards

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 10: Single herbs

Sl. No	Single herbs	Dosage form	Dose	Time	Frequency and Duration	Adjuvants /Anuppanam
1.	<i>Amukkura (Withania somnifera)</i> ³⁹	Dried root powder	2 g	BD	For 48 days	Boiled gruel
2.	<i>Sangan (Azima tetraantha)</i> ⁴⁰	Dried powder of root bark	2 g	BD	For 48 days	Ghee or Honey

Table 11: Compound formulation

Sl. No.	Compound formulations	Dosageform	Dose	Time	Duration and frequency	Adjuvants/ Aṇupāṇam
Chooranam / Medicinal powder						
1.	<i>Thiriphala Chooranam</i> ⁴¹⁻⁴²	Medicinal powder	2 g	Morning & Night	48 days	Water
2.	<i>Kakkirattan Chooranam</i> ⁴³	Medicinal powder	4-12 g	Morning & Night	48 days	Lemon Juice, Rose water, Tamarind syrup
3.	<i>Asuwathi Chooranam</i> ^{44,45}	Medicinal powder	2 g	Morning & Night	48 days	Honey

2) Recommended Diet & Lifestyle^{46, 47}

A comprehensive programme of lifestyle modification is considered the first option for achieving the goal of obesity management. This involves three essential elements of lifestyle

1. Diet therapy

- The primary focus of diet therapy is to reduce overall calorie consumption.
- A calorie-deficit diet is advised, taking into consideration nutritional requirements.
- The calorie deficit can be instituted through dietary substitutions or alternatives. Examples include choosing smaller portion sizes, eating more fruits and vegetables, consuming more whole-grain cereals and selecting leaner cuts of meat and skimmed dairy products.
- Adequate intake of micronutrients and fibre-rich such as pulses, nuts, chia seeds, flax seeds and whole grains including millets, vegetables and fruits helps to maintain levels of blood glucose, insulin, cholesterol as well as triglycerides. Use of healthy cooking methods like grilling, baking, steaming or sautéing with minimal oil instead of frying is recommended.
- A daily calorie deficit of 500-1000 kcal is commonly recommended which typically results in a weight loss of 0.5-1kg per week. Total calorie intake is 1200-1500 kcal/day for women, 1500-1800 kcal/day for men. These values may vary and should be adjusted to individual needs to avoid nutritional deficiencies. A reduction of half a kilogram body weight per week

is considered to be safe. Approaches of rapid weight loss should be avoided. Consuming higher amounts of protein (15% energy from protein) may be important during typical energy-deficient weight loss diets (i.e. 500 to 750 kilo calorie per day deficit) to preserve muscle mass. Nevertheless, the protective effect of higher protein diets on muscle mass is compromised if the energy deficit is more than 40% of daily energy needs and the dietary proteins are oxidised for energy production. Weight reducing diet should be nutrient-rich and nutritionally balanced, with adequate intake of micro-nutrients and fibre rich foods.

- The Yogic diet, popularly known as Satvik diet is the most preferred diet in obese condition. Satvik diet contains more of fresh fruits and vegetables in its natural form, soup etc. Rajasic foods like fried food items, spicy foods, soft drinks and beverages, fast foods etc, should be limited.⁴⁶
- Shift to healthy snacking such as fruits, vegetables and sprouts instead of cakes, biscuits and fried snacks.
- Have regular meals at fixed interval.
- Siddha culinary medicine – As given in preventive management

2. Physical Activity

- A combination of dietary modification and increased physical activity or exercise is the most effective behavioural approach for the treatment of obesity. The most important role of exercise appears to be in the maintenance of weight loss.^{47,48}
- At least 150 minutes aerobic physical activity (e.g., brisk walking) per week (equivalent to 30 minutes per day for 5 days of the week) for initial weight loss, increasing to around 200 to 300 minutes per week to maintain body weight and prevent weight regain is recommended.⁴⁹ Exercise intensity and duration should be increased gradually over a period of time.^{49,50}
- Exercise for weight reduction goes beyond being simply physically active during the day, both in term of type and duration of activity or exercise.
- However, initiating type and duration of exercise and gradual increase in physical activity needs to be undertaken with due consideration of the overall health condition, including systemic complications of the individual patient.
- Cittar yōkam- As given in the preventive management.
- Yoga practices can reduce weight and also manage stress, endocrinal imbalances and other factors associated with obesity. Yoga or physical exercises are suggested to be undertaken under the supervision of a trained therapist.

3. Behavioural therapy

- Cognitive behavioural therapy can change and reinforce new dietary and physical activity behaviours.
- Strategies include self-monitoring techniques (e.g., journaling, weighing and measuring food and activity); stress management; stimulus control (e.g., using smaller plates, not eating in front of the television or in the car); social support; problem solving; and cognitive restructuring to help patients develop more positive and realistic thoughts about themselves.

- When recommending any behavioural lifestyle change, the patient should be asked to identify what, when, where and how the behavioural change will be performed.⁵¹
- Encourage breast feeding as the child who gets proper breast feeding is less likely to develop obesity in the later age.

3) Restricted Diet & Lifestyle³⁰

- Avoid overeating and/or eating foods with *mantham* (Dullness) and *thinmai* (Bulkiness) *gunam* in large quantities.
 - Eg: Tubers like potato, topiaco, milk and milk products
- Avoid starchy foods and sugar-sweetened beverages
- Avoid *tamasa* character foods (*Thamasa* food generates heaviness in the body and dullness of mind.): Leftovers, processed, canned foods, fast food or food with additives and colourings
- Avoid deep-fried foods
- Do not eat on the run or while watching TV
- Quit drinking alcohol and smoking

Follow up: Every 15 days or as per need Review should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their activities of daily living and quality of life.
- Monitoring of signs and symptoms, diet, daily activity, change in weight, anthropometry
- Assessment of energy balance
- Assessment of motivation levels to continue with lifestyle modifications
- Monitoring the long-term course of the condition.
- Discussing the person's knowledge of the condition, any concerns they have, their personal preferences and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.
- Self-management support.

Referral criteria

- Non-response to treatment, no change in weight, anthropometry despite negative energy balance.
- Sudden loss or gain of more than 10% body weight.
- Uncontrolled endocrinal profile.
- Morbid obesity where it is difficult to insinuate lifestyle changes.
- Evidence of an increase in severity/complications
- Diagnostic uncertainty
- Co-morbidities, such as cardiac disease.
- Substantial impact on their quality of life and activities of daily living.

II. At Level 2:

(CHC/Small hospitals (10-20 bedded hospitals with basic facilities such as routine, investigation, X-ray)

Clinical Diagnosis: Same as level 1

- Clinical assessment of body fat percentage

Investigations:

- 24-hour urine free cortisol
- ECG and Chest X-ray
- Respiratory function tests
- Test For Insulin Resistance (OGTT, Fasting plasma insulin)
- Serum Electrolytes
- USG whole abdomen and pelvis

Management

Along with level 1 medications including detoxification treatment any of the following medicines can be used. For new cases at this level, medications listed for Level-1 may also be considered, but the comprehensive set of symptoms exhibited by the patient remains the key factor in determining the appropriate treatment.

