



सत्यमेव जयते  
**Ministry of Ayush**  
Government of India

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

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



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



सत्यमेव जयते

**प्रतापराव जाधव**  
**PRATAPRAO JADHAV**



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



देव का प्रकृति पाठशाला अभियान  
(॥ शरीर स्वस्थ है, तब तक जगत् स्वस्थ है ॥)



स्वतः और समाज के लिए योग  
Yoga for self and society

### MESSAGE

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<sup>th</sup> April, 2025  
New Delhi



वैद्य राजेश कोटेचा  
सचिव

Vaidya Rajesh Kotecha  
Secretary



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## FOREWORD

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

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

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

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

(Rajesh Kotecha)

New Delhi.  
23.04.2025



प्रो.(डॉ.) अतुल गोयल

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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)



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## 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

<b>OHS</b>	Obesity Hypoventilation Syndrome
<b>OSA</b>	Obstructive Sleep Apnea
<b>PCOS</b>	Polycystic Ovarian Syndrome
<b>PUFA</b>	Polyunsaturated Fatty Acid
<b>RA factor</b>	Rheumatoid Arthritis factor
<b>RBSK</b>	Rashtriya Bal Suraksha Karyakaram
<b>RSSDI</b>	Research Society for the Study of Diabetes in India
<b>SF</b>	Synovial Fluid
<b>SM - S</b>	Sphingomyelin
<b>T2DM</b>	Type 2 Diabetes Mellitus
<b>TC</b>	Total Cholesterol
<b>TDS</b>	Three times a day
<b>TG</b>	Triglyceride
<b>TSH</b>	Thyroid stimulating hormone level (TSH).
<b>USG</b>	US: ultrasonography / Ultrasonography (USG)
<b>UTI</b>	Urinary Tract Infection
<b>VLDL</b>	Very Low Density Lipoprotein
<b>WAGR syndrome</b>	Wilms tumor, aniridia, genitourinary malformations and a range of developmental delays
<b>WC</b>	Waist Circumference
<b>WHO</b>	World Health Organisation
<b>WHR</b>	Waist-Hip Ratio
<b>YLD</b>	Years Lived with Disability
<b>YLL</b>	Years of life lost

## GLOSSARY

S. No.	Term	Term or Concept in English	Description
1.	<i>Ābzan</i>	Sitz bath	Method of treatment in which patient sits in warm water or medicated liquid obtained by boiling drugs in water.
2.	<i>Aghdhiya Radiyya</i>	Diet producing bad humours	The foods which produce bad quality of humours.
3.	<i>Al-Ghidhā' al-Dasim</i>	Fatty diet	Diet which has hot and wet temperament but heat to lesser degree and wetness to greater degree than sweet diet.
4.	<i>Balgham</i>	Phlegm	One of the four humours, which is white in colour, bears cold and moist temperament and is next to sanguine humour in excellence.
5.	<i>Balgham Ghalīẓ</i>	Thick phlegm	A more viscous, pathological form of phlegm.
6.	<i>Burūdat</i>	Coldness	One of the two active properties naturally associated with matter.
7.	<i>Dalk</i>	Massage	Massage with techniques ranging from light, moderate, and deep pressure; regimen involving manual manipulation of muscles.
8.	<i>Dam</i>	Sanguine; one of the four humours	One of the four humours, which is characterized by hot and moist temperament, red colour, sweet taste and no odour; it is the best of all the four varieties of humours.
9.	<i>Dhayābītus / Dūlābiya / Mu'aṭṭisha / Dawwāriyya / Parkāriyya</i>	Diabetes mellitus	A chronic metabolic disorder characterized by increased blood sugar, polyuria, polydipsia, and weight loss.
10.	<i>Dhayābītus Ḥārr</i>	Diabetes due to predominance of excess heat on kidneys	A condition in which liver and kidneys due to their <i>Sū'-i-Mizāj Ḥārr</i> (morbid hot temperament) fail to make necessary alteration to the fluids taken orally and it is excreted without any apparent change.
11.	<i>Ḍimād</i>	Poultice	Semi-solid preparation of crude drugs meant for local application.
12.	<i>Ḍu'f al-Kabid</i>	Hepatic insufficiency	A morbid state characterized by decreased appetite, skin discoloration, emaciation and diarrhoea similar in colour to raw-meat washings
13.	<i>Ḍu'f-i-Gurda</i>	Renal insufficiency / Renal debility	Weakness of kidney due to alteration in its parenchyma or due to abnormal temperament, calculus, inflammation, etc.

S. No.	Term	Term or Concept in English	Description
14.	<i>Faşd</i>	Venesection	Bloodletting through venesection is a mode of regimenal therapy for complete evacuation of morbid matter, leading to moderation of all humours.
15.	<i>Fuḍlāt</i>	Waste material(s) of body	Materials which are not required in the body and need to be excreted, e.g., urine, excrement, sweat, etc.
16.	<i>Ghalaba'-i-Balgham</i>	Predominance of phlegm	Qualitative imperfection or quantitative excess of phlegmatic humour in the body.
17.	<i>Ghalaba'-i-Burūdat</i>	Predominance of coldness	A state in which cold temperament overwhelms normal bodily functions.
18.	<i>Ghalaba'-i-Ḥarārat Mufriṭ bar Gurda</i>	Predominance of excess heat on kidneys	Excessive heat affecting kidney function, often associated with polyuria or nephritis.
19.	<i>Haḍm Kabiḍī</i>	Hepatic digestion	Second stage of food digestion taking place in the liver to form chyle.
20.	<i>Ḥammām</i>	Therapeutic bath / Turkish bath	Turkish bath, constructed as per specific guidelines, where temperatures of different rooms are different for therapeutic purposes like cleansing, reducing viscosity of matter and elimination and diversion of morbid matter.
21.	<i>Ḥarārat Gharīzī</i>	Innate heat	Heat of the body regulated by <i>medicatrix naturae</i> to maintain life.
22.	<i>Hijāma</i>	Cupping therapy	Mode of regimenal therapy in which horns (nowadays cups) are used with or without scarification for diversion and evacuation of morbid matter from blood; may be without scarification or with scarification (blood-letting).
23.	<i>Ḥuqna</i>	Enema	Administration of warm water or liquified drugs or medicinal preparation through anal canal in the bowel; administration of liquid drugs through anal canal used to eliminate or getting rid of superfluities, vitiated humours and waste materials from intestine.
24.	<i>Idrār</i>	Diuresis	To induce increased flow of urine/ menstruation/ sweat/milk, etc.
25.	<i>'Ilāj bi'l Dawā'</i>	Pharmacotherapy	Treatment of disease with help of medicines/drugs.
26.	<i>'Ilāj bi'l Tadbīr</i>	Regimenal therapy	Regimenal therapy is modification in <i>Asbāb Sitta Ḍarūriyya</i> (six essential factors) and application of regimens for maintenance of health as well as for management of diseases.
27.	<i>Imtilā'</i>	Plethora	Quantitative or qualitative repletion of blood vessels.

S. No.	Term	Term or Concept in English	Description
28.	<i>Ishāl</i>	Purgation	Evacuation of morbid matter from body through intestines as a regimen; the term does not cover <i>Ishāl</i> mentioned under diseases.
29.	<i>Iṣlāḥ-i-Sū'-i-Mizāj</i>	Correction of morbid temperament	Principles of treatment for correction of an abnormal temperament that leads to morbidity.
30.	<i>Kimād</i>	Fomentation	Powder of drugs tied in a piece of cloth (bag) and used for local fomentation after heating/cooling it.
31.	<i>Laṭūkh</i>	Epithem	Medicated preparation for external application, such as poultice.
32.	<i>Masālik-i-Rūḥ</i>	Pathways of pneuma	Routes through which pneuma disperses in the body.
33.	<i>Mizāj Ḥārr Raṭb</i>	Hot and moist temperament	Temperament in which the hot and moist qualities dominate the other two qualities, i.e., cold and dry.
34.	<i>Mundij</i>	Concoctive	Drug which modifies and prepares morbid humours for evacuation from body.
35.	<i>Mundij-o-Mushil Therapy</i>	Concoctive and purgative therapy	Sequential therapy involving concoction (maturation of morbid matter) followed by its evacuation.
36.	<i>Mushil</i>	Purgative	Drug which helps in expulsion of morbid humours in form of loose stools.
37.	<i>Naṭūl</i>	Douche	Pouring medicated liquid on a body part, typically for pain relief.
38.	<i>Niqris</i>	Gout	Pain and inflammation of big toe/heel; a specific type of swelling, inflammation and pain occurring commonly in small joints.
39.	<i>Niqris Balghamī</i>	Gout of phlegmatic origin	A specific type of swelling, inflammation and pain, caused by predominance of <i>Balgham</i> , occurring commonly in small joints, especially in great toe; but sometimes it may also affect wrist and fingers of hand.
40.	<i>Niqris Damawī</i>	Gout of sanguine origin	A specific type of swelling, inflammation and pain, caused by predominance of <i>Dam</i> , occurring commonly in small joints, especially in great toe; but sometimes it may also affect wrist and fingers of hand.
41.	<i>Niqris Ṣafrāwī</i>	Gout of bilious origin	A specific type of swelling, inflammation and pain, caused by predominance of <i>Ṣafrā'</i> , occurring commonly in small joints, especially in great toe; but sometimes it may also affect wrist and fingers of hand.

S. No.	Term	Term or Concept in English	Description
42.	<i>Niqris Sawdāwī</i>	Gout of melancholic origin	A specific type of swelling, inflammation and pain, caused by predominance of Sawdā', occurring commonly in small joints, especially in great toe; but sometimes it may also affect wrist and fingers of hand.
43.	<i>Pāshoya</i>	Footbath	Lukewarm decoction of drugs used for immersion/irrigation of affected foot.
44.	<i>Qay'</i>	Emesis	To induce vomiting (as a regimen) in order to evacuate gastric contents; the term also covers morbid condition known as vomiting.
45.	<i>Quwwat Jādhiba</i>	Absorptive faculty	Faculty which serves the nutritive faculty and absorbs beneficial material into the body.
46.	<i>Riyāḍat</i>	Exercise	Activity involving physical effort, to maintain or improve health.
47.	<i>Rūḥ</i>	Pneuma	Light gaseous substance obtained from the interaction of inspired air with subtle humours found in organs and fluids of the body and help faculties in their functions.
48.	<i>Ruṭūbat</i>	Moistness	One of the two passive physical properties naturally associated with matter.
49.	<i>Ṣafrā'</i>	Yellow bile; one of the four humours	One of the four humours, which is yellow in colour, has hot and dry temperament and is next to phlegm in excellence.
50.	<i>Sawdā'</i>	Black bile / black humour / melancholic humour	One of the four humours, which is black in colour and has cold and dry temperament
51.	<i>Shaḥm</i>	Fat	A white and soft organ consisting of oily material found commonly around membranes.
52.	<i>Shamūm</i>	Inhalation	Inhalation of drugs which may be in dry or liquid form so that volatile substances reach nasal cavity and respiratory tubes.
53.	<i>Siman Mufriṭ</i>	Obesity	Excessive amount of body fat or weight gain which may interfere with routine life.
54.	<i>Sū'-i-Mizāj</i>	Morbid temperament	Derangement or imbalance of temperament either in terms of four physical properties or qualitative or quantitative predominance of humours.
55.	<i>Sū'-i-Mizāj Bārid Raṭb Māddī</i>	Morbid cold and moist temperament with substance	Morbid cold and moist temperament associated with predominance of cold and wet substances.

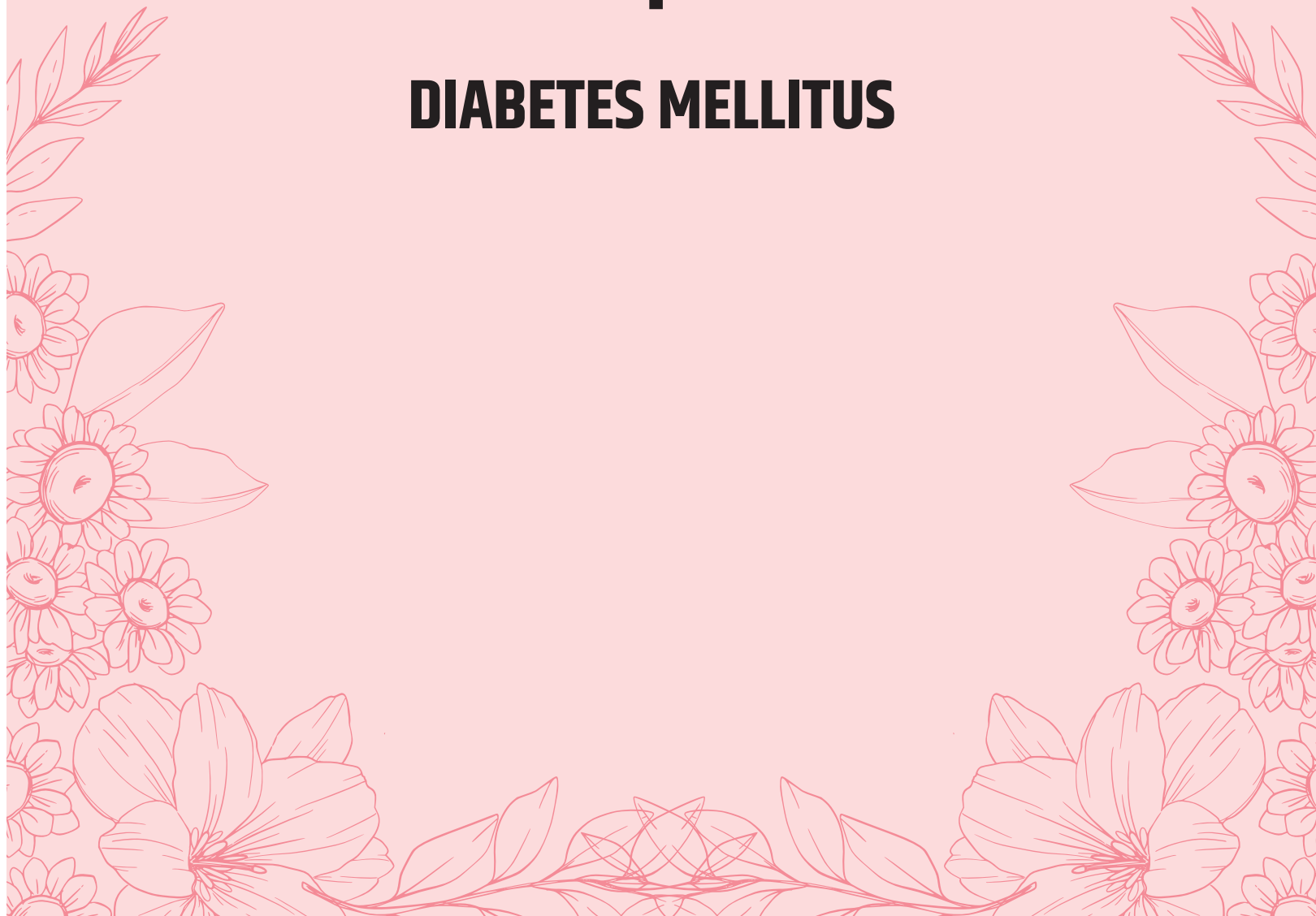
S. No.	Term	Term or Concept in English	Description
56.	<i>Sū'-i-Mizāj Māddī</i>	Morbid temperament associated with substance	Morbid temperament in which change in four physical properties.
57.	<i>Sū'-i-Mizāj Sāda</i>	Simple morbid temperament	Morbid temperament in which only change in four physical properties, i.e. hotness, coldness, dryness and wetness/ moistness takes place.
58.	<i>Sū'-i-Mizāj-i-Jigar Bārid</i>	Cold morbid temperament of liver	A morbid state characterized by diarrhoea, puffiness of face, decreased thirst, whitish skin and lips, slow pulse and white urine of thick consistency.
59.	<i>Sūdād (Sudad al-Kabid)</i>	Obstructions of liver	A morbid state characterized by pallor, weight loss, decreased quantity of blood in body, heaviness without pain in liver and soft and whitish stool.
60.	<i>Ta'dīl-i-Mizāj</i>	Moderation of abnormal temperament	Moderation of temperament or bringing abnormal temperament to normal temperament.
61.	<i>Ta'dīl-i-Mizāj-i-Gurda</i>	Correction of temperament of kidneys	Moderation of temperament of kidneys.
62.	<i>Ta'dīl-i-Sū'-i-Mizāj</i>	Moderation of abnormal temperament	Moderation of temperament or bringing abnormal temperament to normal temperament.
63.	<i>Ta'rīq</i>	Inducing diaphoresis	Sweating as a regimen is usually done to remove morbid matter through skin.
64.	<i>Ṭabī'at</i>	Medicatrix Naturae	Natural power for self-preservation; the power endowed by nature to every individual for self-preservation; it regulates normal functions and is the administrator, protector and healer of the body.
65.	<i>Tabrīd</i>	Cooling	Cooling of body/part of body; a method of treatment in which coldness is produced/ generated or heat is reduced in body by drugs or regimen.
66.	<i>Tadhīn</i>	Oiling	Application of hot or cold oil on body part.
67.	<i>Taftīḥ-i-Sudad</i>	Inducing deobstruction	Process of removing an obstruction by use of deobstruents.
68.	<i>Taḥlīl</i>	Dissolution	Dispersion of disease-causing matter accumulated in an organ or body part; this term does not cover <i>Taḥlīl</i> mentioned under Pharmacology and pharmacy.
69.	<i>Tahlīl-o-Talyīn</i>	Resolution and softening	To resolve the inflammation and soften the joints.
70.	<i>Taḥlīl-i-Mādda</i>	Dissolving morbid matter	Dispersion of disease-causing matter accumulated in an organ or body part.

S. No.	Term	Term or Concept in English	Description
71.	<i>Tahzīl</i>	Inducing weight loss	To induce weight loss.
72.	<i>Tajfīf</i>	Inducing dryness	Process of producing dryness in body or part of body.
73.	<i>Tajfīf-i Badan</i>	Producing dryness in the body	Process of producing dryness in body or part of body.
74.	<i>Taltīf</i>	Process of refining / attenuation	Act of refining of any thick viscid matter.
75.	<i>Talyīn-i-Ṭabī'at</i>	Inducing laxation	Process to soften intestinal content or to evacuate excrement from intestine.
76.	<i>Tanqiya</i>	Cleansing of morbid matter/ humour from body	Induced elimination of morbid material from the body, usually done after proper concoction.
77.	<i>Taqlīl-i-Ghidhā'</i>	Reducing dietary intake	Reducing the intake of diet in order to conserve power of digestive faculty.
78.	<i>Taqwiyat-i-Badan</i>	Toning up of body	Process which strengthens and revitalize the body.
79.	<i>Taqwiyat-i-Gurda</i>	Toning up of kidneys	Process which strengthens the kidney and improves its function.
80.	<i>Taqwiyat-i-Kabid</i>	Toning up of liver	Process which tones up liver and improves its function.
81.	<i>Tarqīq-i-Akhlāt / Mawād</i>	Diluting humours or morbid matter	Making humours or disease-causing matter dilute.
82.	<i>Tartīb-i-Badan</i>	Moistening of body or part of body	Process of moistening body or part of body by drugs or regimen.
83.	<i>Tashaḥḥum-i-Kabid</i>	Fatty liver disease	A disease of the liver characterised by fatty infiltration.
84.	<i>Tashaḥḥum-i-Kabid Ghayr Khamrī</i>	Non Alcoholic Fatty liver disease	A disease of the liver characterised by fatty infiltration without alcohol intake.
85.	<i>Taskhīn-i-Badan</i>	Calefaction of body or its part	Warming / heat production in body / part of body; a method of treatment in which heat is produced/ generated in body by drugs or regimens.
86.	<i>Taskīn-i-Alam</i>	Analgesia	To relieve pain by using drugs, changing temperament of pain site or some other means.
87.	<i>Taskīn-i-Tishnagī</i>	Quenching of thirst	A management approach aimed at reducing excessive thirst.
88.	<i>Ṭilā'</i>	Liniment	A kind of medicated oil or a thin medicinal preparation applied locally.

CHAPTER

1

# DIABETES MELLITUS





# DIABETES MELLITUS

Diabetes mellitus disorder (TM2) SP60

Type 2 diabetes mellitus (ICD-11 for mortality and morbidity statistics: 5A11)<sup>2</sup>

*Dhayābītus/ Dūlābiya/ Mu'aṭṭisha/ Dawwāriyya/ Parkāriyya*  
(National Unani Morbidity Code: G-2)<sup>1</sup>

## CASE DEFINITION:

Diabetes Mellitus is a chronic disorder resulting from aberrations in insulin secretion, insulin action, or both. Long term damage, dysfunction, and failure of different organs resulting in this condition is attributed to the persistent hyperglycaemia state<sup>3</sup> Type 2 Diabetes Mellitus previously referred as non-insulin-dependent diabetes accounts for approximately 90 – 95% of all diabetes cases. The condition also known as adult-onset diabetes is due to insulin resistance and relative insulin deficiency<sup>3,4</sup>.

## Unani medicine's perspective:

***Dhayābītus Hārr*** is a condition wherein liver and kidneys due to their Sū'-i-Mizāj Hārr (morbid hot temperament) fail to make necessary alteration to the fluids taken orally and it is excreted without any apparent change<sup>5</sup>.

## 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<sup>6,7</sup>
- 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<sup>6</sup>.
- 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)<sup>8</sup>.
- 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<sup>6</sup>.
- 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<sup>6</sup>.
- 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<sup>9</sup>.
- The ICMR-INDIAB survey reported 26.6% of Indians above 20 years having dysglycaemia with 11.4% suffering from diabetes and 15.3% suffering from a pre-diabetic state<sup>10,11</sup>.
- Several non-modifiable risk factors like age, ethnicity, genetic predisposition, family history of diabetes, and modifiable factors like sedentary lifestyle, obesity, unhealthy diet, stress, intrauterine environment, environmental pollutants, etc. are associated with the incidence of the disease.

- The COVID-19 pandemic has resulted in a significant rise of new-onset of diabetes mellitus in all age groups especially during the post-acute phase of the disease<sup>10</sup>. The pandemic shows an increase of 14.4% of new onset of diabetes mellitus including T2DM among the hospitalized patients<sup>12</sup>.

