



सत्यमेव जयते

Ministry of Ayush
Government of India

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

AYUSH VERTICAL
DIRECTORATE GENERAL OF HEALTH SERVICES
Government of India



सत्यमेव जयते

Ministry of Ayush
Government of India

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

**AYUSH VERTICAL
DIRECTORATE GENERAL OF HEALTH SERVICES
Government of India**

STANDARD TREATMENT GUIDELINES
ON
MANAGEMENT OF
METABOLIC DISORDERS
IN
AYURVEDA SYSTEM OF MEDICINE

© **Ayush Vertical, Directorate General of Health Services**
April, 2025

ISBN: 978-81-974231-8-5

Publisher:
Ayush Vertical, Directorate General of Health Services, New Delhi
April, 2025

Disclaimer: All possible efforts have been made to ensure the correctness of the contents. However, the Ministry of Ayush shall not be accountable for any inadvertent errors in the content. Corrective measures shall be taken up once such errors are brought into notice.

राज्य मंत्री (स्वतंत्र प्रभार)
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



देश का प्रकृति धर्म अभियान
॥ आयुष्यं शरीरं वा, अथवा आयुष्यं वा ॥



स्वतः और समाज के लिए योग
Yoga for self and society

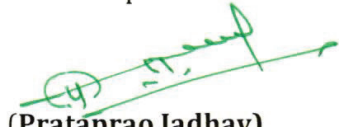
India has a rich legacy of traditional healthcare systems that offer time-tested approaches to health and well-being. In recent years, there has been a growing recognition of the role Ayush can play in addressing contemporary health challenges through holistic approach.

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

Room No. : 101, Ayush Bhawan, 'B' Block, GPO Complex, INA, New Delhi-110023

Tel : 011-24651955, 011-24651935 E-mail : minister-ayush@nic.in

Room No. 250-A, Nirman Bhawan, Maulana Azad Road, New Delhi-110011, Tel. : 011-23061551, 23061016

Residence : 23, Ashoka Road, New Delhi-110001, Tel.: 011-23740412, 23345478, Fax : 011-23740413

वैद्य राजेश कोटेचा
सचिव
Vaidya Rajesh Kotecha
Secretary



भारत सरकार
आयुष मंत्रालय
आयुष भवन, 'बी' ब्लॉक, जी.पी.ओ. कॉम्प्लेक्स,
आई.एन.ए. नई दिल्ली-110023
Government of India
Ministry of Ayush
Ayush Bhawan, B-Block, GPO Complex,
INA, New Delhi-110023
Tel. : 011-24651950, Fax : 011-24651937
E-mail : secy-ayush@nic.in

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

Prof. (Dr.) Atul Goel

MD (Med.)

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)

CONSULTANT EDITOR

PROF. (DR.) ATUL GOEL

Director General, Directorate General
of Health Services

EXECUTIVE EDITORS

VAIDYA RABINARAYAN ACHARYA

Director General
Central Council for Research in Ayurvedic Sciences

DR. A. RAGHU

Deputy Director General (Ayush)
Ayush Vertical, DGHS

EDITORIAL REVIEW BOARD

Dr. Ramesh Agarwal

Professor, Department of Medicine
Lady Hardinge Medical College, New Delhi

Dr. Anupam Prakash

Professor, Department of Medicine
Lady Hardinge Medical College, New Delhi

Dr. M.R.V Nampoothiri

Retd. Director, Directorate of Medical Education, Kerala

Dr. Mukesh E

Professor, Dept. of Roganidana, VPSV Ayurveda College, Kottakkal, Kerala

NATIONAL EXPERT COMMITTEE FOR STANDARD TREATMENT GUIDELINES

S. No.	Expert Name	Designation and Institution
1.	Prof. (Dr.) Atul Goel	Director General, DGHS, MoHFW
2.	Prof. Rabinarayan Acharya	Director General, CCRAS, New Delhi
3.	Dr. M.R.V Nampoothiri	Retd. Director, Directorate of Medical Education, Kerala
4.	Dr. Mukesh E	Professor, Dept. of Roganidana, VPSV Ayurveda College, Kottakkal, Kerala
5.	Dr. Subhash Kaushik	Director General, CCRH, New Delhi
6.	Dr. Radha Das	Former Advisor (Homoeopathy), Ministry of Ayush
7.	Dr. Girish Gupta	Homoeopathy consultant, Chairman, Scientific Advisory Committee, Central Research Institute for Homoeopathy, Lucknow, Uttar Pradesh
8.	Dr. N. Zaheer Ahmed	Director General, CCRUM, New Delhi
9.	Prof. Mohd Anwar	Deptt of Ilaj bit Tadbeer, AKTC. AMU, Aligarh
10.	S.M. Arif Zaidi	Former Dean SUMER, Jamia Hamdard & M.S. Majeedia Hospital (Unani), Jamia Hamdard
11.	Prof. Saiyad Shah Alam	Director, NIUM, Bangalore
12.	Dr. N.J Muthukumar	Director General, CCRS, Chennai
13.	Prof. Meena Kumari	Former Director, NIS, Chennai
14.	Dr. A. Rajendra Kumar	Research officer (Siddha), S-III, CCRS, Hqrs., Chennai.
15.	Dr. G. Sivaraman	Managing Director, Arogya Health Care Pvt. Ltd., & Member, Tamil Nadu Planning Commission, Govt. of Tamil Nadu
16.	Dr. Subhash Singh	Director, NIH, Kolkatta
17.	Dr. Kousthubha Upadhayaya	Advisor (Ayurveda) Ministry of Ayush, New Delhi
18.	Dr. Sangeeta A. Duggal	Advisor (Homoeopathy) Ministry of Ayush, New Delhi
19.	Dr. M.A. Qasmi	Adviser (Unani), Ministry of Ayush, New Delhi
20.	Dr. A. Raghu	DDG-Ayush, DGHS
21.	Dr. Ghazala Javed	Assistant Director, CCRUM
22.	Dr. Varanasi Roja	Research Officer, CCRH
23.	Dr. Vishal Chadha	Medical Officer Govt. of NCT of Delhi
24.	Dr. Pavana M	Medical officer (Panchakarma) Govt. Ayurveda Panchakarma Hospital Alappuzha, Kerala
25.	Dr. Roshni Rajan	Domain Expert(Ayurveda) Ayush vertical, DGHS, MoHFW
26.	Dr. Rahul Singh	SRF(Ayurveda) Ayush vertical, DGHS, MoHFW

SUBJECT EXPERTS/ CONTRIBUTORS

- Dr. N. Srikanth, Deputy Director General
Central Council for Research in Ayurvedic Sciences, New Delhi
- Dr. BCS Rao, Assistant Director
Central Council for Research in Ayurvedic Sciences, New Delhi
- Dr. Shruti Khanduri, Research Officer (Ayurveda)
Central Council for Research in Ayurvedic Sciences, New Delhi
- Dr. Sulochana Bhatt, Assistant Director
Central Ayurveda Research Institute, Bengaluru.
- Dr Raghavendra Naik, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bengaluru.
- Dr. M.N. Shubhashree, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bengaluru.
- Dr. Thejaswini C, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bengaluru.
- Dr. Bhavya B.M, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bengaluru.
- Dr. Rajendra Kumar, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bengaluru.
- Dr. V G Huddar Additional Professor
Kaya Chikitsa AIIA, New Delhi
- Dr. Rakesh Narayanan V, Research Officer (Ayurveda)
Central Council for Research in Ayurvedic Sciences, New Delhi
- Dr. Neha Dubey, Research Officer (Ayurveda)
Central Council for Research in Ayurvedic Sciences, New Delhi
- Dr. Banamali Das, Research Officer (Ayurveda)
Central Ayurveda Research Institute, Bhubaneswar

TABLE OF CONTENTS

S.No.	Chapters	Page No.
I	Abbreviations	ii
II	Glossary	v
1.	Diabetes Mellitus	1
2.	Dyslipidemia	25
3.	Gout	43
4.	Non Alcoholic Fatty Liver Disease	63
5.	Obesity	81

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	Three times a day
TG	Triglyceride
TSH	Thyroid stimulating hormone level (TSH).
USG	US: ultrasonography / Ultrasonography (USG)
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

S. No.	Term	Description
1.	<i>Abhyanga</i>	Procedure to induce oleation carried out by external application of an unctuous substance for therapeutic purposes and healthy well-being.
2.	<i>Adhithana</i>	<i>Adhithana</i> refers to the foundation of a disease. It is the specific location or site within the body where a disease originates or manifests.
3.	<i>Agni</i>	<i>Agni</i> is broadly referred to the factor responsible for digestive function of the body, which also denotes the metabolism at cellular level and is also concerned with body temperature. Its function is the transformation and absorption of ingested food into energy.
4.	<i>Agnideepana</i>	Stimulation of digestive fire
5.	<i>Agnikarma</i>	Thermal Microcautery
6.	<i>Agnimandya/ Agnivikriti</i>	Decreased/ Deranged digestive power
7.	<i>Ajirna/ Ajeerna</i>	Indigestion, weak digestion
8.	<i>Ama dosha</i>	<i>Ama</i> refers to toxic, undigested, or improperly metabolized substances that accumulate in the body due to poor digestive and metabolic process.
9.	<i>Amapachana</i>	<i>Amapachana</i> refers to the process of digesting and eliminating <i>Ama</i>
10.	<i>Angasya Vakrikarana</i>	Disfigurement of the joints.
11.	<i>Anupa Mamsa</i>	Meat of aquatic animals
12.	<i>Anutsaha</i>	Lack of enthusiasm
13.	<i>Apatyanimittaja</i>	Conditions caused by Indulgence in incompatible food habits and unrecommended daily activities
14.	<i>Apya</i>	Water
15.	<i>Arshas</i>	Haemorrhoids
16.	<i>Asana Sukha</i>	Sedentary habits
17.	<i>Avaranajanya</i>	Conditions caused by obstruction of <i>Vata</i>
18.	<i>Bahubaddha Medas</i>	Inconsistency in adipose tissue and muscular tissue leading to increase or their volume
19.	<i>Basti</i>	Therapeutic procedure of giving medication per rectum through specially designed basti instrument
20.	<i>Basti</i>	Urinary bladder
21.	<i>Beejadosha</i>	Genetic factors
22.	<i>BhojanottaraJalapana</i>	Intake of water after food
23.	<i>Bhojanottara Snana</i>	Bath after intake of food
24.	<i>Bijadosha</i>	Genetic predisposition to disease
25.	<i>Brimhan</i>	Nourishment

S. No.	Term	Description
26.	<i>Chimchimayata</i>	Tingling and burning sensations
27.	<i>Churna</i>	Powder
28.	<i>Deepana pachana</i>	Process by which the metabolic fire is enhanced
29.	<i>Dhamani</i>	<i>Dhamani</i> refers to structures similar to arteries in modern anatomy, which are responsible for carrying <i>rasa</i> (nutrient fluid) and <i>Rakta</i> throughout the body. <i>Dhamanis</i> are described as having pulsations and are essential for the nourishment and functioning of the body
30.	<i>Dhatu</i>	<i>Dhatu</i> refers to the seven fundamental tissues in the body that form the basis of its structure and function. These seven tissues of our body includes the <i>rasa</i> , <i>Rakta</i> , <i>Mamsa</i> , <i>Meda</i> , <i>Asthi</i> , <i>Majja</i> and <i>Shukra</i> .
31.	<i>Dhatu Parinama</i>	Process of formation of <i>Dhatu</i>
32.	<i>Dhatukshayajanya</i>	Conditions caused by depletion of major structural body components
33.	<i>Dhatvagni Paka</i>	<i>Dhatvagni Paka</i> refers to the process of digestion and metabolism at the tissue level. The term “ <i>Dhatvagni</i> ” combines “ <i>Dhatu</i> ” (tissues) and “ <i>Agni</i> ” (digestive fire), indicating the specific metabolic processes that occur within each of the seven tissues (<i>Dhatus</i>) in the body
34.	<i>Dhatwagni</i>	Digestive power
35.	<i>Divaswapana</i>	Sleeping in day time
36.	<i>Dosha</i>	Regulatory functional factors of the body
37.	<i>Dushti</i>	<i>Dushti</i> refers to the vitiation of the body’s <i>doshas</i> , <i>dhatu</i> s, and <i>srotas</i> . This vitiation leads to the development of diseases and imbalances within the body
38.	<i>Dushya</i>	Bodily elements affected by the <i>dosha</i> and leading to disease
39.	<i>Dvidoshaja</i>	Vitiation of two <i>doshas</i> (<i>vata-pitta</i> , <i>pitta-kapha</i> or <i>vata-kapha</i>).
40.	<i>Eka doshaja</i>	Vitiation of only one <i>dosha</i> (<i>vata</i> , <i>pitta</i> or <i>kapha</i>).
41.	<i>Gambhir Vatarakta</i>	It is a stage which involves the <i>Gambhira</i> (deep seated) <i>Dhatu</i> such as <i>Meda</i> (adipose tissue), <i>Asthi</i> (bone) and <i>Majja</i> (bone marrow).
42.	<i>Godhuma</i>	Wheat
43.	<i>Guggulu</i>	Tablet containing resin of <i>Commiphora wightii</i> as main content
44.	Hyperglycemia	High blood sugar
45.	Impaired Fasting Glucose (IFG)	Condition where fasting blood glucose is elevated above normal levels but not high enough to be classified as diabetes
46.	Impaired Glucose Tolerance (IGT):	Condition where blood glucose levels are higher than normal after eating, but not high enough to be classified as diabetes
47.	<i>Kamala</i>	It is a Liver disorder, which refers to a condition similar to jaundice.
48.	<i>Kapha dusti</i>	Vitiation of <i>Kaphadosha</i>

S. No.	Term	Description
49.	<i>Kapha-Meda nashaka</i>	<i>Kapha-Meda Nashaka</i> refers to substances or practices that help reduce excess <i>Kaphadosha</i> and <i>Meda</i> (fat) in the body.
50.	<i>Karshana</i>	Depletion
51.	<i>Kleda</i>	Water metabolites of the body
52.	<i>KoshtaShuddhi</i>	Clearness of stomach and bowels, as after copious evacuations
53.	<i>Koshthanga</i>	<i>Koshthanga</i> refers to the internal organs of the body. Essentially, <i>Koshthanga</i> includes the organs housed within the body's cavities, such as the stomach, liver, spleen, intestines, and other vital organs
54.	<i>Kwatha</i>	Hot decoction
55.	<i>Lekhana</i>	Therapeutic action which reduces or scrapes away unwanted substances out from the body
56.	<i>Madhumeha/ Kshaudrameha</i>	Types of <i>Prameha</i> characterized by abnormal excess glucose in urine and other variations
57.	<i>Madhura Mutra Pravritti</i>	Increased glucose content in urine
58.	<i>Madhura Rasa Sevana</i>	Intake of sweet substances
59.	<i>Madhura Snigdha Basti</i>	Enema with sweet unctuous drugs
60.	<i>Madya Sevana</i>	Intake of alcohol
61.	<i>Mamsa</i>	Muscle tissue
62.	<i>Mandagni</i>	Depressed/ transforming metabolic entity
63.	<i>Margavarodha</i>	Obstruction
64.	<i>Masha</i>	Blackgram, Urad dhal
65.	<i>Meda</i>	Fat tissue
66.	<i>Medodusti</i>	Derangement of function of <i>medodhatu</i>
67.	<i>Medoroga</i>	Excessive accumulation of <i>medodhatu</i>
68.	<i>Medovahasrotasa</i>	Channel to carry <i>meda dhatu</i>
69.	<i>Medovahasrotoviddha</i>	Derangement of function of <i>medovahasrotasa</i>
70.	<i>Medovruddhi</i>	Aggravation of <i>Meda</i>
71.	<i>Mithya Ahara Vihara</i>	Faulty Food habits and regimen
72.	<i>Mōhaḥ</i>	Confusion/delirium
73.	<i>Mulasthanā</i>	Place of origin/source/ <i>Utpattisthana</i>
74.	<i>Mutra Pariksha</i>	Urine examination
75.	<i>Nasya Karma</i>	Nasal medication therapy
76.	<i>Nidana</i>	Cause of the disease
77.	<i>Nidana Parivarjana</i>	<i>Nidana Parivarjana</i> refers to the practice of avoiding or eliminating the causative factors of a disease.
78.	<i>Nidanarthakara Rogas</i>	Diseases which cause another diseases

S. No.	Term	Description
79.	<i>Panchakarma</i>	It consists of five therapeutic procedures of body purification and detoxification namely <i>Vamana</i> (emesis), <i>Virechana</i> (purgation), <i>Basti</i> (enema), <i>Nasya</i> (nasal therapy), and <i>Raktamoksha</i> (blood letting). used to clean the body of toxic materials left by disease and poor nutrition
80.	<i>Pandu</i>	<i>Pandu</i> refers to a condition similar to anemia in conventional system of medicine. It is characterized by pallor (paleness) of the skin, mucous membranes and nail bed and other symptoms related to a deficiency in blood and its components.
81.	<i>Parshva</i>	Fatty deposition in flanks
82.	<i>Pathya</i>	The dietary and behavioural practices helpful in treating the disease or maintaining health
83.	<i>Prabhoota Mutrata</i>	Excessive micturition
84.	<i>Prajnaparadha</i>	Intellectual Blemish
85.	<i>Prameha</i>	A group of disorder due to altered renal output basically characterised by frequent, excess and turbid urine.
86.	<i>Rasa- Rakta prasadana</i>	<i>Rasa Rakta Prasadana</i> refers to the purification and nourishment of the blood (<i>Rakta</i>) and plasma (<i>Rasa</i>). This concept emphasizes maintaining the health and balance of these vital fluids to ensure overall well-being.
87.	<i>Sāda</i>	Exhaustion or tiredness of body
88.	<i>Sahaja Prameha</i>	Conditions caused by genetic factors
89.	<i>Samprapti</i>	Pathogenesis of the disease
90.	<i>Samprapti Nirdharan</i>	Determining the pathogenesis of a disease
91.	<i>Samsarjana</i>	Dietary regimen for restoration of <i>agni</i> after <i>shodhana procedure</i>
92.	<i>Sandhi</i>	Joints of body
93.	<i>Sandhi-Asthi-Majja Chinndni</i>	Cutting like pain in <i>Sandhi-Asthi-Majja</i>
94.	<i>Sannikrishta Karana</i>	<i>Sannikrishta Karana</i> refers to the proximal or immediate causes of a disease. These are the factors that directly lead to the aggravation of the <i>doshas</i> (<i>Vata</i> , <i>Pitta</i> , and <i>Kapha</i>) and the manifestation of disease.
95.	<i>Sannipataja</i>	Simultaneous vitiation of all the three <i>doshas</i>
96.	<i>Santarpanajanyavyadhi/ Santarpanotthavikara</i>	Refers to diseases caused by over-nourishment or excessive intake of food and nutrients.
97.	<i>Shariraja Kleda Dushti</i>	Vitiation of water metabolites
98.	<i>Shirodhara</i>	It is a classical Ayurvedic therapeutic procedure of slowly and steadily streaming of medicated oil or other liquids (decoction/ milk/buttermilk) on the forehead.
99.	<i>Shothahara</i>	Refers to substances or treatments that reduce inflammation and swelling (<i>Shotha</i>).
100.	<i>Shukra</i>	Semen

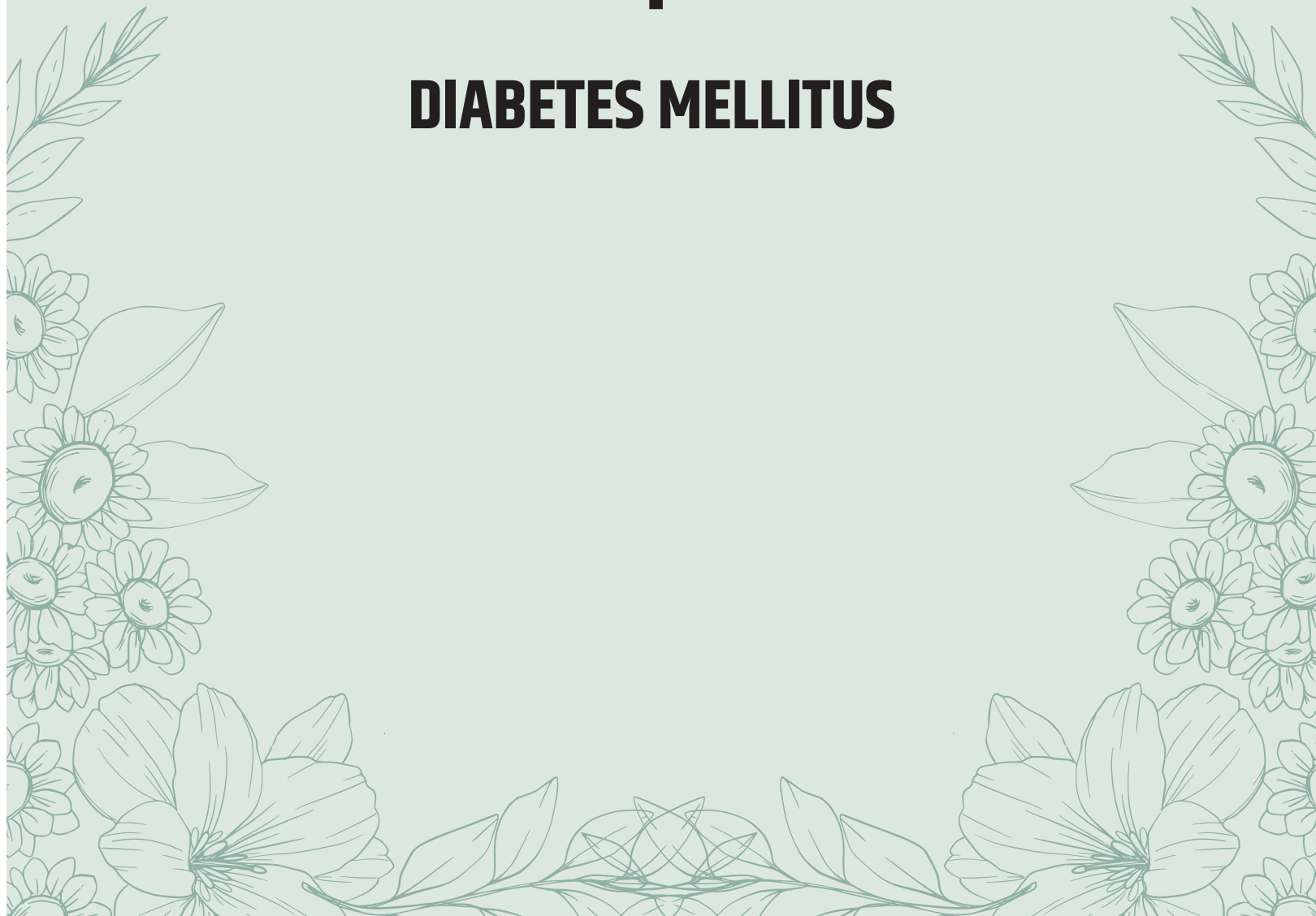
S. No.	Term	Description
101.	<i>Shwayathu</i>	Marked swelling of the skin
102.	<i>Sira Ayama</i>	Dilatation of the vessels
103.	<i>Snehapana</i>	Administration of appropriate dose of ghee, oil or any other unctuous substance internally for achieving oleation.
104.	<i>Snigdhangata</i>	Oiliness of the body
105.	<i>Sphika, Udara and Stana</i>	Buttocks, abdomen and breast
106.	<i>Sphikalambanam</i>	Pendulous fatty enlargement of hips
107.	<i>Sphuranam</i>	Throbbing sensation
108.	<i>Srotas</i>	<i>Srotas</i> are channels or pathways in the body responsible for the transportation of various substances, including nutrients, waste products, and energy.
109.	<i>Srotas Pariksha</i>	Examination of body channels
110.	<i>Srotorodha</i>	Obstruction of the <i>srotas</i>
111.	<i>Srotoshodhana</i>	Refers to the purification and cleansing of the body's channels or pathways
112.	<i>Sthoulya Roga</i>	<i>Sthoulya Roga</i> refers to or excessive accumulation of body fat. It is also known as <i>Medo Roga</i> due to the involvement of <i>Meda Dhatu</i> (fat tissue) in its pathology.
113.	<i>Svedana</i>	Procedure adopted to induce sweating
114.	<i>Tailabhyanga</i>	Oil massage
115.	<i>Tridoshas</i>	Three regulatory functional factors (<i>Vata</i> , <i>Pitta</i> and <i>Kapha</i>) of the body
116.	<i>Ubhayashrita Vatarakta</i>	Presence of symptoms of both <i>Uttana</i> and <i>Gambhira</i> stages of <i>Vatarakta</i>
117.	<i>Udaravruddhi</i>	Pendulous fatty enlargement of abdomen
118.	<i>Udbhava sthana</i>	Refers to the site of origin of a disease.
119.	<i>Udvartana</i>	<i>It is an Ayurvedic therapeutic procedure involving the application/massage of herbal powders or pastes (Dry or oily) to the body.</i>
120.	<i>Uttana Vatarakta</i>	It is a stage which involves the <i>Uttana</i> (superficial) <i>dhatu</i> such as <i>Rasa</i> (tvak), <i>Rakta</i> (blood) and <i>Mamsa</i> (Muscle tissue).
121.	<i>Vatagati</i>	Functional element that control motility and coordination in the body
122.	<i>Vataja Prameha</i>	A type of <i>Prameha</i> caused by vitiation of <i>Vata dosha</i>
123.	<i>Vati</i>	Tablet
124.	<i>Vibandha</i>	Constipation
125.	<i>Virechana</i>	<i>Virechana</i> is a therapeutic purgation process used to cleanse the body of toxins, particularly targeting the expulsion of excess <i>Pittadosha</i> . It is one of the five main procedures in <i>Panchakarma</i> , the comprehensive detoxification and rejuvenation therapy in <i>Ayurveda</i>
126.	<i>Vridhicha Keshanam</i>	Excessive growth of hair
127.	<i>Yakrit</i>	Liver