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 12: Single herbs

Sl. No	Single herb	Dosage form	Dose	Time	Frequency and Duration	Adjuvants /Anupānam
1.	<i>Vilvam (Aegle marmelos)</i> ⁵¹	<i>Kudineer /</i> Decoction of root or root bark	30ml-45ml	BD before food	48 days	--
2.	<i>Nilavagai (Cassia italica)</i> ⁵²	Leaf powder	1-2 g	BD	48 days	Honey
3.	<i>Nilavagai (Cassia italica)</i> ⁵²	Whole plant powder	800 mg – 1000 mg	BD	48 days	Honey
4.	<i>Karisalai (Eclipta prostrata)</i> ⁵²	Leaf Powder	5 g	OD on an empty stomach	48 days	Water
5.	<i>Kollu (Macrotyloma uniflorum)</i> ⁵³	Powder	2 tablespoons	BD after food	According to the discretion of Physician	Water

6.	<i>Neermulli (Hygrophilla auriculata)</i> ⁵⁴	Root powder	2 g	BD	For 48 days	Honey
7.	<i>Ilanthai (Ziziphus mauritiana)</i> ⁵⁴	Karkam / medicinal Paste	2 g	BD	For 48 days	Gruel
8.	<i>Ilanthai (Ziziphus mauritiana)</i> ⁵⁴	Leaves soaked in water overnight	30ml – 45ml	OD	For 48 days	The water should be taken next day morning

Table 13: Compound formulations

Sl. No.	Compound formulations	Dosageform	Dose	Time	Duration and Frequency	Adjuvants / <i>Aṇupāṇam</i>
Kudineer / Decoction						
1.	Powder of <i>Nerunjil (Tribulus terrestris)</i> , <i>Neermulli (Hygrophilla auriculata)</i> , <i>Sombu (Pimpinella anisum)</i> , <i>Kothamalli (Coriandrum sativum)</i> in equal ratio ³⁵	The decoction of this powder	30ml- 45ml	BD after food	twice daily	Milk
Chooranam / Medicinal powder						
2.	<i>Pungu (Pongamia Pinnata)</i> and <i>Vengai (Pterocarpus marsupium)</i> ⁵⁴	Medicinal powder	2 g	BD	48 days	with water
Chenduram / Red calx						
3.	<i>Chunnaloga Chenduram</i> ⁵⁵	Red calx	200 mg 360 mg	BD	48 days	Honey
4.	<i>Ekku Chenduram</i> ⁵⁵	Red calx	488 mg	BD	48 days	<i>Thiriphala Chooranam</i> , Palm Sugar, Honey
5.	<i>Velvanga Chenduram</i> ⁵⁶	Red calx	135 g to 260 g	BD	48 days	<i>Moongil Ilai</i> juice (Tender bamboo leaf juice)
Nei / Medicated Ghee						
6.	<i>Megasanjeevi Nei</i> ³⁰	Medicated ghee	6 – 12 g	BD	48 days	Sugar or Puffed rice flour

Siddha *Puramaruttuvam* (External therapies)

Podithirmirthal (powder massage)²⁰

- *Kollu podi* (Horse gram powder)
- *Manjal Podi* or *Ilai Karkam*
- (*Turmeric* or leaf paste)
- *Thiriphala Chooranam*
- *Puttru man* (Termite mound soils)

15 minutes 1 hr depending on level of obesity

1. Recommended Diet & Lifestyle

2. Restricted Diet & Lifestyle

As per Level 1

3. Follow Up

Every 15 days or as per the need

4. Referral Criteria

- Same as mentioned at Level 1 and any of these
- Psychological imbalance
- Suspected life-threatening complications such as heart failure

III. At Level 3:

(Ayush hospitals attached with teaching Institution, District Level/Integrated/State Ayush Hospitals, Allopathic hospitals also having tertiary care facilities either standalone or integrative management facilities).

Clinical Diagnosis:

Same as levels 1 & 2. Confirm diagnosis and severity with the help of the following investigations:

Treadmill Test or Exercise stress Test to evaluate the efficacy of functioning of heart during exercise

Management

Along with level 1& 2 medications including detoxification treatment any of the following medicines can be used. For patients referred from Level 1 or 2, the treatment provided at those levels may be continued if it suits the current condition. Alternatively, the case may be reassessed to identify the underlying causes of obesity, symptoms the treatment will be adjusted accordingly. For new cases at this level, the complete set of symptoms presented by the patient will be the primary factor guiding treatment decisions.

(**Note:** Administration of medicine, dosage, drug holiday and treatment duration may vary according to the condition of patient, disease severity. This solely depends upon the discretion of the treating Siddha Physician).

Table 14: Single herbs

Sl. No	Single drug	Dosage form	Dose	Time	Frequency and Duration	Adjuvants /Anupānam
1.	<i>Nathaisoori (Spermacoce hispida)</i> ⁵⁷	Dried root powder	1-2 g	BD	48 days	Warm water
2.	<i>Karuveppilai (Murraya koenigi)</i> ⁵⁷	Dried leaf powder	1-2 g	BD	48 days	Warm Water
3.	<i>Seenthil (Tinospora cardifolia)</i> ⁵⁷	Dried leaf powder	1-2 g	BD	48 days	Warm Water

Table 15: Compound formulations

Sl. No.	Compound formulation	Dosage form	Dose	Time	Duration and Frequency	Adjuvants /Anupānam
Kudineer/Decoction						
1.	<i>Mantharaiver Kudineer</i> ³⁰	Decoction	60 ml	BD before food	48 days	NA
Chooranam /Medicinal powder						
1.	<i>Thirikatuku Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	Depending upon the severity of the disease condition.	Warm water
2.	<i>Nilavagai Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
3.	<i>Karisalai karpa Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
4.	<i>Kukkilathi Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
5.	<i>Karunai kizhangu Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
6.	<i>Nathaisoori Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
7.	<i>Kazharchi Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
8.	<i>Karunjeeraga Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water
9.	<i>Asoka pattai Chooranam</i> ³⁰	Medicinal powder	1-2 g	BD after food	-do-	Warm water