### Unani medicine's perspective: (Etiology and Pathology)

#### ➤ Etiology

- *Sū-i Mizāj Hārr Mufrit* (Excessive hot morbid temperament)<sup>5</sup>*Ghalaba'-i-Harārat Mufrit bar Gurda* (Predominance of heat on kidneys)<sup>5</sup>
- *Ḍu'f-i-Gurda* (Weakness of kidneys) and its inability to retain and digest the fluid received from the liver.
- *Ḍu'f-i-Kabid* (Weakness of liver) due to excess of heat.<sup>13</sup>

#### ➤ Pathology

*Quwwat Jādhiba* (Absorptive faculty) is the faculty through which the kidneys absorb fluids from blood and form the urine. Their absorptive faculty gets enhanced in case of predominance of hot morbid temperament making them more demanding for fluids to cool down their abnormal heat. As a result, an increased amount of fluids from liver and other organs comes towards the kidneys resulting in continuous demand of water by the body i.e. thirst.<sup>5</sup>

### CLINICAL PRESENTATION

The presentation of T2DM to the clinician is quite varied and a majority is discovered incidentally during regular blood testing for routine check-up, pre-surgery checkup, dental care, or any medical procedure. The classical presentation of T2DM like polyuria, polydipsia, and fatigue is observed mainly in older individuals. Often recurring bacterial and fungal infections, blurred vision, and delayed wound healing is classically observed in patients especially in older individuals. With a majority of the cases being asymptomatic, the patient may present to the clinician with a macrovascular complication of coronary heart disease, peripheral vascular disease, and cerebrovascular disease or a microvascular one of diabetic nephropathy, retinopathy, nephropathy or diabetic foot ulcer. In recent years, cancers (hepatocellular, pancreatic, colorectal, etc.), infections, Non-Alcoholic Fatty Liver Disease including steatohepatitis and cirrhosis, obstructive sleep apnoea, affective disorders, dementia, erectile dysfunction, and functional disability at the workplace is also considered as emerging complications of T2DM. In severe cases especially in older individuals, hyperosmolar coma is observed especially during medications for major events like myocardial infarction or stroke<sup>14</sup>.

### CLINICAL EXAMINATION

The assessment of a patient with Type 2 diabetes shall first involve the diagnosis and confirmation of the type of diabetes by blood glucose and HbA1c evaluation. Additional evaluation includes the evaluation of the diabetes complications, presence of co-morbidities, and overall health status. The clinician must explore behavioural factors (eating pattern, calorie counting, physical activities, sleep behaviour, addictions), medications and vaccinations, technology use, and social life assessment. A comprehensive physical examination of the patient must be conducted with special emphasis on fundoscopic examination, skin examination, foot examination, cognitive function, mental state examination, and bone health assessment.<sup>15</sup>

## DIFFERENTIAL DIAGNOSIS

Table 1

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

## SUPPORTIVE INVESTIGATIONS

### Essential:

- Blood Sugar Profile: Fasting Blood sugar (FBS)  $\geq$  126 mg/dL, Post-prandial Blood sugar (PPBS)  $\geq$  200 mg/dL, Glycated Haemoglobin HbA1c  $\geq$  6.5%
- Complete haemogram, urine examination for glucose, proteins, ketone bodies, and microscopic examination of urine for pus cells.

### Advanced:

- Oral Glucose Tolerance Test
- 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<sup>16</sup>

**Table 2**

<i>Criteria of diagnosis of Diabetes among non-pregnant individuals</i>
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≥126mg/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≥200mg/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 a 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 ≥5.7%-6.4%

**PRINCIPLES OF MANAGEMENT:**

**Red Flag signs:**

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

1. Severe cardiovascular disease, including valvular and ischemic heart disease.
2. Severe associated infective morbidity like pneumonia, tuberculosis, sepsis, etc.
3. Advanced stages of malignancy
4. Visual loss due to diabetic retinopathy
5. Severe motor or autonomic dysfunction
6. Severe renal dysfunction with severely reduced GFR
7. Diabetic ketoacidosis
8. Hypoglycemia
9. CVA
10. Hyponatremia
11. Hyperosmolar non-ketotic coma

**A) Preventive management<sup>19</sup>**

Prevention of diabetes includes approaches for primary, secondary, and tertiary management of the condition. The primary measures shall target persons with obesity/increased BMI. A targeted 7% weight loss and moderate physical exercise may be useful for prevention or reversal of the disease. Trials also suggest that individualized low-calorie diet plan and lifestyle/behavioural therapy results in prevention or delay of Type 2 diabetes mellitus and related cardiovascular morbidity. Opportunistic screening must be conducted for the following criteria.

**Table 4**

Persons with age of 18 years and above
Persons with a high BMI ( $\geq 25 \text{ kg/m}^2$ )
Women with a history of gestational diabetes
First- or second- degree relative with diabetes
Hypertensive individuals
Sedentary lifestyle
Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovarian syndrome, small-for- gestational age birth weight)
If results are normal, testing should be repeated at a minimum of 3-year intervals, with consideration of more frequent testing depending on initial results and risk status.

**Yoga and Pranayama <sup>42, 20</sup>**

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

**Table 5: 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)		

Criteria	Yoga Techniques	Approximate duration	Effects
	<i>Vakrasana</i> (spinal twist)		
	<i>Paschimottasana</i> (seated forward bend)		
	<i>Bhujangasana</i> (cobra pose)		
	<i>Naukasana</i> (boat pose)		
	<i>Pavanamuktasana</i> (wind releasing pose),		
	<i>Setubandhasana</i> (Bridge pose)		
	<i>Sarvangasana</i> (shoulder stand)		
	<i>Surya namaskara</i>	Slow speed, 3–7 rounds according to an individual's capacity	Stimulates insulin production through brain signalling Significantly decreases hip circumference, exerting beneficial effects on glycaemic 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 minutes	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</i> , <i>surya mudra</i> , <i>prana mudra</i> , <i>apana mudra</i> , <i>gyana mudra</i>	15–45 minutes	Promote deep relaxation and eliminate stress. Boost metabolic rates, promote weight loss, and reduce sugar levels.

Criteria	Yoga Techniques	Approximate duration	Effects
Shuddhi kriya (cleansing processes)	Kapalbhati (frontal brain purification)	5 rounds, 120 strokes	Abdominal pressure created during exhalation improves the efficiency of $\beta$ -cells of the pancreas Helps in the production of insulin and controlling glucose levels in the blood
	Agnisara kriya (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
	Vaman dhauti (stomach cleansing)	Once a week	Increases glucose uptake, minimizes insulin resistance, and promotes the function of insulin by reducing levels of circulating free fatty acids in the body
	Full shankhprakashalana (intestine cleansing)	Once a year	Significantly reduces blood glucose levels, Increases insulin production
	Laghu shankhprakashalana (short cleansing)	Every 40 day	
Dhyana (Meditation)	Meditation	10 minutes or more	Beneficial psychological effects, such as faster reactions to stimuli and being less prone to various forms of stress

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

## Unani medicine's perspective

The general line of treatment as mentioned in classics:

- *Taskīn-i-Tishnagī* (Quenching of thirst)<sup>12,21</sup>
- *Tabrīd* (Cooling of body)<sup>13</sup>
- *Tabrīd-i-Kulya* (Cooling of kidneys)<sup>21</sup>
- *Ta'dīl-i-Mizāj-i-Gurda* (Correction of temperament of kidneys)<sup>22</sup>
- *Tartīb-i-Badan* (Moistening of body)<sup>12</sup>
- *Taqwiyat-i-Gurda* (Strengthening / toning up of kidneys)<sup>22</sup>
- *Taqwiyat-i-Badan* (Strengthening / toning up of body)<sup>12</sup>
- *Ta'rīq* (Inducing diaphoresis) to divert the matter towards skin.<sup>12</sup>
- *Talyīn-i-Ṭabī'at* (Inducing laxation)<sup>12</sup>

The main line of treatment is '*Ilāj bi'l Dawā'* (Pharmacological Treatment) [IUMT-7.1.10] and '*Ilāj bi'l Tadbīr* (Regimenal Therapy) [IUMT-7.2.0]. '*Ilāj bi'l Tadbīr* (Regimenal Therapy) includes application of *Dimad* (Poultice) [IUMT-6.2.52]<sup>12</sup>, *Hijāma* (Cupping) [IUMT-7.2.30]<sup>23</sup>, *Faṣd* (Venesection) [IUMT-7.2.6]<sup>12,13</sup>, *Qay'* (Inducing emesis)[IUMT-7.2.3]<sup>22</sup>, *Ābzān* (Sitz bath)

[IUMT-6.2.96] with cold water<sup>12</sup> *Huqna Mutawassita* (Moderate enema) [IUMT-6.2.163]<sup>12;21, 24</sup>, *Hammām* [IUMT-7.2.70] which induces dryness<sup>25</sup>, *Tadhīn* (Oiling) [IUMT-6.2.116]<sup>12</sup>, *Shamūm* (Inhalation) [IUMT-6.2.101]<sup>12</sup>, *Naṭūl* (Douche) [IUMT-6.2.95]<sup>12</sup>, *Kimād* (Fomentation) [IUMT-6.2.31]<sup>12</sup> etc.

- **For prevention of progression:** Avoiding the causes that may lead to *Dhayābītus Ḥār* e.g., sedentary lifestyle, obesity, mental stress, tobacco use, etc.

**Dos & Don'ts**

**Table 6**

Dos	Don'ts
<ul style="list-style-type: none"> <li>• <i>Mā' al-Sha'īr</i> (Barley water) of thick consistency<sup>12</sup></li> <li>• <i>Mā' al-Jubn</i> (Whey)<sup>12</sup></li> <li>• <i>Mā' al-Qara'</i> (Juice of fruit of <i>Cucurbita maxima</i> Duchesne.)<sup>12</sup></li> <li>• <i>Mā' al-Khiyār</i> (Cucumber juice)<sup>12</sup></li> <li>• <i>Āb-i-Khurfa</i> (Juice of plant of <i>Portulaca oleracea</i> L.)<sup>12</sup></li> <li>• Chilled buttermilk prepared with goat milk/cow milk<sup>12,25</sup></li> <li>• Fruits<sup>12</sup> (As per the instruction of Unani physician)</li> <li>• Rest in a cold /cold wet atmosphere<sup>12</sup></li> <li>• Bathing with lukewarm water<sup>12</sup></li> <li>• Increased water intake<sup>21</sup></li> </ul>	<ul style="list-style-type: none"> <li>• Sweet dishes<sup>12</sup></li> <li>• Salty dishes<sup>12</sup></li> <li>• Spicy dishes<sup>12</sup></li> <li>• Rigorous physical activity resulting in exhaustion<sup>12</sup></li> <li>• Indulgence in excessive sexual activity<sup>12</sup></li> </ul>

**B) Interventions for disease management:**

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

- OPD level management
- Advice **Dos &Don'ts**
- Referral criteria

**Clinical Diagnosis:**

Type 2 Diabetes mellitus presents at the clinic in an adult with either the classical presentation of polydipsia, polyuria, fatigue, or often as an incidental discovery of raised blood glucose levels during a routine health check-up. There may be an increase in occurrences of bacterial and fungal infections and pruritus vulva in women. In many cases, any complication of the disease may be the initial presenting symptom of the disease. Patients may also present with levels of prediabetes on incidental discovery. The diagnosis is 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 ≥ 6.5%.
- Urine examination for glucose, proteins, ketone bodies, and microscopic examination for pus cells.
- Blood for serum creatinine, lipid profile and liver function tests.

## Management:

### OPD level management:

#### Single drugs/Compound Formulations for oral administration

Table 7

S. No.	Drug	Dosage form	Dose (per day)	Time of administration	Duration and Frequency	Badraqa (vehicle)
1.	<i>Āmla</i> ( <i>Phyllanthus emblica</i> L.) <sup>26,27</sup> [Dried fruit]	Powder	3-5 g.	As directed by physician	As prescribed by physician	Water
2.	<i>Rayḥān</i> ( <i>Ocimum sanctum</i> L.) <sup>26,28</sup>	Leaf juice	5-6 mL.	As directed by physician	As prescribed by physician	
3.	<i>Zanjabīl</i> ( <i>Zingiber officinale</i> Roscoe) <sup>26,29</sup>	Powder	5-10 g in divided dose	As directed by physician	As prescribed by physician	Water
4.	<i>Qurṣ-i-Zayābitus Sāda</i> <sup>30</sup>	Tablet	5-10 g in divided dose	As directed by physician	As prescribed by physician	Water
5.	<i>Safūf-i- Zayābitus Sāda</i> <sup>30</sup>	Powder	5-10 g in divided dose	As directed by physician	As prescribed by physician	Water

#### Formulations for local application:

##### Ḍimād (Poultice):

Application of Ḍimād containing following ingredients on renal area<sup>5</sup>:

Ṣandal Safayd (*Santalum album* L.), Kāfūr (Camphor), Gul Surkh (*Rosa damascena* Mill.), Rose water

##### Ḍimād (Poultice):

Application of Ḍimād containing following ingredients on renal area<sup>5</sup>:

Flour of 'Adas (Lentil) and rose water

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. 'Ilāj bi'l Tadbīr (Regimenal Therapy), described under principles of management may be recommended as per assessment of the physician about the condition of the patient and the stage of disease. The duration of the treatment may vary from patient to patient. The physician may decide the dosage (per dose) and duration of the therapy based on the clinical findings and response to the therapy.

**Recommended and restricted diet & lifestyle**—Same as described under preventive management

**Follow up (With duration)** –1st Follow-up after 15 days followed by 2nd follow-up on the 30th day.

#### Reviews should include:

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.

- Management of T2DM in terms of diet, exercise, and other interventions.
- Discussing the person's knowledge of the condition, any concerns they have, their personal preferences, and their ability to access services.
- Reviewing the effectiveness and tolerability of all treatments.
- Self-management support.
- Monitoring the long-term course of the condition with periodic review.

### **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/SHCO (10-20 bedded hospital) with basic facilities and routine investigations]

- Management with single or compound drugs for internal and external use.
- Management with '*Ilāj bi'l Tadbīr* (Regimenal Therapy) as described under the principles of management and as per the assessment of physician about the condition of the patient and stage of disease.
- '*Ilāj bi'l Ghidhā*' (Dieto-therapy) and life-style modifications
- Clinical Follow-up
- Referral criteria

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

**Investigations:** Same as Level 1.

Supportive investigations to assess organ involvement includes:

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

### **Management:**

Patients referred from Level- 1, may be continued the same treatment, if appropriate for the presenting complaints. For new patients at this level, the management described in Level- 1,

may also be considered while giving prescriptions. At this level, the patient may be preferably treated in the indoor department.

**Table 8: Single drugs/Compound Formulations for oral administration**

S. No.	Drug	Dosage form	Dose per day	Time	Duration & Frequency	Badraqa (vehicle)	Precaution/Contraindication
1.	<i>Jāmun</i> ( <i>Syzygium cumini</i> L.) <sup>26,31</sup> seed	Powder	5-10 g.	As directed by physician	As prescribed by physician	Water	Nothing specific (NS)
2.	<i>Hulba</i> ( <i>Trigonella foenum-graecum</i> L.) <sup>26,32,33</sup> seed	Powder	3.5-7 g.	As directed by physician	As prescribed by physician	Water	NS
3.	<i>Habba al-Sawdā'</i> / <i>Kalonjī</i> ( <i>Nigella sativa</i> L.) <sup>26,34,35</sup> seed	Powder	1-2 g.	As directed by physician	As prescribed by physician	Water	NS
4.	<i>Dār Chīnī</i> ( <i>Cinnamomum cassia</i> (L.) J.Presl) <sup>26,36</sup> Stem bark	Powder	1-2 g.	As directed by physician	As prescribed by physician	Water	NS
5.	<i>Qurṣ-i-Zayābitus Khāṣ</i> <sup>30</sup>	Tablet	1-2 g.	As directed by physician	As prescribed by physician	Water	NS
6.	<i>Safūf-i-Zayābitus Dūlābī</i> <sup>30</sup>	Powder	3-6 g.	As directed by physician	As prescribed by physician	Water	NS

**‘Ilāj bi’l Tadbīr (Regimenal therapy):**

**Ḍimād (Poultice):**

Application of Ḍimād containing following ingredients on renal area<sup>12</sup>:

Ṣandal (*Santalum album* L.), Gulnār (*Punica granatum* L.), Tukhm-i-Kāhū (*Lactuca sativa* L.), Gil Armanī (Armenian bole), Barg-i-Mughīlān (Leaves of *Acacia nilotica* (Linn.) Willd.), Āb-i Kāsnī Sabz (Juice of *Cichorium intybus* L.)

**Ḍimād (Poultice):**

Application of Ḍimād containing following ingredients on renal area<sup>12</sup>:

Ārd-i-Jav (Flour of seed of *Hordeum vulgare* L.) 12 g, Gil Armanī (Armenian bole) 06 g, ‘Adas Muqashshar (Dehusked lentil) 06 g, Gul Surkh (*Rosa damascena* Mill.) 06 g, Āb-i-Barg-i-Khurfa (Juice of leaves of *Portulaca oleracea* L.)

**Ḍimād (Poultice):**

Application of Ḍimād containing following ingredients on renal area<sup>12</sup>:

Ṣandal (*Santalum album* L.), Gulnār (*Punica granatum* L.), Aqāqiyā (Extract of pods of *Acacia nilotica* (Linn.) Willd.), Gil Armanī (Armenian bole), Ārd-i-Jav (Flour of seed of *Hordeum vulgare* L.), Āb-i Kāsnī Sabz (Juice of *Cichorium intybus* L.)

**Huqna (Enema):**

*Huqna* with fresh milk, *Roghan-i-Kadū* and almond oil.<sup>12</sup>

**Recommended and restricted diet & Lifestyle**

Same as described under preventive management

**Follow-up (with duration):**

The follow-up may be done once a fortnight. The patients who are responding well may be treated as per the assessment of physician.

**Referral criteria:**

- ✓ Same as level 1

**At Level 3:** (Ayush hospitals attached with teaching institutions/ Ayush research institutions having indoor facility/ District level/ state level Ayush hospitals/ Tertiary care Allopathic hospitals having Ayush facilities)

- Management with single or compound drugs for internal and external use
- Management with '*Ilāj bi'l Tadbīr*' (Regimenal Therapy) described under principles of management as per the assessment of physician about the condition of the patient and stage of disease
- '*Ilāj bi'l Ghidhā*' (Dieto-therapy) and lifestyle modifications
- Clinical Follow-up
- Referral criteria

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

**Investigations:** Same as Levels 1 and 2.

Additional Investigations may be done as follows:

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

**Management:**

Patients referred from Level 1 or 2, may continue the same treatment, if appropriate for the presenting complaints. For new patients at this level, the management described at Level-1 and Level-2, may also be considered while giving prescriptions. At this level, the patient may be preferably treated in the indoor department.

## Single drugs/Compound Formulations for oral administration

**Table 9**

S. No.	Drug	Dosage form	Dose per day	Time	Duration & Frequency	Badraqa (vehicle)	Precaution/Contraindication
1.	<i>Kundur (Boswellia serata</i> Roxb. ex Colebr.) <sup>37</sup>	Powder	1-3 g.	As directed by physician	As prescribed by physician	Water	NS
2.	<i>Şibr (Aloe vera</i> L.) <sup>26,38</sup>	Powder	1/2-1 g.	As directed by physician	As prescribed by physician	Water	NS
3.	<i>Tukhm-i-Katān (Linum usitatissimum</i> L.) <sup>26,39</sup> seed	Mucilage	5-10 g. of seed to obtain mucilage	As directed by physician	As prescribed by physician	-	NS
4.	<i>Tamar Hindī (Tamarindus indica</i> L.) <sup>25</sup> pulp	Decanted water	20-40 gm in divided doses	As directed by physician	As prescribed by physician	-	NS
5.	<i>Ṭabāshīr (Bambusa bambus</i> Druce.) <sup>12</sup>	Powder	3.5-7 g.	As directed by physician	As prescribed by physician	-	NS
6.	<i>Post-i-Darakht-i-Gūlar (Ficus racemosa</i> L.) <sup>12</sup> stem bark	Powder	6 g.	As directed by physician	As prescribed by physician	Water	NS
7.	<i>Safūf-i-Zayābītus Qawī</i> <sup>30</sup>	Powder	3-5 g.	As directed by physician	As prescribed by physician	Water	NS
8.	<i>Qurş-i-Zayābītus</i> <sup>40</sup>	Tablet	4.5 g. in divided dose	As directed by physician	As prescribed by physician	Water	NS
9.	<i>Qurş-i-Kāfūr</i> <sup>40</sup>	Tablet	10.5 g. in divided dose	As directed by physician	As prescribed by physician	Water	NS
10.	<i>Roghan-i-Bādām</i> <sup>41</sup>	Oil	5-10 mL.	As prescribed by physician	As prescribed by physician	-	NS

### **‘Ilāj bi’l Tadbīr (Regimenal therapy):**

#### **Shamūm (Inhalation):**

- Shamūm (Inhalation) with *Kāfūr* (Camphor).<sup>5</sup>

#### **Shamūm (Inhalation):**

- Shamūm (Inhalation) with *Nīlofar (Nymphaea lotus* L.).<sup>5</sup>

#### **Ḍimād (Poultice):**

Application of *Ḍimād* containing following ingredients on renal area<sup>42</sup>:

*Daqīq al-Sha‘īr* (Flour of *Hordeum vulgare* L.), vinegar, *Roghan-i-Gul*

**Ḍimād (Poultice):**

Application of Ḍimād containing following ingredients on renal area<sup>12,25</sup>:

Equal quantity of Ṣandal (*Santalum album* L.), Aqāqiyā (Extract of pods of *Acacia nilotica* (Linn.) Willd.), Kāfūr (Camphor), Banj (*Hyoscyamus niger* L.) mixed with Rose water.

**Tadhīn (Oiling):**

- Application of Roghan-i-Gul on renal area.<sup>5</sup>

**Kimād (Fomentation):**

- Keeping of cloth soaked with chilled vinegar and rose water on low back.<sup>42</sup>

**Naṭūl (Douche):**

- Naṭūl with almond oil on lower back at frequent intervals.<sup>41</sup>

**Ḥuqna (Enema):**

- Ḥuqna with rose water and mucilage of seed of *Plantago ovata* Forsk.<sup>25</sup>
- Ḥuqna (Enema) with juice of Khurfa (*Portulaca oleracea* L.), Bartang (*Plantago major* L.) and egg white.<sup>12</sup>

**Riyāḍat (Exercise):<sup>43</sup>**

- People with diabetes may be advised to follow advice on physical activity as for the general population.
- Supervised and structured exercise programmes may be of particular benefit in type 2 diabetes.
- American Diabetes Association recommends that all adults with diabetes reduce sedentary time (avoiding periods >90 minutes) and do either 150 minutes per week of moderate-intensity exercise or 75 minutes per week of vigorous-intensity exercise.
- Muscle-strengthening (resistance) exercise is recommended on 2 or more days of the week. Of course, older individuals and those with disabilities may not be able to follow these recommendations in full.

**Recommended and restricted diet & Lifestyle**

Same as described under preventive management

**Follow-up (with duration):**

The follow-up may be done once a fortnight. The patients who are responding well may be treated as per the assessment of physician.