CHAPTER

1

DIABETES MELLITUS



DIABETES MELLITUS

ICD-11: 5A11

ICD-11 TM2: SP60

ICD-10: E11 to E11.9

Madhumeha/kshaudrameha (National Ayurveda Morbidity code: EF -2.4.4)

CASE DEFINITION

Diabetes Mellitus is a chronic disorder resulting from aberrations in insulin secretion, insulin action, or both. The persistent hyperglycemic state in this condition leads to long term damage, dysfunction, and failure of various organs. Type 2 Diabetes Mellitus, previously referred to as non-insulin-dependent diabetes, accounts for approximately 90 – 95% of all diabetes cases. This condition, also known as adult-onset diabetes, arises due to insulin resistance and relative insulin deficiency.^[1,2]

Definition: *Prameha*³

प्रमेह (*Prameha*) : *Prameha* Roga is described as a condition involving abnormal changes in the three *Doshas* (*Kapha*, *Pitta* and *Vata*) and *Dushyas* like *Medas* (fat tissue), *Rakta* (blood tissue), *Shukra* (semen), *Ambu* (body fluid), *Vasa* (muscle fat), *Lasika* (lymph), *Majja* (bone marrow tissue), *Rasa* (essence of food), *Ojas* (essence of seven *dhatu*s) and *Mamsa* (muscle tissue). *Prameha* is considered as one among the eight *Maharogas* (disorders with serious consequences) and is categorized into twenty types. It is characterized by *Prabhuta Mutrata* (excessive urination i.e. frequent urination with increased quantity) and *Aavila Mutrata* (turbidity in urine). These twenty sub-types of *Prameha* are differentiated based on *Mutra pariksha* (Urine examination), *purvarupa* (prodromal symptoms) and specific *lakshana* (clinical features).

Madhumeha is classified as a *Vataja Prameha*, whereas Diabetes Mellitus predominantly presents with *Kaphaja Prameha* characteristics. The end stage of any type of *Prameha*, if not managed properly, leads to the *Madhumeha* stage. Additionally, any type of *Prameha* that presents with complications can be referred to as *Madhumeha*.

मधुमेहः/क्षौद्रमेह *Madhumeha/ Kshaudrameha*: is one of the *Vataja Prameha*, characterized by excessive passage of *kashaya mutra* (astringent urine), *madhura mutra* (urine excess glucose), *rooksha mutra* (non-unctuous urine), *paandu mutra* (pale urine), *madhusamam mutra* (urine resembling honey), *kshaudra rasa tulya mutra* (urine resembling honey water) and *kshaudra varna tulya mutra* (honey coloured urine)⁴ with features of *purvarupa* (prodromal symptoms) and specific premonitory symptoms of *Madhumeha*.

INTRODUCTION (incidence/prevalence,mortality/morbidity)

- 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^[5,6].
- 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^[5].

- 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)^[7].
- 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^[5].
- 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^[5].
- 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^[8].
- The ICMR-INDIAB survey conducted reported 26.6% of Indians above 20 years having dysglycemia with 11.4% suffering from diabetes and 15.3% suffering from a pre-diabetic state^[9].
- 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 led to a significant rise in new-onset of diabetes mellitus across all age groups, particularly during the post-acute phase of the disease^[10]. Studies indicate a 14.4% increase in new-onset of diabetes mellitus, including T2DM among the hospitalized patients^[11].

Aetiology and risk factors as per Ayurveda:

Madhumeha is a type of *vatajaprameha*, which occurs either independently due to *Sahaja prameha* (genetic cause) or due to *Apatyanimitta japrameha* (improper management of other 19 types of *prameha*) and is difficult to manage or incurable.⁶⁻⁹ There are specific causative factors responsible for the pathogenesis of different types of *Prameha* i.e. *Kaphaja*, *Pittaja* and *Vataja Prameha*.

Nidana for Madhumeha has been described separately in the classical literatures, and can be broadly classified into two categories:

- Based on Aetiological factor:
 1. Sahaja (inherent predisposition)
 2. Apatyanimittaja (Indulgence in incompatible food habits and unrecommended daily activities)
 3. Vikaravighata bhava abhava (destruction of the bodily components related to the carbohydrate metabolism. This includes all conditions that destructs pancreatic cells and also the antibodies targeting the organ)
- Based on Pathogenesis:
 1. आवरणजन्यवातप्रकोपजमधुमेह(दोषावृतपथ) (Avaranajanya Vataprakopaja Madhumeha-due to obstructive pathogenesis)
 2. धातुक्षयजन्यवातप्रकोपजमधुमेह (Dhatukshayajanya Vataprakopaja Madhumeha-due to depletion of dhatus)

Samprapti/Pathogenesis:

Due to excess intake of food that are *guru* (heavy for digestion), *snigdha* (unctuous/slimy), *amla* (sour) and *lavana* (salty); *navannapana* (intake of newly harvested grains); over indulgence in the pleasure of *nidraasyasukhani* (sedentary lifestyle and eating habits), *tyaktavyayama* (lack of exercise), *chinta* (excess of mental stress or grief), *sanshodhanamakurvata* (not undergoing purificatory therapies),¹³ there will be alleviation of *tridoshas* leading to the manifestation of two broad classifications as *Avaranajanya/ Apatyanimittaja* (obstructive/ Indulgence in incompatible food habits and unrecommended daily activities) and *Dhatukshayajanya/ Sahaja prameha* (factors responsible for the depletion of the body tissues/due to genetic factors). In *Avaranajanya* and *Apatyanimittaja Madhumeha*, the vitiated *Kapha Dosha* (*dosha* responsible for regulating body fluids and keeping the body constituents cohesive) and *pitta dosha* (*dosha* responsible for regulating body temperature and metabolic activities), in the presence of *mandagni* (depressed metabolic activity), cause *malarupivruddhi* of *medas* (abnormal increase in the fat tissue) and *mamsa* (muscle tissue). This leads to *sharirajakledadushti* (vitiation of the water metabolites of body), which obstructs the passage of *vata dosha* (*dosha* responsible for movement and cognition) and carries *Oja* to *Basti* (Urinary Bladder) and manifests as *madhumeha* which will be of *sthula* type (obese). *Dhatukshayajanya Madhumeha* causes aggravation of *vata* and manifests as *krisha* type (thin and asthenic individual) due to loss of *Oja* (essence of all seven *dhātus*).¹²⁻¹⁵

CLINICAL PRESENTATION

The presentation of T2DM varies widely. In majority of the cases, it is discovered incidentally during routine blood tests, pre-surgical check-ups, dental visits, or other medical procedures. The classical presentation of T2DM like polyuria, polydipsia, and fatigue are commonly observed in older individuals. Recurrent bacterial and fungal infections, blurred vision, and delayed wound healing are also frequently seen. 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, or cerebrovascular disease or microvascular complications like diabetic nephropathy, retinopathy, Neuropathy or diabetic foot ulcers. In recent years, T2DM has been linked to emerging complications like 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. In severe cases, especially in older individuals, hyperosmolar coma is observed especially during medications for major events like myocardial infarction and stroke.

CLINICAL EXAMINATION

The assessment of a patient with Type 2 Diabetes Mellitus shall first involve the diagnosis and confirmation of the type of diabetes through blood glucose levels and HbA1C evaluation. Further assessment includes the evaluation of the diabetes-related complications, presence of co-morbidities, and overall health status. The clinician must also consider behavioural factors (eating pattern, calorie counting, physical activities, sleep behaviour, addictions), medications and vaccinations, technology use, and social life. A comprehensive physical examination of the patient must be conducted with special emphasis on fundoscopic examination, skin & foot examination, cognitive function assessment, mental state examination, and bone health assessment.

DIFFERENTIAL DIAGNOSIS

Table 1:

Condition	Differential features
Type 1 Diabetes Mellitus ^[16]	• 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]	• 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]	• 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 ^[17]	• 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 ^[8]	• Develops due to side effects of glucocorticoids used as anti-inflammatory or immunosuppressive purposes • Mostly observed with oral and injected glucocorticoids
Acromegaly ^[19]	• 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	• Circulating glucocorticoids results in increased glucose levels in the blood. • Cortisol levels after dexamethasone suppression test aids in the diagnosis

Complications: ²⁰

Most of the morbidity and mortality associated with diabetes arises due to its complications, which can be broadly classified into acute and chronic complications. Acute complications include hypoglycemic and hyperglycemic emergencies whereas chronic complications include microvascular disease (retinopathy, nephropathy and neuropathy), macrovascular disease (coronary artery disease, cerebrovascular disease and peripheral vascular disease) and diabetic foot.

Prognosis/Sadhya – Asadhya Vivechana:^{21,22}

¹*Madhumeha*, which is a type of *vataja prameha*, if seen in *krishavan* (lean built), and that which has occurred either independently due to *Sahaja karana*(inherited/ genetic cause) or if associated with *upadravas* (complications) is considered as *Asadhya* (incurable) for the management. It is also incurable, when there is *dhatu kshaya* with involvement of *Gambhira*

1 सर्वैवप्रमेहास्तुकालेनाप्रतिकारिणः॥ मधुमेहत्वमायान्ति तदाऽसाध्या भवन्ति हि॥ मा.नि.33/23

and *Sara bhuta Dhatus* like *Majja, Vasa, Oja and Lasika* and when *vatakara nidanas* (*vata dosha* related causes) are present, . It is *Yapya* (manageable with continuous care), if it is *nava* (of recently origin) and due to *Apatyanimittaja* (improper management of other 19 types of *prameha*).

SUPPORTIVE INVESTIGATIONS

Essential:

- Blood Sugar Profile: Fasting Blood sugar (FBS) ≥ 126 mg/dL, Post-prandial Blood sugar (PPBS)≥ 200 mg/dL, Glycated Haemoglobin HbA1C ≥ 6.5%.
- Complete haemogram, urine examination for glucose, proteins, ketone bodies, and microscopic examination for pus cells.

Advanced:

- Blood for serum creatinine, lipid profile and liver function tests.
- Serum electrolytes, Blood urea , Urine microalbumin,
- Creatinine clearance, ACR
- Electro-cardiography
- Chest skiagram- Postero-anterior view
- Ophthalmoscopic examination
- 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)

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^[23]

Table 2:

Criteria of diagnosis of Diabetes among non-pregnant individuals
HbA1C ≥ 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 ≥ 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 ≥ 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 criteria for specific detection of type 2 diabetes mellitus are 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%

Clinical Diagnostic criteria as per Ayurveda

The diagnosis of Prameha can be made when prodromal symptoms of Prameha are manifested along with excess urination.

प्रमेह-रूपम् /प्रमेह-सामान्यलक्षण² (General/cardinal Symptoms of Prameha)

Table:4

NAMC Code	Symptoms	Interpretation
EF2-	प्रभूतमूत्रता	excessive urination (frequent urination with increased quantity)
	आविलमूत्रता	turbid urine

प्रमेह-पूर्वरूपम्³) Prodromal symptoms of Prameha)

Table 5:

S.N.	Symptoms	Meaning
1 (a)	प्रचुरमूत्रता	Increase in the frequency of urination or increase in urine output.
1 (b)	मूत्र-अभीक्ष्णता	increase in the frequency of urination

- सामान्यलक्षणंतेषांप्रभूताविलमूत्रता। दोषदृष्ट्याविशेषेऽपितत्संयोगविशेषतः (Va.Ni. 10/7)
तत्राविलप्रभूतमूत्रलक्षणाःसर्वेएवप्रमेहाभवन्ति ॥६॥ S.Ni 6/6
- तेषांपूर्वरूपाणि- हस्तपादतलदाहःस्निग्धपिच्छिलगुरुतागात्राणामधुरशुक्लमूत्रतातन्द्रासादःपिपासादुर्गन्धश्चासस्तालुगलजिह्वादन्तेषुमलोत्पत्तिर्जटिलीभावःकेशानांवृद्धिश्चनखानाम्॥५॥ S.Ni 6/5
त्रयस्तुखलुदोषाःप्रकुपिताःप्रमेहानभिनिर्वर्तयिष्यन्तइमानिपूर्वरूपाणिदर्शयन्ति; तद्यथा-जटिलीभावकेशेषु, माधुर्यमास्यस्य, करपादयोःसुप्ततादाहौ, मुखतालुकण्ठशोषं, पिपासाम्, आलस्यं, मलंकाये, कायच्छिद्रेषूपदेहं, परिदाहंसुप्ततांचाङ्गेषु, षट्पदपिपीलिकाभिश्चशरीरमूत्राभिसरणं, मूत्रेचमूत्रदोषान्, विस्त्रंशरीरगन्धं, निद्रां, तन्द्रांचसर्वकालमिति। Ch. Ni. 4/47
स्वेदोऽङ्गगन्धःशिथिलाङ्गताचशय्यासनस्वप्नसुखेतिश्च।
हृन्नेत्रजिह्वाश्रवणोपदेहोघनाङ्गताकेशनखातिवृद्धिः॥१३॥
शीतप्रियत्वंगलतालुशोषोमाधुर्यमास्येकरपाददाहः। भविष्यतोमेहगदस्यरूपंमूत्रेऽभिधावन्तिपिपीलिकाश्च॥१४॥ Ch.chi. 6/13-14
स्वेदोऽङ्गगन्धःशिथिलत्वमङ्गशय्यासनस्वप्नसुखाभिषङ्गः। हृन्नेत्रजिह्वाश्रवणोपदेहोघनाङ्गताकेशनखातिवृद्धिः॥३८॥
शीतप्रियत्वंगलतालुशोषोमाधुर्यमास्येकरपाददाहः। भविष्यतोमेहगणस्यरूपंमूत्रेऽभिधावन्तिपिपीलिकाश्च॥३९॥ A.hr Ni 10/38-39
दन्तादीनामलाढ्यत्वंप्राग्रूपपाणिपादयोः। दाहश्चिक्कणतादेहेतुत्वाद्वास्यंचजायते॥५॥ (Madhav Nidan)

S.N.	Symptoms	Meaning
2 (a)	आविलमूत्रता	turbidity in urine/turbid appearance of urine
2 (b)	मूत्रवैवर्ण्यम्	altered colour in urine
2 (c)	मधुरशुक्लमूत्रता	urine abnormalities in colour and taste, specifically sweet and whitish urine
2 (d)	मूत्रदौर्गन्ध्यम्	foul-smelling urine
2 (e)	षट्पदपिपीलिकाभिः चशरीरमूत्राभिसरणम्	Gathering of ants on or towards excreted urine.
3 (a)	मुखतालुकण्ठशोषम्	dryness of mouth, palate and throat
3 (b)	पिपासा/तृट	Increased Thirst
4 (a)	करपाददाह/हस्तपादतलदाहः	burning sensation in palm/soles
4 (b)	अंगेषुपरिदाहः	burning sensation all over the body
4 (c)	करपादयोःसुप्तताः	numbness in hands and feet
4 (d)	अंगेषुसुप्तताम्	numbness in body parts
5.	गात्राणाम्गुरुता	the feeling of heaviness in the body
6 (a)	निद्रां, तन्द्रांचसर्वकालम्	always feeling sleepy and drowsy
6 (b)	आलस्यम्	laziness
6 (c)	शय्यासनस्वप्नसुखेरतिः	preference for sleeping, resting and lying down always
6 (d)	शिथिलाङ्गता /सादः	laxity of muscles and body in general
7.	श्वासदौर्गन्ध्यम्	bad breath/foul smell in inhaled breath
8 (a)	विस्त्रंशरीरगन्धम्	musty body odour
8 (b)	स्वेदः	excessive sweating
9.	कायच्छिद्रेषूपदेहम्/ तालु-गल-जिह्वा-दन्तेषुमलो- त्पत्तिः/दन्तादीनामलाढ्यत्वम्/ हृन्नेत्रजिह्वाश्रवणोपदेह	deposition of grime or dirt or appearance of coating over throat-palate-tongue and teeth
10	आस्यमाधुर्यम्	sweet taste in the mouth
11 (a)	जटिलीभावःकेशानाम्	matted hair
11 (b)	वृद्धिः चनखानाम्	excessive growth of nails
11 (c)	वृद्धिः चकेशानाम्	excessive growth of hairs
12.	शीतप्रियत्वम्	preference for cold temperature and cold things

मधुमेहः/क्षौद्रमेहः/Madhumeha/ Kshaudrameha

⁴The main diagnostic feature of *Madhumeha* is passing urine which is astringent, sweet, pale and unctuous, in excess and there will be smell of the entire body resembling like honey. ¹²⁻¹⁴

4 मधुरंयच्चमेहेषुप्रायोमध्वमेवेति ॥
सर्वेऽपिमधुमेहाख्यामाधुर्याच्चतनोरतः ॥ मानि 33/26

Table 6: Differential Diagnosis as per Ayurveda: *Prameha*, *Mutrakrichhra*, *Mutraghata*

S.N.	Presentation	<i>Prameha</i>	<i>Mutrakrichhra</i>	<i>Mutraghata</i>
1.	Prominent feature	Excessive and frequent urination	Difficulty in urination is more prominent	Obstruction is more prominent
2.	Urine quantity	Increased	Decreased	Decreased
3.	Urine frequency	Increased	Increased	Decreased
4.	Pain	May be present or may not be present depend upon type of <i>Prameha</i>	Present	Present
5.	Burning	Specifically in <i>haridrameha</i> (type of <i>pittajaprameha</i>)	Present	Present
6.	Difficulty in micturition	May be present or may not be present	Present	Present
7.	Flow of urine	Depends on type of <i>prameha</i>	Slow flow	Slow flow
8.	Excreted urine colour	Specific urine colors according to different type of <i>prameha</i>	White, yellow or red colour urine	Yellow or red colour

PRINCIPLE OF MANAGEMENT

Red Flag signs:

Before initiating treatment, it is essential to assess the following conditions, that may require specialised management/consultation through modern medicine.

1. Severe infective co-morbidities like pneumonia, tuberculosis, sepsis, etc.
2. Advanced-stage malignancies
3. Visual impairment due to diabetic retinopathy
4. Severe motor or autonomic dysfunction
5. Severe renal dysfunction with markedly reduced GFR
6. Diabetic ketoacidosis
7. Hypoglycemia
8. CVA
9. Hyponatremia
10. Hyperosmolar non-ketotic coma

Line of Treatment:

⁵Management of *Madhumeha* requires a multicentric approach, including both preventive and therapeutic aspects, focusing on the different stages of the disease. Acharya Charaka has emphasized treatment based on *Rogabala* and *Rogibala*. Diabetic patients who are obese, strong, with an excess of *doshas* should be treated with *shodhanachikitsa* (bio-cleansing therapy), whereas for lean and thin diabetic patients, *brumhana chikitsa* (nourishing therapy) is advised.²⁴ *Sushruta* in the context of '*Pramehapidaka chikitsa*' has mentioned the different stages of *Madhumeha* and their respective treatment such as in *Poorvavastha*, *Vyaktavastha*, *Bhedavastha* and *Upadrava*.²⁵

5 स्थूलः प्रमेही बलवानी है कः कुशस्तथैकः परिदुर्बलश्च ।
संबृंहणं तत्र कुशस्य कार्यं संशोधनं दोषबलाधिकस्य ॥ (च.चि. 6/ 10)

Table 7: Stages of Management of Madhumeha (Avasthanusara chikitsa)²⁵

Sl. No	Stages	Pathogenesis	Predominant Dosha	Line of Treatment	Medicine/Dosage
1	Stage 1: <i>Poorvavastha/Amavastha</i>	Appearance of pre-monitory symptoms	<i>Kapha</i>	<i>Apatarpana</i> (depleting procedures), <i>Vataharakashaya</i> (decoctions) and <i>Chaga mutra</i> (urine of goat)	<i>Trikatuchurna</i> 1-2 gms/ <i>Panchakola Phanta</i> 50ml before meals 7-15 days
2	Stage II: <i>Vyaktavastha</i>	<i>Sweda</i> (sweat), <i>mutra</i> (urine) and <i>sleshma dosha</i> (<i>dosha</i> regulating body fluids) acquires <i>madhura bhava</i> (sweet taste)	<i>Kapha</i> or <i>Pitta</i> or <i>Vata</i>	<i>Samshodhan</i> therapies (bio-cleansing therapy), <i>Brimhana</i> (nourishing), <i>Rasayana</i> (rejuvenating therapies)	• <i>Nishamalaki churna</i> 3gms twice daily, • <i>Chandraprabha vati</i> 250mg twice daily 3 months and can be continued • <i>Ashwagandha churna</i> 3gms with milk at bed time daily 3 months and can be continued
3	Stage III: <i>Bhedavastha</i>	<i>Involvement of mamsa</i> (muscle tissue) and <i>rakta dosha</i> (blood tissue) causing <i>shopha</i> (inflammatory conditions) and other <i>upadravas</i> (complications)	<i>Vata</i> and associated <i>dosha</i> based on predominant symptoms	<i>Raktamoksha-na</i> (venesection) along with afore-said remedies and measures should be resorted	Based on <i>dosha dushya</i> involvement and level of management medicines can be decided along with diet and lifestyle advocacies
4	Stage IV: <i>Upadrava</i>	Aggravation of <i>shopha</i> and other <i>upadravas</i> causing <i>ruja</i> (excessive pain) and <i>vidaha</i> (severe burning sensation).	<i>Vata</i> and associated <i>dosha</i> based on predominant symptoms	<i>Shastra chikitsa</i> (surgical measures) and <i>vranahara chikitsa</i> (treatment of ulcer)	Based on <i>dosha dushya</i> involvement and level of management medicines can be decided along with diet and lifestyle advocacies
5	Stage V: <i>Asadhyaavastha</i>	<i>Upadravas</i> such as <i>vrana</i> (ulcer) invades the deeper structures and become <i>utsangi</i> (accumulated into deeper tissues) and causes abscesses which are incurable	<i>Vata</i> and associated <i>dosha</i> based on predominant symptoms		Based on <i>dosha dushya</i> involvement and level of management medicines can be decided along with diet and lifestyle advocacies

Prevention Management:

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 prediabetes 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.¹⁴

Ayurveda advocates *nidanaparivarjana*(Restricting disease causing factors), adoption of *pathyaahara* and *vihara*(healthy diet and life style regimen), abandonment of *apathyaahara* and *vihara* (unrecommended diet and life style regimens).