Sl. No.	Compound formulation	Dosage form	Dose	Time	Duration and Frequency	Adjuvants / <i>Aṇupāṇam</i>
Maathirai / Tablet						
10.	<i>Veppampoo Maathirai</i> ³⁰	<i>Maathirai</i>	1-2	BD after food	-do-	Warm water
Chenduram /Red calx						
11.	<i>Kaantha Chenduram</i> ³⁰	Red calx	100-200 mg	BD after food	48 days	Honey
12.	<i>Ayakantha Chenduram</i> ³⁰	Red calx	100 - 200 mg	BD after food.	48 days	Honey
13.	<i>Aya Chenduram</i> ³⁰	Red calx	-100-200 mg	BD after food	48 days	Honey
14.	<i>Ekku Chenduram</i> ³⁰	Red calx	-100-200 mg	BD after food	48 days	Honey
Parpam / White calx						
15.	<i>Kungiliya Parpam</i> ³⁰	White calx	200 - 400 mg	BD after food	48 days	Warm water
16.	<i>Silasathu Parpam</i> ³⁰	White calx	200 - 400 mg	BD after food	48 days	Warm water
17.	<i>Palakarai Parpam</i> ³⁰	White calx	65 - 130 mg	BD after food	48 days	Warm water
Karpam / Rejuvenating drugs						
18.	<i>AyasambaraKarpam</i> ³⁰	Rejuvenating drug	100- 200 mg	BD after food	48 days	Honey
19.	<i>Ayabringaraja Karpam</i> ³⁰	Rejuvenating drug	100- 200 mg	BD after food	48 days	Honey

Siddha puramaruthuvam (Siddha external therapies)

Podi Thimirthal (Powder Massage): 30

- *Kollu Podi (Macrotyloma uniflorum)* Podi Thimirthal for 7 days
- *Pottru man* (Termite mound soils)

Vedhu (Steaming):

- *Nochi* (Five-leaved chaste tree-*Vitex negundo*) leaves
- *Manjal* (Turmeric-*Curcuma longa*) powder
- *Elumichai* (Lemon - *Citrus limon*) seed

Suttigai (Cautery cauterization)

- *Kānthe suttigai* (Sun bath)- 30 to 45 minutes with herbal poultice/ day for 48 days

1) **Recommended diet and lifestyle:** Same as Levels 1& 2

2) **Restricted diet and lifestyle:** Same as Levels 1& 2

3) **Follow up:** Every 15 days or as per need

4) **Referral Criteria**⁵⁰

Same as mentioned in Level 2 and any of these

- Morbid obesity not responding to treatment
- Uncontrolled hypertension
- Worsening Hypertriglyceridemia
- Worsening insulin resistance and hyperglycaemia
- Suspected Cardiac arrhythmias
- Suspected Polycythaemia
- Other modalities can be considered depending on the case and to rehabilitate properly.

Table 16: Siddha culinary medicine to be added²⁸

Vegetables	<ul style="list-style-type: none"> • <i>Veḷḷari</i> / Cucumber (<i>Cucumis sativus</i>) • <i>Vellai pōsani</i> / Pumpkin (<i>Cucurbita pepo</i>) • <i>Suraikkāi</i> / Bottle gourd (<i>Lagenaria siceraria</i>) • <i>Vālai thandu</i> / Banana stem (<i>Musa paradisiaca</i>) • <i>Murungai kai</i> / Drumstick (<i>Moringa oleifera</i>) • <i>Vendaikkāi</i> / Ladies finger (<i>Abelmoschus esculentus</i>) • <i>Kērai</i> / Green leafy vegetable • <i>Iṅgi</i> / Ginger (<i>Zingiber officinale</i>) • <i>Pōndu</i> / Garlic (<i>Allium sativum</i>) • <i>Chinna vengāyam</i> / Small onion (<i>Allium parvum</i>) • <i>Kovakāi</i> / Coccinia (<i>Coccinia grandis</i>)
Any one or more vegetables can be included in daily diet in soup, salad, veg curry, etc.	
Fruits	<ul style="list-style-type: none"> • <i>Nellikāi</i> / Gooseberry (<i>Ribes grossularia</i>) • <i>Elumitchai</i> / Lemon (<i>Citrus limon</i>) • <i>Pappāli</i> / Papaya (<i>Carica papyra</i>) • <i>Koyya</i> / Guava (<i>Psidium guajava</i>)
A bowel of fruit salad of the above fruits can be included daily	
Millets	<ul style="list-style-type: none"> • <i>Keḷvaragu</i> / Finger millet (<i>Eleusine coracana</i>) • <i>Thinai</i> / Foxtail millet (<i>Setaria italic</i>) • <i>Kuthiraivāli</i> / Barnyard millet (<i>Echinochloa frumentacea</i>) • <i>Sāmai</i> / Little millet (<i>Panicum sumatrense</i>)
These millets may be taken in the form of various culinary preparations like <i>Idly</i> , <i>Pongal</i> , <i>Porridge</i> , <i>Dōcai</i> , <i>Aṭai</i> , <i>Variety rice</i> etc.	

RBSK_BMI for Age

WHO Simplified field tables- BMI for age 6 to 18 years (z-scores)

Refer any child whose BMI for age and sex is $><3$ SD.