**Referral criteria:**

- ✓ Same as Level 2, with
- ✓ Any condition or serious complication beyond the scope of Unani treatment

## REFERENCES

1. National Unani Morbidity Codes. Available from: <http://namstp.ayush.gov.in/#/Unani> [accessed on 18th January, 2024]
2. <https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/119724091> [accessed on 18th January, 2024]
3. 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>
4. 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/>
5. Khan MA. *Ikseer-i Azam*. Vol. III, 3<sup>rd</sup>edn. Lucknow: Matba' Namee Munshee Naval Kishor; 1906; 446-54.
6. 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>
7. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels, Belgium: 2021. Available at: <https://www.diabetesatlas.org>5. Sarwar N, Gao P, Kondapally Seshasai SR, Gobin R, Kaptoge S, Di Angelantonio E, et al. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. Lancet [Internet] 2010 [cited 2024 Jan 17];375(9733):2215–22. Available from: <https://pubmed.ncbi.nlm.nih.gov/20609967/>
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. Antaki Da'ud b. 'Umar. *Tazkira uli'l Albab*. Vol. II. New Delhi: Central Council for Research in Unani Medicine; 2009; 195.
14. Campbell I, Edinburgh F. Epidemiology and Clinical Presentation of Type 2 Diabetes. Value in Health [Internet] 2000 [cited 2024 Jan 19];3(SUPPL. 1):3–6. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1046/j.1524-4733.2000.36014.x>
15. Tamez-Pérez HE, Quintanilla-Flores DL, Rodríguez-Gutiérrez R, González-González JG, Tamez-Peña AL. Steroid hyperglycemia: Prevalence, early detection and therapeutic recommendations: A narrative review. World J Diabetes [Internet] 2015 [cited 2024 Jan 23];6(8):1073. Available from: <https://pubmed.ncbi.nlm.nih.gov/2615447/>
16. American Diabetes Association. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2024. Diabetes Care [Internet] 2024 [cited 2024 Jan 19];47(Supplement\_1):S20–42. Available from: <https://dx.doi.org/10.2337/dc24-S002>

17. Vedantam D, Poman DS, Motwani L, Asif N, Patel A, Anne KK. Stress-Induced Hyperglycemia: Consequences and Management. *Cureus* [Internet] 2022 [cited 2024 Jan 23]; 14(7). Available from: /pmc/articles/PMC9360912/
18. Ferraù F, Albani A, Ciresi A, Giordano C, Cannavò S. Diabetes Secondary to Acromegaly: Physiopathology, Clinical Features and Effects of Treatment. *Front Endocrinol (Lausanne)* [Internet] 2018 [cited 2024 Jan 23];9(JUL):358. Available from: /pmc/articles/PMC6043782/
19. American Diabetes Association. 3. Prevention or Delay of Type 2 Diabetes and Associated Comorbidities: Standards of Medical Care in Diabetes—2022. *Diabetes Care* [Internet] 2022 [cited 2024 Feb 29];45(Supplement\_1):S39–45. Available from: <https://dx.doi.org/10.2337/dc22-S003>
20. 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.
21. Ibn Sina. *Al-Qanun fi'l-Tibb*. Vol. III (Part II). New Delhi: Jamia Hamdard; 1411 H., pp. 783-85. (from old file)
22. Baha al-Dawla. *Khulasa al-Tajarib*. Delhi: Matba Muhammadi o Ahmadi; 1866; 548-49.
23. Azza A. Abd EL-Hady, Ph.D., S. K. A. M., MOHSEN M. HELMY, M.D., B. H. E. P. Effect of Cupping Therapy on Glycemic Control in Type II Diabetic Patients. *The Medical Journal of Cairo University*, 2018; 86(March): 63-67. doi: 10.21608/mjcu.2018.55032.
24. Ibn Hubal. *Kitab al-Mukhtarati'l-Tibb*. Vol. III. 1<sup>st</sup>edn. Hyderabad: Da'ira al-Ma'arif al-Usmaniyya; 1363 H.; 425-427.
25. Razi Muhammad b. Zakariyya. *Kitab al-Hawi fi'l-Tibb*. Vol. X. Hyderabad: Daira al-Maarif al-Usmaniyya; 1961;189-215.
26. Anonymous. AYUSHMAN BHARAT, AYUSH Health and Wellness Centres Orientation Guidelines for Community Health Officers under Unani stream. New Delhi: Ministry of Ayush, Govt. of India; 2021: 143.
27. Usharani P, Fatima N, Muralidhar N. Effects of *Phyllanthus emblica* extract on endothelial dysfunction and biomarkers of oxidative stress in patients with type 2 diabetes mellitus: a randomized, double-blind, controlled study. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy*. 2013 Jul 26:275-84.
28. Agrawal P, Rai V, Singh RB. Randomized placebo-controlled, single blind trial of holy basil leaves in patients with noninsulin-dependent diabetes mellitus. *Int J Clin Pharmacol Ther*. 1996 Sep;34(9):406-9. PMID: 8880292.
29. Arablou T, Aryaeian N, Valizadeh M, Sharifi F, Hosseini A, Djalali M. The effect of ginger consumption on glycemic status, lipid profile and some inflammatory markers in patients with type 2 diabetes mellitus. *International journal of food sciences and nutrition*. 2014 Jun 1;65 (4):515-20.
30. Anonymous. National Formulary of Unani Medicine, Part I. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2006: 46, 246, 247
31. G. Shivaprakash, M. R.S.M. Pai, M. Nandini, K. Reshma, D. A. Sahana, K. Rajendran, A. Shirwaikar, M. R. Adhikari, J. Ganesh. Antioxidant potential of *Eugenia jambolana* seed; A randomized clinical trial in type 2 diabetes mellitus. *International Journal of Pharma and Bio Sciences*. 2011; 2(2): 220-228.
32. Neelakantan, N., Narayanan, M., de Souza, R.J. et al. Effect of fenugreek (*Trigonella foenum-graecum*L.) intake on glycemia: a meta-analysis of clinical trials. *Nutr J* 13, 7 (2014). <https://doi.org/10.1186/1475-2891-13-7>
33. Najdi RA, Hagraas MM, Kamel FO, Magadmi RM. A randomized controlled clinical trial evaluating the effect of *Trigonella foenum-graecum* (fenugreek) versus glibenclamide in patients with diabetes. *Afr Health Sci*. 2019 Mar;19(1):1594-1601. doi: 10.4314/ahs.v19i1.34. PMID: 31148988; PMCID: PMC6531936.
34. Bamosa AO, Kaatabi H, Lebdaa FM, Elq AM, Al-Sultanb A. Effect of *Nigella sativa* seeds on the glycemic control of patients with type 2 diabetes mellitus. *Indian J PhysiolPharmacol*. 2010 Oct-Dec;54(4):344-54. PMID: 21675032.

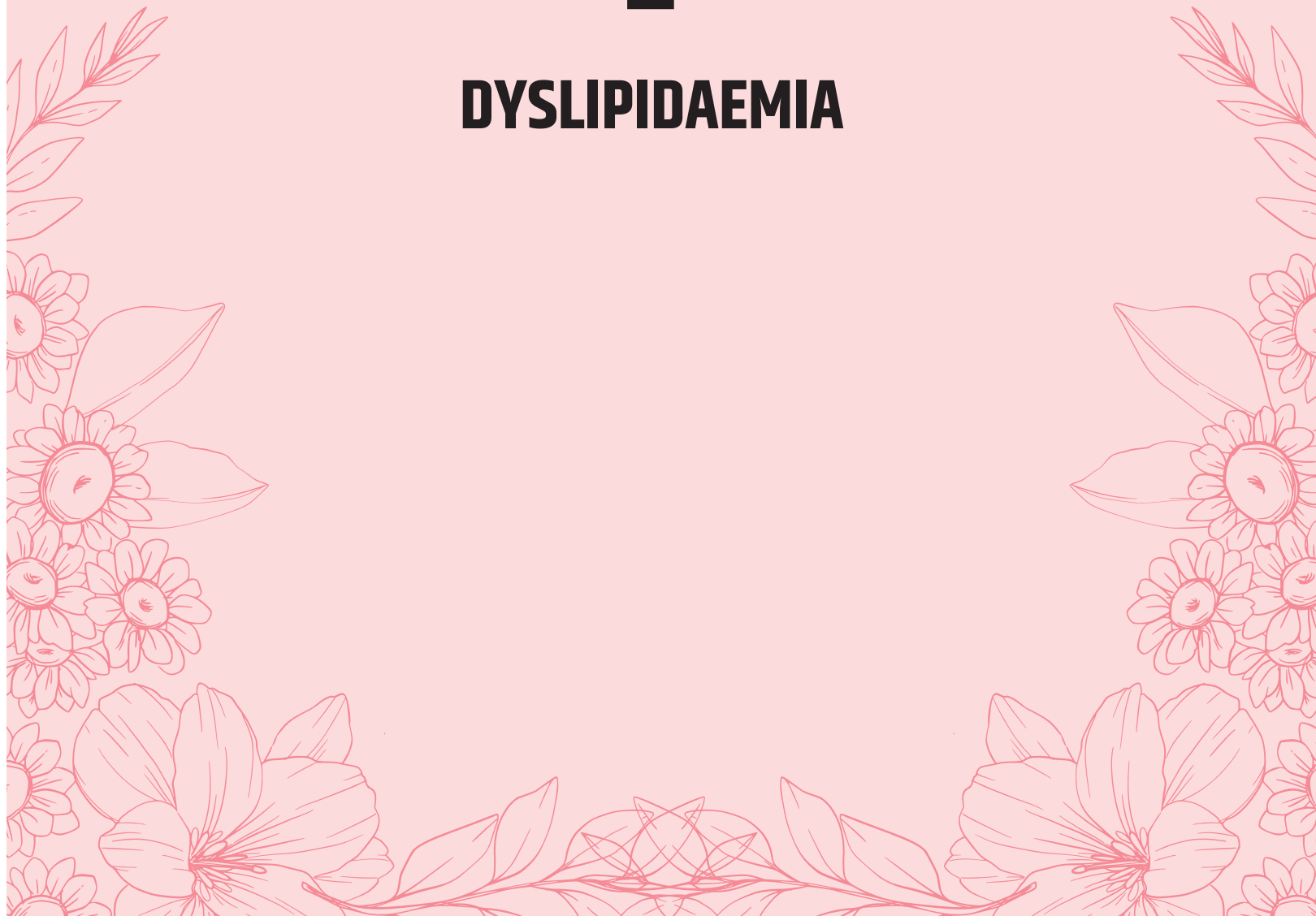
35. Kooshki A, Tofighiyan T, Rastgoo N, Rakhshani MH, Miri M. Effect of *Nigella sativa* oil supplement on risk factors for cardiovascular diseases in patients with type 2 diabetes mellitus. *Phytother Res*. 2020 Oct;34(10):2706-2711. doi: 10.1002/ptr.6707. Epub 2020 Jun 8. PMID: 32510754.
36. Khan A, Safdar M, Ali Khan MM, Khattak KN, Anderson RA. Cinnamon improves glucose and lipids of people with type 2 diabetes. *Diabetes care*. 2003 Dec 1;26(12):3215-8.
37. Azadmehr A, Ziaee A, Ghanei L, Huseini HF, Hajiaghaee R, Tavakoli-Far B, Kordafshari G. A randomized clinical trial study: anti-oxidant, anti-hyperglycemic and anti-hyperlipidemic effects of olibanum gum in type 2 diabetic patients. *Iranian journal of pharmaceutical research: IJPR*. 2014;13(3):1003.
38. Zhang Y, Liu W, Liu D, Zhao T, Tian H. Efficacy of Aloe Vera Supplementation on Prediabetes and Early Non-Treated Diabetic Patients: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Nutrients*. 2016 Jun 23;8(7):388. doi: 10.3390/nu8070388. PMID: 27347994; PMCID: PMC4963864.
39. Thakur G, Mitra A, Pal K, Rousseau D. Effect of flaxseed gum on reduction of blood glucose and cholesterol in type 2 diabetic patients. *International journal of food sciences and nutrition*. 2009 Jan 1;60(sup6):126-36.
40. Kabiruddin M. *Al-Qarabadeen*. New Delhi: Central Council for Research in Unani Medicine; 2006; 931, 932.
41. Ibn Zuhr. *Kitab al-Taysir fi'l Mudawa wa'l Tadbir* (Urdu translation). New Delhi: Central Council for Research in Unani Medicine; 1986; 160-62.
42. Harrani Sabit b. Qurra. 1928, *Kitab al-Zakhira fi Ilm al-Tibb*. Cairo:al-Matba al-Amiriyya; 1928; 109-110.
43. Harrani Sabit b. Qurra. 1928, *Kitab al-Zakhira fi Ilm al-Tibb*. Cairo:al-Matba al-Amiriyya; 1928; 109-110.



CHAPTER

2

# DYSLIPIDAEMIA





# DYSLIPIDAEMIA

(ICD 10 Code: E78.5)<sup>1</sup>

(ICD 11 Code: 5C80.Z)<sup>2</sup>

*Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia)

## CASE DEFINITION

Dyslipidaemias 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), and High triglycerides<sup>3</sup>.

## 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 Africa<sup>4</sup>. In India specific, the prevalence of hypercholesterolemia varies from 10 to 15 % in rural to 25–30 % in urban populations<sup>5</sup>.
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)<sup>6-9</sup> while some studies had shown that non-HDL-C predicts CV risk better than LDL-C<sup>10</sup>.
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 %, LDL cholesterol >100 mg/dl: 50-55 %, triglycerides >150 mg/dl: 30-40 % and low HDL cholesterol: 60-70 %. Most national studies have reported higher prevalence of hypercholesterolemia in most Southern and a few North Indian states, more in urban than rural areas, whereas the prevalence of high triglycerides and low HDL cholesterol is similar throughout the country<sup>11</sup>.

## Unani Medicine's Perspective: (*Etiology and Pathophysiology*)

In Unani Medicine, disturbance in the levels of *Shaḥm* (fat) in blood is termed as *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia) which is generally characterized by increase in *Shaḥm* (fat) and may be correlated with *Siman-i Mufrit* (obesity) in terms of clinical features, complications and risk factors as well as treatment.<sup>12-15</sup> The obese persons are more susceptible to the development of dyslipidaemia than lean and thin individuals.<sup>16</sup> Jālīnūs (Galen) mentioned that an obese person may die earlier than lean and thin people.<sup>17</sup>

Obesity leads to fatal disorders due to excess of *Balgham* (phlegm) and less amount of *dam* (blood).<sup>18</sup> Accordingly, like *Siman-i Mufrit* (obesity), the presence of high levels of *Shaḥm* (fat) in blood is due to excessive *Burūdat* (coldness) and *Ruṭūbāt* (wetness) in the body. The causes which increase the *Burūdat* (coldness) and *Ruṭūbāt* (wetness) in the body are cold foods and drinks, cold medicines, rest, sleep, excess of food, sedentary lifestyle, *Ḥammām* (therapeutic bath) after meals, etc.<sup>19</sup>

Another cause of dyslipidemia may be correlated with increased viscosity of the blood that is a manifestation of disturbances in *Haḍm Kabidī* (hepatic digestion). Furthermore, the disturbances in *Haḍm Kabidī* may be caused by cold temperament of liver. The obstruction due to presence of viscous matter or any inflammation may also cause disturbances in *Haḍm Kabidī*.<sup>20</sup>

### Etiology

- *Sū'-i-Mizāj-i-Jigar Bārid Sāda* (simple morbid cold temperament of liver without substance)
- *Sū'-i-Mizāj-i-Jigar Bārid Māddī* (morbid cold temperament of liver with substance)
- Excessive consumption of *Thaqīl Ghidhā'* (high calorie diet): Intake of high saturated fat (animal fat) diet and high trans-fat diet, e.g. fried and processed foods, red meat, cheese, egg yolks, etc.
- Lifestyle: excessive alcohol intake, smoking, physical inactivity, excessive sleep, *Ḥammām* (turkish bath) after meals
- Excessive consumption of *Ghidhā' Bārid* (diet of cold temperament), and *Mashrūbāt Bārida* (cold drinks)
- Overweight and *Siman-i Mufrit* (obesity)<sup>19,21</sup>
- *Ghalaba'-i Balgham* (predominance of phlegmatic humour)<sup>22</sup>
- *Ghalaba'-i Burūdat* (predominance of coldness)<sup>22</sup>

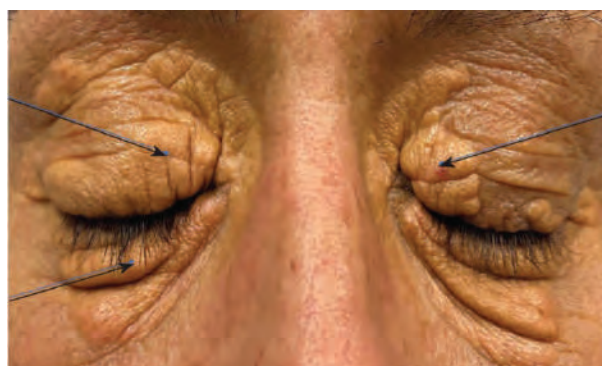
### Pathophysiology

The arteries, veins and other tributaries become narrower in persons with *Siman-i Mufrit* (obesity) leading to narrowing of *Masālik-i-Rūḥ* (routes of the pneuma) in the body. It is therefore the *Rūḥ* (pneuma) becomes unable to travel in the body freely which leads to exhaustion of *Ḥarārat Gharīzī* (innate heat) and dominance of *Burūdat* (coldness) in the body. Further, in obese persons, the quality and quantity of blood becomes low in the body with dominance of *Balgham* (phlegm) that causes thickness of blood, narrowing and rupture of blood vessels, haemorrhage, paralysis leading to sudden death. In females, obesity causes menstrual disorders, lower chances of pregnancy, infertility and miscarriage.<sup>22</sup> In case of any disease, the drugs used for treatment are unable to reach the affected site due to narrowing of vascular routes.<sup>22,23</sup>

### CLINICAL PRESENTATION<sup>24,25</sup>

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

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



- Arcus senilis (gray or white ring around the eye's cornea that is caused by cholesterol depositing in the corneal margin).
- Lipemia retinalis (milky appearance in the retinal vessels due to high blood triglyceride levels with blurred vision).
- 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).
- 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.
- 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.
- Non- Alcoholic Fatty Liver Disease / Metabolic Dysfunction Associated Steatohepatitis (MASH).

**DIFFERENTIAL DIAGNOSIS** 26,27,28

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

**Table 1**

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 oedema, 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 the 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 pruritus, 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

S. No.	Disease condition	Findings
8.	Niemann Pick Disease Type C	Lipidosis due to an 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 a significant intracellular buildup of cholesteryl esters and triglycerides.
10.	Cerebrotendinous xanthomatosis	A rare autosomal recessive genetic condition caused by a mutation in the CYP27A1 gene, resulting in a lack of the mitochondrial enzyme sterol 27-hydroxylase. This enzyme is required to convert cholesterol into chenodeoxycholic acid, a bile acid.

## SUPPORTIVE INVESTIGATIONS <sup>29,30,31</sup>

### Essential:

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

**Table 2: WHO's Classification of Adults according to BMI <sup>32</sup>**

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	
Preobese	25.00-29.99	Increased
Obese class I	30.00-34.99	Moderate
Obese class II	35.00-39.99	Severe
Obese class III	≥40.00	Very severe

**Table 3: Standard BMI classification in Asian adults:**

Classification	BMI (kg/m <sup>2</sup> )	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

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. Liver biopsy
- 15. Waist hip ratio, waist circumference, skin fold thickness
- 16. Plasma leptin
- 17. Upper Abdominal Ultrasound

**DIAGNOSTIC CRITERIA**<sup>29, 33, 34</sup>

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

**Table 4: Diagnostic biochemical parameters for dyslipidaemia in adults**

Levels of risk	TC	LDL-C	TG	HDL-C
<b>Mild-to-moderate risk</b>				
Levels	200-239 mg/dL	130-194 mg/dL	175-499 mg/dL	25-35 mg/dL
<b>Severe risk</b>				
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.

**Classification**<sup>29,24</sup>

Dyslipidaemias are mainly classified into two types:

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

**Secondary:** Secondary dyslipidaemia 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 dyslipidaemia gives 5 categories, according to Frederickson phenotype (World Health Organization) <sup>29</sup>:

- 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.

## PRINCIPLES OF MANAGEMENT

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

### Red Flags <sup>35,36</sup>

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

### Unani Medicine's Perspective

The general line of treatment as mentioned in classics includes:

- *Iṣlāḥ-i-Sū'-i-Mizāj* (correction of morbid temperament) <sup>22</sup>

- *Ta'dīl-i-Sū'-i-Mizāj* (moderation of abnormal temperament): modulation in temperament should be done in *Sū'-i-Mizāj-i-Jigar Bārid Sāda wa maddi* (simple morbid cold temperament of liver with or without substance) <sup>37</sup>.
- *Tarqīq-i- Akhlāt/Mawād* (diluting of humours/morbid body fluids)<sup>22</sup>
- *Tajfīf* (inducing dryness) <sup>38</sup>
- *Taqlīl-i-Ghidhā'* (reducing dietary intake) <sup>38</sup>
- *Taqlīl-i- Nawm* (reducing sleep hours)
- *Ta'rīq* (diaphoresis) <sup>38</sup>
- *Tadhīn* (oiling) with resolvent *rghans* (oils) <sup>38</sup>
- *Tanqiya* (cleansing of morbid matter/ humour from body) In *Sū'-i-Mizāj-i-Jigar BāridMāddī* (morbid cold temperament of liver with substance), elimination of *Mādda* (matter) may be done with *Munḍij o Mushil* (concoctive-purgative) therapy. <sup>38,39</sup>
- After *Munḍij o Mushil* (concoctive-purgative) therapy, *Mā' al-Uṣūl* of the following drugs in the form of decoction may be given:
  - *PostBekh-i-Kabar* (Root bark of *Capparis spinosa*, L.), *Bekh-i-Izkhar* (Root of *Andropogon jwarancusa*, Jones.), *Post Bekh-i-Bādyān* ( root bark of *Foeniculum vulgare*, Gaertn.), *Bādiyān* (*Foeniculum vulgare*, Gaertn.), *PostBekh-i- Karafs* (Root bark of (*Apium graveolens* L.)), *Tukhm-i-Karafs* (Seeds of *Apium graveolens* L.), *Anīsūn* (*Pimpinella anisum* L.), *Bālchad* (*Nardostachys jatamansi* (D. Don) DC), *Parsiyāoshān* (*Adiantum capillis-veneris*) , and *Maṣṭagī* (*Pistacia lentiscus* L.) <sup>40</sup>
- In case of *Sudad al-Kabid* (obstructive liver disease), *Mufattiḥ-i-Sudad* (deobstruent drugs), such as *Afsantīn* (*Artemisia absinthium* L.), *Anīsūn* (*Pimpinella anisum* L.), *Kāsnī* (*Cichorium intybus* L.), *Branjāsaf* (*Achillea millefolium* L), *Kabar* (Root bark of *Capparis spinosa*, L.), *Uṣṭukhuddūs* (*Lavandula stoechas* L.), *Ajwāin* (*Trachyspermum ammi*), etc. may be used. <sup>38</sup>
- In case of *Burūdat-i-Jigar* (coldness of liver) due to *Burūdat-i-Kulya* (coldness of kidney), treatment of kidney may also be considered along with the treatment of liver <sup>38</sup>
- *Mulayyin* (laxative) and *mushil* (purgative) drugs are useful<sup>23</sup>.
- Hot water may be used.<sup>22</sup>
- Low-calorie diet may be used<sup>17,19,21</sup>
- Oily food, fried food and red meat may be avoided.
- Spices, including *Zīra Siyāh* (*Carum carvi* L.), *Rāi* (*Brassica nigra* L.), *Filfil Siyāh* (*Piper nigrum* L.) and *Lehsun* (*Allium sativum* L.) may be used<sup>19</sup>.
- Alcohol may be avoided <sup>41</sup>

#### A. Preventive management<sup>27</sup>

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

- Screening for dyslipidaemia regularly, especially for people with a family history or other risk factors. The frequency and type of screening depend on the individual's age, sex, and health status, but generally, a lipid profile test is recommended every 4 to 6 years for adults and every 2 years for children and adolescents.