Apathya & Pathya ahara / Unhealthy & Healthy Dietary regimens

Apathya- Excessive consumption of food -*atyashana*, inadequate and inappropriate food intake – *vishamashana*, consumption of food before the digestion of the previous meal – *Adhyashana* are the root causes. Foods to be avoided in order of priority.

Table 8: Apathyaahara (Unhealthy dietary regimens)^{13,25}

Sweets	Sugarcane and its byproducts such as jaggery, sugar, sugar products, honey, soft drinks, <i>payasa</i> / puddings made of jaggery /sugar,dairy foods
Processed food	Excessive consumption of heavy diets, fatty foods which increase body weight and lipids / cholesterol such as fried food, bakery products
Beverages	Newly prepared wine, different kinds of fermented beverage, <i>krishra</i> (gruel), aerated drinks / carbonated beverages / fizzy drinks
Non vegetarian food	Flesh of domestic / aquatic animals and of marshy places, water of rivers and tanks during rains and floods
Fruits	Mango, banana, custard apple, jackfruit, oranges, apples
Vegetables	Potato
Newly harvested grains	<i>Shali</i> (freshly harvested rice), <i>navinadhanya</i> (freshly harvested grains), <i>masha</i> (vigna mungo), <i>nishpava</i> (dolichos lablab)

Table 9: Pathya ahara (Healthy dietary regimens)

Diet should include all necessary components for complete nourishment

Time		Diet with method of preparation and quantity	
Early morning drink	5-6 am	Juice of <i>Jambu</i> (<i>Syzigiumcumini</i>) or <i>kapitha</i> (<i>Feronia limonia</i>) or <i>amalaki</i> (<i>Phyllanthus embilica</i>) or <i>bilva</i> (bael - <i>Aegle marmelos</i>) without any added sweeteners such as sugar, honey or jaggery Or Methi water	50-100 gm of fruit di-luted in 100ml of water 1/4tsp of seeds soaked overnight in 100 ml of water

Time		Diet with method of preparation and quantity	
Breakfast	8- 9 am	Oats / Dalia or bisibelebath or roti etc prepared from Millets with nuts-based chutney or vegetable curry	Quantity sufficient
Snack break	11-11.15 am	Overnight soaked green gram with vegetable salads	One small bowl
Lunch	1-2 pm	1 Roti prepared with any of the below mentioned cereal and two types of vegetable curry and salad, 1 cup of buttermilk	Quantity sufficient
Snack break	5-6 pm	1 bowl Fruit salad or fist full of nuts	As advised
Dinner	8-9 pm	1 Roti prepared with any of the millets bowl of old unpolished red rice and two types of vegetable curry and salad	Quantity sufficient

Apathya & Pathya Vihara/ Unhealthy & Healthy Life Style Regimens²⁵⁻²⁷

Table 10: Apathya Vihara (Unhealthy Life Style regimens)

Physical activities	Physical inactivity, avoidance of exercises, laziness
Sleep	Sleep during daytime and awakening at night, long duration of sleep
Habits	Consumption of alcohol, use of tobacco, smoking, excess use of sugar and its products, suppression of urge to void urine, excessive bio-cleansing therapy

Table 11:Pathya Vihara (Healthy Life Style Regimens)

Physical activities	Practicing regular exercises, <i>yogasanas</i> such as <i>vajrasana</i> , <i>paschimottanasana</i> , <i>ardhamatsyendrasana</i> and <i>halasana</i> , practice of pranayama, calorie consuming activities (brisk walking, swimming, cycling, etc) for about 30 minutes regularly
Sleep	Adequate sleep for 6-8 hrs at night
Habits	Practice of <i>sadvrutta</i> , <i>dinacharya</i> and <i>ritucharya</i>

Yoga and Pranayama²⁸

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 glycemic control.

Table 12: The general guidelines of yoga recommended for T2DM patients

Criteria	Yoga Techniques	Approximate duration	Effects
Asanas (yoga postures)	<i>Trikonasanam</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 type 2 diabetes
	<i>Tadasana</i> (palm tree pose)		
	<i>Vakrasana</i> (spinal twist)		
	<i>Paschimottasana</i> (seated forward bend)		

Criteria	Yoga Techniques	Approximate duration	Effects
	<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 signalling Significantly decreases hip circumference, exerting beneficial effects on glycemic outcomes
<i>Pranayama</i> (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
	<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

Criteria	Yoga Techniques	Approximate duration	Effects
	Full <i>shankhaprakshalana</i> (intestine cleansing)	Once a year	Significantly reduces blood glucose levels, Increases insulin production
	<i>Laghu shankhaprakshalana</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 practised under the guidance of a a registered practitioner, qualified yoga instructor,orphysiotherapist

Curative Interventions:

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

1. Clinical Diagnosis
2. OPD level management
3. Preferable and restricted diet & lifestyle
4. Follow up
5. Referral criteria

Clinical Diagnosis:

Type 2 Diabetes mellitus often presents at the clinic in adults either with 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,the disease may first be identified through one of its complications as the initial presenting symptom.. . Patients may also present with levels of prediabetes on incidental discovery. The diagnosis will be made by the following investigations:

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

In the initial stages of the disease (purvavastha and vyaktavastha of madhumeha), when the patient presents with mildly raised blood sugar levels and not associated with complications, a provisional diagnosis can be made based on history and clinical presentation, as *Sthula* or *Krishna subtypes*. Patients diagnosed as *Sthula* and having at least moderate physical strength and blood sugar level within the range (FBS >110 up to 180 PPBS > 200 up to 280) may be treated at this level. ¹⁸

OPD level management:

Table 13: Initial Advocacy^{18,25}

Drug	Dosage form	Dose (per day)	Time of administration	Duration	Adjuvant/ Anupana	Indication
<i>Nishamalaki churna</i>	Powder	3-6 gm three divided doses	Before meal	30 days and can be continued	Lukewarm water	<i>Pramehaghna</i>
<i>Asanadi kwatha</i>	Decoction	10-15 ml twice a day	Before meal	30 days and can be continued	Lukewarm water	<i>Pramehaghna</i>

Table 14: Late advocacy^{18,25}

Drug	Dosage form	Dose (per day)	Time of administration	Duration	Adjuvant/ Anupana	Indication
<i>Katakakhadiradi kashayam</i> or <i>Nishakatakadi kashayam</i>	Decoction	15-30 ml twice a day	Before meal	30 days and can be continued	Lukewarm water	<i>Prameha</i>
<i>Chandraprabha vati</i>	Tablets	250 mg thrice a day	Before meal	30 days and can be continued	Water	<i>Prameha</i> , <i>Sarvarogahara</i>
<i>Arogyavardhinivati</i>	Tablet	1-2 tablet thrice a day	Before meal	30 days	Lukewarm water	<i>Sarvarogahara</i> , <i>Hepatotonic</i>
<i>Shilajatuprayoga</i>	Tablet	250-500mg	Before meal	30 days	Lukewarm water	<i>Prameha</i> , <i>Sarvarogahara</i>
<i>Phalatrikadikwath choorna</i>	Decoction	15-30 ml twice a day	Before meal	30 days and can be continued	Lukewarm water	<i>Prameha</i>

*Higher option of medicines to be advocated in non-responding cases with compound formulations and combinations depending upon the presenting complaints, clinical findings and associated co-morbidities.

Preferable Diet & Lifestyle

It can be followed as mentioned above in the Prevention management. Maintaining a balanced and adequate nutrition intake is essential for sustaining optimal body weight, normal growth, and development. The diet should focus on including high fibre foods like vegetables, fruits and lean proteins, while limiting the intake of carbohydrates and fats. Ayurvedic dietary recommendations like *Yava mantha* and *peya* may be advised for drinking as per individual requirement. Practice yoga and exercise regularly to achieve weight loss, maintaining glycaemic control and managing stress. There should also be restrictions in other behavioural changes like, smoking, tobacco and alcohol.

Dietary recommendations ^{18,25}

Table 15: The dietary components that are beneficial in the prevention and treatment of Diabetes

Type of Diet	Name
Cereals	<i>Yava</i> (Barley - <i>Hordeum vulgare</i>), <i>godhuma</i> (wheat), <i>purana shali</i> (old rice), <i>kodrava</i> (grain variety – <i>Paspalum scrobiculatum</i>) <i>uddalaka</i> (according to <i>dhanvantari nighantu</i> forest variety of <i>kodrava</i>), <i>kangu</i> (<i>Seteria italica</i>), <i>madhulika</i> (<i>Elusine coracana</i>), <i>shyamaka</i> (<i>Echinochloea frumentacea</i>), <i>jurnahva</i> (<i>Sorghum vulgare</i>), <i>vajranna</i> (<i>Pennisatum typhoides</i>)
Pulses	<i>Adhaki</i> (red gram- <i>Cajamus cajan</i>), <i>kulattha</i> (horse gram) and <i>mudga</i> (green gram), <i>masura</i> (lentils), <i>makushtha</i> (moth bean/acpnite bean), <i>chanaka</i> (<i>Cicer arietinum</i>) should be taken with bitter and astringent leafy vegetables.
Vegetables	<i>Navapatola</i> (young <i>Tricosanthus dioica</i>), young vegetables variety of banana, <i>tanduleyaka</i> (<i>Amaranthus spinosus</i>), <i>vastukam</i> (bathuva), all bitter vegetables (<i>tiktasakam</i>) like <i>methika</i> (fenugreek leaves), <i>karavellaka</i> (bitter gourd), <i>bimbi</i> (ivy gourd), <i>shigru</i> fruits and leaves (drum stick), <i>vrintaka</i> (brinjal), <i>raktavrintaka</i> (tomato), <i>putiha</i> (mint leaves), <i>suran</i> (amorphophallus), Curry leaves, <i>mulaka</i> (radish), <i>kushmanda</i> (ash gourd), <i>kritavedhana</i> (ridge gourd), <i>alabu</i> (bottle gourd)
Fruits	<i>Jambu</i> (<i>Syzygium cumini</i>), <i>kapitha</i> (<i>Feronia limonia</i>), <i>amlaki</i> (<i>Phyllanthus embilica</i>), <i>bilva</i> (bael - <i>Aegle marmelos</i>), <i>dadima</i> (pomegranate - <i>Punica granatum</i>), <i>naranga</i> (orange - <i>Citrus aurantium</i>), <i>parushaka</i> (falsa - <i>Gravia asiatica</i>), <i>udumbara</i> (cluster Fig – <i>Ficus racemosa</i>) etc fruits
Flesh	Birds like <i>kapota</i> (pigeon), Chicken, <i>titira</i> (grey francolin)
Oils Condiments	<i>Atasi</i> (<i>Linum usitatisimum</i>), <i>sarshapa</i> (mustard). <i>haridra</i> (turmeric), <i>maricha</i> (pepper), <i>tvak</i> (cinnamon), <i>lashuna</i> (garlic), <i>shunthi</i> (ginger), <i>methika</i> (fenugreek), <i>dhanyaka</i> (coriander), <i>jeeraka</i> (cumin seeds)

Lifestyle recommendations:^{18,25} Ayurvedic texts suggest long walks, swimming, hard labour like pulling carts, digging wells, serving animals etc. All this involves muscular activity, which will help in maintaining muscle tone and peripheral utilization of glucose. Following norms should be followed for exercise:

- Exercise should be initiated at low intensity and should be gradually increased
- It should not be done after eating heavy meals
- It should be done regularly at fixed timings.
- Before exercise, a person should have taken proper sleep, his diet should have been digested properly

Restricted Diet & Lifestyle:

Diet:^{18,25}

Excessive consumption of sweets, fruit salad, sugarcane, high-glycemic index fruits like mango, watermelon, chikoo, dates, jack fruits, custard apple, banana and grapes, cashew nuts, cold drinks and oily or fried foods containing hydrogenated ghee, over indulgence in meat, especially that of wet-land animals, eating food before complete digestion of previous food, and consuming food at improper times and in varied quantity.

Lifestyle:^{18,25}

Day time sleep, especially immediately after a heavy meal, an irregular sleep pattern i.e. sleeping less than 5 hours or more than 10 hours in a day or in an improper way, lack of or infrequent exercise.

Follow Up (With duration): Since T2DM is a multifactorial condition associated with complications, follow-up visits are recommended every 15 days or as needed for effective management of the condition.

Reviews should include

- Blood glucose - FPG and 2 hr PPPG at least once a month and more often if values are not within the ideal target range
- HbA1c at least every 6-12 months and more often (every 3 months) if values are not within the ideal target range, or if tight control is being attempted
- Clinical examination needs to be done during every visit- atleast once in every 3 months
- Screening for long term complications like retinopathy, nephropathy, neuropathy, peripheral vascular disease (PVD) and coronary artery disease (CAD) at least once a year, or more often if needed
- Regular monitoring and management of weight, waist circumference, blood pressure, and lipid levels
- Routine examination of foot & patient education on proper foot care to be done in every visit
- Discourage tobacco use and excess consumption of alcohol
- Urine glucose monitoring is recommended. Urine examination for estimation of ketones should be done if blood glucose level is greater than 400 mg/dl.

Referral Criteria:

- Nonresponse to treatment
- Target organ involvement and investigations
- Complications of diabetes mellitus including all macrovascular, microvascular, and emerging complications
- Complications related to glycemic control including uncontrolled hyperglycemia and frequent hypoglycemic 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 and X-ray)

Clinical Diagnosis

The clinical diagnosis of T2DM should be done same as level 1. The case referred from Level 1, or a fresh case must be evaluated thoroughly for any complications. *Vyaktavstha* and *bhedavastha* of *madhumeha* can be treated at this level.

- Investigations: Same as Level 1.
- Supportive investigations to assess organ involvement includes:
- Serum electrolytes
- Blood urea
- Urine microalbumin, creatinine clearance, ACR
- Electro-cardiography
- Chest skiagram- Postero-anterior view
- Ophthalmoscopic examination

OPD/IPD level management:

In addition to the management mentioned in Level 1, a few of the following drugs may be added as per the requirement and status of the patient. *Rasaushadhi* (Herbo-mineral drugs) or herbal drugs of potential pharmaco-vigilance importance can be used at this level. Patient may be kept under observation while prescribing these medicines.¹⁸

Patients who are eligible for *shodhana* (bio-purificatory procedures) should be assessed for the predominant dosha involvement. If the patient is not eligible for *shodhana* and if *bala*(strength) is less, *shamana* (palliative therapy) line of treatment can be adopted.

Santarpana chikitsa (nourishing therapy): In case of *pitta* predominance, if *prameha* seen in *krisha* type (lean) and with *durbala* (lower strength) and *adhikadoshabala*, *santarpana* or *brumhana chikitsa* can be adopted.

- Preparations of *yava* (barley -*Hordeum vulgare*) along with honey, extracted juice of *amalaki* (gooseberry - *Embilica officinalis*) mixed with *haridra* – (turmeric - *Curcuma longa* linn.) powder and pure honey can be advised.
- Extracted juice of *guduchi* stem (heart leaved moonseed - *Tinospora cordifolia*) with extracted juice of *amalaki* and pure honey can be advised.

Shamana chikitsa(palliative therapy): Along with *Kashtaushadhi* (herbal drugs), *Rasaushadhi* (Herbo-mineral drugs) should be administered.

Table 16: Drugs to be administered for palliative management^{18,25}

Drug	Dosage form	Dose (per day)	Time of administration	Duration	Adjuvant/ Anupana	Indication
<i>Nishamalaki churna</i>	Powder	3-6 gm three divided doses	Before meal	30 days and can be continued	Lukewarm water	<i>Prameha</i>
<i>Katakakhadiradi kashayam</i> or <i>Nishakatakadi kashayam</i>	Decoction	15-30 ml twice a day	Before meal	30 days and can be continued	Lukewarm water	<i>Prameha</i>
<i>Chandraprabha vati</i> or <i>Arogyavardhini vati</i>	Tablets	250 mg thrice a day	Before meal	30 days and can be continued	Water	<i>Prameha</i> , <i>Sarvarogahara</i>
<i>Vasanthakusumakara rasa</i> or <i>Trivangabhasma</i>	Tablet	125-250 mg BD	After meal	15-30 days	Lukewarm water	<i>Prameha</i> , and its <i>upadrava</i> (-complications) <i>Rasayana</i> and <i>dhatuposhaka</i>
<i>Darvyadi ghrta</i> <i>Triphala ghrta</i>	Ghrta	5-10ml	After meal	15-30 days	Lukewarm water	<i>Prameha</i>

Purification/ other procedures (As per applicability)

Shodhana chikitsa (bio-purificatory procedures): As mentioned earlier in *madhumeha chikitsa*, it has very limited applicability in diabetic patients. However, based on *Rogabala* and *Rogibala*, (strength of disease and patient), *Shodhana Chikitsa* (bio-cleansing therapy) may be considered, particularly in patients with the *Sthula* (obese) type who have a strong built and *adhikadoshabala* (more doshas)..

Internal Therapeutic procedures

- **Vamana** (medically induced emesis): *Madanaphala yoga* (15 gms) along with *vamanopaga* (emetics) such as *madhuyashti phanta* or *saindhava jala* can be utilized to elimination of vitiated *kapha dosha* as well as *pitta dosha* (to an extent).
- **Virechana** (medically induced purgation): *Virechana yoga* (10 gm) can be used to eliminate mildly increased *pitta-kapha dosha* and in case of *pitta* predominance. *Nitya virechana* (medicated purgation using mild laxative): *Triphala churna* (10 gms) at night after meal for a period of 7 days can be advised to maintain the balance of *tridoshas* and aids in weight loss.
- **Basti** (medicated enema): *Asthapana basti* (medicated decoction enema): *surasadigana kashaya* with *mahoushadha*, *bhadradraru*, *musta*, *madhu* and *saindhava* in case of *vata* dominance.
- **Nasya karma** (nasal medication): *Nasya* using *triphaladi taila* would be effective in managing excessive sleep and lethargy.

External Therapeutic procedures

- **Sarvanga ruksha udwarthana** (rubbing with medicated dry powder): In the context of diabetic patients where there is *bahuabaddhamedas*, *udvartana*²⁹ should be adopted as the *dravikritamedas* undergoes *shoshana* (depletion), particularly in obese individuals. *Triphala churna* (Q.S) or *yavachurna* or *kolakulatha churna* can be advised for a period of 7 days. This aids in weight loss and maintains stability of the body.

Preferable and Restricted Diet & Lifestyle: As recommended in preventive and level 1 management.

Follow Up:

Once in every 15 days or as needed, for effective management of the condition.

Referral Criteria:

- Same as mentioned earlier at level 1
- Acute exacerbation of the disease
- Advanced stage of the disease
- Increased severity of complications

At Level 3: (Ayush hospitals attached with Teaching Institution, District Level/Integrated/ State Ayush Hospitals)

1. Management with single herbs and compound formulations
2. Bio-purification procedures and external therapeutic procedures
3. Preferable and restricted diet & lifestyle
4. Follow up
5. Referral criteria

Clinical Diagnosis:

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

Investigations: Same as Level 1 and 2.

Additional Investigations may be done as follows:

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

Management of *Bhedavastha* and *Upadrava*:

All patients referred from level 2 should be treated at this level. Patients having HbA1c above 9 should be treated at this level.

In addition to the management of Level 1 and Level -2, if needed other Panchakarma procedures can be performed.

Table 17: Single/ Compound formulations effective in the management of T2DM³⁰

Drug	Dosage form	Dose (per day)	Time	Duration	Adjuvant/ Anupana	Indication
<i>Nyagrodhadichurna</i>	Powder	3-6 gm	After meal	15-30 days	Lukewarm water	<i>Prameha</i> and its complications
<i>Swarnamakshika Bhasma</i>	Tablet	250 mg	After meal	15-30 days	Honey	<i>Prameha</i> , <i>vrana</i> , <i>rasayana</i> , <i>balya</i> and has <i>yogavahi</i> property

Purification/ other procedures (as per applicability)¹⁸

Shodhana chikitsa (bio-purificatory procedures): As mentioned earlier in *madhumeha chikitsa*, it has very limited applicability in diabetic patients. However, based on *Rogabala* and *Rogibala*, (strength of disease and patient), Shodhana Chikitsa (bio-cleansing therapy) may be considered, particularly in patients with the *Sthula* (obese) type, who have a strong built and *adhikadoshabala* (more doshas).

Internal Therapeutic procedures

- **Vamana**(medically induced emesis): *Madanaphala yoga* (15 gms) along with *vamanopaga* (emetics) such as *madhuyashtiphanta* or *saindhavajala* can be utilized to elimination of vitiated *kapha dosha* as well as *pitta dosha* (to an extent).
- **Virechana** (medically induced purgation): *Virechana yoga* (10 gm) can be used to eliminate mildly increased *pitta-kapha dosha* and in case of *pitta* predominance. *Nitya virechana* (medicated purgation using mild laxative): *Triphala churna* (10 gms) at night after meal for a period of 7 days can be advised to maintain the balance of *tridoshas* and aids in weight loss.
- **Basti** (medicated enema): *Asthapana basti* (medicated decoction enema): *surasadigana kashaya* with *mahoushadha*, *bhadradraru*, *musta*, *madhu* and *saindhava* in case of *vata* dominance.
- **Nasya karma** (nasal medication): *Nasya* using *triphaladi taila* would be effective in managing excessive sleep and lethargy.

External Therapeutic procedures

- **Sarvangarukshaudwarthana** (rubbing with medicated dry powder): In the context of diabetic patients where there is *bahuabaddamedas*, *udvartana*²⁹ should be adopted as the *dravikritamedas* undergoes *shoshana* (depletion)), particularly in obese individuals. *Triphalachurna* (Q.S) or *yava churna* or *kolakulatha churna* can be advised for a period of 7 days. This aids in weight loss and maintains stability of the body.

Preferable and Restricted Diet & Lifestyle:

Same as level 1, along with modifications in diet and exercise should be made as per the strength and built of the concerned patient. Moderately nourishing food which do not aggravate *Kapha* and *Meda* can be advised

Follow Up: Once in every 15 days or earlier as per the need.

Referral Criteria:

- Same as mentioned earlier at level 2
- Increased severity of complications
- Patients whose co-morbidities are not controlled at the Level 3 setting and needs urgent intervention at higher centers.