BMI-for-age GIRLS 5 to 19 years (z-scores)							Age in		BMI-for-age BOYS 5 to 19 years (z-scores)						
-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD	Year: Month	Months	-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD
11.8	12.7	13.9	15.2	16.9	18.9	21.3	5:01	61	12.1	13	14.1	15.3	16.6	18.3	20.2
11.8	12.7	13.9	15.2	16.9	18.9	21.4	5:02	62	12.1	13	14.1	15.3	16.6	18.3	20.2
11.8	12.7	13.9	15.2	16.9	18.9	21.5	5:03	63	12.1	13	14.1	15.3	16.7	18.3	20.2
11.8	12.7	13.9	15.2	16.9	18.9	21.5	5:04	64	12.1	13	14.1	15.3	16.7	18.3	20.3
11.7	12.7	13.9	15.2	16.9	19	21.6	5:05	65	12.1	13	14.1	15.3	16.7	18.3	20.3
11.7	12.7	13.9	15.2	16.9	19	21.7	5:06	66	12.1	13	14.1	15.3	16.7	18.4	20.4
11.7	12.7	13.9	15.2	16.9	19	21.7	5:07	67	12.1	13	14.1	15.3	16.7	18.4	20.4
11.7	12.7	13.9	15.3	17	19.1	21.8	5:08	68	12.1	13	14.1	15.3	16.7	18.4	20.5
11.7	12.7	13.9	15.3	17	19.1	21.9	5:09	69	12.1	13	14.1	15.3	16.7	18.4	20.5
11.7	12.7	13.9	15.3	17	19.1	22	5:10	70	12.1	13	14.1	15.3	16.7	18.5	20.6
11.7	12.7	13.9	15.3	17	19.2	22.1	5:11	71	12.1	13	14.1	15.3	16.7	18.5	20.6
11.7	12.7	13.9	15.3	17	19.2	22.1	6:00	72	12.1	13	14.1	15.3	16.8	18.5	20.7
11.7	12.7	13.9	15.3	17	19.3	22.2	6:01	73	12.1	13	14.1	15.3	16.8	18.6	20.8
11.7	12.7	13.9	15.3	17	19.3	22.3	6:02	74	12.2	13.1	14.1	15.3	16.8	18.6	20.8
11.7	12.7	13.9	15.3	17.1	19.3	22.4	6:03	75	12.2	13.1	14.1	15.3	16.8	18.6	20.9
11.7	12.7	13.9	15.3	17.1	19.4	22.5	6:04	76	12.2	13.1	14.1	15.4	16.8	18.7	21
11.7	12.7	13.9	15.3	17.1	19.4	22.6	6:05	77	12.2	13.1	14.1	15.4	16.9	18.7	21
11.7	12.7	13.9	15.3	17.1	19.5	22.7	6:06	78	12.2	13.1	14.1	15.4	16.9	18.7	21.1
11.7	12.7	13.9	15.3	17.2	19.5	22.8	6:07	79	12.2	13.1	14.1	15.4	16.9	18.8	21.2
11.7	12.7	13.9	15.3	17.2	19.6	22.9	6:08	80	12.2	13.1	14.2	15.4	16.9	18.8	21.3
11.7	12.7	13.9	15.4	17.2	19.6	23	6:09	81	12.2	13.1	14.2	15.4	17	18.9	21.3
11.7	12.7	13.9	15.4	17.2	19.7	23.1	6:10	82	12.2	13.1	14.2	15.4	17	18.9	21.4
11.7	12.7	13.9	15.4	17.3	19.7	23.2	6:11	83	12.2	13.1	14.2	15.5	17	19	21.5
11.8	12.7	13.9	15.4	17.3	19.8	23.3	7:00	84	12.3	13.1	14.2	15.5	17	19	21.6
11.8	12.7	13.9	15.4	17.3	19.8	23.4	7:01	85	12.3	13.2	14.2	15.5	17.1	19.1	21.7
11.8	12.8	14	15.4	17.4	19.9	23.5	7:02	86	12.3	13.2	14.2	15.5	17.1	19.1	21.8
11.8	12.8	14	15.5	17.4	20	23.6	7:03	87	12.3	13.2	14.3	15.5	17.1	19.2	21.9
11.8	12.8	14	15.5	17.4	20	23.7	7:04	88	12.3	13.2	14.3	15.6	17.2	19.2	22
11.8	12.8	14	15.5	17.5	20.1	23.9	7:05	89	12.3	13.2	14.3	15.6	17.2	19.3	22
11.8	12.8	14	15.5	17.5	20.1	24	7:06	90	12.3	13.2	14.3	15.6	17.2	19.3	22.1
11.8	12.8	14	15.5	17.5	20.2	24.1	7:07	91	12.3	13.2	14.3	15.6	17.3	19.4	22.2
11.8	12.8	14	15.6	17.6	20.3	24.2	7:08	92	12.3	13.2	14.3	15.6	17.3	19.4	22.4
11.8	12.8	14.1	15.6	17.6	20.3	24.4	7:09	93	12.4	13.3	14.3	15.7	17.3	19.5	22.5
11.9	12.9	14.1	15.6	17.6	20.4	24.5	7:10	94	12.4	13.3	14.4	15.7	17.4	19.6	22.6
11.9	12.9	14.1	15.7	17.7	20.5	24.6	7:11	95	12.4	13.3	14.4	15.7	17.4	19.6	22.7
11.9	12.9	14.1	15.7	17.7	20.6	24.8	8:00	96	12.4	13.3	14.4	15.7	17.4	19.7	22.8
11.9	12.9	14.1	15.7	17.8	20.6	24.9	8:01	97	12.4	13.3	14.4	15.8	17.5	19.7	22.9
11.9	12.9	14.2	15.7	17.8	20.7	25.1	8:02	98	12.4	13.3	14.4	15.8	17.5	19.8	23
11.9	12.9	14.2	15.8	17.9	20.8	25.2	8:03	99	12.4	13.3	14.4	15.8	17.5	19.9	23.1
11.9	13	14.2	15.8	17.9	20.9	25.3	8:04	100	12.4	13.4	14.5	15.8	17.6	19.9	23.3
12	13	14.2	15.8	18	20.9	25.5	8:05	101	12.5	13.4	14.5	15.9	17.6	20	23.4
12	13	14.3	15.9	18	21	25.6	8:06	102	12.5	13.4	14.5	15.9	17.7	20.1	23.5
12	13	14.3	15.9	18.1	21.1	25.8	8:07	103	12.5	13.4	14.5	15.9	17.7	20.1	23.6
12	13	14.3	15.9	18.1	21.2	25.9	8:08	104	12.5	13.4	14.5	15.9	17.7	20.2	23.8
12	13.1	14.3	16	18.2	21.3	26.1	8:09	105	12.5	13.4	14.6	16	17.8	20.3	23.9
12.1	13.1	14.4	16	18.2	21.3	26.2	8:10	106	12.5	13.5	14.6	16	17.8	20.3	24
12.1	13.1	14.4	16.1	18.3	21.4	26.4	8:11	107	12.5	13.5	14.6	16	17.9	20.4	24.2
12.1	13.1	14.4	16.1	18.3	21.5	26.5	9:00	108	12.6	13.5	14.6	16	17.9	20.5	24.3
12.1	13.2	14.5	16.1	18.4	21.6	26.7	9:01	109	12.6	13.5	14.6	16.1	18	20.5	24.4
12.1	13.2	14.5	16.2	18.4	21.7	26.8	9:02	110	12.6	13.5	14.7	16.1	18	20.6	24.6
12.2	13.2	14.5	16.2	18.5	21.8	27	9:03	111	12.6	13.5	14.7	16.1	18	20.7	24.7
12.2	13.2	14.6	16.3	18.6	21.9	27.2	9:04	112	12.6	13.6	14.7	16.2	18.1	20.8	24.9
12.2	13.3	14.6	16.3	18.6	21.9	27.3	9:05	113	12.6	13.6	14.7	16.2	18.1	20.8	25