- Adopting a healthy lifestyle by eating a balanced diet with plenty of fruits, vegetables, whole grains, lean proteins, and healthy fats, such as omega-3 fatty acids from fish, nuts, and seeds. Avoid foods high in cholesterol, saturated fats, trans fats, added sugars, and salt. If possible, engage in physical activity for at least 150 minutes weekly.
- Maintaining a healthy weight and body mass index, quitting smoking, and limiting alcohol intake are all recommended.
- Comorbidities such as diabetes, hypertension, hypothyroidism, chronic kidney disease, or liver disease can affect lipid levels or increase the risk of cardiovascular disease; therefore, it is important to remain compliant with any medications.

**Table 5: Common Yoga Protocol**

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

Sr. No.	Name of Posture/Procedure		
20.	Sasankasana	Hare Posture	
21.	Balasana	Child Pose	
22.	Uttana Mandukasana	Stretched Up Frog Posture	
23.	Vakrasana	Spinal Twist Posture	
24.	Paschimottanasana	Seated Forward Bend	
25.	Simhasana	Lion Pose	
26.	Marjarasana	Cat Pose	
Prone Positions			
27.	Makarasana	Crocodile Posture	
28.	Bhujangasana	Cobra Pose	
29.	Salabhasana	Locust Posture	
30.	Dhanurasana	Bow Pose	
Supine Positions			
31.	Chatuspadasana Setubandhaasana	Bridge Posture	
32.	Uttanapadasana	Raised Leg Posture	
33.	Matsyasana	Fish Pose	
34.	Ardhahalasana	Half Plough Pose	
35.	Pavanmuktasana	Wind Releasing Posture	
36.	Markatasana	Monkey Pose	
37.	Shavasana	Corpse Body Posture	
38.	Kapalbhati	Forceful Rapid Ex- halations	Sukhasana/Padmasana/Vajrasana 1 inhalation :20-30 exhalation
Breathing Exercises			
39.	Anuloma-Viloma/ Nad- ishodhana 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.	Shitali Pranayam	Cooling breath	Jnana Mudra or Dhyan Mudra or Anjali Mudra (Na- maste Pose) Inhale through Tongue Tube and exhale through nostrils
41.	Bhramari Pranayam	Humming bee breath	Sanmukhi Mudra IMRL Thumb-Eye Nose Mouth Ear
42.	Dhyana	Meditation	Jnana Mudra or Dhyan Mudra or Anjali Mudra Tip of thumb to Tip of IF Other fingers straight/relaxed

## B. Interventions

**LEVEL 1:** At solo Unani physician's clinic/PHC (Optimal Standard of Treatment where Technology and Resources are Limited)

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

## Management

- Since dyslipidaemia is a lifestyle disorder, the most important part of management includes:
  - Lifestyle modification
  - Diet restriction
  - Physical exercise

### A. *'Ilāj bi'l Tadbīr* (Regimenal Therapy)

#### Lifestyle Modification

Lifestyle modification is the cornerstone of long-term control of dyslipidaemia. Therapeutic lifestyle changes, including exercise, weight loss, and dietary modifications, are the first step in the treatment of all patients with dyslipidaemia. Lifestyle modifications can reduce LDL-C levels by about 10 mg/dL. Healthy lifestyle habits are characterized by a healthy diet, regular physical activity, and smoking avoidance<sup>42</sup>.

#### i. Physical Activity

- Physical activity is an important component of lifestyle modification in patients with dyslipidaemia.
- Physical activity may be included in daily activities, such as walking to school, using the stairs, doing housework (hanging out washing, ironing, and dusting) and yard work, and engaging in sports.
- Exercise may also be done on a regular basis, although the resulting changes in LDL and HDL cholesterol are modest<sup>43</sup>
- Moderate-intensity Physical Activity: Regular moderate aerobic exercise, e.g.
  - Brisk walking for ≥30 minutes daily 3-5 days per week (150 minutes/week)<sup>42</sup>
  - Gentle swimming
  - Social tennis
- *Riyāḍat* (Exercise):
  - It is effective in the treatment of dyslipidaemia, and it plays an important role in the maintenance of weight loss.
  - Regular exercise for at least 30 minutes daily 5 days per week can contribute to weight loss and prevention of weight regain, consequently reduction in lipids.
- Playing outdoor games

#### ii. Behavior Modification (Behavioral Lifestyle Change)

All persons with risk factors for ischaemic heart disease should be encouraged to make the following lifestyle changes as appropriate:

- Reducing sedentary behavior (e.g. commuting by bicycle rather than bike/car, choosing the stairs, walking to do errands, etc.)
- Waking up early morning
- Avoid excess sleep and day sleep
- Stop smoking
- Avoid alcohol consumption <sup>42</sup>

**B. ‘Ilāj bi’l Ghidhā’ (Diet Therapy)**

- Maintaining Body Weight:
  - Maintain ideal body weight, i.e. BMI <25 kg/m<sup>2</sup>
  - Weight reduction in the overweight patients, i.e. BMI >25 kg/m<sup>2</sup>
  - Moderate weight reduction (10% of body weight) can significantly improve the lipid profile and lower risk<sup>42,43</sup>.
- To improve dietary habits, the following composition of the optimal diet is recommended under the supervision of a dietician:
  - Low-calorie diet
  - Low Saturated Fat (Animal Fat) and High Polyunsaturated Fat (Plant Fat) Diet
  - Unrefined Carbohydrate Diet
- High-fibre diet with adequate fresh fruits and vegetables <sup>42</sup>.
  - Reduced cholesterol to <200 mg/day and saturated fats (especially trans fats) to <7% of total calories
  - Increased plant stanols, sterols, and soluble fibre (20-30 g/day)
  - Adoption of a Mediterranean diet<sup>43</sup>

**Table 6: Diet Therapy and Lifestyle Modification**

Dos	Don'ts (Disease Aggravating Factors)
<ul style="list-style-type: none"> <li>• Maintaining a diet rich in whole grains, fruits, vegetables, and dietary fibre</li> <li>• Consumption of whole-grain cereals, e.g., brown rice, and Chapati prepared with whole wheat, barley, maize, millets [Jowar (Sorghum), Bajra (Pearl Millet), Ragi (Finger Millet), etc.]</li> <li>• Consumption of fat-free or low-fat dairy products (skimmed dairy products)</li> <li>• Consumption of Foods with low energy density include soups, oatmeal, and lean meats</li> <li>• Keeping added sugars and saturated fat intake to &lt;10% of daily calories</li> <li>• Decreasing sodium intake to &lt;2300 mg/d</li> <li>• Green Tea (<i>Camellia sinensis</i>)</li> <li>• <i>Roghan Zaytūn</i> (Olive Oil)</li> <li>• <i>Sirka</i>: Vinegar</li> </ul>	<ul style="list-style-type: none"> <li>• Overeating</li> <li>• Consumption of fried foods and other foods with added fats and oils</li> <li>• High-sugar drinks and sugar-sweetened beverages</li> <li>• Dry foods and high-fat foods such as cheese, egg yolks, potato chips, and red meat.</li> <li>• Excessive intake of coconut &amp; ground nut</li> <li>• Consumption of whole milk, curd, fermented and bakery items</li> <li>• Consumption of high glycemic index foods (rice, corn, sugar, white bread, white pasta)</li> <li>• Consumption of refined foods such as refined flour</li> <li>• Sedentary Lifestyle</li> <li>• Alcohol Consumption</li> <li>• Smoking</li> <li>• Excess sleep and day sleep</li> </ul>

Dos	Don'ts (Disease Aggravating Factors)
<ul style="list-style-type: none"> <li>A minimum content of: <ul style="list-style-type: none"> <li>40-50 g protein daily minimizes muscle degradation,</li> <li>Minimum 20 g of fat helps to avoid gallstone formation, with</li> <li>sufficient magnesium to avoid constipation</li> </ul> </li> <li>Fluid intake of at least 2.5 L/day to avoid dehydration, which can cause orthostatic hypotension, headache, constipation and nausea.</li> <li>Waking up early morning</li> <li>Regular Exercises</li> <li>Brisk Walking</li> <li>Swimming, Playing Outdoor Games, etc.</li> </ul>	

## OPD Level Management

In patients with *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia), two or more of the following forms of medications may be given along with diet restriction:

**Table 7: Medicines for *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia) at Level 1**

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
1.	<i>Juntiyāna</i> ( <i>Gentiana kurroo</i> Royle) <sup>19,22, 41</sup>	Powder	1-2 gm	After meal	15 days to 1 month	water
2.	<i>Tukhm Sudāb</i> ( <i>Ruta graveolens</i> L.) <sup>19,22,41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
3.	<i>Zarāwand Mudharaj</i> ( <i>Aristolochia rotunda</i> L.) <sup>19,22,41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
4.	<i>Fatrāsaliyūn/ Karafs Kohī</i> ( <i>Prangos pabularia</i> Lindl.) <sup>19,22, 41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
5.	<i>Sandrūs</i> ( <i>Vateria indica</i> L.) <sup>19,22</sup>	Powder	1-2 gm	After meal	15 days to 1 month	water
6.	<i>Luk Maghsūl</i> (Lac) <sup>19,22,41</sup>	Powder	0.5-2 gm	Empty stomach	15 days to 1 month	Water/ vinegar
7.	<i>Tukhm Karafs</i> ( <i>Apium graveolens</i> L.) <sup>19,22, 41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
8.	<i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.) <sup>19,22,41</sup>	Powder	5-7 gm	After meal	15 days to 1 month	water
9.	<i>Mur Makkī</i> ( <i>Commiphora myrrha</i> Engl.) <sup>22</sup>	Powder	0.5-1 gm	After meal	15 days to 1 month	water
10.	<i>Muqil</i> ( <i>Commiphora mukul</i> Hook ex Stocks) <sup>21</sup>	Powder	1-1.5 gm	After meal	15 days to 1 month	water

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
11.	<i>Safūf-i-Muhazzil</i> <sup>22,44,45</sup>	Powder	5 - 10 gm	Morning empty stomach	15 days to 1 month	water
12.	<i>Dawāul Kurkum</i> <sup>22</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
13.	<i>Majūn Falāfilī</i> <sup>19,22,23</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
14.	<i>Itrifal Saghīr</i> <sup>19,21,22,23</sup>	Semi-Solid	10-15 gm in two divided doses	After meals	15 days to 1 month	water
15.	<i>Jawārish Kamūnī</i> <sup>19,22</sup>	Semi-Solid	10-15 gm in two divided doses	After meals	15 days to 1 month	water

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. 'Ilāj bi'l Tadbīr (Regimenal Therapy) described above may be recommended as per assessment of physician about the condition of the patient and status of dyslipidaemia. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

### Follow Up

- Every month or as recommended by the physician

### Reviews should include:

- Monitoring the person's condition and the ongoing impact of the condition on their activities of daily living (ADL) and quality of life (QoL)
- Monitoring the long-term course of the condition
- Management of dyslipidaemia in terms of physical activity, e.g., exercise, sports, etc.
- Discussing the person's knowledge of the condition, concerns, personal preferences, and ability to access services
- Reviewing the effectiveness and tolerability of all treatments. If the patient is improving, continue treatment, and if not, review the totality for further prescription.
- Self-management support

### 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.

**LEVEL 2:** CHC/ small hospitals (10-20 bedded hospitals with basic facilities, such as routine investigations, X-ray, ECG and 2D Echo)

### Clinical Diagnosis

- Same as level 1.
- The case referred from Level 1, or a fresh case reporting directly should be evaluated thoroughly for any complications.

### Investigations

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

- High sensitivity C-reactive protein.
- Apolipoprotein B (ApoB), apolipoprotein A1
- Lipoprotein(a)
- Glycosylated haemoglobin (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 and/or treatment mentioned at this level.
- Diet Therapy and Lifestyle Modification: Same as level 1
- In patients with dyslipidaemia, two or more of the following medications may be given along with diet restriction.

**Table 8: Medicines for *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia) at Level 2**

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
1.	<i>Junṭiyāna</i> ( <i>Gentiana kurroo</i> Royle) <sup>19,22,41</sup>	Powder	1-2 gm	After meal	1-2 months	water
2.	<i>Tukhm Sudāb</i> ( <i>Ruta graveolens</i> L.) <sup>19,22,41</sup>	Powder	3-5 gm	After meal	1-2 months	water
3.	<i>Zarāwand Mudharaj</i> ( <i>Aristolochia rotunda</i> L.) <sup>19,22,41</sup>	Powder	3-5 gm	After meal	1-2 months	water
4.	<i>Fatrāsaliyūn/ Karafs Kohī</i> ( <i>Prangos pabularia</i> Lindl.) <sup>19,22, 41</sup>	Powder	3-5 gm	After meal	1-2 months	water

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
5.	<i>Sandrūs</i> ( <i>Vateria indica</i> L.) <sup>19,22</sup>	Powder	1-2 gm	After meal	1-2 months	water
6.	<i>Tukhm Karafs</i> ( <i>Apium graveolens</i> L.) <sup>19,22</sup>	Powder	3-5 gm	After meal	1-2 months	water
7.	<i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.) <sup>19,22</sup>	Powder	5-7 gm	After meal	1-2 months	water
8.	<i>Mur Makkī</i> ( <i>Commiphora myrrha</i> Engl.) <sup>22</sup>	Powder	0.5-1 gm	After meal	1-2 months	water
9.	<i>Bilādur</i> ( <i>Semicarpus anacardium</i> L.) <sup>21</sup>	Powder	0.5-1 gm	After meal	1-2 months	water
10.	<i>Muqil</i> ( <i>Commiphora mukul</i> Hook ex Stocks) <sup>21</sup>	Powder	1-1.5 gm	After meal	1-2 months	water
11.	<i>Sufūf-ī-Muhazzil</i> <sup>22, 44,45</sup>	Powder	5 - 10 gm	Morning empty stomach	1-2 months	water
12.	<i>Dawāul Kurkum</i> <sup>22</sup>	Semi-Solid	2-3 g in 2 divided doses	After meal	1-2 months	water
13.	<i>Majūn Falāfilī</i> <sup>19,22,23</sup>	Semi-Solid	2-3 g in 2 divided doses	After meal	1-2 months	water
14.	<i>Anqaruya</i> <sup>19,22</sup>	Semi-Solid	2-3 gm	Morning empty stomach	1-2 months	water
15.	<i>Dawāul Luk</i> <sup>22,23</sup>	Semi-Solid	2-3 gm in 2 divided doses	After meal	1-2 months	water
16.	<i>Asānāsiya</i> <sup>19,22</sup>	Semi-Solid	2-3 g in 2 divided doses	After meal	1-2 months	water
17.	<i>Amrosiya</i> <sup>19,22</sup>	Semi-Solid	3-7 g in 2 divided doses	After meal	1-2 months	water
18.	<i>Itrifal</i> <sup>19,21,22,23</sup>	Semi-Solid	10-25 g in 2 divided doses	Bed time	1-2 months	water
19.	<i>Jawārish Kamūnī</i> <sup>19,22</sup>	Semi-Solid	10-15 g in 2 divided doses	After meal	1-2 months	water

- *Tiryāq-i Adviya* (drugs which eliminate toxicity and preserve the life properties) are also useful in the management of the Dyslipidaemia<sup>22</sup>.
- Treatment of Secondary Causes: Dyslipidaemia must not be treated in isolation; the following secondary causes must be addressed:
  - *Siman-i Mufriṭ* (Obesity)
  - *Qillat-i-Darqiyyat* (Hypothyroidism)
  - *Dhayābīṭus Ḥār* (Type 2 Diabetes Mellitus)

## Follow Up

- Every month or as recommended by the Physician

## Referral Criteria

The following patients may be referred to higher centers for better management.

- Same as level 1, with
- Patients not responding to the above-mentioned management and need further management in the form of 'Ilāj bi'l Tadbīr (Regimenal Therapy) procedures
- Psychological imbalance
- Any red flag signs.
- Signs of CVD as stroke, transient ischaemic attack, and angina.

**LEVEL 3:** (Unani Hospitals attached with Teaching Institution, District Level/ Integrated/ State Unani Hospitals, Tertiary Care Allopathic Hospitals having Unani Facilities, multiple Departments/ Facilities for Diagnosis & Interventions, and Additional Facilities, including Dieticians, Counselling, and Physiotherapy Unit)

## Clinical Diagnosis

- Same as level 1 and 2.
- The case referred from Level 1 or 2, or a fresh case reporting directly should be evaluated thoroughly for any complications.
- Plasma Leptin
- Treadmill Test or Exercise stress Test to evaluate the efficacy of the functioning of the heart during exercise

## Management

- Same as level 1 and 2 and/or treatment mentioned at this level
- *Diet Therapy and Lifestyle Modification:* Same as level 1
- In patients with dyslipidaemia, two or more of the following medications may be given along with diet restriction.

**Table 9: Medicines for *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia) at Level 3**

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
1.	<i>Juntyāna</i> ( <i>Gentiana kurroo</i> Royle) <sup>19,22,41</sup>	Powder	1-2 gm	After meal	15 days to 1 month	water
2.	<i>Tukhm Sudāb</i> ( <i>Ruta graveolens</i> L.) <sup>19,22,41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
3.	<i>Zarāwand Mudharaj</i> ( <i>Aristolochia rotunda</i> L.) <sup>19,22, 41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)
4.	<i>Fatrāsaliyūn/ Karafs Kohī (Prangos pabularia Lindl.)</i> <sup>19,22, 41</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
5.	<i>Sandrūs (Vateria indica L.)</i> <sup>19,22</sup>	Powder	1-2 gm	After meal	15 days to 1 month	water
6.	<i>Luk Maghsūl (Lac)</i> <sup>19,22</sup>	Powder	0.5-2 gm	Empty stomach	15 days to 1 month	Water/ vinegar
7.	<i>Tukhm Karafs (Apium graveolens L.)</i> <sup>19,22</sup>	Powder	3-5 gm	After meal	15 days to 1 month	water
8.	<i>Marzanjosh (Origanum vulgare L.)</i> <sup>19,22</sup>	Powder	5-7 gm	After meal	15 days to 1 month	water
9.	<i>Chirchita (Achyranthes aspera L.)</i> <sup>22</sup>	Powder	1-3 gm	After meal	15 days to 1 month	water
10.	<i>Mur Makkī (Commiphora myrrha Engl.)</i> <sup>22</sup>	Powder	0.5-1 gm	After meal	15 days to 1 month	water
11.	<i>Bilādur (Semicarpus anacardium L.)</i> <sup>21</sup>	Powder	0.5-1 gm	After meal	15 days to 1 month	water
12.	<i>Muqil (Commiphora mukul Hook ex Stocks)</i> <sup>21</sup>	Powder	1-1.5 gm	After meal	15 days to 1 month	water
13.	<i>Sufūf-ī-Muhazzil</i> <sup>22,44,45</sup>	Powder	5 - 10 gm	Morning empty stomach	15 days to 1 month	water
14.	<i>Dawāul Kurkum</i> <sup>22</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
15.	<i>Majūn Falāfilī</i> <sup>19,22,23</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
16.	<i>Anqaruya</i> <sup>19,22</sup>	Semi-Solid	2-3 gm	Morning empty stomach	15 days to 1 month	water
17.	<i>Dawāul Luk</i> <sup>22,23</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
18.	<i>Asānāsiya</i> <sup>19,22</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water
19.	<i>Amrosiya</i> <sup>19,22</sup>	Semi-Solid	3-7 gm in two divided doses	After meal	15 days to 1 month	water
20.	<i>Itrifal Saghīr</i> <sup>19,21,22,23</sup>	Semi-Solid	10-15 gm in 2 divided doses	After meals	15 days to 1 month	water
21.	<i>Jawārish Kamūnī</i> <sup>19,22</sup>	Semi-Solid	10-15 gm in two divided doses	After meal	15 days to 1 month	water

## **‘Ilāj bi’l Tadbīr (Regimenal Therapy)**

### **1. Riyāḍat (Exercise)**

- It is effective in the treatment of *Khalal Shaḥmiyyāt al-Dam* (Dyslipidaemia), as it plays an important role in the maintenance of weight loss.
- Regular exercise for at least 30 minutes daily 5 days per week can contribute to weight loss and prevention of weight regain, leading to reduction in lipids.
- *Riyāḍat Hathītha* (rigorous and rapid exercise)
- *Riyāḍat Kathīra* (exercise for a prolonged duration)<sup>19,21,22,23, 41</sup>

### **2. Dalk (Massage)**

- *Dalk* (massage): *Dalk Ṣulb* (massage with firm/ strong pressure), and *Dalk Kathīr* (prolonged massage) are recommended with *Rovghan Ḥārr* (hot oils).<sup>19,22,23,41</sup>
- *Dalk* (massage) by *Roghan Shibbat* and *Roghan Qust*<sup>23, 41</sup>
- *Dalk* (massage) by *Roghan* made up of drugs like *Bekh Qisā al-Ḥimār* (root of *Momordica charantia* L.), *Beikh Khatmī* (root of *Althaea officinalis* L.), *Juntyāna* (*Gentiana kurroo* Royle)<sup>19</sup>

### **3. Hammām (Therapeutic Bath)**

- *Hammām Shamsī* (Sun Bath)
- *Hammām Zaytī* (Oil Bath) twice a week
- Steam Bath once a week
- *Hammām* (therapeutic bath), especially *Hammām Ḥārr* (hot bath)
- *Hammām Kibrītī* (sulphur bath)
- *Hammām Būraqī* (borax bath)<sup>22,23,41</sup>