REFERENCES

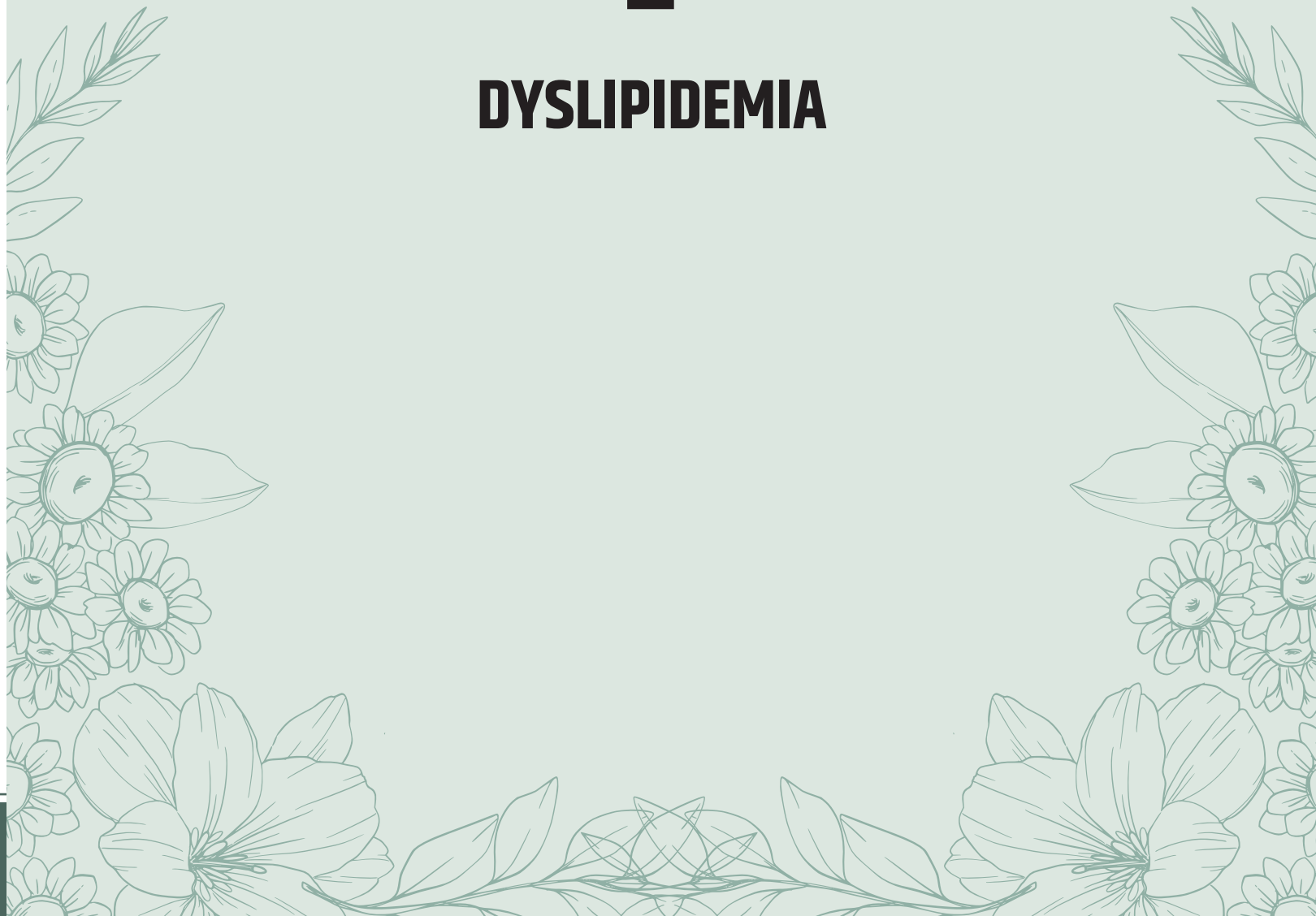
1. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care [Internet] 2014 [cited 2024 Jan 17];37(Supplement_1):S81–90. Available from: <https://dx.doi.org/10.2337/dc14-S081>
2. Petersmann A, Müller-Wieland D, Müller UA, Landgraf R, Nauck M, Freckmann G, et al. 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/>
3. National AYUSH Morbidity and Standardized Terminologies Electronic Portal. Morbidity Codes- Ayurveda: Prameha [Internet]. Hyderabad: CCRAS, Ministry of AYUSH, Government of India; 2017 Oct 17 [cited 2024 May 22]. Available from: <https://namstp.ayush.gov.in/#/Ayurveda>
4. National AYUSH Morbidity and Standardized Terminologies Electronic Portal. Morbidity Codes- Ayurveda: Madhumeha [Internet]. Hyderabad: CCRAS, Ministry of AYUSH, Government of India; 2017 Oct 17 [cited 2024 May 22]. Available from: <https://namstp.ayush.gov.in/#/Ayurveda>
5. 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>
6. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels, Belgium: 2021. Available at: <https://www.diabetesatlas.org>
7. 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/>
8. 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/>
9. 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/>
10. 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>
11. 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: <https://pubmed.ncbi.nlm.nih.gov/34102081/>
12. 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>
13. Acharya JT, editor. Charaka Samhita of Agnivesha: Ayurveda Deepika Commentary of Chakrapanidatta, Nidana Sthana. 1st ed., Ch.4, Ver 37. Varanasi: Chaukambha Sanskrit Sansthan; 2016.p.215.
14. Acharya JT, editor. Charaka Samhita of Agnivesha: Ayurveda Deepika Commentary of Chakrapanidatta, Sutra Sthana. 1st ed., Ch.17, Ver 81-82. Varanasi: Chaukambha Sanskrit Sansthan; 2016.p.103.
15. Madhavakara. Madhava Nidanam; Translated into English by Dr. K. R. Srikantha Murthy, Roga Vinischayam. Uttarardham, Pramehanidana, Ch.33, Ver.1-4. Varanasi: Chaukamba Oriental Publisher & Distributor, Krishnadas Academy; 1987. P.181-182.
16. Bhishagacharya Harishastri editor. Astanga Hridayam of Vriddhavagbhata, Nidana Sthana, 11th ed. Ch. 10, Ver. 3-4, Varanasi: Chaukamba Orientalia; 2022. p.502.

17. [Guidelines], American Diabetes Association. Standards of medical care in diabetes- 2011. *Diabetes care*. 2011. Jan. 34. Suppl 1. S11-61.
18. David B. Sacks, Mark Arnold, George L. Bakris, David E. Bruns, Andrea R. Horvath, Åke Lernmark, Boyd E. Metzger, David M. Nathan, M. Sue Kirkman; Guidelines and Recommendations for Laboratory Analysis in the Diagnosis and Management of Diabetes Mellitus. *Diabetes Care* 1 October 10) 46 ;2023): e151–e199
19. Ministry of Ayush. Ayurvedic Standard Treatment Guidelines. 1st ed. New Delhi. Government of India. 2017; p. 79-85.
20. Indian Council of Medical Research. ICMR Guidelines for Management of Type 2 Diabetes 2018. New Delhi. ICMR_GuidelinesType2diabetes2018_0.pdf
21. Peter P. Harms, Amber A. van der Heijden, Femke Rutters, Hanno L. Tan, Joline W.J. Beulens, Giel Nijpels, Petra Elders, Prevalence of ECG abnormalities in people with type 2 diabetes: The Hoorn Diabetes Care System cohort, *Journal of Diabetes and its Complications*, Volume 35, Issue 2, 2021, 107810, ISSN 1056-8727, <https://doi.org/10.1016/j.jdiacomp.2020.107810>.
22. Madhavakara. Madhava Nidanam; Translated into English by Dr. K. R. Srikantha Murthy, Uttarakhand, Pramehanidana, Ch.33, Ver.23-36. Varanasi: Chaukamba Oriental Publisher & Distributor, Krishnadas Academy; 1987: p.185-186.
23. Bhavamishra. Bhavaprakash; Translated into English by Dr. K. R. Srikantha Murthy, Madhyakhanda, Vol.2, Pramehapidakaadhikara, Reprint, Ch.38, Ver. 24-26. Varanasi: Chaukamba Oriental Publisher & Distributor, Krishnadas Academy; 2002: 484, 497.
24. 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>
25. Acharya JT, editor. Charaka Samhita of Agnivesha: Ayurveda Deepika Commentary of Chakrapanidatta, Chikitsa Sthana. 1st ed., Ch.6, Ver 10. Varanasi: Chaukambha Sanskrit Sansthan; 2016.p.449.
26. Central Council For research in Ayurvedic Sciences. Guidelines for prevention and management of Diabetes. New Delhi: Available from: http://www.ccras.nic.in/sites/default/files/Guidelines_for_prevention_and_management_of_Diabetes.pdf
27. Dr. Subhas Kumar Sahani, Dr. Ramnihar Tapsi Jaiswal and Dr. Manohar Ram, A critical review on madhumeha (dm type-2) a life style disorder, *world journal of pharmaceutical and medical research*, wjpmr, 2023, 9(11), 85-89, ISSN 2455-3301
28. Mohit Sharma, Prem Prakash Vyas, Harish Kumar Singhal. Role of Pathya-Apathya in the Management of Prameha (Type-2 Diabetes Mellitus). *AYUSHDHARA*, 2023;10(Suppl 2):38-43. DOI:10.47070/ayushdhara.v10iSuppl2.1211
29. Raveendran AV, Deshpandae A, Joshi SR. Therapeutic Role of Yoga in Type 2 Diabetes. *Endocrinol Metab (Seoul)*. 2018 Sep;33(3):307-317. doi: 10.3803/EnM.2018.33.3.307. Epub 2018 Aug 14. PMID: 30112866; PMCID: PMC6145966.
30. Bhishagacharya Harishastri editor. Astanga Hridayam of Vriddhaghata, Sutra Sthana, 11th ed. Ch. 2, Ver. 15, Varanasi: Chaukamba Orientalia; 2022. p.28.
31. Gill D. et.al. Prameha-Hara Yogas (Anti diabetic formulations) in Bhaishajya Ratnavali and their utility. *Annals Ayurvedic Med*. 2022;11(1) 73-90, DOI:10.5455/AAM.115564.
32. Ayurvedic Management of selected Geriatric Disease Conditions, 1st ed. Central Council for Research in Ayurveda and Siddha, Department of Ayush, Ministry of Health and family welfare, Government of India. 2011; p.81-88
33. National List of Essential Ayush Medicines (NLEAM). 1st ed. Ministry of AYUSH, AYUSH Bhawan, B Block, GPO Complex, INA, New Delhi – 110023: Ministry of AYUSH, Government of India, New Delhi. www.ayush.gov.in; 2022. In.
34. Ministry of Ayush. Ayurvedic Standard Treatment Guidelines. 1st ed. New Delhi. Government of India. 2017; p. 79-85.

CHAPTER

2

DYSLIPIDEMIA



DYSLIPIDEMIA

ICD 10:E78.5

ICD 11: 5C81

Medorogah (National Ayush Morbidity Code:EF-3)

CASE DEFINITION

Dyslipidemias 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 triglycerides¹.

The clinical features seen in Dyslipidaemia are varyingly learnt from the signs and symptoms as described in *Medoroga*, *Medovridhi*, *Medovahasrotoviddha*. The signs and symptoms such as *Mēdastusarvabhūtānāmudarēṣvasthiṣusthitam* [tendency of fat to accumulate in the abdomen and in the bony prominences], *Daurgandhya* [bad odour], *Kṣuta* [voracious appetite], *Tr̥ṣā* [thirst], *Kṣudraśvāsa* [dyspnoea on exertion], *kasa* (cough) [difficulty/hard breathing], *alpeṣpiceshtitesvasham* [dyspnoea on little exertion], *Snigdhangata* [unctuousness of body parts], *Sthulashophata* [non pitting oedema], *Sāda* [exhaustion or tiredness of body], *Svapna* [sleepiness], and *Ayathōpacayōtsāhaḥ* [vigour is not in proportion to his body bulk]. Many *Prameha purvarupa* symptoms are also exhibited in Dyslipidamia conditions.

Shrama [exhaustion/fatigue]/ *Alpachestitha Shrama* and *Swasha*: These two symptoms in combination can be seen as a symptoms of high Cholesterol level as deposition of cholesterol in vessels can lead to such symptoms.

INTRODUCTION (Incidence/Prevalence, Morbidity/Mortality)

1. 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 Africa². In India specific, the prevalence of hypercholesterolemia varies from 10 to 15 % in rural to 25–30 % in urban populations³.
2. Dyslipidaemia 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) ⁴⁻⁷ while some studies had shown that non-HDL-c predicts CV risk better than LDL-C ⁸.
3. 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 %, 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 country⁹.

Classification¹⁰⁻¹³

Dyslipidemias 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)¹³:

- 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.

In Ayurveda, Dyslipidemia is described as *Medoroga* or disorder of fat metabolism which is also linked to conditions like *Sthaulya* (Obesity). Dyslipidemia is primarily considered a result of an imbalance in the *Kapha* dosha, which is responsible for structure, stability, and lubrication in the body. An increase in *Kapha* leads to the excessive accumulation of *Medo Dhatu* (fat tissue), manifesting as *Medoroga*.¹⁴

Clinical presentation of Dyslipidemia is also seen in *Medovruddhi* which results in deposition of fats in various body parts, particularly in abdomen and in flanks, difficulty in breathing, dyspnoea on little exertion, bad odour.¹⁵

Dyslipidemia can also be linked with *Medovaha Srotas Vidha or Dushti*, characterized by excessive sweating, oily skin & organs, dryness of oral cavity, excessive thirst, gross swelling or enlargement of body parts due to the accumulation of fats.¹⁶

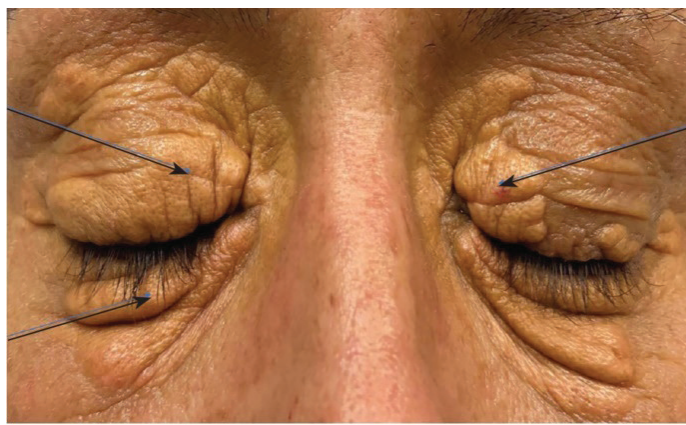
Similar to the classification of Dyslipidaemia such as caused by primary and secondary factors, the causative factors as described in Ayurveda also include those due to *Bijaswabhawaja* (dyslipidemia due to genetic factors) and those due to conditions like *Prameha* (Diabetes), *Medovruddhi* (obesity), and *Dhatvagnimandya* (Hypothyroidism). **Irrespective of the weight and built of the person it can manifest in both obese and non-obese persons.** The

dyslipidaemia may also associate with *Pleeha-abhivridhi*. This can be attributed to the decrease level of alpha and beta lipoprotein and more deposition of lipids in spleen.

CLINICAL PRESENTATION ^{17,18}

Dyslipidemias 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 retinalis (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 / Metabolic Dysfunction Associated Steatohepatitis(MASH).

DIFFERENTIAL DIAGNOSIS¹⁹⁻²¹

Table 1: Several disease conditions remain as secondary causes for dyslipidemia. They are as follows:

S. 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, 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, EUS, magnetic resonance, cholangiopancreatography (MRCP), or direct cholangiography
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, 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.	Cerebrotendinousxanthomatosis	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.

SUPPORTIVE INVESTIGATIONS^{10, 22,23}

Essential:

- **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 24

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

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

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

BMI classification for Asians must be used in indian context.

Reference: World Health Organization, author. The Asia-Pacific perspective: redefining obesity and its treatment. WHO; 2000.

Advanced:

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

1. Apolipoprotein B (ApoB), apolipoprotein A1
2. Lipoprotein(a)
3. Treadmill Test.
4. High sensitivity C-reactive protein.
5. Glycosylated hemoglobin (HbA1c).
6. Fasting blood glucose (FBS).
7. Thyroid stimulating hormone level (TSH).
8. Liver function tests.
9. Serum creatinine.
10. Creatine kinase.
11. Urine analysis.
12. Homocysteine levels.
13. Fundoscopy
14. Waist hip ratio, waist circumference, skin fold thickness
15. Plasma leptin
16. Upper Abdominal Ultrasound

DIAGNOSTIC CRITERIA¹⁰⁻¹²

Dyslipidemia is often diagnosed with routine screening tests. Dyslipidemia is diagnosed by measuring serum lipids. Routine measurements (lipid profile) include total cholesterol (TC), triglyceride (TGs), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (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

Levels of risk	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	≥ 499 mg/dL	< 25 mg/dL

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

Principle of Management

Red Flags^{25,26}

- 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 dyslipidemias (FH & High Lp)
- Clinical evidence of atherosclerotic CAD
- Atherosclerotic disease in other vascular beds
- Heterozygous Familial Hypercholesterolemia (HeFH) with ASCVD, or coronary imaging showing >50 % stenosis in 2 coronary vessels.
- Total cholesterol ≥ 220 mg/dL or LDL cholesterol ≥ 190 mg/dL in individual.
- Tendon Xanthomas
- Uncontrolled co-morbidities

Prevention Management

Diet- A healthy diet is essential for managing Dyslipidemia, as it can help lower LDL cholesterol, reduce triglycerides, and increase HDL cholesterol.

Table 5:

Take/ Pathya	Avoid/Apathya
Low calorie diet	High calorie diet
Wholesome balanced food	Ultra-processed food
Diet with low salt & trans-fat	Diet with high salt, sugar & trans-fat

Take/ Pathya	Avoid/Apathya
Non-Saturated Fats or Monounsaturated Fats- Mustard oil, Groundnut oil, Coconut oil(edible oil from plant origin) Avocados, Nuts, Lean meats, Skinless Poultry(Jangala Mansa-Meat of arid animals) and low-fat or non-fat dairy products (Go-Ghrita -Cow's ghee), Go-Dugdha Cow's milk) etc.	Saturated and Trans Fats- Red meat, full-fat dairy products, butter, Partially hydrogenated oils, baked goods, snacks, margarines etc.
Polyunsaturated Fats- Fish (like salmon, mackerel, and sardines),	
Omega-3 Fatty Acids- Flaxseeds, and Sunflower seeds, Chia seeds, Walnuts etc.	
Fiber rich diet- at least 25-30 grams of fibre daily. Variety of whole grains, vegetables, and fruits. Soluble Fiber sources: Oats, barley, beans, lentils, cowpea, Rajma, apples, and citrus fruits. Insoluble Fiber Sources: Whole grains, vegetables, and fruits with skin.	Low Fiber Processed food diet, Soft drinks, Fast food, canned soups, and salty snacks. Alcohol
Whole grains- Brown rice, Red Rice, Black Rice, wheat, quinoa.	Refined grain- White Rice, White flour/ Maida. People who take white rice as staple diet can take good quantity of vegetables with it.
Quality Protein intake- Soy protein, Low fat paneer	High-cholesterol foods, Fatty food, Sodium
Early dinner	Late night dinner
Freshly cooked food	Refrigerated / stale food
Nitya Sevaniyadravyas • Shashtika Shali (Oryza sativum L.) • Yava /Barley(Hordeum vulgare L.) • Mudga (Vigna radiata L.) • Saindhava (Sodii chloridum) • Amalaki (Phyllanthus emblica L.) • Honey	Dried Meat (Vallura) Dried Vegetables (Shushka Shakha) Coagulated/Fermented Milk (kūrcika) Cream Cheese (Kilata) Pork (sukara) Black gram/Black lentil (Masha)

Life Style- Exercises

To effectively manage Dyslipidemia, incorporating a variety of exercises that target different aspects of fitness is beneficial. The focus should be on aerobic exercise, resistance training, and, optionally, high-intensity interval training (HIIT) to improve lipid metabolism and cardiovascular health.

Following exercises can help manage Dyslipidemia-

1. Aerobic Exercises- Aerobic exercise is key for improving cardiovascular health and managing lipid levels. It helps increase HDL cholesterol, lower LDL cholesterol, and reduce triglycerides. It includes following activities-

- **Walking:** A brisk walk for 30-60 minutes most days of the week can be very effective.
- **Jogging or Running:** Offers a higher intensity workout; aim for 20-30 minutes per session, 3-5 times per week.
- **Cycling:** Either stationary or outdoor cycling, cycling can be done for 30-60 minutes, 3-5 times a week.
- **Swimming:** Provides a full-body workout and is easy on the joints; swim for 30-60 minutes, 3-5 times a week.
- **Dancing:** An enjoyable way to boost heart rate up; consider 30 minutes of dancing several times a week.

Guidelines for use- At least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity exercise per week. Moderate intensity where one can talk but not sing comfortably, or vigorous intensity where talking is challenging. People with sedentary lifestyle can start with these exercises and later on switch these with resistance training.

2. Resistance Training- It helps build muscle mass, which can improve metabolic rate and support better lipid metabolism. It includes following activities-

- **Weight Lifting:** Focus on major muscle groups (legs, back, chest, arms) with exercises like squats, deadlifts, bench presses, and rows. Aim for 2-3 sessions per week.
- **Bodyweight Exercises:** Include push-ups, squats, lunges, and planks. These can be done 2-3 times a week.
- **Resistance Bands:** Use bands for exercises like bicep curls, shoulder presses, and leg lifts. These can be incorporated 2-3 times a week.

Guidelines for use- At least 2 days a week, with rest days in between to allow muscle recovery. 2-4 sets of 8-12 repetitions for each exercise can be performed. Active individuals doing aerobic exercises can start resistance training for additional benefits.

3. High-Intensity Interval Training (HIIT)- It involves alternating between short bursts of intense exercise and periods of lower intensity or rest. It can be effective in improving cardiovascular fitness and managing lipid levels. It includes following activities-

- **Interval Running:** Alternate between 1 minute of sprinting and 2 minutes of walking or jogging. Repeat for 20-30 minutes.
- **Circuit Training:** Perform exercises like jumping jacks, burpees, and mountain climbers at high intensity for 30-60 seconds, followed by a short rest, then repeat.
- **Bike Sprints:** Alternate between 30 seconds of high-speed cycling and 1-2 minutes of slower cycling.

Guidelines for use- 1-2 times a week, depending on fitness level and recovery. Recommended for highly active individuals who are doing exercise regularly.

Moderate intensity continuous training is being considered safe and effective for cardiac patients, with increasing evidence that HIIT is well-tolerated for selected cardiac patients³⁰.

However, caution must be taken in performing any of the Aerobic exercises, Resistance training or High intensity interval training if there is any prevailing Heart ailments.

Table 6: Common Yoga Protocol

Sr. 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.	Samasthiti	Standing Alert Posture
6.	Tadasana	Palm Tree Posture
7.	Vrksasana	Tree Posture
8.	Uttanasanan	Standing Forward Bend
9.	Pada-Hastasana	Hand to Feet Posture
10.	Ardha Chakrasana	Half Wheel Pose
11.	Trikonasana	Triangle Pose
Sitting Yoga Positions		
12.	Visramasana	Long Sitting Posture
13.	Sukhasana	Easy Pose
14.	Padmasana	Lotus Pose
15.	Dandasana	Stick/Staff Pose
16.	Bhadrasan	Gracious Pose or Butterfly Pose
17.	Vajrasana	Thunderbolt Pose
18.	Ushtrasana	Camel Pose
19.	Ardha-Ushtrasana	Half Camel Pose
20.	Sasankasana	Hare Posture
21.	Balasana	Child Pose
22.	UttanaMandukasana	Stretched Up Frog Posture
23.	Vakrasana	Spinal Twist Posture

Sr. No.	Name of Posture/Procedure		
24.	<i>Paschimottanasana</i>	Seated Forward Bend	
25.	<i>Simhasana</i>	Lion Pose	
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>Shavasan</i>	Corpse Body Posture	
38.	<i>Kapalbhati</i>	Forceful Rapid Exhalations	Sukhasana/Padmasana/Va-jrasana 1 inhalation :20-30 exhalation
Breathing Exercises			
39.	Anuloma-Viloma/ Nadishodhana Pranayam/Suryabhedan	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>ShitaliPranayam</i>	Cooling breath	<i>Jnana Mudra or Dhyan 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 Dhyan Mudra or Anjali Mudra</i> <i>Tip of thumb to Tip of index finger</i> <i>Other fingers straight/relaxed</i>

Curative Interventions

At Level 1- Solo physician clinic, health clinic, PHC (optimal standard of treatment where technology and resources are limited)

Clinical diagnosis: Understanding the signs and symptoms of dyslipidemia is crucial for timely intervention and prevention of 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. A pertinent social history would include tobacco use or specific details about diet. Diagnosis of dyslipidemia is primarily based on investigations such as **fasting lipid profile**. However other investigations may be advised based on the clinical presentation.