BMI-for-age GIRLS 5 to 19 years (z-scores)							Age in		BMI-for-age BOYS 5 to 19 years (z-scores)						
-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD	Year:	Months	-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD
12.2	13.3	14.6	16.3	18.7	22	27.5	9:06	114	12.7	13.6	14.8	16.2	18.2	20.9	25.1
12.3	13.3	14.7	16.4	18.7	22.1	27.6	9:07	115	12.7	13.6	14.8	16.3	18.2	21	25.3
12.3	13.4	14.7	16.4	18.8	22.2	27.8	9:08	116	12.7	13.6	14.8	16.3	18.3	21.1	25.5
12.3	13.4	14.7	16.5	18.8	22.3	27.9	9:09	117	12.7	13.7	14.8	16.3	18.3	21.2	25.6
12.3	13.4	14.8	16.5	18.9	22.4	28.1	9:10	118	12.7	13.7	14.9	16.4	18.4	21.2	25.8
12.4	13.4	14.8	16.6	19	22.5	28.2	9:11	119	12.8	13.7	14.9	16.4	18.4	21.3	25.9
12.4	13.5	14.8	16.6	19	22.6	28.4	10:00	120	12.8	13.7	14.9	16.4	18.5	21.4	26.1
12.4	13.5	14.9	16.7	19.1	22.7	28.5	10:01	121	12.8	13.8	15	16.5	18.5	21.5	26.2
12.4	13.5	14.9	16.7	19.2	22.8	28.7	10:02	122	12.8	13.8	15	16.5	18.6	21.6	26.4
12.5	13.6	15	16.8	19.2	22.8	28.8	10:03	123	12.8	13.8	15	16.6	18.6	21.7	26.6
12.5	13.6	15	16.8	19.3	22.9	29	10:04	124	12.9	13.8	15	16.6	18.7	21.7	26.7
12.5	13.6	15	16.9	19.4	23	29.1	10:05	125	12.9	13.9	15.1	16.6	18.8	21.8	26.9
12.5	13.7	15.1	16.9	19.4	23.1	29.3	10:06	126	12.9	13.9	15.1	16.7	18.8	21.9	27
12.6	13.7	15.1	17	19.5	23.2	29.4	10:07	127	12.9	13.9	15.1	16.7	18.9	22	27.2
12.6	13.7	15.2	17	19.6	23.3	29.6	10:08	128	13	13.9	15.2	16.8	18.9	22.1	27.4
12.6	13.8	15.2	17.1	19.6	23.4	29.7	10:09	129	13	14	15.2	16.8	19	22.2	27.5
12.7	13.8	15.3	17.1	19.7	23.5	29.9	10:10	130	13	14	15.2	16.9	19	22.3	27.7
12.7	13.8	15.3	17.2	19.8	23.6	30	10:11	131	13	14	15.3	16.9	19.1	22.4	27.9
12.7	13.9	15.3	17.2	19.9	23.7	30.2	11:00	132	13.1	14.1	15.3	16.9	19.2	22.5	28
12.8	13.9	15.4	17.3	19.9	23.8	30.3	11:01	133	13.1	14.1	15.3	17	19.2	22.5	28.2
12.8	14	15.4	17.4	20	23.9	30.5	11:02	134	13.1	14.1	15.4	17	19.3	22.6	28.4
12.8	14	15.5	17.4	20.1	24	30.6	11:03	135	13.1	14.1	15.4	17.1	19.3	22.7	28.5
12.9	14	15.5	17.5	20.2	24.1	30.8	11:04	136	13.2	14.2	15.5	17.1	19.4	22.8	28.7
12.9	14.1	15.6	17.5	20.2	24.2	30.9	11:05	137	13.2	14.2	15.5	17.2	19.5	22.9	28.8
12.9	14.1	15.6	17.6	20.3	24.3	31.1	11:06	138	13.2	14.2	15.5	17.2	19.5	23	29
13	14.2	15.7	17.7	20.4	24.4	31.2	11:07	139	13.2	14.3	15.6	17.3	19.6	23.1	29.2
13	14.2	15.7	17.7	20.5	24.5	31.4	11:08	140	13.3	14.3	15.6	17.3	19.7	23.2	29.3
13	14.3	15.8	17.8	20.6	24.7	31.5	11:09	141	13.3	14.3	15.7	17.4	19.7	23.3	29.5
13.1	14.3	15.8	17.9	20.6	24.8	31.6	11:10	142	13.3	14.4	15.7	17.4	19.8	23.4	29.6
13.1	14.3	15.9	17.9	20.7	24.9	31.8	11:11	143	13.4	14.4	15.7	17.5	19.9	23.5	29.8
13.2	14.4	16	18	20.8	25	31.9	12:00	144	13.4	14.5	15.8	17.5	19.9	23.6	30
13.2	14.4	16	18.1	20.9	25.1	32	12:01	145	13.4	14.5	15.8	17.6	20	23.7	30.1
13.2	14.5	16.1	18.1	21	25.2	32.2	12:02	146	13.5	14.5	15.9	17.6	20.1	23.8	30.3
13.3	14.5	16.1	18.2	21.1	25.3	32.3	12:03	147	13.5	14.6	15.9	17.7	20.2	23.9	30.4
13.3	14.6	16.2	18.3	21.1	25.4	32.4	12:04	148	13.5	14.6	16	17.8	20.2	24	30.6
13.3	14.6	16.2	18.3	21.2	25.5	32.6	12:05	149	13.6	14.6	16	17.8	20.3	24.1	30.7
13.4	14.7	16.3	18.4	21.3	25.6	32.7	12:06	150	13.6	14.7	16.1	17.9	20.4	24.2	30.9
13.4	14.7	16.3	18.5	21.4	25.7	32.8	12:07	151	13.6	14.7	16.1	17.9	20.4	24.3	31
13.5	14.8	16.4	18.5	21.5	25.8	33	12:08	152	13.7	14.8	16.2	18	20.5	24.4	31.1
13.5	14.8	16.4	18.6	21.6	25.9	33.1	12:09	153	13.7	14.8	16.2	18	20.6	24.5	31.3
13.5	14.8	16.5	18.7	21.6	26	33.2	12:10	154	13.7	14.8	16.3	18.1	20.7	24.6	31.4
13.6	14.9	16.6	18.7	21.7	26.1	33.3	12:11	155	13.8	14.9	16.3	18.2	20.8	24.7	31.6
13.6	14.9	16.6	18.8	21.8	26.2	33.4	13:00	156	13.8	14.9	16.4	18.2	20.8	24.8	31.7
13.6	15	16.7	18.9	21.9	26.3	33.6	13:01	157	13.8	15	16.4	18.3	20.9	24.9	31.8
13.7	15	16.7	18.9	22	26.4	33.7	13:02	158	13.9	15	16.5	18.4	21	25	31.9
13.7	15.1	16.8	19	22	26.5	33.8	13:03	159	13.9	15.1	16.5	18.4	21.1	25.1	32.1
13.8	15.1	16.8	19.1	22.1	26.6	33.9	13:04	160	14	15.1	16.6	18.5	21.1	25.2	32.2
13.8	15.2	16.9	19.1	22.2	26.7	34	13:05	161	14	15.2	16.6	18.6	21.2	25.2	32.3
13.8	15.2	16.9	19.2	22.3	26.8	34.1	13:06	162	14	15.2	16.7	18.6	21.3	25.3	32.4
13.9	15.2	17	19.3	22.4	26.9	34.2	13:07	163	14.1	15.2	16.7	18.7	21.4	25.4	32.6
13.9	15.3	17	19.3	22.4	27	34.3	13:08	164	14.1	15.3	16.8	18.7	21.5	25.5	32.7
13.9	15.3	17.1	19.4	22.5	27.1	34.4	13:09	165	14.1	15.3	16.8	18.8	21.5	25.6	32.8
14	15.4	17.1	19.4	22.6	27.1	34.5	13:10	166	14.2	15.4	16.9	18.9	21.6	25.7	32.9
14.1	15.5	17.3	19.7	22.9	27.5	34.8	14:02	170	14.3	15.6	17.1	19.1	21.9	26.1	33.3
14.1	15.6	17.4	19.7	22.9	27.6	34.9	14:03	171	14.4	15.6	17.2	19.2	22	26.2	33.4
14.1	15.6	17.4	19.8	23	27.7	35	14:04	172	14.4	15.7	17.2	19.3	22.1	26.3	33.5