### **4. Other Regimenal Therapy Procedures**

- *Ābzan* (Sitz bath) in *Mā’ al-Ma’dinī* (water containing minerals), such as *Mā’ al-Kibrītī* (sulphur-containing water), *Mā’ al-Shabbī* (alum-containing water)<sup>19</sup>.
- *Ṭilā’* (liniment) by *Shūkrān* (*Conium maculatum* L.), *Banj* (*Hyoscyamus niger* L.), *Adhān* (oils), and *Marūkhāt* (oily drugs)<sup>22</sup>

### **Follow Up**

- Ever Month or as recommended by the Physician

### **Referral Criteria<sup>46</sup>**

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

## REFERENCES

1. Anonymous (2019). ICD-10 Version: 2019. Chapter IV. Endocrine, nutritional and metabolic diseases (E00-E90). E78-Disorders of lipoprotein metabolism and other lipidaemias. WHO. <https://icd.who.int/browse10/2019/en#/E70-E90>
2. Anonymous (2024). ICD-11 for Mortality and Morbidity Statistics (MMS)-WHO. 2024-01. [https://icd.who.int/ct/icd11\\_mms/en/2024-01](https://icd.who.int/ct/icd11_mms/en/2024-01)
3. de Ferranti SD, Newburger JW. Dyslipidemia in children and adolescents: Definition, screening, and diagnosis. UpToDate, Waltham, MA, USA. 2020.
4. 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]
5. Gupta R, Rao RS, Misra A, Sharma SK. Recent trends in epidemiology of dyslipidemias in India. *Indian Heart J.* 2017;69(3):382-392. doi: 10.1016/j.ihj.2017.02.020
6. Mohamed-Yassin MS, Baharudin N, Abdul-Razak S, Ramli AS, Lai NM. Global prevalence of dyslipidaemia in adult populations: a systematic review protocol. *BMJ Open.* 2021;11(12): e049662. Published 2021 Dec 3. doi:10.1136/bmjopen-2021-049662
7. Collins R, Reith C, Emberson J, et al. Interpretation of the evidence for the efficacy and safety of statin therapy. *Lancet* 2016; 388:2532–61.
8. Ference BA, Ginsberg HN, Graham I, et al. Low-Density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement 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.
9. Silverman MG, Ference BA, Im K, et al. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis. *JAMA* 2016; 316:1289–97.
10. Ramjee V, Sperling LS, Jacobson TA. Non-high-density lipoprotein cholesterol versus apolipoprotein B in cardiovascular risk stratification: do the math. *J Am Coll Cardiol* 2011; 58:457–63
11. Sawhney JP, Ramakrishnan S, Madan K, Ray S, Jayagopal PB, Prabhakaran D, et al. CSI clinical practice guidelines for dyslipidemia management: executive summary. *Indian Heart Journal.* 2024 Mar 1;76:S6-19.
12. Jālīnūs (2008). *Kitāb fi'l Mizāj* (Urdu Translation by Syed Zillur Rahman). Ibn Sina Academy, Aligharh, pp. 138-149.
13. Nafīs I (1906). *Mu'ālajāt al-Nafīsī*. Munsī Nawāl Kisorē, Lucknow, pp. 537-539.
14. Hussain MI, Alam S. Relation between dyslipidemia and obesity: A concise review. *Int. J. Appl Res.* 2019; 5(10): 175-179.
15. Nasir A, Siddiqui MY, Mohsin M, Ahmad MA, Iqbal MN. Hyperlipidaemia (*Fart-e-Tadassum fid-Dam*) in the Light of Unani System of Medicine. *Internationale Pharmaceutica Scientia*, 2013; 3(4): 1-8.
16. Buqrat (1832). *Al-Fuṣūl al-Buqrāṭiyya* (Arabic translation by Hunayn ibn Ishgaq). Education press, Calcutta, pp. 2.
17. Rāzī, Abū Bakr Muhammad ibn Zakariyyā (1999). *Kitāb al-Ḥāwī fi'l Ṭibb*, CCRUM, New Delhi, Vol. VI, pp. 133-139, 184-201.
18. Qamarī M (2003). *Ghinā Munā*. Central Council for Research in Unani Medicine, New Delhi, pp. 316-322.
19. Jurjānī, Sayyid Ismā'īl(YNM). *Dhakhīra Khwarizm Shāhī* (Urdu translation) Munshi Nawal Kishore Press, Lucknow. Vol-VIII, pp. 24-28.
20. Sazid Alam, Md. Anzar Alam. et al “Approach of understanding dyslipidaemia in unani medicine”, *The pharma innovation*, 2018; 7(12): 235-237).

21. Tabarī, Abū al-hasan Raban (2010). *Firdaws al-Ḥikma fi'l Ṭibb* (Urdu translation), Diamond Publication, Lahore, Lucknow, pp. 597-599.
22. Ibn Sīnā (YNM). *Al-Qānūn fi'l Ṭibb* (Urdu translation), Vol. 4, Munshi Nawal Kishore, New Delhi, pp. 372-381.
23. Kirmānī, Nafīs ibn 'Iwaz (2007). *Sharḥ al-Asbāb wa'l 'Alāmāt, Tarjama'-i-Kabīr*, Volume III & IV, Aijaz publication House, New Delhi, pp. 369-371.
24. 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.
25. Karantas ID, Okur ME, Okur NÜ, Siafaka PI. Dyslipidemia Management in 2020: An Update on Diagnosis and Therapeutic Perspectives. *Endocr Metab Immune Disord Drug Targets.* 2021;21(5):815-834.
26. 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.
27. Pappan N, Awosika AO, Rehman A. Dyslipidemia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; March 4, 2024.
28. Loscalzo J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Harrison's principles of internal medicine. 2022 May.
29. Berberich AJ, Hegele RA. A modern approach to dyslipidemia. *Endocrine reviews.* 2022 Aug 1;43(4):611-53.
30. 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>
31. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American association of clinical endocrinologists and american college of endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease. *Endocr pract.* 2017;23(suppl 2):1-87. Doi: 10.4158/ep171764.appgl
32. World health organization. Obesity: preventing and managing the global epidemic: report of a WHO consultation.
33. 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.
34. 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>
35. 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.
36. 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.
37. Jurjānī, Sayyid Ismā'īl (2010). *Dhakhīra Khwarizm Shāhī* (Urdu translation by Hadi Hasan), Vol. 1 Part 1, Vo. 2 Part 6. Idāra Kitāb al-Shifā', New Delhi, pp. 113-114, 372.

38. Khān, M A (1290H). *Iksīr-i-A'zam* Vol VI *Matba Nizami*, Kanpur pp. 575-578.
39. Arzānī, Muhammad Akbar (YNM). *Ṭibb-i-Akbar* (Translated by Husain Muhammad). *Idāra Kitāb al-Shifā'*, New Delhi, p. 441.
40. Kabīruddīn, Muhammad (2006). *Al-Qarābādīn*. 2<sup>nd</sup> ed. CCRUM, New Delhi, p. 1010.
41. Khān, Muhammad Sharīf (1939). *'Ilāj al-Amrāḍ* (Urdu translation by Hkm. Muhammad Kabiruddin), Vol. 2, *Daftarul Masih*, Karol Bagh, New Delhi, pp. 438-439, 444.
42. Goldman L, Schafer AI (2020). *Goldman-Cecil Medicine*, 26<sup>th</sup> edition, Elsevier, Inc., U.S., pp. 1355-1364.
43. Bender JR, Russell KS, Rosenfeld LE, Chaudry S (2011). *Oxford American Handbook of Cardiology*, 1<sup>st</sup> edition, Oxford University Press, New York, p. 274
44. Anonymous (2006). *National Formulary of Unani Medicine (NFUM)*, Vol. I, 1<sup>st</sup> edition, Dept. of AYUSH, Ministry of Health & Family Welfare, Govt. of India, New Delhi, p. 239.
45. Zillur Rahman, HS (1991). *Kitāb al-Murakkabāt*, Publication division, AMU Aligarh, pp. 15, 16, 19, 23, 24, 92.
46. Olefsky JM. Obesity. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, editors. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw-Hill Education; 1994. p. 446-452.



CHAPTER

3

**GOUT**





# GOUT

ICD 10 code: M10.9

ICD 11 code: FA25.2

## Niqris (Gout) (NUMC: L-7)

Gout disorder (TM2) **SP14**

### CASE DEFINITION<sup>1,2,3</sup>

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.

### INTRODUCTION (Incidence/ Prevalence, Morbidity/Mortality/Risk Factors)<sup>4,5,6</sup>

- 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.<sup>7,8,9,10</sup>

### Unani medicine's perspective: (Etiology, Pathology, Risk factors, Clinical Presentation and Prognosis)

**Niqris (Gout):** It is a type of joint pain which involves the ankle joint and toes, especially the great toe<sup>11</sup> and causes excruciating pain that eventually renders a person unable to move or walk.<sup>12</sup>

#### Etiology:

- *Sū'-i-Mizāj* (morbid temperament) of joints; especially *Sū'-i-Mizāj Māddī* (morbid temperament associated with substance) involving *Dam* (sanguine) [*Niqris Damawī*], *Ṣafrā'* (yellow bile) [*Niqris Ṣafrāwī*], *Balgham* (phlegm) [*Niqris Balghamī*], *Sawdā'* (black bile)

[*Niqris Sawdāwī*]. It is rarely caused by *Sū'-i-Mizāj Sāda* (simple morbid temperament).<sup>13</sup> Mainly it is caused by predominance of *Ṣafrā'* (yellow bile) [*Niqris Ṣafrāwī*] or *Balgham Ghalīz* (phlegm) [*Niqris Balghamī*] in blood.<sup>12</sup>

- Accumulation of viscous phlegm in the joint.
- Intake of *Aghdhiya Radiyya* (diet providing little nourishment)<sup>13</sup>

### Pathology:

Gout occurs due to presence of plethora (*Imtilā'*) in the body while all the organs of the body remain healthy and have uniformity in strength. In such condition, the waste products gathering within them are expelled by each organ to the nearest organ until it reaches to farthest one, which is the feet. These waste products remain there until removed by the body itself through their maturation by *medicatrix naturae* (*Ṭabī'at*) followed by their resolution and subsequent expulsion or through drugs or regimens.<sup>7</sup> The accumulation of the waste products is further facilitated by the structural weakness of these joints which may be congenital or caused by excessive physical movement, trauma and sexual activity when the stomach is full.<sup>14</sup>

### Risk Factors:<sup>13</sup>

- Weakness of joints which may be congenital or acquired
- Plethora (*Imtilā'*) due to excessive intake of foods and drinks
- Excessive grief and sorrow
- Sedentary lifestyle
- Luxurious life
- Hereditary
- Excessive intake of alcohol
- Menopause
- Season – Spring and autumn

### Clinical Presentation:

**Pain:** The pain usually starts in the big toe, but it may also occur in other joints especially when the disease persists for a long duration.<sup>15</sup> In some patients, the pain starts from the heel and sole, which may radiate to the thigh.<sup>16</sup> Other inflammatory signs, such as heat, redness,<sup>17</sup> heaviness, and the pulsating nature of the pain over the affected joint, occur in cases where the sanguine and choleric humours are involved, whereas constant pain and a less burning sensation occur in cases where phlegmatic humour is involved in the progression of the disease.<sup>16</sup>

**Swelling:** Swelling over the affected joint is also an important sign of gout.<sup>17,18</sup> The joint becomes rigid and may turn into a stony structure when morbid matters are accumulated there for a long duration <sup>15</sup>.

### Prognosis:

- The prognosis of *Niqris* is good when immature humours are present in the body and the consistency of the urine is viscid. In cases where the urine output is very low and the consistency of urine is thin, the patient is about to develop inflammation of joints.<sup>17</sup>
- *Niqris Ṣafrāwī* (gout due to the involvement of morbid humour of bilious origin) may be

dangerous when managed improperly.<sup>19</sup> The disease may subside within 40 days in case *Nigris* is due to the involvement of the hot nature of morbid matters.<sup>15</sup>

### CLINICAL EXAMINATION<sup>5</sup>

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

- **Intense joint pain:** Gout usually affects the large joint of your big toe, but it can occur in any joint. Other commonly affected joints include the ankles, knees, elbows, wrists and fingers. The pain is likely to be most severe within the first four to 12 hours after it begins.
- **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. 1<sup>20</sup>:** (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. 2<sup>21</sup>:** 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 a few joints

## DIFFERENTIAL DIAGNOSIS: 22,23,24,25,26,27,28

The following diseases may be considered in the differential diagnosis of acute gout

**Table 1**

Clinical Conditions	Differential Features
Septic arthritis	<ul style="list-style-type: none"> <li>• Knee is most commonly involved (may be any joint distribution)</li> <li>• Synovial fluid findings: <ul style="list-style-type: none"> <li>◦ WBC Count &gt; 50,000 per mm<sup>3</sup></li> <li>◦ Culture positive</li> <li>◦ Synovial fluid crystals absent</li> <li>◦ Radiography findings- Joint effusion; radiography results otherwise normal early in the disease</li> </ul> </li> </ul>
Trauma	A history of injury may be present.
Pseudogout	<ul style="list-style-type: none"> <li>• Knee, wrist, or first metatarsophalangeal joints are commonly involved.</li> <li>• Synovial fluid findings: <ul style="list-style-type: none"> <li>◦ WBC Count 2,000 to 50,000 per mm<sup>3</sup></li> <li>◦ Culture negative</li> <li>◦ Synovial fluid crystals-Rhomboid shaped, weak positive birefringence</li> <li>◦ Radiography findings-soft tissue swelling, chondrocalcinosis (calcification of cartilage)</li> </ul> </li> </ul>
Rheumatoid arthritis	<ul style="list-style-type: none"> <li>• Arthritis of three or more joint areas</li> <li>• Symmetrical arthritis</li> <li>• Morning stiffness (&gt; 1 hour)</li> <li>• Positive rheumatoid factor</li> <li>• Positive anti-CCP antibody</li> <li>• Elevated ESR and CRP</li> </ul>
Psoriatic arthritis	<ul style="list-style-type: none"> <li>• Onset usually between 25 and 40 years of age</li> <li>• Most commonly in patients with current or previous skin psoriasis (70%)</li> <li>• 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.</li> <li>• HLA-B27 Positive.</li> </ul>
Reactive arthritis	<ul style="list-style-type: none"> <li>• Monoarthritis or oligoarthritis following a recent infection (e.g., urethritis, enteric).</li> <li>• Asymmetric pattern of joint involvement</li> <li>• Symptoms or signs of enthesopathy, Keratoderma blennorrhagica or circinate balanitis</li> <li>• Radiologic evidence of sacroiliitis and/or spondylitis</li> <li>• The presence of human leukocyte antigen (HLA) B27</li> </ul>
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	<ul style="list-style-type: none"> <li>• 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.</li> <li>• 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.</li> </ul>
Tenosynovitis	<ul style="list-style-type: none"> <li>• Centesis of the tenosynovial sheath and microscopic examination should be encouraged in acute tenosynovitis as gout flares may mimic infectious tenosynovitis.</li> </ul>

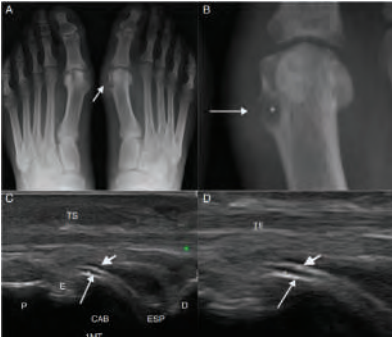
Table 2: Unani Medicine's Perspective:<sup>16</sup>

Characters	Niqris Sawdāwī	Niqris Balghamī	Niqris Şafrāwī	Niqris Damawī
Onset	Gradual	Gradual	Sudden	Abrupt
Nature of pain	Less severe pain	Constant moderate pain	Excruciating	Severe pulsating
swelling	Least marked	Marked	Marked	More marked
Touch	Hard & Cold	Soft & Cold	Hard & warm	Soft & warm
Skin over the joint	Bluish (sometimes)	Whitish	Red tinge to yellowish	Reddish
Aggravating Factors	cold	Cold	Heat	Heat
Relieving Factor	Heat	Heat	Cold	Cold

SUPPORTIVE INVESTIGATIONS<sup>29,30,31</sup>

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

Table 3

Investigations	findings
Essential	
Serum urate concentration	Serum uric acid level may go down in a few cases during an acute attack (serum uric acid levels ≤6 mg/dL)
Advanced	
X ray	<p>X-ray has low sensitivity for the 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.</p>  <p>Fig. 3<sup>26</sup></p>
Ultrasonography (USG)	Characteristic for the diagnosis of gout is the “double contour signal”, which is characterized by an irregular linear hyper echoic layer on the superficial margin of the anechoic hyaline cartilage and parallel to the bone cortex, without a posterior acoustic shade.

Investigations	findings
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<sup>5,32</sup>

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 2015<sup>33</sup>

STEP 1- Entry Criterion: If yes, Classification criteria required for positive diagnosis  
 $\geq 1$  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.

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. **Asymptomatic Hyperuricemia should not be treated** but lifestyle modifications like dietary changes and increased exercise may be advised.

### Unani Medicine's Perspective:

The general line of treatment as mentioned in classics:

- *Taskīn-i-Alam* (analgesia)<sup>13</sup>
- *Tanqiya* (evacuation of causative morbid matters)<sup>13</sup>
- *Ta'dīl-i-Mizāj* (moderation of abnormal temperament)<sup>13</sup>
- *Tahlīl o Talyīn* (to resolve the inflammation and soften the joints)<sup>15,16,13</sup>
- *Taqlīl-i-Ghidhā'* (dietary control)<sup>13</sup>

In Unani medicine, '*Ilāj bi'l Tadbīr* (regimenal therapy) [IUMT-7.2.0] and '*Ilāj bi'l Dawā'* (pharmacotherapy) [IUMT-7.1.10] are considered the core mode of treatment in cases of *Niqris* (gout). '*Ilāj bi'l Tadbīr* (regimenal therapy) [IUMT-7.2.0], includes *Qay'* (emesis)[IUMT-7.2.3]<sup>13</sup>, *Naṭūl* (douche)[IUMT-6.2.95]<sup>13</sup>, *Ḍimād* (poultice)[IUMT-6.2.52]<sup>13</sup>, *Ṭilā'* (liniment) [IUMT-6.2.53]<sup>13</sup>, *Huqna* (enema) [IUMT-6.2.159]<sup>13</sup>, *Dalk* (massage) [IUMT-7.2.92]<sup>13</sup>, *Pāshoya* (footbath) [IUMT-6.2.97]<sup>13</sup>, *Idrār* (diuresis) [IUMT-7.1.169]<sup>13</sup>, *Faṣd* (venesection) [IUMT-7.2.6]<sup>13</sup>, *Ishāl* (purgation) [IUMT-7.2.2]<sup>34</sup>, *Hijāma* (cupping) [IUMT-7.2.30]<sup>35</sup>, *Ḥammām* (therapeutic bath / turkish bath) [IUMT-7.2.70]<sup>12</sup>, *Mundij-o-Mushil* therapy [MM Therapy] (concoctive and purgative therapy) [IUMT-6.1.134] & [IUMT- 6.1.146]<sup>12</sup>, etc.

### (A) Preventive management:

Primary, secondary, and tertiary prevention strategies are necessary to prevent increasing incidence of Gout and Hyperuricemia resulting from increasing incidence of lifestyle disorders.

Primary prevention strategies include maintaining serum uric acid levels within normal limit, achieving and maintaining a normal weight, avoiding alcohol consumption, adherence to Dietary Approaches to Stop Hypertension (DASH)-style diet, and to avoid use of diuretics. Weight loss is required for obesity<sup>36</sup>

- **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.