Table 7:

Sr. No.	Drugs	Dosage form	Dose/ day	Time	Duration	Adjuvants/Anupana
1.	Shunthi Churna ²⁷	Powder	2-3gm	In 2-3 divided doses, before meal	1-3 months	Luke warm water
2.	Vidanga Churna ²⁸	Powder	5-10 gm	In 2-3 divided doses, before meal	1 month	Luke Warm water
3.	Haritaki Churna	Powder	3-6 gm	1-2 divided doses, Before meal or at bed time.	1-3 months	Luke Warm water
4.	Navaka Guggulu ²⁹	Tablet	500mg-1 gm	BD	1-3 months	Luke Warm water or Shunthisiddha Jala
5.	Triphala churna	powder	3-5gm	OD/BD	1-3 months	Luke Warm water
6.	Trikatu churna	powder	1-2 gm	BD	1-3 month	Luke Warm water
7.	Medohar Guggulu	Tablet	500mg-1 gm	Twice daily, before meal	1-3 months	Luke Warm water
8.	Triphala Guggulu	Tablet	1- 3 gms	in 2-3 divided doses	1-3 months	Luke Warm water
9.	Phalatrikadi Kwatha	Decoction	25-50ml	Twice daily, before meal	1-3 month	Luke Warm water
10.	Musta churna	powder	3-6 gm	Thrice daily, before meal	1-3 month	Luke Warm Water
11.	Vrikshamla churna	powder	3-5gm	Twice daily, before food	1-3 month	Luke Warm Water

The drugs are having Agnidipana, Amapachana, Srotoshodhana, kapha-medanashaka, rasa-raktaprasadana, virechaka property.

Duration of intake of medicine can be up to 3 months to 1 year depending upon the treatment response and patient conditions.

Rasayana like Brahma Rasayana and Pippali Rasayana and others can also be added.

Purification/other procedures

- Udvartana: In kaphavruddhi/In Medovruddhi/Obese conditions.
- Lekhana: therapeutic scrapping in ati kapha and atimeda conditions

Follow Up (With duration)

- Every 15 days or earlier as per the need.
- Reviewing the effectiveness and tolerability of all treatments provided.
- Monitoring of the person's symptoms, diet and activity level.
- Monitoring the long-term course of the disease condition.
- Explaining the importance of regular activity and following proper diet regimen
- Discussing the patient's knowledge of the condition and addressing any concerns
- Self-management support: Encourage active participation in managing the conditions through lifestyle modifications and adherence to treatment plans.

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, ECG and 2D Echo)

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. In advanced stages which lead to complications these investigations may also be suggested at level 2 or at level 3 settings to investigate complications or exclude other differential diagnosis 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: 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 the patient's symptoms is the sole indication guide for treating each patient.

Additional Medicines that can be given at level 2

Table 8:

Sr. No.	Drugs	Dosage form	Dose/day	Time	Duration	Adjuvants
1.	Arogyavardhini vati	Tablet	250-500 mg	Once/twice daily	1 month	Warm water or Honey or Adrak Swarasa
2.	Lashunadi Vati	Tablet	500mg	BD	1 month	Warm water or Ark pudina or Ark Ajwain

Duration of intake of medicine can be up to 3 months to 1 year depending upon the treatment response and patient conditions.

Recommended diet and lifestyle: Same as Level 1

Restricted diet and lifestyle: Same as Level 1

Follow-up (every 14 days or earlier as per the need)

Referral criteria

- Same as mentioned earlier at Level 1, plus
- Psychological imbalance
- Any red flag signs.
- Signs of CVD as stroke, transient ischaemic attack, and angina.

At Level 3- Ayush hospital attached to teaching institute, district level/ integrated state Ayush hospital, tertiary care hospital, tertiary care allopathic hospital having Ayush facilities), multiple departments/ facilities for diagnosis and interventions. Must provide additional facilities like dieticians, counselling, exercise therapy).

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 exercise

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 identification of causes, and the 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.

Table 9:

S. No.	Drugs	Dosage form	Dose	Time	Duration	Adjuvants	Indications
1	Vyoshadi Guggulu (A.H)	Tablet	1 gm	TID (A/F)	1-3 months	Lukewarm water	Associated with ama and vatavikara like pain dominant conditions
2.	Ayaskriti	Arishta	20 ml	BD (A/F)	1 month	Warm water	Vibandha, deepanapachana, medohara, Hridya
3.	Sthoulyahara Kashaya (Sahasra Yoga)	Kashaya	15-20ml	BD (B/F)	1 month	60ml lukewarm water	Medovridhi/ Sthoulya
4.	Medohara Vidangadi Lauham	Tablet	250-500 mg	OD/BD (A/F)	1 month	Warm water	Pandu associated with Medovridhi
5.	Tryushanadi Lauham	Tablet	500-750 mg	BD (A/F)	1 month	Warm water/honey	Medovridhi with diabetes, skin disease, digestive disorders
6	Asanadi Kashaya	Kashaya	15 -20 ml	BD (B/F)	1 month	60ml lukewarm water	Medovridhi/ Sthoulya

Rasayasna like Brahma Rasayana and Pippali Rasayana and others can also be added

Purification/ other procedures

- Udvarthana: In kaphavrudhi/Medovruddhi/Obese conditions
- Lekhaneya Basti
- Virechana

Referral Criteria

- Same as mentioned earlier at Level 2, plus
- 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 dyslipidemias (FH& High Lp(a))
- Suspected Polycythemia
- Other modalities may be considered depending on the case and to rehabilitate properly

REFERENCES

1. de Ferranti SD, Newburger JW. Dyslipidemia in children and adolescents: Definition, screening, and diagnosis. UpToDate, Waltham, MA, USA. 2020.
2. World Health Organization. Global health observatory data repository Geneva: World Health Organization;2018.
Available: <https://apps.who.int/gho/data/view.main.2467?lang=en> [Accessed 3 Sep 2024]
3. 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
4. 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
5. 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.
6. 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 4 Mohamed-Yassin M-S, et al. *BMJ Open* 2021;11:e049662. doi:10.1136/bmjopen-2021-049662 Open access from the European atherosclerosis Society consensus panel. *Eur Heart J* 2017; 38:2459–72.
7. 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.
8. 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
9. 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.
10. Berberich AJ, Hegele RA. A modern approach to dyslipidemia. *Endocrine reviews.* 2022 Aug 1;43(4):611-53.
11. 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.
12. Carmena R. Primary Mixed Dyslipidemias, Editor(s): Ilpo Huhtaniemi, Luciano Martini, Encyclopedia of Endocrine Diseases (Second Edition), Academic Press, 2019, Pages 314-319, ISBN 9780128122006, <https://doi.org/10.1016/B978-0-12-801238-3.65333-3>
13. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. *Ann Intern Med.* 1971 Sep;75(3):471-2. Lugo-Somolinos A, Sánchez JE. Xanthomas: a marker for hyperlipidemias. *Bol Asoc Med P R.* 2003 Jul-Aug;95(4):12-6.
14. KashinatahSashtri and Gangasahay Pande. Charaka Samhita: Translated into Hindi. 1st ed. Sutra Sthana, Chapter 21, Verse 21/10. Varanasi: Chowkhamba Vidya Bhawan; 1962.
15. Shastri Ambika Dutta, Sushrut Samhita, Purvardha, Edition: Reprint 2010, Sutra Sthana, 15/14 Varanasi: Chaukhambha publication; 2012.
16. Shastri Ambika Dutta, Sushrut Samhita, Purvardha, Edition: Reprint 2010, ShariraSthana, 9/12 Varanasi: Chaukhambha publication; 2012.
17. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. *Ann Intern Med.* 1971 Sep;75(3):471-2. Lugo-Somolinos A, Sánchez JE. Xanthomas: a marker for hyperlipidemias. *Bol Asoc Med P R.* 2003 Jul-Aug;95(4):12-6.
18. Karantas ID, Okur ME, Okur NÜ, Siafaka PI. Dyslipidemia Management in 2020: An Update on Diagnosis and Therapeutic Perspectives. *EndocrMetab Immune Disord Drug Targets.* 2021;21(5):815-834.

19. 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.
20. Pappan N, Awosika AO, Rehman A. Dyslipidemia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; March 4, 2024.
21. Loscalzo J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Harrison's principles of internal medicine. 2022 May.
22. 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>
23. 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. *Endocrpract*. 2017;23(suppl 2):1-87. Doi: 10.4158/ep171764.appgl
24. World health organization. Obesity: preventing and managing the global epidemic: report of a WHO consultation.
25. 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.
26. 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.
27. Ministry of Ayush. Essential List of Medicine for Ayurveda. Page 21. Available from: https://eaushadhi.gov.in/ayush/download/external/Notification/Essential_Ayurvedic_Medicines.pdf Accessed January 29, 2025.
28. Ministry of Ayush. Essential List of Medicine for Ayurveda. Page 21. Available from: https://e-aushadhi.gov.in/ayush/download/external/Notification/Essential_Ayurvedic_Medicines.pdf Accessed January 29, 2025.
29. Ministry of Ayush. Essential List of Medicine for Ayurveda. Page 13. Available from: https://e-aushadhi.gov.in/ayush/download/external/Notification/Essential_Ayurvedic_Medicines.pdf Accessed January 29, 2025.
30. A Clinical Guide for Assessment and Prescription of Exercise and Physical Activity in Cardiac Rehabilitation. A CSANZ Position Statement Verdicchio, Christian et al. *Heart, Lung and Circulation*, Volume 32, Issue 9, 1035 – 1048
31. National List of Essential Ayush Medicines (NLEAM). 1st ed. Ministry of AYUSH, AYUSH Bhawan, B Block, GPO Complex, INA, New Delhi – 110023: Ministry of AYUSH, Government of India, New Delhi. www.ayush.gov.in; 2022. In.

CHAPTER

3

GOUT



GOUT

ICD 10 code: M10

ICD-11 TM 2: SP14

Vatarakta (National Ayurveda Morbidity Code: ED-8)

CASE DEFINITION^{1,2,3}

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.

Vatarakta: This disorder is characterized by chronic joint and body pain along with stiffness, swelling over joints due to vitiated *Vatadosha* as well as *Raktadhatu*.

Introduction (incidence/ prevalence, morbidity/mortality/risk factors)^{4,5,6}

- 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.
- 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, menopause.^{7,8,9,10}

Vatarakta is a disease produced by the vitiation of *Vata* and *Rakta*. *Vata* as well as *Rakta* get aggravated due to their own aggravating factors. When this *Vatadosha* gets obstructed due to aggravated *Raktadhatu* in its path, it gets more vitiated, causing further vitiation of *Raktadhatu*, resulting in the development of the disease *Vatarakta*. *Adhyavata*, *Khudha-Vata*, *Vatabalasa* and *Vatashonita* are the names used to refer the illness *Vatarakta*. According to Acharya Charaka, vitiated *Vata* and *Rakta* affect the joints of hands, feet, fingers and other joints slowly. Hands and feet are the sites of first attack and there after whole body comes under the grip of the disease.

CLINICAL EXAMINATION¹¹

The signs and symptoms of gout usually 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.
- **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. 1¹²: (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).



Fig. 2¹³: 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.

DIFFERENTIAL DIAGNOSIS^{14,15,16,17,18,19,20}

Table 1: The following diseases must be considered in differential diagnosis of acute gout:

Condition	Differential Features
Septic arthritis	• Knee is most commonly involved (may be any joint distribution) • Synovial fluid findings: ◦ 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	• Knee, wrist, or first metatarsophalangeal joints are commonly involved. • Synovial fluid findings: ◦ 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	• 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	• 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.
Reactive arthritis	• 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	• 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	• 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	• Centesis of the tenosynovial sheath and microscopic examination should be encouraged in acute tenosynovitis as gout flares may mimic infectious tenosynovitis.

Table 2:Differential diagnosis of Vatarakta

Features:	Vatarakta	Sandhigatavata	Amavata
Ama Lakshna	Not pathognomonic	Not pathognomonic	Present
Jwara	Present	Absent	Present
Vedana (Pain)	Mushika Damsavata Vedena (Pain like rat bite) Episodic (<i>Punahpunah Utpatti & Shamana</i>)	<i>Prasarane Akunchane</i> (mainly during move- ments of affected joint)	<i>Vrischika Damshavat</i> <i>Sanchari</i> (Intolerable pain which persists even during rest)
Shopha (Swelling)	Inflammatory changes involving single joint or multiple small joints and occurring in episodes.	<i>Vatapurna Druti Sparsha</i> (Feel of air being trapped in the affected joint)/soft swelling	<i>Sarvanga Sandhigata</i> (Inflammatory changes on the affected joint usually involving multiple joints)
Joints afflicted (<i>Sandhi</i>)	Smaller Joints mainly of hand and foot	Usually, asymmetrical weight bearing joints	Multiple and symmetrical small joints

SUPPORTIVE INVESTIGATIONS^{21,22,23}

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 listed in **Table 3**:

Test	Comment
Essential	
Serum urate concentration	Level may go down in few cases during an acute attack (serum uric acid levels ≤ 6 mg/dL)
Advanced	
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. Fig. 3²³

Test	Comment
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.
Synovial fluid examination	Presence of MSU crystals in the synovial fluid (SF) by polarizing microscopy
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	Hyperlipidemia, diabetes mellitus, hypothyroidism, and possibly hyperthyroidism is 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^{5,11}

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).
- **Acute gouty attack:** Classically, it produces an acute mono-arthritis 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 2015²⁴

STEP 1- Entry Criterion: If yes, Classification criteria required for positive diagnosis

≥ 1 episode of swelling, pain or tenderness in a peripheral joint/ bursa

STEP2- 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 4:

	Criteria	Categories	Score
Clinical	Pattern of joint/bursa involvement during symptomatic episode(s) ever	Ankle or midfoot	1
		Involvement of the first metatarsophalangeal joint	2
	Characteristics of symptomatic episode(s) ever 1. Erythema overlying affected joint 2. Cannot bear touch or pressure to the affected joint 3. Great difficulty with walking or inability to use the affected joint	One characteristic	1
		Two characteristics	2
		Three characteristics	3
	Time course of episode(s) ever • Time to maximal pain < 24 hours • Resolution of symptoms in ≤ 14 days • Complete resolution (to baseline level) between symptomatic episodes	One typical episode	1
		Recurrent typical episodes	2
	Clinical evidence of tophus	Present	4
Laboratory	Serum urate: measured by the uricase method (mg/dL)	< 4	-4
		6 to < 8	2
		8 to < 10	3
		≥ 10	4
	Synovial fluid analysis of a symptomatic (ever) joint or bursa	Monosodium urate crystal negative	-2
Imaging	Imaging evidence of urate deposition in symptomatic (ever) joint or bursa: ultrasound evidence of double-contour sign or DECT demonstrating urate deposition	Present (either modality)	4
	Imaging evidence of gout-related joint damage: conventional radiography of the hands or feet shows at least one erosion	Present	4
Total			23

A threshold score of ≥ 8 classifies an individual as having gout.

According to Charaka, *Vatarakta* is classified according to *Avastha* and *Dosha*.^{25,26}

➤ *Avastha Bheda*²⁷

Uttana Vatarakta

One which involves the *Uttana dhatu* such as *Rasa (tvak)*, *Rakta* and *Mamsa*. Characterised by *Kandu* (Itching), *Daha* (Burning sensation), *Ruja* (Pain), *Sira Ayama* (Dilatation of the vessels), *Toda* (Pricking pain), *Sphuranam* (Throbbing sensation), *Kunchana* (Contraction),

Shyava Twak (Cyanosis of the skin), *Rakta Twak* (Reddish coloration of skin), *Bheda* (Splitting type of pain), *Gourava* (Heaviness), *Suptata* (Numbness).

Gambhira Vatarakta

One which involves the *Gambhira Dhatu* such as *Meda*, *Asthi*, *Majja*. *Gambhira vatarakta* is Characterised by *Svayathu Stabdata* (Swelling, which is fixed), *Svayathu Kathinya* (Swelling with indurations), *Arti* (Deep pain), *Shyavata* (Black discoloration), *Tamra Twak* (Coppery discoloration of skin), *Daha* (Burning sensation), *Toda* (Pricking sensation), *Sphurana* (Throbbing sensation), *Paakavaan* (Inflammation which leads to ulceration).

Ubhayashrita Vatarakta

When there is presence of symptoms of both *Uttana* as well as *Gambhira Vatarakta*, it is called as *Ubhayashrita Vatarakta*. This is characterised by *Ruja* (Pain), *Vidaha* (Burning sensation), *Sandhi-Asthi-Majja Chinndni* (Cutting like pain in *Sandhi-Asthi-Majja*), *Angasya Vakrikarana* (Disfigurement of the joints), *Khanjatwa* (Lameness), *Pangutwa* (Paraplegia), *Vatasya Sarva Shareera Charana* (Vitiated Vata moves all over the body).

➤ **Dosha bheda:**

According to the involvement of *Dosha*, one more classification of *Vatarakta* has been discussed. Classical texts give the classification as: *Eka doshaja*, *Dvidoshaja* and *Sannipataja*. Hence classification will be as follows, *Vataja*, *Raktaja*, *Pittaja*, *Kaphaja*, *Dvandvaja* and *Sannipataja*.

Table 5: Lakshana of different types of Vatarakta.

Sr. No.	Vataja Vatarakta	Pittaja Vatarakta	Kaphaja Vatarakta	Raktaja Vatarakta	Dvandavaja Vatarakta
1.	<i>Ayama</i> (mainlyin Sira)	<i>Vidaha</i> (Burning sensation)	<i>Staimitya</i>	<i>Shwayathu</i>	<i>Vata-Pittaja</i>
2.	<i>Shoola</i> (Pain)	<i>Vedana</i> (Pain)	<i>Gauravama</i>	<i>Atiruka</i>	<i>Pitta-Kaphaja</i>
3.	<i>Sphurana</i> (Throbbingpain)	<i>Murcha</i>	<i>Sneha Snigdhata</i>	<i>Toda</i>	<i>Vata-Kaphaja</i>
4.	<i>Toda</i> (Prickingpain)	<i>SwedaAdhikya</i> (Excessives weating)	<i>Supti</i>	<i>Tamra Varna</i>	
5.	<i>Shotha Shyavata</i> (Bluishcolor)	<i>TrishnaAdhikya</i> (Excessivethirst)	<i>Manda Vedana</i>	<i>Chimchimayata</i>	
6.	Changeincolor of <i>Shotha</i> and <i>Vridhhi</i> or <i>Hani</i>	<i>Mada</i> (Narcosis)	<i>Shitalta</i>	<i>Snigdha Ruksha Sama Abhava</i>	
7.	<i>Ruksha</i>	<i>Bhrama</i> (Giddiness)	<i>Kandu</i>	<i>Kandu</i>	

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.

Prevention Management

- Nidana Parivarjana:

Avoidance of causative factors e.g. unhealthy diet and lifestyle, postural causes etc

- Diet and Life Style

First line of treatment in all disorders is correction of Agni by avoiding the causes and a few modifications in diet and lifestyle.

Table 6: Pathya Apathya^{28,29}

Dos	Don'ts (Disease aggravating factors)
Intake of Properly cooked and fresh food. Intake of food in appropriate quantity.	Excessive intake of dried/preserved/frozen foods,
Old cereals like wheat, red rice and barley	Refined foods such as bakery products made of white flour.
Pulses like green gram, chick pea, red lentil	Excessive intake of pulses like black gram, horse gram and white pea.
Fruits like <i>Draksha</i> (grapes, Dried Grapes) and Ash gourd.	Cold beverages like cold drinks, liquor and cold water.
Vegetables like bitter gourd, elephant-foot yam and pointed gourd	Excessive intake of radish, flat beans and betel leaf. Vegetables prepared with less or no oil or ghee. Excessive intake of meat of aquatic/marine animals (<i>Anupa Mamsa</i>)
Spices like rock salt, black pepper and asafoetida.	Excessive intake of sour and pungent food, acrid (<i>Atikatu</i>)
Judicious intake of milk, Ghee and butter made from indigenous Cow or Goat	Excessive intake of Curd, Curd products and sugarcane juice.
Herbs like <i>Kakamachi</i> , <i>Shatavari</i> , <i>Vastuka</i> (<i>Bathua</i>), <i>Upodika</i> (<i>Malabar Spinach/poi</i>) <i>Tanduliya</i> (<i>Amaranth greens /Chaulai</i>), <i>Dhatrphala</i> (<i>Amla/Indian gooseberry</i>), <i>Shringavera</i> (<i>Ginger</i>),	Incompatible food items like having fruits with milk.
Internal and external oiling (<i>Snehana</i>) in Vata dominant condition, Deep muscular massage (<i>Mardana</i>), <i>Upanaha</i> ,	Suppression of natural urge especially of hunger, bowel, urine and emotions.

Dos	Don'ts (Disease aggravating factors)
Affected parts are given Sheeta or Ushna Parisheka(Pouring of cool/hot decoction over body) according to the Doshic involvement Medicated Poultice (<i>Pradeha</i>), Gentle massage (<i>Mridusamvahana</i>).	
Appropriate exercises and adequate amount of rest (<i>sukhashayana</i>).	Day time sleeping (<i>Diwaswapna</i>), excessive exercise (<i>Vyayama</i>), activities causing increase in body temperature (<i>Santapa</i>) like Sun bath (<i>Aatapa Sevana</i>) and excessive indulgence in sexual intercourse (<i>Maithuna</i>).

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.

Curative Interventions

At Level 1: (Solo Physician Clinic/Health & Health Clinic/PHC (Optimal Standard of treatment in 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

In the initial stage when the patient is having mild symptoms and slightly elevated uric acid, following medicines may be given along with diet restriction and lifestyle modification.