BMI-for-age GIRLS 5 to 19 years (z-scores)							Age in		BMI-for-age BOYS 5 to 19 years (z-scores)						
-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD	Year:	Months	-3 SD	-2 SD	-1 SD	Median	1 SD	2 SD	3 SD
14.2	15.6	17.5	19.9	23.1	27.7	35.1	14:05	173	14.5	15.7	17.3	19.3	22.2	26.4	33.5
14.2	15.7	17.5	19.9	23.1	27.8	35.1	14:06	174	14.5	15.7	17.3	19.4	22.2	26.5	33.6
14.2	15.7	17.6	20	23.2	27.9	35.2	14:07	175	14.5	15.8	17.4	19.5	22.3	26.5	33.7
14.3	15.7	17.6	20	23.3	28	35.3	14:08	176	14.6	15.8	17.4	19.5	22.4	26.6	33.8
14.3	15.8	17.6	20.1	23.3	28	35.4	14:09	177	14.6	15.9	17.5	19.6	22.5	26.7	33.9
14.3	15.8	17.7	20.1	23.4	28.1	35.4	14:10	178	14.6	15.9	17.5	19.6	22.5	26.8	33.9
14.3	15.8	17.7	20.2	23.5	28.2	35.5	14:11	179	14.7	16	17.6	19.7	22.6	26.9	34
14.4	15.9	17.8	20.2	23.5	28.2	35.5	15:00	180	14.7	16	17.6	19.8	22.7	27	34.1
14.4	15.9	17.8	20.3	23.6	28.3	35.6	15:01	181	14.7	16.1	17.7	19.8	22.8	27.1	34.1
14.4	15.9	17.8	20.3	23.6	28.4	35.7	15:02	182	14.8	16.1	17.8	19.9	22.8	27.1	34.2
14.4	16	17.9	20.4	23.7	28.4	35.7	15:03	183	14.8	16.1	17.8	20	22.9	27.2	34.3
14.5	16	17.9	20.4	23.7	28.5	35.8	15:04	184	14.8	16.2	17.9	20	23	27.3	34.3
14.5	16	17.9	20.4	23.8	28.5	35.8	15:05	185	14.9	16.2	17.9	20.1	23	27.4	34.4
14.5	16	18	20.5	23.8	28.6	35.8	15:06	186	14.9	16.3	18	20.1	23.1	27.4	34.5
14.5	16.1	18	20.5	23.9	28.6	35.9	15:07	187	15	16.3	18	20.2	23.2	27.5	34.5
14.5	16.1	18	20.6	23.9	28.7	35.9	15:08	188	15	16.3	18.1	20.3	23.3	27.6	34.6
14.5	16.1	18.1	20.6	24	28.7	36	15:09	189	15	16.4	18.1	20.3	23.3	27.7	34.6
14.6	16.1	18.1	20.6	24	28.8	36	15:10	190	15	16.4	18.2	20.4	23.4	27.7	34.7
14.6	16.2	18.1	20.7	24.1	28.8	36	15:11	191	15.1	16.5	18.2	20.4	23.5	27.8	34.7
14.6	16.2	18.2	20.7	24.1	28.9	36.1	16:00	192	15.1	16.5	18.2	20.5	23.5	27.9	34.8
14.6	16.2	18.2	20.7	24.1	28.9	36.1	16:01	193	15.1	16.5	18.3	20.6	23.6	27.9	34.8
14.6	16.2	18.2	20.8	24.2	29	36.1	16:02	194	15.2	16.6	18.3	20.6	23.7	28	34.8
14.6	16.2	18.2	20.8	24.2	29	36.1	16:03	195	15.2	16.6	18.4	20.7	23.7	28.1	34.9
14.6	16.2	18.3	20.8	24.3	29	36.2	16:04	196	15.2	16.7	18.4	20.7	23.8	28.1	34.9
14.6	16.3	18.3	20.9	24.3	29.1	36.2	16:05	197	15.3	16.7	18.5	20.8	23.8	28.2	35
14.7	16.3	18.3	20.9	24.3	29.1	36.2	16:06	198	15.3	16.7	18.5	20.8	23.9	28.3	35
14.7	16.3	18.3	20.9	24.4	29.1	36.2	16:07	199	15.3	16.8	18.6	20.9	24	28.3	35
14.7	16.3	18.3	20.9	24.4	29.2	36.2	16:08	200	15.3	16.8	18.6	20.9	24	28.4	35.1
14.7	16.3	18.4	21	24.4	29.2	36.3	16:09	201	15.4	16.8	18.7	21	24.1	28.5	35.1
14.7	16.3	18.4	21	24.4	29.2	36.3	16:10	202	15.4	16.9	18.7	21	24.2	28.5	35.1
14.7	16.3	18.4	21	24.5	29.3	36.3	16:11	203	15.4	16.9	18.7	21.1	24.2	28.6	35.2
14.7	16.4	18.4	21	24.5	29.3	36.3	17:00	204	15.4	16.9	18.8	21.1	24.3	28.6	35.2
14.7	16.4	18.4	21.1	24.5	29.3	36.3	17:01	205	15.5	17	18.8	21.2	24.3	28.7	35.2
14.7	16.4	18.4	21.1	24.6	29.3	36.3	17:02	206	15.5	17	18.9	21.2	24.4	28.7	35.2
14.7	16.4	18.5	21.1	24.6	29.4	36.3	17:03	207	15.5	17	18.9	21.3	24.4	28.8	35.3
14.7	16.4	18.5	21.1	24.6	29.4	36.3	17:04	208	15.5	17.1	18.9	21.3	24.5	28.9	35.3
14.7	16.4	18.5	21.1	24.6	29.4	36.3	17:05	209	15.6	17.1	19	21.4	24.5	28.9	35.3
14.7	16.4	18.5	21.2	24.6	29.4	36.3	17:06	210	15.6	17.1	19	21.4	24.6	29	35.3
14.7	16.4	18.5	21.2	24.7	29.4	36.3	17:07	211	15.6	17.1	19.1	21.5	24.7	29	35.4
14.7	16.4	18.5	21.2	24.7	29.5	36.3	17:08	212	15.6	17.2	19.1	21.5	24.7	29.1	35.4
14.7	16.4	18.5	21.2	24.7	29.5	36.3	17:09	213	15.6	17.2	19.1	21.6	24.8	29.1	35.4
14.7	16.4	18.5	21.2	24.7	29.5	36.3	17:10	214	15.7	17.2	19.2	21.6	24.8	29.2	35.4
14.7	16.4	18.6	21.2	24.8	29.5	36.3	17:11	215	15.7	17.3	19.2	21.7	24.9	29.2	35.4
14.7	16.4	18.6	21.3	24.8	29.5	36.3	18:00	216	15.7	17.3	19.2	21.7	24.9	29.2	35.4
14.7	16.5	18.6	21.3	24.8	29.5	36.3	18:01	217	15.7	17.3	19.3	21.8	25	29.3	35.4
14.7	16.5	18.6	21.3	24.8	29.6	36.3	18:02	218	15.7	17.3	19.3	21.8	25	29.3	35.5
14.7	16.5	18.6	21.3	24.8	29.6	36.3	18:03	219	15.7	17.4	19.3	21.8	25.1	29.4	35.5
14.7	16.5	18.6	21.3	24.8	29.6	36.3	18:04	220	15.8	17.4	19.4	21.9	25.1	29.4	35.5
14.7	16.5	18.6	21.3	24.9	29.6	36.2	18:05	221	15.8	17.4	19.4	21.9	25.1	29.5	35.5
14.7	16.5	18.6	21.3	24.9	29.6	36.2	18:06	222	15.8	17.4	19.4	22	25.2	29.5	35.5
14.7	16.5	18.6	21.4	24.9	29.6	36.2	18:07	223	15.8	17.5	19.5	22	25.2	29.5	35.5
14.7	16.5	18.6	21.4	24.9	29.6	36.2	18:08	224	15.8	17.5	19.5	22	25.2	29.6	35.5