### Unani Medicine's Perspective:

According to Unani physicians, gout can be prevented through following modifications:

- Change sedentary life style
- Avoid frequent coitus<sup>13,34</sup>
- Avoid overeating<sup>13</sup>
- Avoid to consume meat<sup>5</sup>and alcohol<sup>13</sup>

**Table 5**

Dos <sup>12</sup>	Don'ts (Disease aggravating factors) <sup>12</sup>
<ul style="list-style-type: none"><li>• Intake of decreased quantity of food and drinks;</li><li>• Intake of small amount of dietary substance producing humours of good quality in body;</li><li>• Intake of bird's meat;</li><li>• Intake of small fish;</li><li>• Intake of peeled almond with sugar and Pistachio with deseeded raisins;</li><li>• Intake of grapes, figs, apple, pomegranate, quince, etc.</li></ul>	<ul style="list-style-type: none"><li>• Intake of dietary substance producing bad humour;</li><li>• Intake of salted fish,salted meat and dried meat;</li><li>• Intake of milk;</li><li>• Excessive intake of walnut, dates, etc</li><li>• Intake of apricot, peach, mulberry, unripe sour apple, all unripe fruits;</li><li>• Intake of <i>Jirjīr</i> (<i>Eruca sativa</i> Mill.), <i>Jangalī Tulsī</i> (<i>Ocimum basillicum</i> L.), <i>Kurrāth</i> (<i>Allium ampeloprasum</i> L.), <i>Karafs</i> (<i>Apium graveolens</i> L.), mint, etc. and excessive intake of chicory.</li><li>• Sedentary lifestyle</li></ul>

**(B) Interventions:**

**At Level 1-** Solo Physician Clinic/ Health Clinic/ PHC (Optimal standard of treatment where technology and resources are limited)

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

**Recommended diet and life style:**

- Skimmed milk, vegetables, soybeans, vegetable sources of proteins, cherries<sup>37</sup>, apple, pear, fig, etc.<sup>38</sup>
- Suitable physical exercise
- Weight loss in case of excess body weight or obesity<sup>37</sup>

**Restricted diet and life style:**

- Fat and purine containing diets
- Alcohol
- Soft drinks<sup>39</sup>
- Excessive coitus<sup>13,34</sup>
- Overeating<sup>13</sup>

**Unani Medicine's Perspective:**

**OPD level management** – If the patient shows mild features of *Niqris* (Gout), two or more of following forms of medications may be given along with diet restrictions:

**Single drugs and Compound formulations for internal/ external use**

**Table 6**

S. No.	Drugs	Dosage form	Dose per day	Time	Duration	Badraqa (Vehicle)	Precaution/ Contraindication
1.	<i>Sūranjān</i> ( <i>Colchicum autumnale</i> L.) <sup>40</sup>	Powder	3 g in two divided doses	After meal	10-15 days	Water	Nothing specific (NS)
2.	<i>Sanā</i> ( <i>Cassia angustifolia</i> Vahl.) <sup>41</sup>	Powder	5-10 g in two divided doses	After meal	10-15 days	Water	Pregnancy
3.	<i>Tukhm-i-Karafs</i> ( <i>Apium graveolens</i> L.) <sup>41</sup>	Powder	3-5 g once in a day	After meal	10-15 days	Water	NS
4.	<i>Ḥabb-i-Sūranjān</i> <sup>41</sup>	Pills	250-500 mg	After meal	10-15 days	Water	NS
5.	<i>Ma'jūn-i-Sūranjān</i> <sup>41</sup>	Semi-solid preparation	5-10 g. in two divided doses	After meal	10-15 days	Water	Diabetes Mellitus Type I&II

**Formulations for local application:**

**Roghan (oil)**

- *Roghan-i-Mālkanganī*<sup>41</sup> for external use for 10-15 days or may be use as directed by the physician.
- *Roghan-i-Sūranjān*<sup>41</sup> for external use for 10-15 days or may be use as directed by the physician

**Ḍimād (Poultice) for Niqris caused by hot morbid humours:**

- *Ḍimād* prepared with extracted water of *Ḥayy al-Ālam* (*Sempervivum arboretum* L.) and *Ṣandal Safayd* (*Santalum album* L.) is applied over the affected joints.<sup>13</sup>
- *Ḍimād* prepared with extracted water of *Mako* (*Solanum nigrum* L.) and *Ṣandal Safayd* (*Santalum album* L.) is applied over the affected joints.<sup>13</sup>

**Naṭūl (Douche) for Niqris caused by hot morbid humours:**

- *Naṭūl* with decoction or extracted water of *Karnab* (cabbage).<sup>13</sup>
- *Naṭūl* with cold water on affected joint.<sup>13</sup>

**Naṭūl (Douche) for Niqris caused by cold morbid humours:**

- *Naṭūl* with decoction of turnip.<sup>13</sup>
- *Naṭūl* with decoction of *Kurrāth* (*Allium ampeloprasum* L.).<sup>13</sup>

**Pāshoya (Footbath) for Niqris caused by cold morbid humours:**

- *Pāshoya* with decoction of *Kathūth* (seed of *Cuscuta reflexa* Roxb.).<sup>13</sup>

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. *‘Ilāj bi’l Tadbīr* (Regimenal therapy) described under principles

of management may be recommended as per assessment of physician about the condition of the patient and stage of disease. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

**Follow Up** (7 days or as recommended by the Physician)

**Reviews<sup>42</sup> should include:**

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

**Referral Criteria:**

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

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

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

**Investigations:** The diagnosis would be primarily clinical along with some investigations which will be necessary to investigate complications or exclude other differential diagnoses as follows:

- Serum urate concentration
- Complete blood count/ESR
- Renal function Test
- Fasting lipids, glucose, and thyroid functions
- Urinary urate excretion

**Management:** Same as Level 1 and/ or treatment mentioned at this level.

**Table 7: Single and compound drugs for internal/ external use**

S. No.	Drugs	Dosage form	Dose (per day)	Time of administration	Duration	Badarqa (Vehicle)	Precaution/ Contraindication
1.	<i>Qusṭ</i> ( <i>Saussurea lappa</i> C.B. Clarke) <sup>43</sup>	Powder	2-3 g once a day	After meal	15-30 days	Water	NS
2.	<i>Tagar</i> ( <i>Valerianawallichii</i> DC.) <sup>43</sup>	Powder	2-5 g once a day	After meal	15-30 days	Water	NS
3.	<i>Safūf-i-Chob Chīnī</i> <sup>44</sup>	Powder	2-6 g in two divided doses	After meal	15-30 days	Water	NS
4.	<i>Safūf-i- Sūranjā</i> <sup>44</sup>	Powder	5-10 g in two divided doses	After meal	15-30 days	Water	NS
5.	<i>Ḥabb-i-Shifā</i> <sup>44</sup>	Pills	250-500 mg in two divided doses	After meal	15-30 days	Water	NS
6.	<i>Ḥabb-i-Muntin Akbar</i> <sup>45</sup>	Pills	5-10 g in two to three divided doses	After meal	15-30 days	Water	NS
7.	<i>Majūn-i- Niqris</i> <sup>46</sup>	Semisolid	5 g once a day	After meal	15-30 days	Water	Diabetes Mellitus Type I&II

#### Formulations for local application:

##### Roghan (oil)

- *Roghan Surkh*<sup>44f</sup> for external use for 15-30 days or may be use as directed by the physician

##### Ḍimād (Poultice) for Niqris caused by hot morbid humours:

- Application of poultice of *Khurfa* (*Portulaca oleracea* L.)/*Tuḥlub* (Algae)/*Kāhū* (*Lactuca sativa* L.)/*Aspghol* (*Plantago ovata* Forssk.)/peel of *Kadū* (*Cucurbita maxima* L.)/*Barg-i-Bed* (leaf of *salix tetrasperma* Roxb.)/*Nīlofar* (*Nymphaea alba* L.) over the affected joints.<sup>13</sup>
- Application of *Ḍimād* prepared in the following manner:

Dissolve the *Mom* (wax) in *Roghan-i-Sosan* thereafter add *Lu'āb-i-Ḥulba* (mucilage of seed of *Trigonella foenum-graecum* L.), *Lu'āb-i-Aspghol* (mucilage of seed of *Plantago ovata* Forssk.), *Lu'āb-i-Bazr-i-Katān* (mucilage of seed of *Linum usitatissimum* L.) and *Lu'āb-i-Khaṭmī* (mucilage of *Althaea officinalis* L.) and apply the paste over the affected joint.<sup>13</sup>

**Ḍimād (Poultice) for Niqris caused by cold morbid humours:**

- Application of *Ḍimād* prepared with *Karnab* (cabbage) and saffron over the affected joints.<sup>13</sup>

**Naṭūl (Douche) for Niqris caused by cold morbid humours:**

- *Naṭūl* with decoction of *Bābūna* (*Matricaria chamomilla* L.).<sup>13</sup>

**Dalk (Massage) for Niqris caused by cold morbid humours:**

- *Dalk* with *Roghan-i-Sosan*.<sup>13</sup>

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. '*Ilāj bi'l Tadbīr* (Regimenal therapy) described under principles of management may be recommended as per assessment of physician about the condition of the patient and stage of disease. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

**Management with Mundij-o-Mushil therapy (Concoctive and Purgative therapy):**

In case of *Niqris Ṣafrāwī*, following *Mushil* formulation may be given:

**Table 8**

S. No.	Formulation	Dosage form	Dose	Time	Duration	Badarqa (vehicle)	Precaution/Contraindication
1.	Ālū Bukhāra ( <i>Prunus domestica</i> L.) 10 pieces, <i>Tamar Hindī</i> ( <i>Tamarindus indica</i> L.) 10 g, <i>Shāhtra</i> ( <i>Fumaria officinalis</i> L.) 5 g, <i>Afsantīn</i> ( <i>Artemisia absinthium</i> L.) 5 g, <i>Ṣibr</i> ( <i>Aloe barbadensis</i> Mill.) 5 g, <i>Saqmūniyā</i> ( <i>Convolvulus scammonia</i> L.) 3 g <sup>13</sup>	Decoction	100 ml	Morning before meal	3-5 days	Water	Pregnancy

In case of *Niqris Balghamī*, following formulation may be given:

**Table 9**

S. No.	Formulation	Dosage form	Dose	Time	Duration	Badarqa (Vehicle)	Precaution/Contraindication
1.	<i>Sūranjān</i> ( <i>Colchicum autumnale</i> L.) 5 g, <i>Chirā'ita</i> ( <i>Swertia chirayita</i> (Roxb. ex Flem.) Karst.) 7 g, <i>Shāhatra</i> ( <i>Fumaria officinalis</i> L.) 7 g, <i>Aftīmūn</i> ( <i>Cuscuta reflexa</i> Roxb.) 5 g, <i>Bisfā'ij</i> <i>Fustaqī</i>	Decoction (MM therapy)	100 ml	Morning before meal	7-14 days	Water	Pregnancy

S. No.	Formulation	Dosage form	Dose	Time	Duration	Badarqa (Vehicle)	Precaution/ Contraindication
	( <i>Polypodium vulgare</i> L.) 5g, 'Unnāb ( <i>Zizyphus jujuba</i> Mill.) 5 No., Bādiyān ( <i>Foeniculum vulgare</i> Mill.) 7 g, Bekh-i-Bādiyān ( <i>Foeniculum vulgare</i> Mill. root) 7 g <sup>47</sup>						
2.	Ayārij-i-Fayqrā <sup>47</sup>	Powder	3-5 g	Early morning before food	2-3 days (after MM therapy)	Water	Pregnancy

#### 'Ilāj bi'l Tadbīr (Regimenal therapy):

'Ilāj bi'l Tadbīr (Regimenal therapy) described under principles of management may be recommended as per assessment of physician about the condition of the patient and stage of disease.

**Recommended Diet and Lifestyle:** Same as described under preventive management and level-1

**Restricted Diet and Lifestyle:** Same as described under preventive management and level-1

**Follow Up** (7 days or as recommended by the physician).

#### Referral Criteria

- Same as mentioned earlier at level 1, with
- 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:** (Unani hospitals attached with teaching institution, District Level/Integrated/ State Unani Hospitals, Tertiary care allopathic hospitals having Unani facilities), multiple departments/facilities for diagnosis and interventions. Must provide additional facilities like dieticians, counselling, and physiotherapy unit.

**Clinical Diagnosis:** Same as Level 1 & 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:** Same as level 1 & 2. For the patients referred from Level-1 or 2, treatment given in Level-1 &/or 2 may be continued if appropriate for the presenting condition or the case may

be reassessed for the totality of symptoms and treatment may be given accordingly. For new cases at this level, the totality of symptoms presented by the patient is the sole indicative and guide for treating each patient.

In addition to the level 1 and level 2 management strategies, Unani medicine has a number of specific remedies that can ease pain and other symptoms in patients with end-stage of gout or in those who have not responded to treatment due to a lack of symptoms, co-morbid conditions, or the use of other immune-suppressives, oral hypoglycaemic agents, or anti-hypertensives. Palliative care medications can therefore be provided based on the sphere of action or keynote prescription in these disorders as well as other advanced pathological states.

**Table 10: Single and compound drugs for internal/ external use**

S. No.	Drugs	Dosage form	Dose (per day)	Time of administration	Duration	Badarqa (Vehicle)
1.	<i>Būzīdān (Tanacetum umbelliferum Boiss.)</i> <sup>48</sup>	Powder	3-5 g once a day	After meal	1-3 months	Water
2.	<i>Bābūna (Matricaria chamomilla L.)</i> <sup>48</sup>	Powder	3-5 g once a day	After meal	1-3 months	Water
3.	<i>Nākhūna (pods of Astragalus homosus L.)</i> <sup>48</sup>	Powder	3-5 g once a day	After meal	1-3 months	Water
4.	<i>Asgand (Withania somnifera(L.) Dun.)</i> <sup>48</sup>	Powder	3-5 g once a day	After meal	1-3 months	Water
5.	<i>Zanjībīl (Zingiber officinale Rosc.)</i> <sup>48</sup>	Powder/ Decoction	3-5 g once a day	After meal	1-3 months	Water
6.	<i>Ḥabb-i-Muqīl</i> <sup>48</sup>	Pills	0.5-1 g once a day	After meal	1-3 months	Water
7.	<i>Ḥabb-i-Azārāqī</i> <sup>48</sup>	Pills	125-250 mg in two divided doses	After meal	1-3 months	Water
8.	<i>Ḥabb-i-Asgand</i> <sup>48</sup>	Pills	1-2 g in two divided doses	After meal	1-3 months	Water
9.	<i>Ma'jūn-i-Azārāqī</i> <sup>48</sup>	Semisolid	3-5 g in two divided doses	After meal	1-3 months	Water
10.	<i>Ma'jūn-i-Chobchīnī</i> <sup>48</sup>	Semisolid	5-10 g. in two divided doses	After meal	1-3 months	Water
11.	<i>Ma'jūn-i-Jogrāj Gogul</i> <sup>48</sup>	Semisolid	5-10 g in two divided doses	After meal	1-3 months	Water
12.	<i>Kushta'-i-Ga'odanti</i> <sup>44</sup>	Powder	60-120 mg in two or three divided doses	After meal	1-3 months	Water

**‘Ilāj bi’l Tadbīr (Regimenal therapy)** described under principles of management may be recommended as per assessment of physician about the condition of the patient and stage of disease.

#### **Formulations for local application:**

##### **Roghan (oil)**

- *Roghan-i-Haft Barg*<sup>48</sup> for External use for a period of 1-3 Month or may be used as directed by the physician
- *Roghan-i-Bābūna Sāda*<sup>48</sup> for External use for a period of 1-3 Month or may be used as directed by the physician

##### **Ḍimād (Poultice) for Niqris caused by hot morbid humours:**

- *Ḍimād prepared with fresh juice of Ḥayy al-‘Ālam (Sempervivum arboretum L.) and flour of seed of Hordeum vulgare L. is applied over the affected joints.*<sup>13</sup>

##### **Ḍimād Muḥallil:**

- The paste prepared with the powder of *Iklīl al-Malik* (pods of *Astragalus homosus* L.) 1 part, *Bābūna* (*Matricaria chamomilla* L.) 1 part, *Asgand Nāgorī* (*Withania somnifera* (L.) Dunal) 1 part, *Mako* (*Solanum nigrum* L.) 1 part, *Tukhm-i-Khaṭmī* (*Althaea officinalis* L.) 1 part, *Rewand Chīnī* (*Rheum emodi* Wall. ex Meissn.) 1 part, *Muqil* (*Commiphora mukul* (Hook. ex Stocks) Engl.) ¼ part, and *Āb-i-Mako Sabz* (fresh juice of *Solanum nigrum* L.) or *Āb-i-Barg-i-Sambhālū* (fresh juice of *Vitex negundo* L.), is applied over the affected joint.<sup>45</sup>

##### **Ḍimād:**

- *Ḍimād prepared with the powder of Ushaq (Dorema ammoniacum D. Don.) and Rasavt (Berberis aristata DC) dissolved in alcohol in the equal quantity and mixed with Roghan-i-Zaytūn (olive oil), is applied over the affected joint.*<sup>49</sup>

**Munḍij-o-Mushil therapy (Concoctive and Purgative therapy):** In case of *Niqris Balghamī*, the formulation mentioned at Level 2 may be given. The *Munḍij* therapy may be given for 14-21 days.

**Recommended Diet and Lifestyle:** Same as described under preventive management and level-1

**Restricted Diet and Lifestyle:** Same as described under preventive management and level-1

**Follow Up** (7 days or as recommended by the physician)

##### **Referral Criteria**

- Same as mentioned earlier at level 1, with
- Failure of acute exacerbation to respond to initial medical management.
- Advanced stages of disease like tophus formation, recurrent gout, deformities, and complications.
- Patients need surgical intervention.
- 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 Opin Rheumatol*. 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. Arzani M A.Meezan al-Tibb. Deoband: Matba Qasmi;YNM: 154-156.
12. Razi M. Maqalafi'lNiqris. Al-Iskandariya:Maktaba Iskandariya; 2003: 16, 17, 19, 28-35.
13. Khan MA. Ikseer-i-Azam, Vol. IV. Lucknow: Matba Nami Munshi NawalKishor; 1906: 16-18, 48-52.
14. Majusi Ali b. Abbas.Kamil al-Sana'a al-Tibbiyya (Arabic), Part 1. New Delhi: Central Council for Research in Unani Medicine; 2005: 391-394.
15. Razi Z. Kitab al-Hawi, Vol 11 (Urdu translation by CCRUM). New Delhi: Central Council for Research in Unani Medicine, Dept. of AYUSH, Ministry of H & F. W. Govt. of India; 2004: 75, 83, 86.
16. Sina I. Al-Qanunfi'l-Tibb, Vol. 3 (Arabic). New Delhi: Jamia Hamdard; 1411 H.: 910-924.
17. Qamri Abul Mansoor. Ghina Muna (Arabic). New Delhi: Central Council for Research in Unani Medicine; 2008: 272-284.
18. Baghdadi Abul Hasan. Kitab al-Mukhtaratfi'lTibb, Vol. 4 (Arabic). Hyderabad: Daira al-Maa'arif al-Usmaniyya. 1364 H.: 84-103
19. Weaver JS, Vina ER, Munk PL, Klauser AS, Elifritz JM, Taljanovic MS. Gouty arthropathy: review of clinical manifestations and treatment, with emphasis on imaging. *Journal of Clinical Medicine*. 2021; 11 (1): 166. <https://doi.org/10.3390/jcm11010166>
20. Jelley MJ, Wortmann R. Practical Steps in the Diagnosis and Management of Gout. *BioDrugs*. 2000; 14 (2): 99-107.
21. 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.
22. Eggebeen AT. Gout: An Update. *American Family Physician*.2007;76(6):801-808.
23. Doherty M, Abhishek A. Clinical manifestations and diagnosis of osteoarthritis. Characteristics of specific joint involvement. In up to date. Post TW (Ed), UpToDate, Waltham, MA. (Accessed on August 11, 2022.) Available from: [https://wolterkluwer.ccrhlibrary.in/contents/clinical-manifestations-and-diagnosis-ofosteoarthritis?Search=osteoarthritis&source=search\\_result&selectedtitle=2~150&usage\\_type=default&display\\_ran k=2](https://wolterkluwer.ccrhlibrary.in/contents/clinical-manifestations-and-diagnosis-ofosteoarthritis?Search=osteoarthritis&source=search_result&selectedtitle=2~150&usage_type=default&display_ran k=2)
24. 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
25. 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/>

26. 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/>
27. 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/>
28. 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.
29. Underwood M. Diagnosis and management of gout. *BMJ.* 2006;332(7553):1315–1319.
30. Schlesinger N, Norquist JM, Watson DJ. Serum urate during acute gout. *J Rheumatol.* 2009; 36(6):1287-89
31. 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.
32. Grassi W, Angelis RD. Clinical features of gout. *Reumatismo.* 2011; 63(4):238-245.
33. 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.
34. Tabari A. Firdaus al-Hikmat (Urdu Translation). New Delhi: Idarahkitab al-Shifa; 2010: 293-294.
35. Akhtar H, Rashid M, Siddiqi M, Ahmad QZ. Role of Hijama (Cupping Therapy) in the Management of Nigras (Gouty Arthritis). *Journal of Arthritis.* 2017; 6 (6): 1-8.
36. McCormick N, Rai SK, Lu N, Yokose C, Curhan GC, Choi HK. Estimation of Primary Prevention of Gout in Men Through Modification of Obesity and Other Key Lifestyle Factors. *JAMA Netw Open.* 2020 Nov 2;3(11): e2027421. doi: 10.1001/jamanetworkopen.2020.27421. PMID: 33231639; PMCID: PMC7686865.
37. Jurjani I. Zakhira Khwarizm Shahi, Vol. 6 (Urdu translation by Khan HH). New Delhi: Idarah Kitab al-Shifa; 2010: 647.
38. Wang Y, Li W, Wu H, Han Y, Wu H, Lin Z, Zhang B. Global status and trends in gout research from 2012 to 2021: a bibliometric and visual analysis. *Clinical Rheumatology.* 2023; 42 (5): 1371-1388. DOI: 10.1007/s10067-023-06508-9.
39. Clebak KT, Morrison A, Croad JR. Gout: Rapid Evidence Review. *American Family Physician.* 2020; 102 (9): 533-538.
40. Nabi G. Makhzan-i-Mufradat-o-Murakkabat. New Delhi: Central Council for Research in Unani Medicine; 2007: 151.
41. Anonymous. Essential Drug List (EDL) Unani Medicine. New Delhi: Dept. of AYUSH, Ministry of Health & Family Welfare, Govt. of India; 2013: 37-38, 43-45, 50-51.
42. Ministry of Health & Family Welfare, Government of India. Standard Treatment Guidelines. Management of Osteoarthritis Knee. Macro Graphics Pvt. Ltd. August 2017.
43. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. I. New Delhi: Dept. of AYUSH, Ministry of H & FW, Govt. of India; 2007: 74-75, 86-87
44. Anonymous. Essential Drug List (EDL) Unani Medicine. New Delhi: Dept. of AYUSH, Ministry of Health & Family Welfare, Govt. of India; 2013: 37-38, 43-45, 50-51.
45. Anonymous. National Formulary of Unani Medicine, Part I. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2006: 26, 170-171.
46. Anonymous. National Formulary of Unani Medicine, Part II, Vol. I. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2007: 76.
47. Kabeeruddin M. Bayaz-i-Kabeer, Vol. 1. New Delhi: Idarah Kitab al-Shifa; 2010: 203-204.
48. Anonymous. AYUSHMAN BHARAT, AYUSH Health and Wellness Centres Orientation Guidelines for Community Health Officers under Unani stream. New Delhi: Ministry of Ayush, Govt. of India; 2021: 204, 235
49. Jurjani I. Zakhira Khwarizm Shahi, Vol. 6 (Urdu translation by Khan HH). New Delhi: Idarah Kitab al-Shifa; 2010: 647.

CHAPTER

# 4

## **NON ALCOHOLIC FATTY LIVER DISEASE**





# NON-ALCOHOLIC FATTY LIVER DISEASE

(ICD 10 code: K75.8)

(ICD 11 code: DB92)

*Tashaḥḥum-i-Kabid Ghayr Khamrī* (Non-alcoholic Fatty Liver Disease) (NUMC: F-112) <https://namstp.ayush.gov.in/#/Unani>

## 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<sup>[1]</sup>. An international group of expert consensus statement suggested to change the name to Metabolic Associated Fatty Liver Disease (MAFLD)<sup>[2]</sup>. 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<sup>[3]</sup>.

## INTRODUCTION (incidence/prevalence, mortality/morbidity)

- NAFLD is a spectrum of disorder ranging from Non-Alcoholic Fatty liver to Non-Alcoholic Steatohepatitis (NASH), NASH with fibrosis, NASH- cirrhosis and NASH associated with hepatocellular carcinoma (HCC)<sup>[4,5]</sup>.
- The prevalence of NAFLD in India varies from 9-35% as per the accordance to ultrasonography data<sup>[6,7]</sup>. Studies demonstrated area-wise prevalence data of NAFLD with 16.6 % in Western India, 24.5 % in Eastern India, and 32 % in South India<sup>[6]</sup>.
- 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<sup>[3]</sup>.
- 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<sup>[7]</sup>.
- 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<sup>[7]</sup>.
- NAFLD is known to be associated with several extrahepatic conditions like chronic kidney disease (CKD)<sup>[8]</sup>, cardiovascular diseases<sup>[9-11]</sup>, osteopenia, osteoarthritis<sup>[12]</sup>, obstructive sleep apnoea<sup>[13]</sup>, hypothyroidism<sup>[14]</sup>, and polycystic ovarian syndrome<sup>[15,16]</sup>. NAFLD has also been shown to increase the risk of extrahepatic malignancies like carcinoma colon, gastric cancer, carcinoma pancreas, uterine, and breast conditions<sup>[17]</sup>.
- 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<sup>[18]</sup>.