Table 7: Single drugs/Compound Formulations for internal/external medication

S.No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Anupaana
1.	<i>Guduchi kwatha</i> ³⁰	Decoction	60-80 ml in two divided doses	Before meal	Thrice a day for 2-3 months for aam conditions	Lukewarm Water
2.	<i>Guduchi</i> ³¹ Swarasa	Swarasa	10-20 ml	Before meal/	Thrice a day for 2-3 months for aam conditions	-
3.	Kokilaksham Kashayam	Decoction	20-40 ml	Before meal	Thrice a day for 2-3 months	Lukewarm Water
4.	Laghu Manjisthadi Kwatha	Decoction	20-40 ml	Before meal	Thrice a day for 2-3 months	
5.	<i>Triphala kwatha</i> ³²	Decoction	60-80 ml in two divided doses	Before meal	Thrice a day for 2-3 months for aam conditions	-

S.No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants/ Anupaana
6.	<i>Rasnaerandadi Kvatha</i> ³³	Decoction	60-80 ml in two divided doses	Before meal	Thrice a day for 2-3 weeks	-
7.	<i>Patoladi Kwatha</i>	Decoction	60-80 ml in two divided doses	Before meal	Thrice a day for 2-3 weeks	-
8.	<i>Kaishora Guggulu</i> ³⁴	Vati	1-3 gm in 2-3 divided doses	After meal	Twice/Thrice daily for 2-3 weeks	Warm Water
9.	<i>Amrutadi Guggulu</i>	Vati	500 mg-1 gms in 2-3 divided doses	Before meal	thrice a day, 2-3 weeks	Warm water
10.	<i>Gokshuradi Guggulu</i>	Vati	500 mg-1 gms in 2-3 divided doses	Before meal/ after meal	thrice a day, 2-3 weeks	Warm water
11.	<i>Punarnava Guggulu</i> ³⁵	Vati	1-3 gm in 2-3 divided doses	After meal	2-3 weeks	Warm Water
12.	<i>Nimbadi Churna</i> ³⁶	Powder	1-3 gm	After meal	2-3 weeks	<i>Guduchikvatha</i> , Warm water
13.	<i>Eranda taila</i>	Taila	Q.S	1-2 times	2-3 weeks	-
14.	<i>Pinda taila</i>	Taila	Q.S	1-2 times	2-3 weeks	-
15.	<i>Ksheerbala taila</i>	Taila	Q.S	1-2 times	2-3 weeks	-
16.	<i>Amritadya taila</i>	Taila	Q.S	1-2 times	2-3 weeks	-
17.	<i>Balaguduchyadi taila</i>	Taila	Q.S	1-2 times	2-3 weeks	-
18.	<i>Swayambu guggulu</i>	Vati	1-3 gm in 2-3 divided doses	After meal	2-3 weeks	Warm Water
19.	<i>Mahatiktaka ghrita</i> ,	Ghrita	Q.S	1-2 times	2-3 weeks	-
20.	<i>Guggulutiktaka ghrita</i>	Vati	5-10 gms in 2-3 divided doses	After meal	2-3 weeks	Warm Water

Recommended Diet & Lifestyle

As mentioned earlier

Restricted Diet & Lifestyle

As mentioned earlier

Follow Up

Every 15 days or earlier as per the need and the medicines can be continued upto one month with consultation.

Review should include:

- Monitoring the person's symptoms and the on-going 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.

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

Clinical Diagnosis

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:

1. Serum urate concentration
2. Complete blood count/ESR
3. Renal function Test
4. Fasting lipids, glucose, and thyroid functions
5. Urinary urate excretion

Management

- *Koshta Shuddhi* with any of the following medicines before administration of palliative treatment
 - *Avipattikara Churna*³⁷(5-10-gm) with warm water at bed time
 - *Eranda taila* (Castor oil 10-15 ml at bed time)
- In addition to the medicines mentioned at Level 1, any of the following medicines may be added as appropriate.

Table 8: Compound Formulations for internal/external medication

S.No.	Drugs	Dosage form	Dose	Time	Duration and Frequency	Adjuvants / Anupaana
1.	Brhatmanjistadi Kwatha ⁹	Decoction	60-80 ml in two divided doses	Before meal	thrice a day for 2-3 weeks	-
2.	<i>Simhanada Guggulu</i> ³⁸	Vati	1-3 gm in 2-3 divided doses	After meal	2-3 weeks	Warm water
3.	<i>Panchtikta Guggulu Ghrita</i> ³⁹	Ghee	5-10 gm twice daily	Before meal	2-3 weeks	Milk, warm water
4.	<i>Sukumara Ghrita</i> ⁴⁰	Ghee	10-15 gm twice daily	Before meal	2-3 weeks	Milk, warm water
5.	<i>Jivaniya Ghrita</i> ⁴¹	Ghee	10-12 gm twice daily	Before meal	2-3 weeks	Milk, warm water
6.	Grihadhumadi Lepa	Churna	Q.S	Once daily	15 days	External application
7.	Nagaradi Lepa	Churna	Q.S.	Once daily	15 days	External application
8.	Dashamoola Ksheera	Kshira Paka	Q.S.	Once daily	15 days	External application
9.	Sahacharadi Taila/Pinda Taila / Dhanvantaram Taila/ Ksheerabala Taila	Taila	Q.S.	Once daily	15 days	External application

Panchakarma Procedures/ other Upakrama

- *Lepa – Grihadhumadi*⁴², *Jadamayadi*
- *Parisheka - Dashamoola Ksheera Parisheka, Taila Parisheka* (In case of *Stambha Akshepa Shula*) – *Sahacharadi*⁴³, *Pindataila*⁴⁴, *Dhanvantaram Taila*⁴⁵, *Ksheerabala Taila*
- *Abhyanga with suitable oil as mentioned above as per Ama/Nirama Avastha.*

Recommended Diet & Lifestyle

Same as level 1

Restricted Diet & Lifestyle

Same as level 1

Follow Up

Every 15 days or earlier as per the need.

Referral Criteria

- Same as mentioned earlier at level 1, plus
- 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.

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, sophisticated procedures like *Agnikarma*, *Ksharakarma* etc. (as applicable)

Clinical Diagnosis

Same as level 1 and 2.

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

Management

In addition to the medicines mentioned at Level 1 and Level 2, any of the following medicines may be added as appropriate.

Table 9:

S.No.	Drugs	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ Anupaana
1.	Shilajatu Rasayana	Churna	500 mg – 1 gm	Early morning empty stomach	2-3 months	GuduchiKwatha
2.	Pippali Rasayana	Kshirapaka	3 Pippali in increasing dose upto 33 Pippali and reverse	Early morning empty stomach	22 days	Milk

S.No.	Drugs	Dose form	Dose	Time	Duration and Frequency	Adjuvants/ Anupaana
3.	Madhusnuhi Rasayana	Avaleha	6-12 gm	Once/ twice daily	1-3 months	Warm water
4.	Chyavanaprasha	Avaleha	12-24 gm	morning	1-3 months	warm water/ milk
5.	Dashamulaharitaki Rasayana	Avaleha	5-15 gm	morning	1-3 months	warm water/ milk

Panchakarma Procedures/ other Upakrama

In addition to the management of Level 1 and Level -2, if needed Panchakarma procedures indicated for Vatarakta can be performed..

Shodhana Chikitsa

a. Virechana Karma

- *Deepana pachana* - *Panchkola churna, Trikatu churna*
- *Snehapana* – *Saindhavadi taila*⁴⁶, *Accha Ghrita* (plain ghee) (Up to *Samyak snighdha Lakshana*) maximum for 7 days.
- *Mrudu Abhyanga* & *Svedana*– *Ksirabala Taila, Pinda Taila*⁴⁷ (*Abhyanga*), *Bashpa Sveda* (*Nirgundi patra*)
- *Virechana*–According to *Agni, Koshtha* and availability of medicine like *Trivruta Avaleha, Abhayadi modaka, Eranda taila*.
- *Samsarjana*– As per *Shuddhi lakshana* (3,5 or 7 days)

b. **Rakta Mokshana**- Leech therapy on painful and swollen joint (predominant pitta and Rakta involvement).

c. Basti –

1. *Matrabasti* – *Madhuyasti taila, Brihatsaindhavadi Taila*⁴⁸ in painful condition, *Amavastha*.
2. *TiktaKshira Basti*- *Pitta Rakta* involvement, *Raktaprasadana, Kledaharana, Rasayana*
3. *Yapana Basti* – In *gambhira vatarakta/* No or less response to oral medications (*Guduchyadi Yapana, Madhutailika Basti*)

Recommended Diet & Lifestyle

- Same as level 1 & 2.

Restricted Diet & Lifestyle

- Same as level 1 & 2.

Follow Up

- Every 15 days or earlier as per the need.

Referral Criteria

- Same as mentioned earlier at level 2, plus
- Other modalities can be considered depending on the case.

REFERENCES

1. Neogi T. Clinical practice. Gout. *N Engl J Med*. 2011;364(5):443-452
2. Davidson S, Bouchier I, Edwards C. Davidson's principles and practice of medicine. 21st ed. London: E.L.B.S. and Churchill Livingstone;1991
3. Dalbeth N, Merriman TR, Stamp LK. Gout. *Lancet*. 2016;388(10055):2039-2052.
4. 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.
5. Kumar S, Gupta R, Suppiah R. Gout in women: differences in risk factors in young and older women. *NZMJ*.2012;125(1363):39-45.
6. Paul BJ, James R. Gout: an Asia-Pacific update. *Int J Rheum Dis*. 2017; 20(4): 407-416.
7. Singh JA, Reddy SG, Kundukulam J. Risk factors for gout and prevention: a systematic review of the literature. *Curr OpinRheumatol*. 2011;23(2):192-202.
8. Roddy E, Doherty M. Gout. *Epidemiology of gout. Arthritis Research & Therapy*. 2010; 12(6):223
9. Saag KG, Choi H. Epidemiology, risk factors, and lifestyle modifications for gout. *Arthritis Res Ther*.2006;8(Suppl 1):2
10. 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.
11. Grassi W, Angelis RD. Clinical features of gout. *Reumatismo*.2011; 63(4):238-245.
12. Jelley MJ, Wortmann R. Practical Steps in the Diagnosis and Management of Gout. *BioDrugs*. 2000; 14 (2): 99-107.
13. 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.
14. Eggebeen AT. Gout: An Update. *American Family Physician*.2007;76(6):801-808.
15. 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-fofosteoarthritis?Search=osteoarthritis&source=search_result&selectedtitle=2~150&usage_type=default&display_rank=2
16. Doherty M, Lanyon P, Ralston SH. Musculoskeletal Disorder. In Boon NA, Colledge NR, Walker BR. (Ed.) Davidson's Principles & Practice of Medicine; 21st edition. Philadelphia. Elsevier Ltd. 2010
17. Diagnosis, Osteoarthritis: Care and Management in Adults. Clinical guideline CG177 Methods, evidence, and recommendations. February 2014. National Clinical Guideline Centre, 2014. [cited 02 Apr. 2019]; Available at: <https://www.ncbi.nlm.nih.gov/books/NBK333067/>
18. Abraham S, Patel S. Monoarticular Arthritis. [Updated 2023 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK542164/>
19. Williams CH, Jamal Z, Sternard BT. Bursitis. [Updated 2023 Jul 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK513340/>
20. Pirker IFJ, Rein P, von Kempis J. Important differential diagnosis in acute tenosynovitis. *BMJ Case Rep*. 2019 Jan 10;12(1):bcr-2018-228373. doi: 10.1136/bcr-2018-228373. PMID: 30635314; PMCID: PMC6340559.
21. Underwood M. Diagnosis and management of gout. *BMJ*. 2006;332(7553):1315-1319.
22. Schlesinger N, Norquist JM, Watson DJ. Serum urate during acute gout. *J Rheumatol*.2009; 36(6):1287-89

23. 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.
24. 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.
25. Priya Pathak, Nitu Tegta, Ankush Jagota, Umesh Shukla, Rajika Gupta and T. C. Thakur.
26. An Ayurvedic perspective of Vatarakta. *World Journal of Pharmaceutical and Medical Research.* 2020,6(8), 344-356
27. Vikas Rana, Anjana Mishra, B.L.Mehra. A Comprehensive Study of Vata Rakta w.s.r. to Gout. *International Journal of Ayurveda and Pharma Research.* 2017;5(8):93-96.
28. AcharyaJadavajiTrikamji, Charaka Samhita, Chakrapanidatta, Chikitsasthana 29/19, reprint, AyurvedaDipika,Commentary,Chaukhamba Prakashan:2007
29. Savitri Soni. Vatarakta : An Ayurvedic classical literature review. *J Ayurveda Integr Med Sci* 2023;06:215-229.
30. Akansha Singh, Shivani Mahajan, Amit Tiwari, Ketan Mahajan. A Conceptual Study on Vataraktaw.s.r. to Gouty Arthritis. *Ayushdhara [Internet].* 2024Jul.10;11(3):70-8.
31. AcharyaJadavajiTrikamji, CharakaSamhita, Chakrapanidatta, Chikitsasthana 29, reprint, AyurvedaDipika,Commentary,Chaukhamba Prakashan:2007
32. AcharyaJadavajiTrikamji, CharakaSamhita, Chakrapanidatta, Chikitsasthana 29, reprint, AyurvedaDipika,Commentary,Chaukhamba Prakashan:2007
33. AcharyaVrinda, *Vrindamadhava*,edited and translated byPremvati tewari,1st Edition, ChaukhambhaVisvabharati, Varanasi, 2007
34. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:197
35. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:207
36. Anonymous, The Ayurvedic Formulary of India, Part 2, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003
37. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:321
38. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:309
39. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:223
40. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:270
41. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:301
42. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:301
43. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:485

44. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:456
45. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:404
46. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:394
47. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:458
48. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:404
49. Anonymous, The Ayurvedic Formulary of India, Part 1, Ed. 2nd, Govt. of India, Ministry of Health and Family Welfare, Department of I.S.M. & H., New Delhi, 2003:428
50. National List of Essential Ayush Medicines (NLEAM). 1st ed. Ministry of AYUSH, AYUSH Bhawan, B Block, GPO Complex, INA, New Delhi – 110023: Ministry of AYUSH, Government of India, New Delhi. www.ayush.gov.in; 2022. In.
51. Ministry of Ayush. Ayurvedic Standard Treatment Guidelines. 1st ed. New Delhi. Government of India. 2017; p. 79-85.



CHAPTER

4

NON ALCOHOLIC FATTY LIVER DISEASE



NON-ALCOHOLIC FATTY LIVER DISEASE

ICD-10-CM Diagnosis Code K76.0

ICD 11 CO DE: DB92

Non-Alcoholic Fatty Liver Disease (NAFLD)

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^[1]. An international group of expert consensus statement suggested to change the name to Metabolic-associated Fatty Liver Disease (MAFLD)^[2]. 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^[3].

Specifically, NAFLD has not been mentioned in Ayurveda, but it has its own principles of understanding the etiopathology of the diseases and their treatments. NAFLD although is the disease condition where yakrit (liver) is affected and results into Yakrutodara (Yakriddalyudara) yet the disease can be attributed to agnimandya occurs due to mithya-ahar vihara and Prajnaparadha. Agnimandya is inclusive of poor digestion, poor or disturbed absorption, disturbed metabolism and excretory mechanism^[4].

INTRODUCTION

- 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)^[5,6].
- The prevalence of NAFLD in India varies from 9-35% as per the accordance to ultrasonography data^[7,8]. Studies demonstrated area-wise prevalence data of NAFLD with 16.6 % in Western India, 24.5 % in Eastern India, and 32 % in South India^[7].
- 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^[3].
- 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^[8].
- 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^[8].
- NAFLD is known to be associated with several extrahepatic conditions like chronic kidney disease (CKD)^[9], cardiovascular diseases^[10-12], osteopenia, osteoarthritis^[13], obstructive

sleep apnoea^[14], hypothyroidism^[15], and polycystic ovarian syndrome^[16,17]. NAFLD has also been shown to increase the risk of extrahepatic malignancies like carcinoma colon, gastric cancer, carcinoma pancreas, uterine, and breast conditions^[18].

- 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^[19].

Even though Non-Alcoholic Fatty Liver Disease (NAFLD) has not been directly mentioned in Ayurveda, owing to the clinical presentations, it is similar to symptoms exhibited by Medodusti/Medoroga which is a Santarpanajanya Vyadhi. Heavy fat rich diet, junk food, soft drinks, sedentary lifestyle, Metabolic syndrome (Obesity, Diabetes Mellitus, Dyslipidaemia), drugs (eg: Corticosteroids, Aspirin, Tetracycline) etc. are considered to be the major etiological factors of NAFLD. As per Ayurvedic concept, heavy fat rich diet (Guru and Ati-snigdha), and sedentary lifestyle (Not following Dinacharya and Ritucharya) are responsible for the vitiation of Annavaha, Rasavaha, Raktavaha and Medovahasrotas.^[20]

Ajirna, Sthaulya and Prameha (Diabetes Mellitus) which occurs due to the vitiation of Annavaha, Rasavaha and Medovaha Srotas acts as Nidanarthakara Rogas which may result in the manifestation of Fatty Liver.

Being a Santarpanottha vikara, Nidana and Samprapti of this disease are similar to those of Sthoulyaroga. Initially Agni vikriti leads to the formation of Ama dosha, which vitiates Kapha dosha and Meda dhatu and gets accumulated in the Yakrit. This condition is called as Fatty Liver. Vitiating Kapha dosha and Meda dhatu results in srotorodha resulting into aggravation of Vata dosha. Vitiating Vata dosha further causes Agnivikriti and this cycle gets repeated⁹. Major factors responsible for the etio-pathogenesis of NAFLD include vitiation of Samanavayu, Apanavayu, Pachakapitta, Ranjakapitta, Kledaka kapha, Rasa Rakta Medo dhatu and Pureesha. Annavaha, Rasavaha, Raktavaha and Medovaha are the Srotas involved directly or indirectly in the causation and manifestation of NAFLD^[21].

The Sampraptighatakas of the NAFLD could be:

Dosha:	Vata pradhana Kapha and Vata pradhana Pitta
Dushya:	Meda, Rakta and Rasa
Srotas:	Medovaha, Annavaha, Rasavaha, Raktavaha
Agni:	Jatharagni Mandya, Medodhatvagni-Rasadhatvagni Mandya
Udbhava sthana:	Amashaya
Adhithana:	Koshthanga (Yakrit)

Usually, NAFLD is being presented in two ways:

- NAFLD in obese, which is more common and has better prognosis
- NAFLD in Non-obese, which has reasonably worse prognosis

However, in both the conditions deposition of Meda in Yakrit occurs leading to the formation of Non-alcoholic Fatty Liver Disease (NAFLD), Non-alcoholic steatohepatitis (NASH), Fibrosis or Cirrhosis of Liver condition.

CLINICAL PRESENTATION AND EXAMINATION

In the primary condition of NAFLD, the patients are presented with the symptoms of Ajirna viz. feeling of heaviness in the abdomen and abdominal distension, irregular appetite, irregular bowel habit, body ache and excessive belching. Further, the patients are presented with the symptoms like sour eructation, burning sensation in the chest and throat and sometimes loose stool, which is the specific symptom of formation of Ama dosha. In the further progression of the disease i.e. in case of Fibrosis and Cirrhosis condition, systemic features like pandu, kamala and raktapitta are produced. At last, Udara roga i.e. Yakritdaludara gets produced.

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^[22]. 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 contracture. On clinical examination, mild to moderate hepatomegaly may be the most common finding. Patients of NAFLD may often present with obesity and hypertension^[23]. 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^[24]. Patients with such risk factors must be screened as it has been observed that Metabolic syndrome is closely associated with NAFLD^[25].

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

<i>Macro-vesicular steatosis</i>	<i>Micro-vesicular steatosis</i>
Starvation	Inborn errors of metabolism
Parenteral nutrition	
Abetalipoproteinemia	
Medications like methotrexate and steroids	
Kwashiorkor	
Anorexia nervosa	
Personality Disorders	

SUPPORTIVE INVESTIGATIONS

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^[3]. Non-invasive tests remain the investigation of choice among the physicians and patients alike.

Table 3:

Investigations	Findings	
Essential		
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

Investigations	Findings
Advanced	
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 ^[26] .
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 ^[27]	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 ^[28]	AST and platelet levels	Hepatic fibrosis.
Fibrosis-4 score (Fib-4) ^[29]	AST, ALT, age, and platelets	Hepatic fibrosis
NAFLD fibrosis scores (NFS) ^[30,31]	BMI, Age, AST/ALT ratio, Albumin, and presence of insulin resistance and diabetes	Hepatic fibrosis
BARD score ^[31]	BMI, Age, AST/ALT ratio, and presence of diabetes	Hepatic fibrosis
Magnetic resonance elastography (MRE) and Fibrosis-4 score (MEFIB) ^[32]	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. ^[33]

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^[3,18,19]. 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 conducted to rule out alternate causes of hepatic steatosis.

PRINCIPLES OF MANAGEMENT

Red Flags

- NASH-associated cirrhosis
- End-stage liver disease
- Hepatocellular carcinoma (HCC)
- Uncontrolled co-morbidities
- LSM ≥ 20
- Platelet count $< 150 \times 10^6 / L$
- Portal hypertension
- Hepatic encephalopathy

General line of treatment as mentioned in Ayurveda

The Sannikrishta karana (nearest cause) of NAFLD is Agnivikriti, which causes kapha and medodusti leads to excessive accumulation of Kapha and Meda, thereby causing srotorodha. In such condition, Agnimandya is inclusive of poor digestion, poor or disturbed absorption, disturbed metabolism and disturbed excretory mechanism. As discussed earlier Yakrit is the seat of Pitta dosha and responsible for different types of dhatvagnipaka (metabolic processes). When Yakrit gets affected the agni (Jatharagni, dhatvagni and bhootagni) gets disturbed, which results into many non-communicable diseases including NAFLD. Therefore, the principle of treatment of NAFLD is Agnidipana, Amapachana, Srotoshodhana, kapha-medanashaka, rasa-raktaprasadana, virechaka and Yakritshothahara treatment.

Agnideepana (Stimulation of digestive fire), Amapachana, Srotosodhana (Removal of blockage of channels), pacification of Kapha, Meda and Vata should be the first line of treatment in the management of NAFLD. The principle of treatment implemented is usually similar to that of Sthaulya roga (Atisantarpanajanya vikara).

Judicious administration of all the procedures of treatment ie. **Nidana parivarjana** (both ahara and vihara), **Samsodhana**, and **Samshamana** will help in the setback of fatty changes of liver and prevention of further complications.

For Prevention of NAFLD

Agni is the key factor for manifestation of any kind of disease including NAFLD. Balanced Agni is essential for maintenance of healthy life, and diet play an important role to keep the Agni in balanced condition. One should take diet in proper quantity to maintain the balanced Agni³⁴. Following are the rules for taking diet:

- Diet should be taken as per 'Eight Rules of Eating' (अष्टआहारविधिविशेषआयतन) by Acharya Caraka³⁵.
 - Prakruti (nature of food)
 - Karan (methods of preparation of food)
 - Samyoga (combination of food substances)
 - Rashi (quantity of food)
 - Desha (place where food items are grown)
 - Kala (time of consumption of food)
 - Upyogasamstha (rules of consumption of food)
 - Upabhokta/Upyokta- (the person who consumes food)
- Diets which could be included are Shali, Shastika and Mudga. These are the diets which are light by nature due to the predominance of Vayu and Agni Mahabhoota and can be easily digestible³⁶.
- Therefore, ahara, viz. Food articles with excess unctuous, sweet, heavy and viscous substances, freshly harvested food grains, wines with the flesh of wetland and aquatic animals with cow's milk and its products and the products of jaggery must be avoided.³⁷
- Diet consisting mostly of dry foods, Priyangu (Italian millet) Shyamaka (sanwa millet), Yava (barley), kodrava (common millet), Mudga (green gram) Kulattha (horse-gram) etc., the seed of Adhaki (pigeon pea) mixed with wild snake-gourd and Emblic myrobalan should be used as food followed by Madhu udakam as drink such wines as are eliminative of fat, flesh and Kapha. Regular intake of barley and wheat and consumption of Ayurveda food recipe like Vyoshadi saktu are also effective.³⁷
- One should remain physically active, and avoid day-sleeping, over-indulgence in lounging and lying in soft beds (luxurious and mattress). Exercise, fasting, smoking and sudation are beneficial. Daily exercise or eating only after the previous meal has been digested is also effective³⁷.
- **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.

Interventions:

At Level 1: (Where optimal standard of treatment in situation where technology and resources are limited e.g. Solo Physician clinic/Community wellness centres/ PHC)

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 cases of hepatic steatosis must be ruled out by clinical history and available investigations.