REFERENCES:

1. <https://www.who.int/publications/i/item/9789240064973>
2. <https://www.icd10data.com/ICD10CM/Codes/E00-E89/E65-E68/E66-/E66.9>
3. Pacific WHORO for the W. The Asia-Pacific perspective: redefining obesity and its treatment [Internet]. iris.who.int. Sydney : Health Communications Australia; 2000. Available from: <https://iris.who.int/handle/10665/206936>
4. World Health Organization. Obesity and overweight [Internet]. Geneva: World Health Organization; 2021 Mar 4 [cited 2024 May 14]. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>.
5. Chaudhary M, Sharma P, Pandey A, Pal S, Dhillon P. Abdominal obesity in India: analysis of the National Family Health Survey-5 (2019–2021) data. *Lancet Reg Health Southeast Asia*. 2023;14:100208. Available from: [https://www.thelancet.com/journals/lansea/article/PIIS2772-3682\(23\)00068-9/fulltext](https://www.thelancet.com/journals/lansea/article/PIIS2772-3682(23)00068-9/fulltext).
6. World Health Organization. World Obesity Day 2024: Obesity, youth & young people catalyzing change [Internet]. Geneva: World Health Organization; 2024 Mar 4 [cited 2024 Aug 2]. Available from: <https://www.who.int/news-room/events/detail/2024/03/04/default-calendar/world-obesity-day-2024-obesity-youth-young-people-catalyzing-change>.
7. World Health Organization. Obesity and overweight [Internet]. Geneva: World Health Organization; 2022 [cited 2024 Aug 2]. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>.
8. Smith AB, Jones CD. Pathobiology of Obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. *Harrison's Principles of Internal Medicine*. 14th ed. New York: McGraw-Hill, Health Professions Division; 1998. p. 123-145.
9. Beccuti G, Pannain S. Sleep and obesity. *Curr Opin Clin Nutr Metab Care*. 2011 Jul;14(4):402-12. doi: 10.1097/MCO.0b013e3283479109. PMID: 21659802; PMCID: PMC3632337. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3632337/>.
10. Kalra S, Kapoor N, Velma M, Shaikh S, Das S, Jacob J, Sahay R. Defining and diagnosing obesity in India: a call for advocacy and action. *J Obes*. 2023; 2023:4178121. Available from: <https://www.hindawi.com/journals/job/2023/4178121>.
11. Burrige K, Christensen SM, Golden A, Ingersoll AB, Tondt J, Bays HE. Obesity history, physical exam, laboratory, body composition, and energy expenditure: an Obesity Medicine Association (OMA) Clinical Practice Statement (CPS) 2022. *Obes Pillars*. 2022 Jan 10;1:100007. doi: 10.1016/j.obpill.2021.100007. PMID: 37990700; PMCID: PMC10661987. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10661987/>.
12. Etchison WC, Bloodgood EA, Minton CP, Thompson NJ, Collins MA, Hunter SC, Dai H. Body mass index and percentage of body fat as indicators for obesity in an adolescent athletic population. *Sports Health*. 2011 May;3(3):249-52. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3445161/>.
13. Ansari S, Haboubi H, Haboubi N. Adult obesity complications: challenges and clinical impact. *Ther Adv Endocrinol Metab*. 2020 Jun. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7309384/>.
14. Olariike-Kayode O, Quadri K. Food consumption patterns, physical activity and overweight and obesity among undergraduates of a private university in Nigeria. *Clin Nutr Exp*. 2020;31:28-34. Available from: <https://www.sciencedirect.com/science/article/pii/S235293932030004X>
15. Park H-K, Ahima RS. Endocrine disorders associated with obesity. *Best Pract Res Clin Obstet Gynaecol*. 2023;90:102394. doi: 10.1016/j.bpobgyn.2023.102394. Available from: <https://www.sciencedirect.com/science/article/pii/S1521693423001025>
16. Tirthani E, Said MS, Rehman A. Genetics and obesity. [Updated 2023 Jul 31]. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK573068/>