## Unani Medicine's Perspective: (Etiology, Pathology and Risk Factors):-

The normal *Mizāj* of liver is *Ḥārr Raṭb* (hot and moist temperament).<sup>19,20,21,22</sup> The disturbance in the normal temperament of liver is caused by erratic dietary habits, excessive consumption of fatty and cold food etc., which enables excessive accumulation of fat in liver parenchyma (*Tashaḥḥum-i-Kabid*), which ultimately alters the *Mizāj* of liver. This alteration leads to *Sū' Mizāj al-Kabid al-Bārid / Sū'-i-Mizāj-i-JigarBārid* (cold morbid temperament of liver) resulting in alteration in liver function which ultimately leads to other structural and functional abnormalities.<sup>20,22,23</sup>

### Etiology

- Excessive use of diets having cold temperament
- Staying in excessively cold surroundings
- Drinking cold water on empty stomach in morning
- Use of cold water after *Ḥammām* (therapeutic bath /Turkish bath) and intercourse
- When spleen is not able to absorb *Sawdā'* (black bile), it may also lead to *Sū' Mizāj al-Kabid al-Bārid / Sū'-i-Mizāj-i-JigarBārid* (cold morbid temperament of liver)<sup>22</sup>.

The causes of *Sū' Mizāj al-Kabid al-Bārid* are excessive activity leading to dispersion of innate heat, excessive repose leading to suppression of innate heat, food and drinks in excess (overeating), marked reduction in food (undernutrition), cold foods, drinks, medications, and regimens, undue retention of *fuzlat* (morbid material), obstruction due to accumulation of *fuzlat*, excessive emotions like worry, joy, pleasure, fear and anxiety<sup>21,23, 24</sup>.

### Pathology

*Sū' Mizāj al-Kabid al-Bārid / Sū'-i-Mizāj-i-JigarBārid* (cold morbid temperament of liver) leads to the formation of *sudad* (obstruction) in liver. These obstructions cause *Ḍu'f al-Kabid* (hepatic insufficiency) and disturb the metabolism of food and formation of normal humors. Cold morbid temperament of liver may be simple or with the involvement of matter. In this case cold morbid temperament is accompanied with passive quality of moistness. This favors the predominance of *Balgham* (phlegm) in the form of fat<sup>19</sup>.

## CLINICAL PRESENTATION AND EXAMINATION

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, pruritus, ascites, memory impairment, right upper quadrant discomfort, hepatomegaly, acanthosis nigricans and lipomatosis<sup>25</sup>. 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<sup>26</sup>. 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<sup>27</sup>. Patients with such risk factors must be screened as it has been observed that metabolic syndrome is closely associated with NAFLD<sup>28</sup>

Table 1

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

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

SUPPORTIVE 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<sup>[3]</sup>. Non-invasive tests remain the investigation of choice among the physicians and patients alike.

Table 3

<b>Investigations</b>	<b>Findings</b>
<b>Essential</b>	
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.

Investigations	Findings	
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 liver in addition to Grade1/2 findings
<b>Advanced</b>		
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 <sup>31</sup>	
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 <sup>32</sup>	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 <sup>33</sup>	AST and platelet levels	Hepatic fibrosis.
Fibrosis-4 score (Fib-4) <sup>34</sup>	AST, ALT, age, and platelets	Hepatic fibrosis

Name of score	Measuring components	Utility
NAFLD fibrosis scores (NFS) <sup>35,36</sup>	BMI, Age, AST/ALT ratio, Albumin, and presence of insulin resistance and diabetes	Hepatic fibrosis
BARD score <sup>36</sup>	BMI, Age, AST/ALT ratio, and presence of diabetes	Hepatic fibrosis
Magnetic resonance elastography (MRE) and Fibrosis-4 score (MEFIB) <sup>37</sup>	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.<sup>29</sup>

## 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<sup>3,18,30</sup> 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 a history of significant alcohol intake, hepatitis B and C, and autoimmune hepatitis must be conducted to rule out alternate causes of hepatic steatosis.

### Diagnostic tools in Unani medicine:

- *Nabḍ Baṭī'* (slow pulse), *Nabḍ Layyin* (pulsus mollis / soft pulse), *Nabḍ Mutaḥawit* (pulsus rarus)<sup>23,38,39</sup>
- *Bawl Abyaḍ* (white urine), *Bawl Raḳīq* (urine of thin consistency)<sup>23</sup>
- *Al-Barāz al-Yābis* (dry stool), *Qilla al-Barāz* (lesser quantity of stool), *Barāz Nārī* (markedly yellow stool).<sup>20, 23,39,40</sup>

## PRINCIPLES OF MANAGEMENT

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

### Red Flags

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

- Portal hypertension
- Hepatic encephalopathy
- Weight loss or anorexia

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

### A) Prevention management

Lifestyle interventions including dietary calorie management and exercise constitute the main pillars of NAFLD management. Studies have demonstrated that there is a dose-response relationship between the magnitude of weight loss and the degree of histological improvement of NAFLD. 3-5%,  $\geq 7\%$ , and  $\geq 10\%$  of weight loss has been associated with regression in steatosis, steatohepatitis, and fibrosis respectively<sup>41</sup>. Daily caloric restriction by 30% with cutting down of both carbohydrates and fat in the staple diet. Intermittent fasting (e.g. alternate day fasting, 5:2 fasting with 2 days of severely reduced caloric intake and 5 days of normal consumption) may be a promising approach but sufficient evidence is still not available to routinely recommend such practice<sup>42</sup>. Exercise shall consist of moderate-intensity aerobic exercises such as brisk walking, jogging, running, swimming, etc. supplemented by resistance exercises<sup>43,44</sup>

**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.

### Unani Medicine's Perspective

The general line of treatment according to classical literature: 19, 22, 39,45,46

- *Iṣlāḥ-i-Sū'-i-Mizāj* (correction of morbid temperament)
- *Taskhīn* (calefaction)
- *Taftīḥ-i-Sudad* (inducing deobstruction)
- *Taltīf* (process of refining / attenuation)
- *Tahlīl* (dissolution)
- *Tanqiya* (cleansing of morbid matter)
- *Taqwiyat-i-Kabid* (Liver tonics)

### Interventions

**At level 1-** Solo physician Clinic/ Health clinic/ PHC (Optimal standard of treatment where technology and resources are limited)

### Clinical diagnosis

The diagnosis of NAFLD shall be done at 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)

Recommended diet and lifestyle

Light and easily digestible diet may be prescribed for liver patients such as small bird’s soup, chicken soup, pulses, *Sāgū Dāna / Sābū Dāna* (preparation of *Sābū Dāna*), *Aab nakhud* (decanted water of *Cicer arietinum*), *Dalyā* (frumenty/porridge), *Kishneez* (*Coriandrum sativum*), *Pudina* (*Mentha piperita*)<sup>47</sup>

Riyāḍat Mu’tadila (Moderate exercise)

It has been evidenced that *Riyāḍat Mu’tadila* (moderate exercise), where force as well as movement is moderate, can be beneficial for NAFLD patients.

Weight loss

Weight loss is the mainstay of treatment for NAFLD. NAFLD patients, whether obese or not, should be encouraged and educated to partake in a healthy lifestyle approach, which exists irrespective of weight-loss<sup>48</sup>

Nutrition

A healthy diet i.e. reduction of caloric intake and high-glycaemic index (GI) foods, increased consumption of monounsaturated fatty acids, omega-3 fatty acids, fibres, and specific protein sources such as fish and poultry are suggested to have beneficial effects. Studies suggest that a Mediterranean diet, defined as reduced carbohydrate intake (especially sugars and refined carbohydrates) and increased mono-saturated and omega-3 fatty acid intake, can reduce liver fat and thus positively contribute to the management of NAFLD<sup>49,50</sup>

Restricted diet and lifestyle

Patients should avoid oily, fatty, spicy, fried and foods that take a long time to digest

Unani medicine’s perspective<sup>47</sup>

Table 5

Dos	Don'ts (Disease aggravating factors)
<ul style="list-style-type: none"><li>• Intake of <i>Aghdhiya Laṭīfa</i> (Food items which are easy to digest but have little nutritional value, and produce such a sanguine which is normal in viscosity. e.g. meat of small birds, small fishes, etc)</li><li>• Intake of <i>Aghdhiya Musakhkhina</i> (Food items which increase the metabolism of the body due to their hot temperament or heat producing properties e.g. spices).</li><li>• Liver strengthening food and drinks viz; Amla, Pomegranate, Lemon, Butter milk, plenty of green vegetables etc. are advised.</li></ul>	<ul style="list-style-type: none"><li>• Non-vegetarian food containing high fat content</li><li>• Sedentary lifestyle</li><li>• Hepatotoxic drugs</li><li>• Substance abuse, alcohol abuse and tobacco products</li></ul>

## OPD level management

The symptoms associated with NAFLD may be managed at OPD level using medications along with dietary restrictions and increased physical activity

**Table 6: Single drugs and compound formulations for internal/ external use**

S. No	Drug	Dosage form	Dose (per day)	Time of administration	Duration & Frequency	Badraqa (vehicle)
1	<i>Afsanteen (Artemisia absinthium L.)</i> <sup>51</sup>	Powder Decoction Distillate	2-5 g 7-9 g 70 ml	After meal	Upto 3 Months	Water <i>Araq-i Badiyan</i> <i>Sharbat-i Kasoos</i>
2	<i>Dar Chini (Cinnamomum verum. J. Presl)</i> <sup>51</sup>	Powder	1-2 g	After meal	Upto 3 Months	Water
3	<i>Mako (Solanum americanum Mill.)</i> <sup>52</sup>	Distillate	60-100 ml in 2 divided doses	After meal	Upto 3 Months	Water
4	<i>Rewand chini (Rheum australe D. Don)</i> <sup>53</sup>	Powder	2-7 g	After meal	Upto 3 Months	Water
5	<i>Sumbul-ut-Teeb (Nardostachys jatamansi (D. Don) DC.)</i> <sup>51</sup>	Powder	3-5 g	After meal	Upto 3 Months	Water
6.	<i>Qurs-i Kabidi</i> <sup>54</sup>	Pills	1g twice a day	After meal	Up to 3 Months	Water
7	<i>Jawarish Tamarhindi</i> <sup>55</sup>	Semi-solid preparation	5-10 g	After meal	Up to 3 Months	Water
8	<i>Qurs-i Hummaz</i> <sup>56</sup>	Pills	1g twice a day	After meal	Up to 3 Months	Water
9	<i>Araq-i Kasni</i> <sup>57</sup>	Distillate	60-100 ml in 2 divided doses	After meal	Up to 3 Months	Water
10	<i>Zimad-i Sibr</i> <sup>51</sup>	Paste for local application	Q.S for external use	As directed by the physician	Up to 3 months	--

Note: out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. '*Ilaj bi'l Tadbir* (Regimenal Therapy) described under principles of management may be recommended as per the assessment of the physician about the condition of the patient and stage of disease. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

**Follow up:** 15 days or as recommended by the physician

### Reviews should include

- Monitoring the person's symptoms and the ongoing impact of the condition on their everyday activities and quality of life.

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

### Referral criteria

- Non-response to treatment
- Progression of the disease to NASH, NASH-associated Cirrhosis, or NASH-associated, and Hepatocellular Carcinoma
- Any other hepatic or extra-hepatic complications, such as Gallstone disease commonly seen in older age and higher grade of NAFLD.
- Evidence of an increase in severity/complications
- Co-morbidities, such as cardiac disease.
- Substantial impact on their quality of life and activities of daily living
- Diagnostic uncertainty

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

**Clinical diagnosis:** Same as level 1. The case referred from Level 1, or a fresh case must beevaluated thoroughly for any complications.

### Investigations:

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

**Management:** Same as level 1 and/or treatments mentioned at this level.

**Table 7: Single drugs and compound formulations for internal/ external use**

S. No	Drug	Dosage form	Dose (per day)	Time of administration	Duration & Frequency	Badraqa (vehicle)
1	<i>Asaroon (Asarum europaeum L.)</i> <sup>57</sup>	Powder	2-5 g	After meal	Upto 3 Months	Water
2	<i>Filfil siyah (Piper nigrum L.)</i> <sup>52</sup>	Powder	0.75- 2 g	After meal	Upto 3 Months	Water
3	<i>Tukhm-i Kasni (Chicorium intybus L. seeds)</i> <sup>57</sup>	Arq Extract Powder	60-100 ml 12-24 ml 3-6 gm	After meal	Upto 3 Months	Water
4	<i>Qust (Saussurea costus (Fa;c.) Lipsch.)</i> <sup>51</sup>	Powder	2-3 g	After meal	Upto 3 Months	Water
5	<i>Zafran (Crocus sativus L.)</i> <sup>57</sup>	Powder	1-2 g in divided doses	After meal	Upto 3 Months	Water
6.	<i>Jawarish Zarooni Ambri</i> <sup>58</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water

S. No	Drug	Dosage form	Dose (per day)	Time of administration	Duration & Frequency	Badraqa (vehicle)
7	<i>Qurs-i Pudina</i> <sup>55</sup>	Pills	1g twice a day	After meal	Upto 3 Months	Water
8	<i>Qurs-i Gul</i> <sup>56</sup>	Pills	1g twice a day	After meal	Upto 3 Months	Water
9	<i>Araq-i Mako</i> <sup>52</sup>	Distillate	60-100 ml in 2 divided doses	After meal	Upto 3 Months	Water
10	<i>Jawarish Zanjabil</i> <sup>52</sup>	Semi-solid preparation	7-14 gm in 2 divided doses	After meal	Upto 3 Months	Water
11	<i>Zimad-i Feesaghorus</i> <sup>58</sup>	Paste for local application	Q.S for external use	As directed by the physician	Upto 3 months	--

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. '*Ilaj bi'l Tadbir* (Regimenal Therapy) described under principles of management may be recommended as per assessment of physician about the condition of the patient and stage of disease. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy

**'Ilaj bi'l Tadbir (Regimenal therapy):**<sup>59</sup>

*Riyāḍat* (exercise) in the form of brisk running is highly recommended as it reduces body mass and increases body heat.

*Dalk* (massage) with hot oils or pastes over hepatic region is recommended.

*Ḥammām* (therapeutic bath/turkish bath), preferably with medicated steam, are also recommended.

**Recommended diet and lifestyle:** Same as level 1

**Restricted diet and lifestyle:** Same as level 1

**Follow up:** 15 day or as recommended by the Physician

**Referral criteria:**

- Same as mentioned earlier at level 1, with
- Failure of acute exacerbation to respond to initial medical management

**At level 3** (Unani hospitals attached with teaching institution, District level/ Integrated/ State Unani hospitals, Tertiary care allopathic hospitals having Unani facilities), multiple departments/ facilities for diagnosis and interventions. Must provide additional facilities like dieticians, counselling, and physiotherapy unit.

**Clinical diagnosis:** Same as level 1 & 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 MTTP to rule out abetalipoproteinemia ( only if needed)

**Management:** Same as levels 1&2 and/or treatment mentioned at this level.

At this level care giver should assess the patient for the disease complications and manage accordingly. Screening and treatment of portal hypertension, screening for advanced fibrosis, HCC should be done and the patient should be referred to tertiary care hospital

**Table 8: Single drugs and compound formulations for internal/ external use**

S. No	Drug	Dosage form	Dose (per day)	Time of administration	Duration & Frequency	Badraqa (vehicle)
1	<i>Aftimoon (Cuscuta reflexa Roxb.)</i> <sup>60</sup>	Decoction	40-50 ml	After meal	Upto 3 Months	Water
2	<i>Na'na (Mentha arvensis L.)</i> <sup>61</sup>	Powder Distillate	3-5 g 60-120 ml in divided doses	After meal	Upto 3 Months	Water
3	<i>Gul-i Surkh (Rosa damascena Mill.)</i> <sup>60</sup>	Powder Distillate Oil	5-7 g 20-40 ml Q.S	After meal	Upto 3 Months	Water
4	<i>Qaranful (Syzygium aromaticum (L.) Merr. &amp; L.M. Perry)</i> <sup>51</sup>	Powder Oil	0.5-1 g 1-3 drops	After meal	Upto 3 Months	Water
5	<i>Jawarish Amla Ambri</i> <sup>57</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water
6	<i>Jawarish Darchini Qawi</i> <sup>56</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water
7	<i>Jawarish Jalinoos</i> <sup>56</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water
8	<i>Majun Dabeedul Ward</i> <sup>55</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water
9	<i>Qurs-i Luk</i> <sup>56</sup>	Pills	1g twice a day	After meal	Upto 3 Months	<i>Sikanjbeen-i Asli</i>

S. No	Drug	Dosage form	Dose (per day)	Time of administration	Duration & Frequency	Badraqa (vehicle)
10	<i>Qurs-i Rewand Kabidi</i> <sup>56</sup>	Pills	1g twice a day	After meal	Upto 3 Months	Sikanjbeen-i Asli
11	<i>Qurs-iSumbul-ut-Teed</i> <sup>56</sup>	Pills	1g twice a day	After meal	Upto 3 Months	Sikanjbeen-i Asli
12	<i>Dawa'ul Misk Motadil Jawahar Wali</i> <sup>55</sup>	Semi-solid preparation	5-10 g	After meal	Upto 3 Months	Water

**Note:** out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. 'Ilaj bi'l Tadbir (Regimenal Therapy) described under principles of management may be recommended as per assessment of physician about the condition of the patient and stage of disease. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

**'Ilaj bi'l Tadbir (Regimenal therapy):**<sup>59, 62</sup>

**Riyāḍat (exercise)** in the form of brisk running is highly recommended as it reduces body mass and increases body heat.

**Dalk (massage)** with hot oils or pastes over hepatic region is recommended. Compound formulations such as, *Roghan Afsanteen* may be used.

*Ḥammām* (therapeutic bath/Turkish bath), preferably with medicated steam, are also recommended.

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

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

**Follow-up :**15 days or as recommended by the physician

**Referral criteria:**

- Same as Level 1 & 2, with,
- Hepatic encephalopathy
- Portal hypertension
- Haematemesis or melaena or any condition requiring blood transfusion or critical care management
- Any condition or serious complication beyond the scope of Unani treatment
- 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. Duseja A, Singh SP, Mehta M, Shalimar, Venkataraman J, Mehta V, et al. Clinicopathological Profile and Outcome of a Large Cohort of Patients with Nonalcoholic Fatty Liver Disease from South Asia: Interim Results of the Indian Consortium on Nonalcoholic Fatty Liver Disease. *Metab Syndr Relat Disord* 2022;20(3):166–73.
5. 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>
6. Duseja A, Chalasani N. Epidemiology and risk factors of nonalcoholic fatty liver disease (NAFLD). *Hepatol Int* [Internet] 2013 [cited 2021 Nov 24];7 Suppl 2:S755–64. Available from: <https://pubmed.ncbi.nlm.nih.gov/26202291/>
7. 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.
8. 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>
9. 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>
10. 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/NEJMr0912063>
11. 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.
12. 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>
13. 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>
14. 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>

15. 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.
16. 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](https://journals.lww.com/ijmr/fulltext/2020/51040/fibroscan_as_a_non_invasive_predictor_of_hepatic.12.aspx)
17. 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>
18. 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](https://journals.lww.com/hep/fulltext/2018/01000/the_diagnosis_and_management_of_nonalcoholic_fatty.31.aspx)
19. Tabri AM. Moalajate Buqratiya. Vol 3. New Delhi: CCRUM; 1997. p. 202-217.
20. Jurjani AH. Zakheera Khwarzam Shahi. Vol 6. New Delhi: Idara Kitabus Shifa; 2010. p. 371-374, 377-381
21. Ibn Rushd AM. Kitabul Kulliyat. 2nd ed. New Delhi: CCRUM; 1987. p. 26,35,46,49-50
22. Kabiruddin, M. Al-Iksir. New Delhi: Idara Kitabus Shifa; 2011. p. 481-498.
23. Ibn e Sina. Al Qanoon Fil Tib. Vol 3. Part 1. New Delhi: Idara Kitabus Shifa; YNM. p. 849-852, 854-864.
24. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. III. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 1-3,31-32
25. Pouwels S, Sakran N, Graham Y, Leal A, Pintar T, Yang W, et al. Non-alcoholic fatty liver disease (NAFLD): a review of pathophysiology, clinical management and effects of weight loss. *BMC Endocr Disord* [Internet] 2022 [cited 2024 Aug 30];22(1):1–9. Available from: <https://bmcendocrdisord.biomedcentral.com/articles/10.1186/s12902-022-00980-1>
26. 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: <https://pubmed.ncbi.nlm.nih.gov/16882782/>
27. 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: <https://pubmed.ncbi.nlm.nih.gov/2429802/>
28. 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: <https://pubmed.ncbi.nlm.nih.gov/427027/>
29. 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/>
30. Berzigotti A, Tsochatzis E, Boursier J, Castera L, Cazzagon N, Friedrich-Rust M, et al. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis – 2021 update. *J Hepatol* [Internet] 2021 [cited 2024 Aug 30];75(3):659–89. Available from: <http://www.journal-of-hepatology.eu/article/S0168827821003986/fulltext>
31. 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/>

32. 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>
33. 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.
34. 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.
35. 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>
36. Cichoz-Lach H, Celiński K, Prozorow-Król B, Swatek J, Słomka M, Lach T. The BARD score and the NAFLD fibrosis score in the assessment of advanced liver fibrosis in nonalcoholic fatty liver disease. *Med Sci Monit* [Internet] 2012 [cited 2024 Sep 2];18(12):CR735. Available from: [/pmc/articles/PMC3560810/](https://pubs.scisearch.org/PMCID/PMC3560810/)
37. 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>
38. Al Razi ABMBZ. *Kitab-al Hawi Fit-Tib*. Vol. 7 New Delhi: CCRUM; 2000. p.47-51
39. Baghdadi ABH. *Mukhtarat Fil Tib*. Vol. 3. New Delhi: CCRUM; 2004. p.268-273
40. Arzani HA. *Tib e Akbar*. Vol. 2. (Urdu translation by Hakim Mohammad Hussain).Deoband: Faisal Publications; YNM. p. 439-442.
41. Vilar-Gomez E, Martinez-Perez Y, Calzadilla-Bertot L, Torres-Gonzalez A, Gra-Oramas B, Gonzalez-Fabian L, et al. Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. *Gastroenterology* [Internet] 2015 [cited 2024 Sep 2];149(2):367-378.e5. Available from: <http://www.gastrojournal.org/article/S0016508515004965/fulltext>
42. Memel ZN, Wang J, Corey KE. Intermittent Fasting as a Treatment for Nonalcoholic Fatty Liver Disease: What Is the Evidence? *Clin Liver Dis (Hoboken)* [Internet] 2022 [cited 2024 Sep 2];19(3):101–5. Available from: [https://journals.lww.com/cld/fulltext/2022/03000/intermittent\\_fasting\\_as\\_a\\_treatment\\_for.5.aspx](https://journals.lww.com/cld/fulltext/2022/03000/intermittent_fasting_as_a_treatment_for.5.aspx)
43. Younossi ZM, Corey KE, Lim JK. AGA Clinical Practice Update on Lifestyle Modification Using Diet and Exercise to Achieve Weight Loss in the Management of Nonalcoholic Fatty Liver Disease: Expert Review. *Gastroenterology* [Internet] 2021 [cited 2024 Sep 2];160(3):912–8. Available from: <http://www.gastrojournal.org/article/S0016508520355384/fulltext>
44. Sung KC, Ryu S, Lee JY, Kim JY, Wild SH, Byrne CD. Effect of exercise on the development of new fatty liver and the resolution of existing fatty liver. *J Hepatol* [Internet] 2016 [cited 2024 Sep 2];65(4):791–7. Available from: <http://www.journal-of-hepatology.eu/article/S0168827816302124/fulltext>
45. Firdaus HHAK.Majmaul Behrain.Lucknow:Munshi Naval Kishor;YNM:p.411-413.
46. Ali SA. *Tarjuma Zakheera Sabit Ibn Qurrah Aligarh*: Lithocolour printers; 1987. p.239-240
47. Freidoony L, Kong ID. Practical approaches to the nutritional management of nonalcoholic fatty liver disease. *Integr Med Res*. 2014;3(4):192–7.
48. Nseir W, Hellou E, Assy N. Role of diet and lifestyle changes in nonalcoholic Fatty liver disease. *World J Gastroenterol*. 2014;20(28):9338–44.
49. Romero-Gómez M, Zelber-Sagi S, Trenell M. Treatment of NAFLD with diet, physical activity and exercise. *J Hepatol*. 2017;67(4):829–46.