Investigations

1. Blood for 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:

OPD level management – If the patient has Grade-I Fatty Liver, where the symptoms like heaviness of abdomen, constipation and flatulence are present, two or more of the following forms may be advised along with restriction of diet and physical exercise or yoga and Pranayama.

Table 5:

S.No.	Drugs	Dosage form	Dose	Time	Duration	Adjuvants
1.	Triphala churna	Churna	3-5grams	At Bed time	15 days	Luke warm water
2.	Eranda Bhrista Haritaki	Churna	5 grams	At Bed time	15 days	Luke warm water
3.	Vaiswanar Churna	Churna	1-3 grams	At Bed time	15 days	Luke warm water
4.	Drakshavaleha	Avaleha	6-12 grams	Twice daily after food	15 days	Luke warm water
5.	Phalatrikadi Kashaya	Kashaya	25-50 ml	Morning and evening before/after food	15 days	Luke warm water
6.	Vasa Guduchyadi Kashaya	Kashaya	25-50 ml	Morning and evening after food	15 days	Luke warm water
7.	Pippalyasava	Asava	12-24 ml	After Lunch and Dinner	15 days to 30 days	With equal quantity of water
8.	Arogyavardhini Vati	Vati	250-500 mg	Morning and evening before/after food	15 days	Luke warm water
9.	Chitrakadi Gutika	Gutika	250 -500 mg	Morning and evening after food	15 days	Luke warm water
10.	Siva Gulika	Gulika	1 gram	Morning and evening after food	15 days to 30 days	Luke warm water
11.	Triphala Guggulu	Guggulu	1 -3 gram	Morning and evening Be-fore/ food	15 days to 30 days	Luke warm water

The medicines has Agnidipana, Amapachana, Srotoshodhana, kapha-medanashaka, rasa-raktaprasadana, virechaka and Yakritshothahara property. Considering the status of Agni of the person medicines to be prescribed.

The above medicines may be continued as per follow-up upto three months.

Yoga: As mentioned above.

Restricted Diet and Life style: As mentioned earlier.

Follow up- Every 15 days or as per need.

Reviews should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.
- Standard Treatment Guidelines in Ayurveda on NAFLD
- Monitoring the long-term course of the disease condition.
- Management of NAFLD in terms of Physical exercise, and Shodhana therapy like Virechana
- Discussing the patient's knowledge of the condition and addressing any concerns they have, their personal preferences, and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.

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 bed hospitals with basic facilities such as routine investigations and X-ray

- Management with single herbs and compound formulations for internal and external use
- Bio-purification procedures
- Advice of Pathya Apathya
- Referral criteria
- Clinical Diagnosis – Same as level 1. The case referred from Level 1, or a fresh case must be evaluated thoroughly for any complications

Investigations:

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

Management –

For the patients referred from Level-1, treatment given in level-1 may be continued if appropriate for the presenting condition. For new cases at this level, the medications mentioned for Level-1 may also be considered while giving prescription. Any of the following medicines may be added as appropriate. In-patient management may be opted if necessary.

Table 6:

S.No.	Drugs	Dosageform	Dose	Time	Duration	Adjuvants
1.	Katuki Churna	Churna	3-5 grams	At Bed time	15 days	Luke warm water
2.	Trivrit Churna	Churna	2-3 gms grams	At Bed time	15 days	Luke warm water
3.	Avipattikar Churna	Churna	3-6 grams	At Bed time	15 days	Luke warm water
4.	Eranda BhristaHaritaki	Churna	5 grams	At Bed time	15 days	Luke warm water
5.	Patola Katurohinyadi Kashaya	Kashaya	30 ml	Morning and evening after food	15 days	Luke warm water
6.	Rohitakarista	Arista	12-24 ml	After Lunch and Dinner	15 days to 30 days	With equal quantity of water
7.	Shankha Vati	Vati	250-500 mg mg	Morning and evening after food	15 days	Luke warm water
8.	Kankayana Vati	Vati	500 mg-1 gm	Morning and evening after food	15 days to 30 days	Luke warm water
9.	Agnitundi Vati	Vati	250 mg	Morning and evening after food	15 days	Luke warm water

The above medicines may be continued as per follow-up upto three months.

As per the clinical severity of the patient the medicines of level 1 can also be added at this level

Yoga: As mentioned above

Bio-purification procedures like Panchakarma and other procedures:

- Basti Chikitsa (per rectal administration of lukewarm medicated oils or emulsions):
 - i. Matra Basti (per rectal administration of 60 ml of any of these medicated oils): Pippalyadi Anuvasana Taila, Ksheerabala Taila, Dhanwantaram Taila, Panchatikta Guggulu Ghrita etc.

- ii. Karma vasti or Kala Vasti or Yoga Vasti as appropriate may be given with Dasamulakwatha, Eranda Taila etc.
- Vatanulomana /Nitya Mridu Virechana with mild laxatives like Triphala Churna or Avipattikara Churna or Vaiswanar Churna or Drakshavaleha 5-10 gm daily at night with lukewarm water.

Recommended and restricted Diet & Lifestyle – as mentioned above.

Follow up – Every 15 days or earlier as per the need.

Referral Criteria:

- Same as mentioned earlier at level 1, plus
- Failure of acute exacerbation to respond to initial medical management
- Advanced stages of disease like Liver Cirrhosis

At Level 3: (Ayush hospitals attached with teaching institution, District level/State Ayush Hospitals, Tertiary care allopathic hospitals having Ayush facilities)

- Management with single herbs and compound formulations
- Bio-purification procedures
- Advice of Pathya Apathya
- Referral criteria

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:

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. For new cases at this level, the medications mentioned for Level-1 & 2 may be considered while giving prescription and any of the following medicines may be added as appropriate. Indoor management may be preferred if necessary.

Table 7: Single herbs/ compound formulation for NAFLD in Level 3

S.No.	Drugs	Dosage form	Dose	Time	Duration	Adjuvants
1.	Patoladi Churna	Churna	1-3 grams	At Bed time	15 days	Luke warm water
2.	Dhatri Lauha	Lauha	250-500 mg	Twice daily	15-30 days	Luke warm water
3.	Dasamula Haritaki Lehya	Avaleha	5-10grams	Twice daily	15-30 days	Luke warm water
4.	Chitraka Guda	Guda Paka	5-10grams	Twice daily	15-30 days	Luke warm water

As per the clinical severity of the patient the medicines of level 1 and 2 can also be added at this level

The above medicines may be continued as per follow-up upto three months.

Yoga: As mentioned above

➤ **Bio-purification procedures like Panchkarma and other procedures:**

Same as Level 2

Recommended and restricted Diet & Lifestyle – as mentioned above

Follow up – Every 15 days or earlier as per the need.

Referral Criteria:

- Same as Levels 1 & 2, plus,
- Hepatic encephalopathy
- Portal hypertension
- Haematemesis or melaena or any condition requiring blood transfusion or critical care management
- Other modalities can be considered depending on the case and to rehabilitate properly.

REFERENCES

1. 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.
2. 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>
3. 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.
4. Sanjeev Sharma & Asit Panja. Non-Alcoholic Fatty Liver Disease (NAFLD) and Ayurveda. *Science and Culture*. May-June, 2024, Vol.90, Nos. 5-6
5. 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 RelatDisord* 2022;20(3):166–73.
6. 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>
7. Duseja A, Chalasani N. Epidemiology and risk factors of nonalcoholic fatty liver disease (NAFLD). *Hepatol Int* [Internet] 2013 [cited 2021 Nov 24];7 Suppl2:S755–64. Available from: <https://pubmed.ncbi.nlm.nih.gov/26202291/>
8. 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.
9. 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>
10. 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>
11. 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>
12. 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.
13. 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>
14. 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>

15. Grewal H, Joshi S, Sharma R, Mittal P, Goel A. Non-alcoholic fatty liver disease in patients with hypothyroidism presenting at a rural tertiary care centre in north India. <https://doi.org/10.1177/0049475520945058> [Internet] 2020 [cited 2024 Aug 29];51(2):181–4. Available from: <https://journals.sagepub.com/doi/10.1177/0049475520945058>
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. Chakraborty S, Ganie MA, Masoodi I, Jana M, Shalimar, Gupta N, et al. Fibroscan 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
18. 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>
19. 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
20. Charaka Samhita, Dipika commentary by Chakrapanidatta edited by Vaidya Yadavji Trikamji Acharya, Published by Choukhambha Sanskrit Samsthana, 4th edition, Vimana sthana 5/11-16 & 21, page 251.
21. Remya. E. Non Alcoholic Fatty Liver Disease- An Ayurvedic Pragmatic Approach with Its Management. *International Journal of Ayurvedic & Herbal Medicine* 7(6) Nov.-Dec. 2017 (2948-2955)
22. 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://bmccendocrdisord.biomedcentral.com/articles/10.1186/s12902-022-00980-1>
23. 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/](https://pubmed.ncbi.nlm.nih.gov/14695401/)
24. 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/](https://pubmed.ncbi.nlm.nih.gov/14695401/)
25. Zohara Z, Adekun A, Seffah KD, Salib K, Dardari L, Taha M, et al. The Prospect of Non-Alcoholic Fatty Liver Disease in Adult Patients with Metabolic Syndrome: A Systematic Review. *Cureus* [Internet] 2023 [cited 2024 Sep 2];15(7). Available from: [/pmc/articles/PMC10427027/](https://pubmed.ncbi.nlm.nih.gov/14695401/)
26. 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/>
27. 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>
28. Loaeza-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.

29. 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.
30. 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>
31. 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/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3560810/)
32. 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>
33. 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/>
34. Charaka Samhita, Dipika commentary by Chakrapanidatta edited by Vaidya YadavjiTrikamji Acharya, Published by Choukhambha Sanskrit Samsthana, 4th edition, Sutrasthana 5/3, page 36.
35. Charaka Samhita, Dipika commentary by Chakrapanidatta edited by Vaidya YadavjiTrikamji Acharya, Published by Choukhambha Sanskrit Samsthana, 4th edition, VImanasthana 1/21, page 235
36. Charaka Samhita, Dipika commentary by Chakrapanidatta edited by Vaidya YadavjiTrikamji Acharya, Published by Choukhambha Sanskrit Samsthana, 4th edition, Sutrasthana 5/5-6, page 37
37. Charaka Samhita, Dipika commentary by Chakrapanidatta edited by Vaidya YadavjiTrikamji Acharya, Published by Choukhambha Sanskrit Samsthana, 4th edition, Sutrasthana 23/3-4, page 122
38. National List of Essential Ayush Medicines (NLEAM). 1st ed. Ministry of AYUSH, AYUSH Bhawan, B Block, GPO Complex, INA, New Delhi – 110023: Ministry of AYUSH, Government of India, New Delhi. www.ayush.gov.in; 2022. In.



CHAPTER

5

OBESITY



OBESITY

ICD 11 TM 2 Code 7A02.0

Sthoulya (Obesity) 401 BE-1 Athisthoulya

Synonyms - Sthaulya, Atisthauilya, Sthulata, Sthulatva, Sthavima, Medoroga, Medovridhhi, Medovikara, Medogada, Medodushti, Atipushti, Upachaya etc.

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 $\geq 23\text{kg/m}^2$ as overweight and $\geq 25\text{kg/m}^2$ as Obesity. Obesity is defined as a body mass index (BMI) equal to or greater than the 95th percentile for age and sex.¹

Description of *Sthaulya/Medoroga* in Ayurveda is found to have similarities to Obesity.

Definition

मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः।
अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते॥९॥ (Cha.Su.21/9)

According to Acharya Charaka, due to excessive increase of Meda Dhatu along with Mamsa Dhatu, person who becomes disfigured, lacks enthusiasm, and has pendulous Sphika, Udara and Stana is called Atisthula.²

“Sthoolayativardhateudarraadivridhayayasasthoola”

A person who has bulky body especially in abdominal region is termed as sthula and the state of being sthula is called “Sthoulya”.³

Sthoulya refers to the abnormal & excessive accumulation of Medo dhatu. A sedentary lifestyle, lack of mental & physical exercise and Beeja Dosha i.e. hereditary causes are common etiological factors. A person with an excessive and abnormal increase of Medo dhatu along with Mamsa dhatu, develops a characteristic pendulous appearance of buttocks, belly and breasts and is termed as sthoola. Despite the increased body mass, there is no proportional increase in energy levels.⁴

A. Nidana

a) Aharatmaka Hetu:

Ahara Rasa plays a major role for increasing Meda Dhatu in *Sthaulya*. (Su.Su.15/37)

- **Atisampurnata and Adhyashana:** *Ati Sampurna* and *Adhyasana* can be considered as faulty eating habits. *Ati Sampurna* means *Atibhojana* (excess food intake in a single meal), while *Adhyasana* means *Ajirhashana* (frequent food intake before the digestion of

the previous meal). In *Sthaulya*, *Atimatrabojana* provokes *Tridosha* (Ch.Vi.2/7) as well as leads to *Ama* formation at *Jatharagni* level, whereas *Adhyasana* cause *Ama* formation at *Medodhatvagni* level.

- **Guru and Snigdha Ahara:** *Guru* and *Snigdha* are the properties of *Meda dhatu*. *Meda* is the seat of *Kapha Dosha* and both share similar properties. So, a *Guru-Snigdha Guna* dominant *Ahara* can lead to an increase of both *Kapha* as well as *Meda Dhatu*. This occurs due to *Ashrayashrayibhava* and *Samanya Vriddhi Karanam* concepts.
- **Madhura Rasa Sevana:** Excessive use of *Madhura rasa* is considered as *Meda* aggravating factor and causative factor of *Sthaulya*. *Ikshu* (Sugarcane), *Paya* (Milk), *Guda* (jaggery) are dominant in *Madhura Rasa*. Therefore, excessive consumption of these substances can lead to *Sthaulya*.
- **Gramya Udaka Anupa Mamsa:** *Gramyaudaka* and *Anupa Mamsa* possess *Brimhana karma*, so they increase *Meda* by *Karma Samanya*.
- **Madya Sevana:** Excessive consumption of alcoholic beverages.

b) Viharatmaka Hetu:

- **Avyayama:** *Avyayama* can cause opposite actions of *vyayama* in the body like *Gaurava*, *Sharira Shaithilya*, *Agni Vikriti*, *Alasya*, *Dosha vriddhi*, *Dukha Asahishnuta* etc. *Avyayama* is one of the main cause for aggravation of *Kapha*, which in turn contributes to excessive *Meda Dhatu* formation. This *Kapha-Meda* accumulation forms the key etiopathological pathway of *Sthaulya*.
- **Divaswapna and Atinidra:** *Divaswapna* is a *Kapha* aggravating factor and particularly possess *Abhishyandi* property, which leads to blockage in all body channels. During *Nidra* and *Divaswapna*, physical activity diminishes which further provokes *Kapha* leading to *Meda* deposition.

c) Manasika Hetu:

Achintana, *Harshanitya*, *Mansonivriti*, *Saukhyena* etc. are the psychological factors described by Ayurvedic texts.

d) Anya Hetu:

Beeja Svabhava (Genetic Factor): According to *Charaka*, defect in *Beejabhagavayava* i.e. part of *Beeja*, which resembles with chromosomes and genes may lead to defective development of that organ

B. Rupa

Symptoms of *Sthaulya* may not be observed always, but they appear at various stages in different individuals. Manifestation of *Rupa* is associated with either excessive accumulation of *Meda Dhatu* or diminished nourishment of other *Dhatu* or obstruction in various *Srotasa* by *Medajanya Margaavarodha* or *Ama* or vitiation of *Vata* and *Slesma Dosha*. *Meda Dhatu* attributes to *Guru*, *Snigdha*, *Mridu*, *Manda*, *Sithila*, *Vishyandi*, *Sthula*, *Sandra*, *Slakshna* and *Bahu* properties. So, excessive accumulation of *Meda Dhatu* produces various signs and symptoms in *Sthaulya* patients.

C. Samprapti

Due to obstruction of Srotas by excessive Meda, Vatadosha becomes primarily confined to the stomach. This augments the Agni, causing rapid digestion and an excessive craving for food. As a result, the individual tends to over eat, further causing the over growth of Meda Dhatu, and this vicious cycle leads to Sthoulya.

D. SampraptiGhataka

- **Dosha:**Kapha – Kledaka
 - : Pitta – Pachaka
 - : Vata - Samana, Vyana
- **Dushya:** Meda
- **Agni:** Jatharagni, Dhatvagni (Medodhatvagni)
- **Srotasa:**Medovaha Srotasa
- **Srotodushhti:** Sanga
- **Adhithana:** Vapavahan, Medodharakala
- **Udbhavasthana:**Amashaya
- **Prasara:** Rasayani
- **Rogamarga:** Bahya
- **Ama:** Jatharagnimandhyajanita, Medodhatvagnimandhyajanita
- **Vyaktisthana:** Sarvanga, especially Sphika, Udara, Stana

E. Upadrava

- If Sthaulya is left untreated, it can lead to many diseases..
- **Prameha:** Prameha and Mutrakriccha Upadrava occur due to vitiated Meda, particularly Abaddha Meda. Due to similarity of Nidana and Dosha Dushya, Prameha is most frequent complication of Sthaulya.
- **Mutrakriccha:** Mutrakriccha occurs in Sthula purusha due to loss of water in the body owing to excessive sweating.
- **Jvara:** Jvara in Sthaulya is mainly due to the involvement of Rasavaha Srotasa and production of Ama in Sthaulya Purusha.
- **Ajirna, Atisara, Bhagandara, Arsha and Udara roga:**Upadrava like Ajirna, Atisara, Bhagandara, Arsa, and Udararoga etc. can emerge due to malfunctioning of Agni and formation of Ama owing to Adhyasana and Shleshmavardhaka Ahara in Sthaulya.
- **Vatavikara:** Excessive production of Meda causes Maragavarodha in Srotasa which may lead to Anuloma Kshaya of Uttar Dhatu. As a result of Asthidhatukshaya, Vatavikara manifests.
- **Kasa and Svasa:** Elevated Meda and Ama obstruct the Srotasa. As a result, other Dhatus do not get Poshana from Ahara Rasa. So Alpa prana (low vitality power) results.

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.⁵
- 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 men⁶
- 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.⁷
- 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).⁸ Therefore, Obesity is more effectively defined by assessing its linkage to morbidity and mortality.⁹ The current guidelines, deal with management of both overweight and obesity.

In *Ayurveda*, *Sthaulya* is a predominant metabolic disorder. It is described by *Acharya Charaka* in *Ashta nindita Purusha*. *Charaka* also considered *Sthoulya* as *Sleshma Nanatmaja Vikara* (Ch.S.Su.20/17), *Santarpana Nimittaja Vikara* (Ch.S.Su.23/6) and *Adhika Dosha Yukta Roga* (Ch.S.Su.16/13-16). He also mentioned *Sthula* as *Swedana Ayogya* (C.Su 14/17), while *Acharya Sushruta* considered *Sthula* as *sadatura*, because *Sthaulya* needs regular and continuous care and prevention is the best way of management. In *Ayurveda* classics many times, both the words *medovridhhi* & *Sthaulya* are used as synonyms of each other, but *Acharya Charaka* has clearly established the two conditions as separate entities. The *samprapti* of both *Medovridhhi* and *Sthaulya* is one and the same. However, *medovridhhi* is the first stage of *Sthaulya* i.e. overweight. If *Medovridhhi* continues further, it turns into disease *Sthaulya*. Daily and seasonal health regimens (*swasthavritta* and *ritucharya*) and other modalities, such as detailed instructions on a proper balanced diet and appropriate levels of exercise as per the constitution (*prakriti*) of the person, have been laid out clearly in *Ayurvedic* texts.¹⁰ It is interesting to note that the world is now again focusing on a healthy lifestyle as a key to avoiding risk factors like obesity.¹¹

Agni and Srotas with Reference to Obesity

The concept of biological *Agni* has been described in *Ayurveda* in two references *Agni* & *Pitta*. It is the energy or capacity of the body to convert complex food materials to their constituents and then to build the body tissues (*dhatu*s).¹² The first and foremost level of *Agni* is digestive fire (*jatharagni*).¹³ It digests all types of food in the stomach and the small intestine. The digested and absorbed essence of the food material is called *ahar rasa*, which circulates, providing the substrates for tissues. For each of the seven tissues, there is a special energy or digestive power (*dhatwagni*)¹⁴ assimilating the digested substrates. The lipid precursors are acted upon by fat-specific energy (*medodhatwagni*) for its conversion into adipose tissue (*medadhatu*). The channels and the loci where these conversions take place are called *srotas* or *dhatuvahasrotas*. The quality of all the specific energies depends on the quality of the digestive fire, which is protected and maintained carefully. The impairment of the digestive fire and the specific tissue energies lead to poor availability of the constituents and depletion of tissues (*dhatukshaya*).

With the substrates and energies in balance, all the metabolic activities occur properly in the channels. Defects in the sources with undigested matter block tissue channels. A healthy person is one in whom the activities of humor (dosa), tissue (dhatu), wastes (mala), fire, mind (manas), soul (atma), and senses are harmonious and in balance.¹⁵

CLINICAL EXAMINATION¹⁶

Persons presenting with overweight, or obesity must have a detailed history taken, a clinical examination performed, and appropriate investigations done. This is done to identify the environmental, genetic and lifestyle factors responsible for obesity and at the same time, identify the 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 increase or reduction in body weight over the individual's lifetime (e.g., slow and gradual, rapid and sudden, or a combination) and factors influencing weight change.
- 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).
- 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.
- 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.
- **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 is essential to gather comprehensive information to understand their current activity level, any past injuries or limitations, their exercise preference and lifestyle factors.
- **Sleep Patterns:** Short sleep duration and poor sleep quality may increase the risk of obesity, making it important to record sleep patterns in patients.¹⁷

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.¹⁸

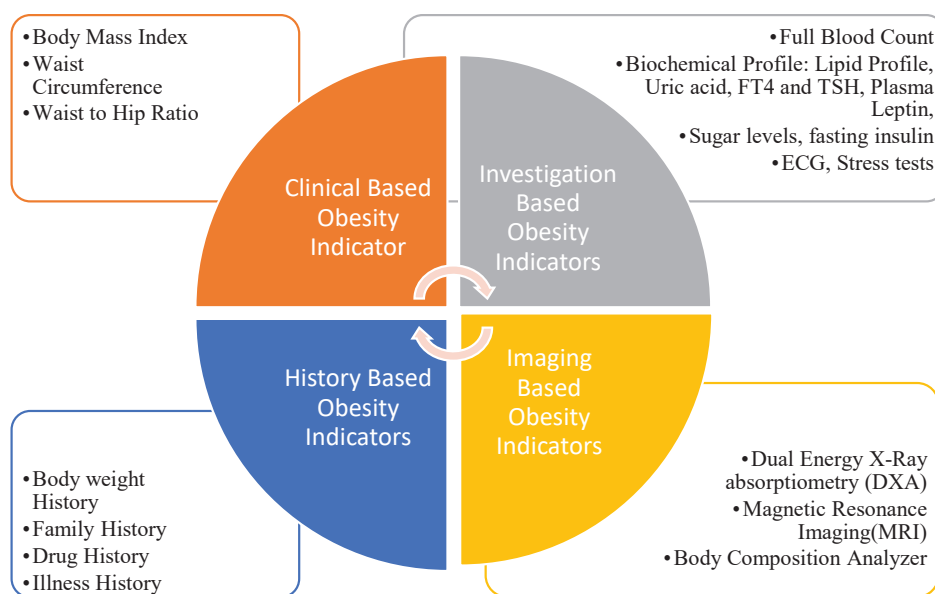


Figure 1 Assessments in overweight and obese persons

Physical Examination¹⁹

- 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 measurements.²⁰
- Tongue examination (Size, Colour, Texture).
- Markers of insulin resistance- Skin tags, acanthosis nigricans.