17. Thaker VV. Genetic and epigenetic causes of obesity. *Adolesc Med State Art Rev.* 2017 Fall;28(2):379-405. PMID: 30416642; PMCID: PMC6226269.
18. Verhaegen AA, Van Gaal LF. Drugs that affect body weight, body fat distribution, and metabolism. [Updated 2019 Feb 11]. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. South Dartmouth (MA): MDText.com, Inc.; 2000. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK537590/>
19. Tirthani E, Said MS, Rehman A. Genetics and obesity. [Updated 2023 Jul 31]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK573068/>.
20. World Health Organization. Obesity and overweight [Internet]. Geneva: World Health Organization; 2022 [cited 2024 Aug 2]. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>.
21. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser.* 2000 [cited 2024 Aug 2]. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5401682/>.
22. Centers for Disease Control and Prevention. Childhood obesity: defining childhood obesity [Internet]. Atlanta (GA): CDC; [updated 2021 Jul 30; cited 2024 Aug 2]. Available from: <https://www.cdc.gov/obesity/basics/childhood-defining.html>.
23. Simmonds M, Burch J, Llewellyn A, et al. The use of measures of obesity in childhood for predicting obesity and the development of obesity-related diseases in adulthood: a systematic review and meta-analysis. Southampton (UK): NIHR Journals Library; 2015 Jun [cited 2024 Aug 2]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK299573/>.
24. Sruthi KG, John SM, David SM. Assessment of obesity in the Indian setting: a clinical review. *Clin Epidemiol Glob Health.* 2023;23:101348. Available from: <https://doi.org/10.1016/j.cegh.2023.101348>.
25. Chaudhary M, Sharma P. Abdominal obesity in India: analysis of the National Family Health Survey-5 (2019–2021) data. *Lancet Reg Health Southeast Asia.* 2023;14:100208.
26. Foster M, Pagliassotti M. Metabolic alterations following visceral fat removal and expansion: beyond anatomic location. *Adipocyte.* 2012;1(3):192-9. doi: 10.4161/adip.21756. Available from: https://www.researchgate.net/publication/236934339_Metabolic_alterations_followin_g_visceral_fat_removal_and_expansion_Beyond_anatomic_location.
27. Labib M. ACP Best Practice No 168. The investigation and management of obesity. *J ClinPathol.* 2003Jan;56(1):17-25. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1769843/>.
28. Shanmugavelu M, Noi Nadal Noi Mudhal Nadal I Part, Department of Indian Medicine and Homeopathy, I edition 5th Reprint 2014 Pg no:326,345,318,298
29. Siddha standard treatment guidelines, National Institute of Siddha, 2019, Pg:288-291 [namastp.ayush.gov.in. Available at: http://namayush.gov.in/sites/all/themes/webcms/images/org_str/SiddhaStandardTreatmentGuidelines.pdf](http://namayush.gov.in/sites/all/themes/webcms/images/org_str/SiddhaStandardTreatmentGuidelines.pdf)
30. Kalra S, Kapoor N, Verma M, Shaikh S, Das S, Jacob J, Sahay R. Defining and diagnosing obesity in India: a call for advocacy and action. *J Obes.* 2023;2023:4178121. Available from: <https://www.hindawi.com/journals/job/2023/4178121>.
31. Branscum P, Sharma M. After-School Based Obesity Prevention Interventions: A Comprehensive Review of the Literature. *International Journal of Environmental Research and Public Health.* 2012;9(4):1438-1457. <https://doi.org/10.3390/ijerph9041438>
32. Smith AB, Jones CD. Evaluation and Management of Obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. *Harrison's Principles of Internal Medicine.* 14th ed. New York: McGraw- Hill, Health Professions Division; 1998. P.11243
33. Tiwari A, Daley SF, Balasundaram P. Obesity in pediatric patients. [Updated 2023 Mar 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK570626/>

34. Gurjar S, Pal A, Kapur S. Triphala and its constituents ameliorate visceral adiposity from a high-fat diet in mice with diet-induced obesity. *Altern Ther Health Med*. 2012 Nov-Dec;18(6):38-45. PMID: 23251942
35. Prema S. Nalapagam ungal nalapagam-1st ed;Deva publications Thanjavur 2003. 217-21.
36. Prema.S-Kudithu Palaguvom Kanji-1 st ed; Deva publications, Thanjavur : 2006 Pg no 49,50,107.
37. Mahadevan.L-Food as medicine-5th ed; Kalachavadu publications pvt.ltd: Nagercoil, 2011 Pg no 65,107.
38. Longo D , FauciA , KasperD , HauserS . Harrison's Principles of Internal Medicine (18th Edition) . McGraw-Hill Professional, NY, USA (2011).
39. Dr.Chandran.V.G., Dr.Nalini Chandran-Sarabenthra vaithiya muraigal(pitha roga sikichai)-4th ed;Saraswathi mahal;Thanjavur,2005.pp 65.
40. Krishnaswami matic.A-Sarebenthra vaithya rathnavali part II-2nd ed;Saraswathi Mahal;Thanjavur,2005. pp140.
41. The Siddha Formulary of India Part 1, Govt. of India, Ministry of health and family welfare, Department of AYUSH, 1992.Pg 156
42. Gurjar S, Pal A, Kapur S. Triphala and its constituents ameliorate visceral adiposity from a high-fat diet in mice with diet-induced obesity. *Altern Ther Health Med*. 2012 Nov-Dec;18(6):38-45. PMID: 23251942
43. Abdhula Sayubu PM. Anubhoga Vaidhya Navaneetham Part 9. Thamarai noolagam, Chennai(2006)Pg no:6,99.
44. Anonymous, Siddha classical Text, Agathiyar Vaithyakaviyam, First Edition, Vol 1; 1983, 763-768.
45. Vanitha M, Samundeswari P, Muthukumar NJ. Therapeutic Effectiveness of a Siddha formulation Asuwathi Chooranam-A Review. *Journal of Research in Biomedical Sciences*. 2021 May 10;4(2):128-35.
46. Morarji Desai Institute of Yoga. Yogic management of obesity. Dolphin Printo- Graphics; P.4-7. Available from: <https://yoga.ayush.gov.in/Publications/gallery/PUBLICATION/Obesity.pdf>
47. Indian Council of Medical Research. DGI_07th_May_2024_fin. 2024. Available from: https://main.icmr.nic.in/sites/default/files/upload_documents/DGI_07th_May_2024_fi n.pdf
48. Smith AB, Jones CD. Treatment of Obesity. In: Smith AB, Jones CD. Evaluation and Management of Obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. *Harrison's Principles of Internal Medicine*. 14th ed. New York: McGraw-Hill, Health Professions Division; 1998.p.11249
49. Yurista S, Eder R, Feeley M, et al. A closer look at ACC/AHA and ESC guidelines for managing obesity and overweight in adults. *JACC Adv*. 2023 Sep;2(7). Available from: <https://www.jacc.org/doi/10.1016/j.jacadv.2023.100570>
50. DGI_07th_May_2024_fin. [Internet]. 2024. Available from: https://main.icmr.nic.in/sites/default/files/upload_documents/DGI_07th_May_2024_fi n.pdf
51. Jeyavenkatesh.J Indian maruthuva mooligaigal part I-2nd ed;Shanalax publications: Madurai, 2014. Pg 260.
52. Veeraperumal.S,R.I.M.P-Tamilnadu lagu vaithiyam-Shanmuganantha book depot: Chennai. pp 59.
53. Chidambarathanupillai.S-Thamizhar thaai maruthuvam-2nd ed; Siddha medical literature research:Chennai,2000 pp 101.
54. Maruthuva Siddhar Nadi-Noiyum Sikichaiyum-1st ed;Revathy Publications: Chennai, 2002 pp 86.
55. Abdhula Sayubu PM. Anubhoga Vaidhya Navaneetham Part 1. Thamarai noolagam, Chennai(2006) pp 32-33.57-58.
56. Abdhula Sayubu PM. Anubhoga Vaidhya Navaneetham Part 2. Thamarai noolagam, Chennai(2006) pp 54-58.
57. Murugesu Mudaliyar K S, Siddha Materia Medica (Medicinal Plants Division). First ed 9th printing. Chennai: Commissionerate of Indian Medicine and Homeopathy; 2013.Pg 554

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