Comorbidities and Complications²¹

Obesity and Overweight are associated with raised risk of disabilities and a number of comorbidities and complications ²² as listed, which must be diagnosed timely.

Table 1: Complications and Comorbidities.

SYSTEM	DISEASES
Respiratory	• Obstructive sleep apnoea (OSA) • Obesity Hypoventilation Syndrome (OHS)
Cardiovascular	• Coronary Heart Disease • Congestive Cardiac Failure • Hypertension
Cerebrovascular	• Stroke

SYSTEM	DISEASES
Gastrointestinal	• Gastroesophageal Reflux Disease • Barrett's Oesophagus • Erosive Oesophagitis • Diverticular Disease • Oesophageal Cancer • Colon Cancer • Abdominal Hernia
Metabolic	• Dyslipidemia • Type 2 Diabetes Mellitus • Hyperinsulinemia • Metabolic Syndrome • Gout • Gestational Diabetes
Hepato-biliary	• NASH (Non-alcoholic steatohepatitis) • Liver Cirrhosis • Hepatocellular Carcinoma • Gallstone • Gall Bladder Cancer
Musculoskeletal	• Osteoarthritis
Cutaneous	• Acanthosis nigricans • Cutaneous fungal and yeast infections • Venous stasis
Reproductive disorders	• Male: gynaecomastia • Female: Menstrual Irregularities, PCOS, Infertility
Cancer	• Male: Liver cancer, Pancreas cancer, Rectum cancer, Prostate • Female: Gall bladder, Bile duct, Breast, Ovary, Uterine, Cervix

DIFFERENTIAL DIAGNOSIS

Obesity is known to be multifactorial, occurring due to complex interactions 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 2: Differential diagnosis

S.No.	Condition	Features
1.	Obesity due to lifestyle factors	• 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 obesity²³

S.No.	Condition	Features
2.	Obesity due to endocrinal conditions ²⁴	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. Some endocrinal disorders associated with obesity are: • Hypothyroidism • Cushing's Syndrome • Insulinoma • Ovarian disorders, hyperovarian syndrome • Hypogonadism in men • Hypothalamic tumours or damage to this part of the brain as a consequence of irradiation, infection, or trauma
3.	Obesity with genetic conditions ²⁵	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 • 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 ^{26, 27}	Weight gain or body fat redistribution are common side effects of many widely used drugs, some of which are given below: • 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

INVESTIGATIONS²⁸

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:

Essential

- Complete Blood Count/ESR
- Fasting lipid profile

- Fasting plasma glucose
- Fasting Insulin levels
- Serum uric acid
- Serum FT4 and TSH
- HbA1c

Advanced

- 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 (Insulin Sensitivity Test, Insulin Tolerance Test)
- Hormonal Assay (FH, LH, Prolactin, Androstenedione, Progesterone 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 square (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 to³⁰; overweight is defined as a BMI greater than or equal to²⁵
- In children aged below 5 years, overweight is 2 standard deviations and obesity is greater than 3 standard deviations above the WHO Growth Reference median²⁹
- 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 median³⁰

The classification of body weight as per BMI in adults and children is given in Tables 3 & 4 respectively.

Table 3: Classification of obesity by BMI in adults³¹

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	III	>50

Table 4: Classification of weight by BMI in adult Asians

Classification	BMI (kg/m ²)
Underweight	<18.5
Normal range	18.5-22.9
Overweight	23-24.9
Obese I	25-29.9
Obese II	≥ 30

(Reference: World Health Organization, author. The Asia-Pacific perspective: redefining obesity and its treatment. WHO; 2000)

Table 5:Classification of BMI in children³²

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 at Annexure-I

Indian BMI cutoff values must be used to diagnose obesity in Indian Context.

The body mass index is a surrogate marker of fatness and additional measurements, such as the waist circumference, are also used to diagnose obesity.³³ Measures of overweight and obesity and their cut off for Indian population are given in Table – 4.

Table 6:Indian cut-offs for Indicators³⁴

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.³⁵

Types of Body Fat Distribution^{36, 37}

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 2)

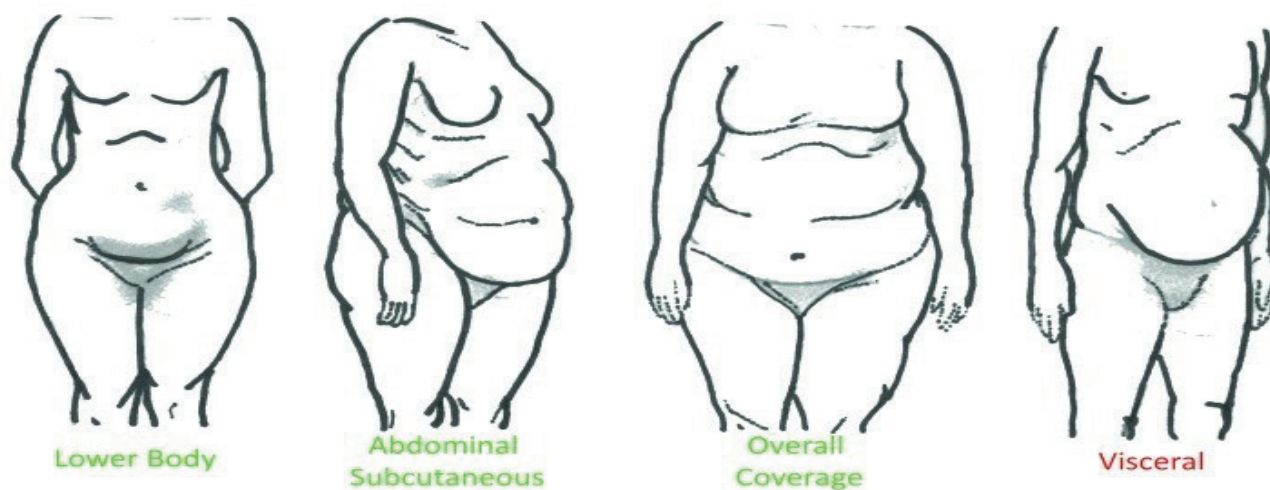


Figure – 2 Body fat distribution is characterized as **Lower body:** fat storage around the buttocks, hips, and thighs; **Abdominal subcutaneous:** subcutaneous fat storage around the stomach and chest; **Overall coverage:** fat accumulation in the arms, breast, thighs, buttocks, lower back, and breast, **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.

PRINCIPLE OF MANAGEMENT

Red Flags

- Unintentional weight gain
- Breathlessness
- Sleep Apnea syndrome
- 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
- Malignancies associated with obesity

Prevention Management

- Diet
- Life Style
- Yoga

PATHYA- APATHYA

Table 7: Pathya Apathya Ahara

AharaVarga	Pathya	Apathya
1.Shuka Dhanya (Cereal grain)	PuranaShali (old rice), Kodrava (Paspalum scrobiculatum),Shyamak (Panicum miliare) , Yava (Hordeum vulgare), Laja (Puffed rice), Navara (Oryza nivara), Kangu(Foxtail millet)	Naveen Dhanya (Shali) (newly harvested rice)
2.Shami Dhanya (Pulses)	Mudga (Phaseolus aureus),Rajamasha (Vigna catiang Walp), Kulattha (Dolichos biflorus), Chanaka (Cicer arietinum), Masur (Lens culinaris), Adhaki (Cajanus cajan), Makusthaka (Phaseolus aconitefolius)	Masha (Vigna mungo)
3.ShakaVarga (Vegetables)	Patola (Trichosanthes Dioica), Patrashaka, Shigru (Moringa oleifera), Vrutaka (Solanum melongena), Vastuka (Chenopodiastru mmurale),Trapusha, Vartaka, Evaruka, Ardraka (Zingiber officinale), Mulaka (Raphanus sativus), Surasa(Tulsi)	KandaShaka (Rhizome Vegetable),
4.PhalaVarga (Fruits)	Jambu (Syzygium cumini), Amalaki (Phyllanthus emblica), Ela (Elettaria cardamomum), Bibhitaki (Terminalia bellirica), Haritaki (Terminalia chebula), Maricha (Piper nigrum), Pippali (Piper longum), ErandKarkati, Narang, Bilvaphala (Aegle marmelos).	Madhura Phala(Sweet Fruits)
5. Drava Varga	Madhu (honey),Takra (buttermilk), Ushnajala (hot water), Tila (Sesamum indicum) & Sarshapa (Brassica juncea) Taila, Medicated Alcoholic preparations	Milk Preparations, Dhadhi, Sarpi), Ikshuvikara Products made from sugar cane
6. MamsaVarga	Rohita Matsya(Rohu Fish) Aanupa, Audaka, Gramya Mamsa Sevana	

Apathya

Sleeping in day time, Sedentary lifestyle, lack of exercise, over eating, repeated eating, consuming cold water,Excess intake of food, excess intake of sweets, fatty food, fried food, red meat etc

Yoga practices for obesity

- 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.



Curative Interventions

The first line of treatment for *Sthaulya* is to avoid those factors which are responsible for the causation of *Sthaulya*. All the factors, having *Snigdha Guna* (unctuousness) dominance in general should be avoided.

Nitya Langhana therapy & *Langhana* even in *Shishir Ritu* (winter season) is advised for the patients of *Sthaulya* by *Vagbhata*.³⁸ (A.S.Su.24/13, A.H.Su.14/13). *Vamana*, *Virechana* etc. are advised for practice according to *Vyadhibala* & *Dehabala* by *Charaka*.³⁹ (Ch.Su.22/18). Amongst *Shadvidha Upakramas*, *Langhana* & *Rukshana* therapies are more suitable for the management of *Sthaulya*.

Vagbhata included all therapies under two main headings i.e. *Langhana* & *Brimhana*. *Langhana*, the line of treatment for *Sthaulya* has been further divided into *Samsodhana* & *Samshamana*.⁴⁰ (A.S.Su.24/13-16, A.H.Su.14/14).

A. Samsodhana:

All *Sthula* patients with *Adhika Dosha* & *Adhika Bala* should be treated with *Samsodhana* therapy, including *Vamana*, *Virechana*, *Niruha*, *Raktamokshana* & *Sirovirechana*.⁴¹ (A.H.Su.14/14). Being a syndromic condition (*Bahudoshasya Laksanam*), *Samsodhana* therapy is highly recommended for *Sthaulya* patients possessing stamina & strength.⁴² (Ch.Su.16/13-16). *Ruksha*, *Ushna* & *Tikshna Basti* are also suggested by *Acharya Charaka*.⁴³ (Ch.Su.21/21-23). *Ruksha Udvartana* is the *BahyaSodhana* indicated for the management of *Sthaulya*.⁴⁴ (A.S.Su.25/65-66). *Snehana Karma* is always restricted for the patients of *Sthaulya*⁴⁵ (Ch.Su.13/53); however on exigency usage of *Taila* is recommended.⁴⁶ (Ca.Su.13/44-46).

B. Samshamana:

Alleviation of *Samana Vayu*, *Pachaka Pitta* & *Kledaka Kapha* along with reduction of *Medo Dhatu* by increasing *Medodhatvagni* is the main goal of treatment in *Sthaulya*.

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

Clinical Diagnosis

The cardinal signs & symptoms of *Sthaulya* are –

The increase of fat & flesh has disfigured by pendulous buttocks, abdomen & breast and that increased bulk reduces the energy. So, the person has less enthusiasm in his physical activity.

- Ayushohrasa* (Diminution of life span)
- Javoparodha* (Lack of enthusiasm)
- KricchaVyavaya* (Difficulty in sexual act)
- Daurbalya* (General debility)
- Daurgandhya* (Foul smelling of body)
- Swedabadha* (Distressful sweating)
- Kshudhatimatra* (Excessive hunger)
- Pipasatiyoga* (Excessive thirst)

Table 8: Management

S.No.	Drugs	Dosage form	Dose	Time	Duration	Adjuvants
1	Vyoshadi Guggulu (A.H)	Tablet	1 gm (2 Tablets of 500mg each)	TID (A/F)	1-3 months	Lukewarm water
2	Vidanga Churna	Powder	5-10 gm	BD (A/F)	1-3 months	Warm water
3	Triphala Churna	Powder	3-5gm	BD (A/F)	1-3 months	Warm water
5	Ayaskriti	Arishta	20ml	BD (A/F)	1-3 months	Warm water
7	Lodhrasava	Arishta	12-24 ml	BD (A/F)	1-3 months	Warm water
9	Guggulutiktaka Kashaya	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
10	Varanadi Kashaya	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
11	Varadi Kashaya	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
12	Asanadi Kashaya	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
13	Triphala Kashaya	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
14	Sthoulyahara Kashaya (Sahasra Yoga)	Kashaya	15ml	BD (B/F)	1-3 months	lukewarm water
17	Navaka Guggulu (B.R. Medoroga)	Tablet	500 mg-1 gm	BD (A/F)	1-3 months	Warm water
19	Medohara Guggulu (Rasatantrasara)	Tablet	500 mg-1 gm	BD (A/F)	1-3 months	Warm water
20	Kanchanara Guggulu	Tablet	500 mg-1 gm	BD (A/F)	1-3 months	Warm water
21	Medohara Vidangadi Lauham	Tablet	250-500 mg	BD (A/F)	1-3 months	Warm water
22	Tryushanadi Lauham	Tablet	500-750 mg	BD (A/F)	1-3 months	Honey
23	Ksharadi lauham	Tablet	500-750 mg	BD (A/F)	1-3 months	Honey
26	Loharishta	Arishta	20ml	BD (A/F)	1-3 months	Warm water
27	Amritadi guggulu	Tablet	500 mg-1 gm	BD (A/F)	1-3 months	Warm water
28.	Musta Churna	Powder	3-6 gm	Twice/Thrice daily, before meal	1-3 months	Warm water
29.	Haritaki Churna	Powder	3-6 gm	BD, Before meal	1-3 months	Warm water

S.No.	Drugs	Dosage form	Dose	Time	Duration	Adjuvants
30	Arogyavardhini vati	Tablet	250-500 mg	Before meal/ twice daily, Before food	1-3 months	Warm water
31.	Brihat Manjisthadi kwat	Decoction	20-40 ml	Twice daily/ Before meal	1-3 months	Warm water
32.	Phalatrikadi Kwatha	Decoction	25-50 ml	Twice daily before meal	1-3 months	Warm water
33.	Triphala Guggulu	Tablet	1-3 gms	Twice daily before meal	1-3 months	Warm water
34.	Shilajatu	Churna	500 mg	Before meal/ Twice daily	1-3 months	Warm water

Preferable Diet & Lifestyle:

Diet as mentioned earlier

Restricted Diet & Lifestyle:

As mentioned earlier

Follow Up

Every month follow up for 6 months

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
- Obesity-associated with the conditions like diabetes, hypertension, coronary artery disease, heart disease, stroke, certain cancers and obstructive apnea
- Substantial impact on their quality of life and activities of daily living.

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

Clinical Diagnosis: Same as level 1

- Clinical assessment of body fat percentage

Management

Purification/ other procedures

Udwarthana (Triphala choorna, Kola Kulatthadi choorna) followed by Bashpa Sweda Lekhaneeya Vasti (Madhu-Honey, Saindhava-Rock salt, Tila taila-Sesamum orientale, Triphala

kwatha-Amalaki (Phyllanthus emblica linn.), Bibhitaki (Terminalia bellerica), Haritaki (Terminalia chebula);Prakshepa Dravya-Gomutra, Yava kshara (alkali preparation of barley), Tuttha-(CuSo4), Kasisa (FeSo4), HinguNiryasa(Ferula narthex.), Shilajatu (black bitumen).

- Virechana
 - Snehapana with Panchtikta Ghrita
 - Sarvanga Abhyanga with Murchitatilataila
 - Sarvangabashpasweda with Dashmoolkwath
 - Virechana with TrivritAvaleha along with Anupana of Triphalakwath/Draksha Kashaya
- Vamana

Preferable Diet & Lifestyle

Same as level 1

Restricted Diet & Lifestyle

Same as level 1

Follow Up

- Every month follow up for 3 months

Referral Criteria

- Same as mentioned earlier at Level 1, plus
- Psychological imbalance
- Suspected life-threatening complications such as heart failure

At Level 3(Ayush hospitals attached with teaching Institution, District Level/Integrated/State Ayush Hospitals)

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 medicines mentioned in level 1 and 2, the following Panchkarma procedures can also be adopted.

Shodhana

Poorvakarma

Snigdha Udwartana is indicated in conditions of obesity associated with dryness-3 to 7 days using Triphala churna with murchitatila taila .

Ruksha Udwartana in obese persons is done using Triphala or Kolakulatthadi churna or Godhuma churna.

It is followed by Snehapana with tikta ghrita like Gugguluthiktaka ghrita, Varanadi ghrita, Triphala ghrita for 3 to 7 days followed by Vishrama kala of 1 day in which Abhyanga, steam, dadhipathya (administration of ahara which increases kapha like curd and sweets are advised).

Next day, abhyanga, steam and vamana are administered if obesity is associated with Kapha Pradhana lakshana.

In case of Pitta dominance, instead of Vamana, virechana is administered with Trivrut lehya.

In case of Vata dominance like more bloating of abdomen, aches and pains, lekhana vasti is given.

In Lekhana vasti, drugs having lekhana properties are used. Yoga vasti is administered for 8 days, kala vasti for 16 days, karma vasti for 30 days.

Purification/ other procedures)as per applicability

- Udwartana
- Lekhaneeya vasti
- Virechana
- Vamana

Preferable Diet & Lifestyle

Diet as mentioned earlier

Restricted Diet & Lifestyle

As mentioned earlier

Follow Up

Every month follow up for 3 months

Referral Criteria⁴⁷

- Same as mentioned earlier at Level 2, plus
- Morbid obesity not responding to treatment
- Uncontrolled hypertension
- Worsening Hypertriglyceridemia
- Worsening insulin resistance and hyperglycaemia
- Suspected Cardiac arrhythmias
- Suspected Polycythemia
- Other modalities can be considered depending on the case and to rehabilitate properly.

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: Month	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: Month	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	25.3	29.6	35.5

REFERENCES

1. Environment factors and Obesity Available at <https://www.ncbi.nlm.nih.gov/books/NBK580543>
2. Shri Taranath Tarak Vaschaspati Bhattacharya, Vachaspatyam 6/5358, Chauhukambha Ayurveda Pratishthan; Reprint; 2016.
3. Vaidya Ravidatta Tripathi, Charak Samhita with vaidymanorama Hindi commentary. Chaukhamba Sanskrit pratishthan Delhi; 2013. Sutrasthana 21/9. 301p.
4. Bhramhasankar Mishra; BhavaprakashUttarardha; Chaukhamba Sanskrit Sansthan; 1997; adhyay 39/2
5. 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>.
6. 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).
7. 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>.
8. 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>.
9. 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.
10. Svoboda, R.E., *Prakriti – Your Ayurvedic Constitution*, 1st ed., Motilal Banarasidas, New Delhi, 1989–1999.
11. Dossey, L., *Meaning and Medicine*, 1st ed., Bantam Books, New York, 1991, p. 66.
12. Munshi, V.D. (translator), *Ashtang Hriday, Sastum Sahityavardhak Mudranalaya*, Ahamdabad, India, 1952, p. 101.
13. Munshi, V.D. (translator), *Ashtang Hriday, Sastum Sahityavardhak Mudranalaya*, Ahmedabad, India, 1952, p. 111.
14. Shastri, P.K. (translator), *Charak Sanhita, Part II*, 2nd ed., Chaukhamba Sanskrit Sanathan, Varansi, India, 1983, p. 380.
15. Acharya, J.T. (translator), *Sushrut Sanhita of Sushrut*, Nirnay Sagar Press, Mumbai, India, 1915, p. 65.
16. 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/>.
17. 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>.
18. Burridge 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/>.
19. 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/>.

20. 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/>.
21. 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/jobe/2023/4178121>.
22. Labib M. ACP Best Practice No 168. The investigation and management of obesity. *J Clin Pathol.* 2003 Jan;56(1):17-25. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1769843/>
23. 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>
24. 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>
25. 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/>
26. Thaker VV. Genetic and epigenetic causes of obesity. *Adolesc Med State Art Rev.* 2017 Fall;28(2):379-405. PMID: 30416642; PMCID: PMC6226269.
27. 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/>
28. 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/>
29. 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>.
30. 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/>.
31. 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/>.
32. 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>.
33. 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/>.
34. 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>.
35. 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.
36. 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_following_visceral_fat_removal_and_expansion_Beyond_anatomic_location.

37. Labib M. ACP Best Practice No 168. The investigation and management of obesity. J Clin Pathol. 2003 Jan;56(1):17-25. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1769843/>.
38. AshtangSangraham (4) Sutrasthana Adhyaya 24/9, 292, VahataorVriddhaVagbhata 19 with the Sasilekha Sanskrit Commentary by Indhu, Edited by Dr. Shiv Prasad Sharma.
39. Charaka Samhita by Agnivesha, revised by Charaka and Dridhabala with Ayurveda- Dipika commentary of Chakrapanidatta edited by Vaidya JadavjiTrikamjiacharya a Sutrasthana 22nd Chp Sl.no18 Pp121. Edited by JadavajiTrikamji,FifthEdition,Published by Chaukhambha Sanskrit Sansthana, Varanasi, 2001.
40. AshtangSangraham (4) Sutrasthana Adhyaya 24/13-16, VahataorVriddhaVagbhata 19 with the Sasilekha Sanskrit Commentary by Indhu, Edited by Dr. Shiv Prasad Sharma.
41. Yadunandana Upadhyaya, Ashtanga Hradayama, Sutra Sthana, 14th chapter, 12 th sloka. P 136.
42. Charaka Samhita by Agnivesha, revised by Charaka and Dridhabala with Ayurveda- Dipika commentary of Chakrapanidatta edited by Vaidya JadavjiTrikamjiacharya a Sutrasthana 16th Chp Sl.no13-16. Edited by JadavajiTrikamji,FifthEdition,Published by Chaukhambha Sanskrit Sansthana, Varanasi, 2001.
43. Vaidya yadavjiTrikamajiAacharya, Charaka Sanhita, Sutra Sthana, 21th chapter, 21 th - 23th sloka. P 117.
44. Ashtanga Samgraha with Hindi Commentary, by Kaviraj Atrideva Gupta, Chowkhamba Krishnadas Academy, Reprint 2005, Vol. 1, Ashtanga Samgraha Su. 25/65-66.
45. Agnivesha, Charaka Samhita, Vidyotinihindi commentary by Pt. Kashi Nath Shastri and Dr. GorakhNathChaturvedi, Reprint 2003, Sutra Sthana (13:53), pg.269, ChaukhambaBharati Academy, Varanasi (2003).
46. Agnivesha, Charaka Samhita, Vidyotinihindi commentary by Pt. KashiNathShastri and Dr. Gorakh Nath Chaturvedi, Reprint 2003, Sutra Sthana (13:44), pg.267, ChaukhambaBharati Academy, Varanasi (2003).
47. 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.
48. National List of Essential Ayush Medicines (NLEAM). 1st ed. Ministry of AYUSH, AYUSH Bhawan, B Block, GPO Complex, INA, New Delhi – 110023: Ministry of AYUSH, Government of India, New Delhi. www.ayush.gov.in; 2022. In.
49. Ministry of Ayush. Ayurvedic Standard Treatment Guidelines. 1st ed. New Delhi. Government of India. 2017; p. 79-85.

AYUSH VERTICAL
DIRECTORATE GENERAL OF HEALTH SERVICES
Government of India