50. Pintó X, Fanlo-Maresma M, Corbella E, Corbella X, Mitjavila MT, Moreno JJ, et al. A Mediterranean diet rich in extra-virgin olive oil is associated with a reduced prevalence of nonalcoholic fatty liver disease in older individuals at high cardiovascular risk. *J Nutr.* 2019;149(11):1920–9.
51. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. I. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 26-27,70-71,74-75,84-85, 86-87
52. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. IV. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 7-8,38-39, 91-92
53. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. II. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 3-4, 91-92
54. Anonymous. National Formulary of Unani Medicine, Part VI. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2011: 32.
55. Anonymous. National Formulary of Unani Medicine, Part V. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2008: 27,66,73,90.
56. Anonymous. National Formulary of Unani Medicine, Part II, Vol. I. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2007: 19,29, 30,32,33,87,94.
57. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. VI. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 95-96,15-16,101-102
58. Anonymous. National Formulary of Unani Medicine, Part IV. New Delhi: Dept. of AYUSH, Ministry of H & F W, Govt. of India; 2006: 40,118.
59. Ahmed NZ, Alam MA, Sheeraz M. Concept and Management of Fatty Liver Disease in Unani Medicine Vis-A-Vis Conventional Medicine-A Review. *Spatula DD.* 2014;4(4):233-41
60. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. III. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 1-3,31-32
61. Anonymous. The Unani Pharmacopoeia of India, Part I, Vol. V. New Delhi: Dept. of Ayush, Ministry of H & FW, Govt. of India; 2007: 54-55.
62. Riyazuddin M, Shahid A. Non-alcoholic fatty liver disease—discussed under the light of Unani medicine. *Journal of Complementary and Integrative Medicine.* 2023 Mar 14;20(1):17-23.

CHAPTER

# 5

## OBESITY





# OBESITY

(ICD 10 code: E66.0-E66.9)  
(ICD 11 code: 5B81.0-5B81.Z)

*Siman Mufrit* (Obesity) (NUMC: M-37)  
Obesity disorder (TM2) **SP64**

## 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/m<sup>2</sup> or higher and BMI between 25 and 30 kg/m<sup>2</sup> is defined as overweight. The WHO Asia -Pacific region defined BMI  $\geq 23\text{kg/m}^2$  as overweight and  $\geq 25\text{kg/m}^2$  Obesity. Obesity is defined as a body mass index (BMI) equal to or greater than the 95<sup>th</sup> percentile for age and sex.<sup>1</sup>

## INTRODUCTION (incidence/ prevalence, morbidity/ mortality)

- In 2022, one in every eight 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.<sup>2</sup>
- As per National Family Health Survey-5 (NFHS-5), one in every four Indians is now having obesity. There are 135 million obese individuals in India. The prevalence of abdominal obesity in the country was found to be 40% in women and 12% in men.<sup>3</sup>
- 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. <sup>4</sup>
- 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).<sup>5</sup> Therefore, obesity is more effectively defined by assessing its linkage to morbidity and mortality.<sup>6</sup> The current guidelines, deal with management of both overweight and obesity.

## Unani Medicine's Perspective: (Etiology, Pathology, Risk Factors and Clinical Presentation)

*Siman Mufrit* (obesity) is a condition in which the body becomes extremely overweight causing hindrance in daily physical activities. It is caused by *Sū'-i Mizāj Bārid Raṭb Māddī* (morbid cold and moist temperament with substance), excessive accumulation of humours which are normally evacuated from the body, sedentary and luxurious lifestyle, excessive rest, excessive sleep and excessive use of those diets that are cold and moist in nature. <sup>7,8</sup>

### Etiology<sup>7,8,9,10</sup>

- *Sū'-i Mizāj Bārid Raṭb Māddī* (morbid cold and moist temperament with substance)
- Excessive and abnormal accumulation of humours which are normally evacuated
- Excessive consumption of *Al-Ghidhā' al-Dasim* (fatty diet)

- Excessive sleep
- Excessive rest
- Excessive consumption of diet of cold temperament
- Use of *Ḥammām* after meals

### **Pathology**

Phlegmatic persons are prone to obesity. Predominance of *Burūdat* (coldness) and *Ruṭūbāt* (wetness) associated with matter in their bodies leads to morbid obesity. Predominance of *Burūdat* (coldness) leads to constriction of vessels resulting into decreased innate heat and obstruction in the flow of pneuma to the organs. Predominance of *Ruṭūbāt* (wetness) leads to *imtilā* (congestion) <sup>7,8,9,10</sup>

**Risk factors-** Children and adolescents, sedentary life style, Excessive sleep, Excessive rest, phlegmatic temperament individuals. <sup>7,8</sup>

### **Clinical presentation:** <sup>7,8,9,10</sup>

- Lethargy
- Excessive sweating
- Muscular Pain
- Breathlessness on exertion
- Difficulty in performing normal physical activities
- Loss of libido
- Nausea
- Joint pain
- Low back pain
- Immobility
- Low production of semen
- Inability of a woman to conceive
- Abortion is common, if conceived
- Obese persons are prone to epilepsy, paralysis, bronchial asthma, cholera, syncope and *Ḥummiyāt Muḥarriqa*.

### **CLINICAL EXAMINATION** <sup>11</sup>

Persons presenting with overweight, or obesity must have a detailed history taken, a clinical examination performed, and appropriate investigations done (Figure – 1). This is done to identify the environmental, genetic and lifestyle factors responsible for obesity and at the same time identify impact of overweight and obesity on the individual, physically, mentally and socially.

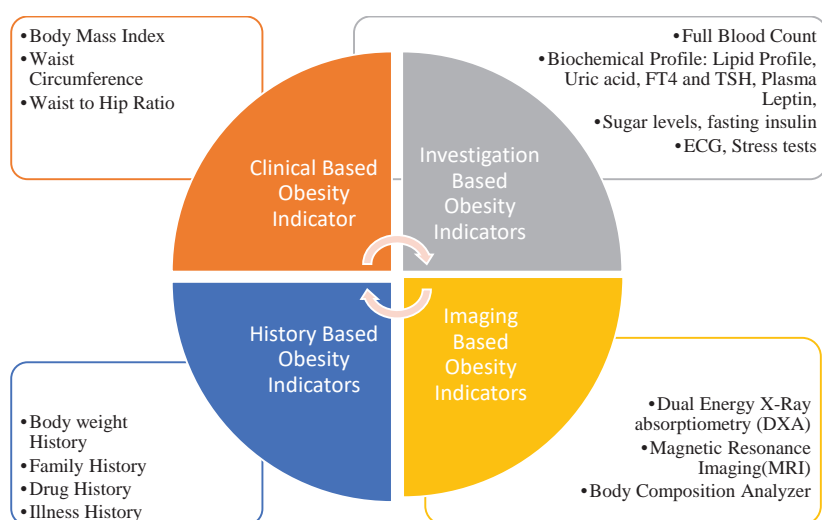
### **Clinical History**

- **Body weight history** in persons who are overweight or present with pre-obesity/obesity may begin with an assessment of body weight increases or reductions over the individual's lifetime (e.g., slow and gradual, rapid and sudden, or a combination) and factors influencing weight change. Short sleep duration and poor sleep quality may increase the risk of obesity, making it important to record sleep patterns in patients<sup>12</sup>

- **A detailed family history** is important and often suggests a genetic predisposition.
- **Drug history** should be taken to identify possible drugs that may be contributing to weight gain, such as steroid hormones, antidepressants (tricyclics), antipsychotics (phenothiazines and butyrophenones), anticonvulsants (valproate and carbamazepine), lithium, and antihyperglycemics (insulin, sulfonylurea, and thiazolidinediones).
- **The psychological aspects of eating behaviour** should be explored, such as loneliness, boredom, or stress. Often obese persons express feelings of low self-esteem and depression. Eating disorders should be particularly sought.
- A thorough **Review of Systems** must be taken to assess any co-morbidities that are directly or indirectly related to obesity, to identify any evidence of endocrine disease as an occult aetiology of obesity.
- A thorough **examination of the patient's present dietary habits** is essential. This evaluation can be conducted by a dietitian. It should involve assessing the total daily calorie intake and determining the percentage of calories derived from fat. Individuals with obesity often show abnormal eating patterns. The eating disorders that have been most frequently studied in individuals with obesity are binge eating disorder and bulimia nervosa.
- **History pertaining to physical activity.** Physically active and fit individuals are considerably less likely to be obese than physically inactive and unfit individuals. Therefore, it's essential to gather comprehensive information to understand their current activity level, any past injuries or limitations, their exercise preference and Lifestyle Factors.

### Clinical and imaging indicators of obesity

Apart from BMI, waist circumference, waist-hip ratio, and skin-fold thickness, the variations in lean muscle mass and body fat percentage are also assessed utilizing the body composition analyzer.<sup>13</sup>



**Figure 1 Assessments in overweight and obese persons**

**Physical Examination<sup>14</sup>**

- 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.<sup>15</sup>
- Tongue examination (Size, Colour, Texture).
- Markers of insulin resistance- Skin tags, acanthosis nigricans.

**Comorbidities and Complications<sup>16</sup>**

Obesity and overweight are associated with increased risk of disabilities ,comorbidities and complications<sup>17</sup> as listed in Table 4, which must be diagnosed timely.

**Table 1: Complications and Comorbidities**

SYSTEM	DISEASES
Respiratory	<ul style="list-style-type: none"><li>• Obstructive sleep apnoea (OSA)</li><li>• Obesity Hypoventilation Syndrome (OHS)</li></ul>
Cardiovascular	<ul style="list-style-type: none"><li>• Coronary Heart Disease</li><li>• Congestive Cardiac Failure</li><li>• Hypertension</li></ul>
Cerebrovascular	<ul style="list-style-type: none"><li>• Stroke</li></ul>
Gastrointestinal	<ul style="list-style-type: none"><li>• Gastroesophageal Reflux Disease</li><li>• Barrett’s Oesophagus</li><li>• Erosive Oesophagitis</li><li>• Diverticular Disease</li><li>• Oesophageal Cancer</li><li>• Colon Cancer</li><li>• Abdominal Hernia</li></ul>
Metabolic	<ul style="list-style-type: none"><li>• Dyslipidaemia</li><li>• Type 2 Diabetes Mellitus</li><li>• Hyperinsulinemia</li><li>• Metabolic Syndrome</li><li>• Gout</li><li>• Gestational Diabetes</li></ul>
Hepato-biliary	<ul style="list-style-type: none"><li>• NASH (Non-alcoholic steatohepatitis)</li><li>• Liver Cirrhosis</li><li>• Hepatocellular Carcinoma</li><li>• Gallstone</li><li>• Gall Bladder Cancer</li></ul>
Musculoskeletal	<ul style="list-style-type: none"><li>• Osteoarthritis</li></ul>
Cutaneous	<ul style="list-style-type: none"><li>• Acanthosis nigricans</li><li>• Cutaneous fungal and yeast infections</li><li>• Venous stasis</li></ul>

SYSTEM	DISEASES
Reproductive disorders	<ul style="list-style-type: none"> <li>Male: gynaecomastia</li> <li>Female: Menstrual Irregularities, PCOS, Infertility</li> </ul>
Cancer	<ul style="list-style-type: none"> <li>Male: Liver cancer, Pancreas cancer, Rectum cancer, Prostate</li> <li>Female: Gall bladder, Bile duct, Breast, Ovary, Uterine, Cervix</li> </ul>

### DIFFERENTIAL DIAGNOSIS

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

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

**Table 2: Differential diagnosis**

S.No.	Condition	Features
1.	<b>Obesity due to lifestyle factors</b>	<ul style="list-style-type: none"> <li>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.</li> <li>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.</li> <li>Snacking and reliance on fast food are recognized as significant contributors to childhood overweight and obesity <sup>18</sup></li> </ul>
2.	<b>Obesity due to endocrinal conditions<sup>19</sup></b>	<p>The mechanisms underlying the development of obesity vary according to the abnormalities of endocrine function, whilst at the same time, increase in body fats also tends to lead to abnormalities in endocrinal functions.</p> <p>Some endocrinal disorders associated with obesity are:</p> <ul style="list-style-type: none"> <li>Hypothyroidism</li> <li>Cushing's Syndrome</li> <li>Insulinoma</li> <li>Ovarian disorders, hyperovarian syndrome</li> <li>Hypogonadism in men</li> <li>Hypothalamic tumours or damage to this part of the brain as a consequence of irradiation, infection, or trauma</li> </ul>
3.	<b>Obesity with genetic conditions <sup>20</sup></b>	<p>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</p> <ul style="list-style-type: none"> <li>Chromosomal rearrangements, monogenic due to mutations in leptin signalling pathways or polygenic i.e. multiple mutations coding for proteins in skeletal and adipose tissues</li> </ul>

S.No.	Condition	Features
		<ul style="list-style-type: none"> <li>• Down's syndrome</li> <li>• Prader-Willi syndrome</li> <li>• WAGR syndrome</li> <li>• SIM1 syndrome</li> <li>• Bardet-Biedl syndrome</li> <li>• Fragile X syndrome</li> <li>• Cohen syndrome</li> <li>• Albright hereditary Osteodystrophy/PHP Type 1 a</li> <li>• Alstrom syndrome</li> <li>• Carpenter syndrome</li> <li>• Chudley-Lowry syndrome, etc.</li> </ul>
4.	<b>Drugs-Induced obesity</b> <sup>21,22</sup>	<p>Weight gain or body fat redistribution are common side effects of many widely used drugs, some of which are given below:</p> <ul style="list-style-type: none"> <li>• <b>Anticonvulsants:</b> Sodium Valproate, Phenytoin</li> <li>• <b>Hypoglycaemics:</b> Insulin, Sulfonylurea (SU), Thiazolidinediones</li> <li>• <b>Beta-Blockers:</b> Atenolol, Metoprolol, Propranolol</li> <li>• <b>Antidepressants:</b> Amitriptyline, Nortriptyline, Imipramine, Desipramine, Dosulepin, Doxepin, Clomipramine</li> <li>• <b>Antipsychotics:</b> Haloperidol, Perphenazine</li> </ul>

## INVESTIGATIONS <sup>23</sup>

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
- Magnetic Resonance Imaging (MRI)
- Test For Insulin Resistance (OGTT, 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 squared (kg/m<sup>2</sup>): weight (kg)/height (m<sup>2</sup>).

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

- Obesity in adults is defined as a BMI greater than or equal to 30; overweight is defined as a BMI greater than or equal to 25
- In children aged below 5 years, overweight is 2 standard deviations and obesity is greater than 3 standard deviations above the WHO Growth Reference median<sup>24</sup>
- 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 median<sup>25</sup>

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

Table 3: Classification of obesity by BMI in adults<sup>26</sup>

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

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

Table 5: Classification of BMI in children <sup>27</sup>

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

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

The body mass index is a surrogate marker of fatness, and additional measurements, such as the waist circumference, are also used to diagnose obesity<sup>28</sup>. Measures of overweight and obesity and their cut-off for Indian population are given in Table 3.

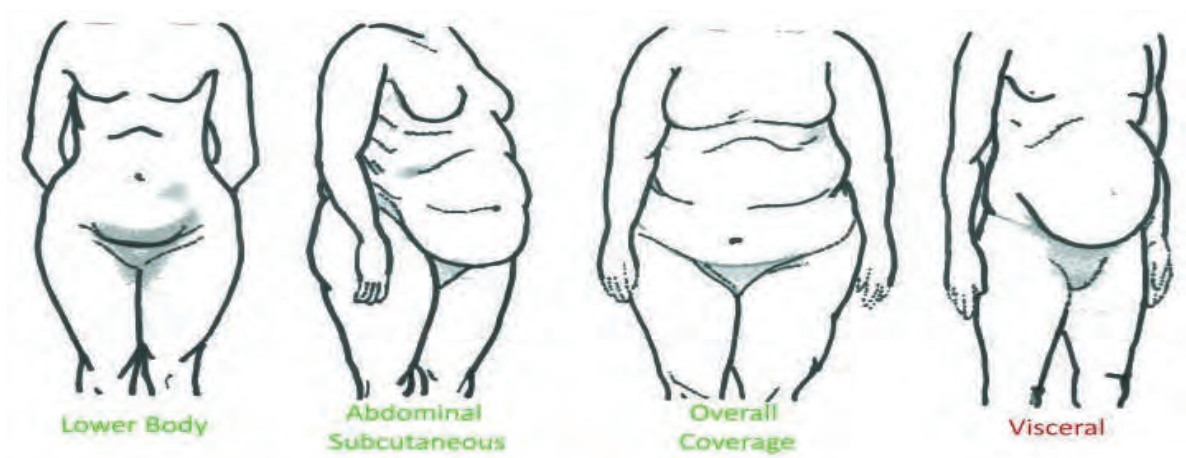
**Table 6: Indian cut-offs for Indicators** <sup>29</sup>

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 <sup>2</sup> )	>23 Overweight, >25 – Obesity	

The 5<sup>th</sup> 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 a healthy BMI also happened to have abdominal obesity.<sup>30</sup>

**Types of Body Fat Distribution** <sup>31,32</sup>

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 1)



**Figure – 1 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.

## PRINCIPLES OF MANAGEMENT

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

### Red Flags

- Unintentional weight gain
- Breathlessness
- Sleep Apnoea syndrome
- Rapid Onset of weight gain.
- Body Mass Index (BMI) greater than 40 kg/m<sup>2</sup> – Morbid obesity
- Weight gain associated with other systemic complications.
- Cardiac arrhythmia and unstable cardiac conditions
- Malignancies associated with obesity

### (A) Prevention management

Measures addressing dietary intake, home nutrition environment, diet knowledge, physical self-concept, and body perception, barriers for exercise are known to prevent obesity particularly in the younger age group<sup>33, 34</sup>

- The primary goals of treatment are to improve obesity-related comorbid conditions, improve quality of life and reduce the risk of developing future obesity-related complications.
- Obesity in children and adolescents also requires an interprofessional team approach. Failure to adequately diagnose and treat overweight/ obesity results in comorbid medical conditions and the likelihood that a child will become an obese adult.<sup>35</sup>
- Patients who present with obesity-related comorbidities and who would benefit from weight-loss intervention should be managed proactively.

### Unani Medicine's Perspective:

The general line of treatment as mentioned in classics:<sup>7,8</sup>

- *Taskhīn-i-Badan* (calefaction of body)
- *Tajfīf-i Badan* (desiccation)
- *Taqlīl-i-Ghidhā'* (to reduce the quantity of food)
- *Taltīf* (refining of thick and viscid matter)
- *Tahlīl-i-Mādda* (dissolving morbid matter)
- *Tahzīl* (to induce weight loss)

A comprehensive plan for the management of obesity in an individual patient may include educational, behavioural, psychosocial, and physical interventions, as well as Unani topical and oral medications (single and compound formulations). A single physical, psychosocial, or pharmacologic intervention may be adequate to control obesity in some patients. While in chronic cases, multiple interventions may be used in sequence or in combination to treat the patients.

'*Ilāj bi'l Dawā*' (pharmacotherapy) and '*Ilāj bi'l Tadbīr*' (regimenal therapy) are considered the mainstay of treatment in the case of obesity. '*Ilāj bi'l Tadbīr*' (regimenal therapy) includes *Riyāḍat* (exercise) [IUMT-7.2.80]<sup>7,8,9,10</sup>, *Dalk* (therapeutic massage [IUMT-7.2.92]<sup>8</sup>, *Laṭūkh* (epithem) [IUMT-6.2.92]<sup>8</sup>, *Ḥammām* (therapeutic bath /Turkish bath [IUMT-7.2.70]<sup>8</sup>, *Idrār* (to induce increased flow of urine/menstruation/ sweat) [IUMT-7.2.4]<sup>7,8</sup>, *Ta'rīq* (inducing diaphoresis) [IUMT-7.2.69]<sup>7,8</sup>

#### **For prevention of progression:**

- Avoiding the causes and risk factors that may lead to obesity, e.g., intake of phlegm-producing diet, sedentary lifestyle, mental stress, etc.
- Correction of humoral and temperamental derangement: obesity is caused by cold and wet morbid temperament that involves phlegmatic matter. The basis of correction of obesity is food and lifestyle modification, along with the causative phlegmatic humour evacuation and the administration of obesity-specific medications.

#### **(B) Interventions**

##### **At Level 1- Solo Physician Clinic/Health Clinic/PHC (Optimal standard of treatment where technology and resources are limited)**

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

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

#### **Laboratory Investigations**

- No specific investigation is required.

#### **Recommended Diet and Lifestyle<sup>36,37</sup>**

Overweight and obesity care involves attention to three essential elements of lifestyle:

- Dietary habits,
- Physical activity, and
- Behaviour modification.

#### **1. Diet Therapy**

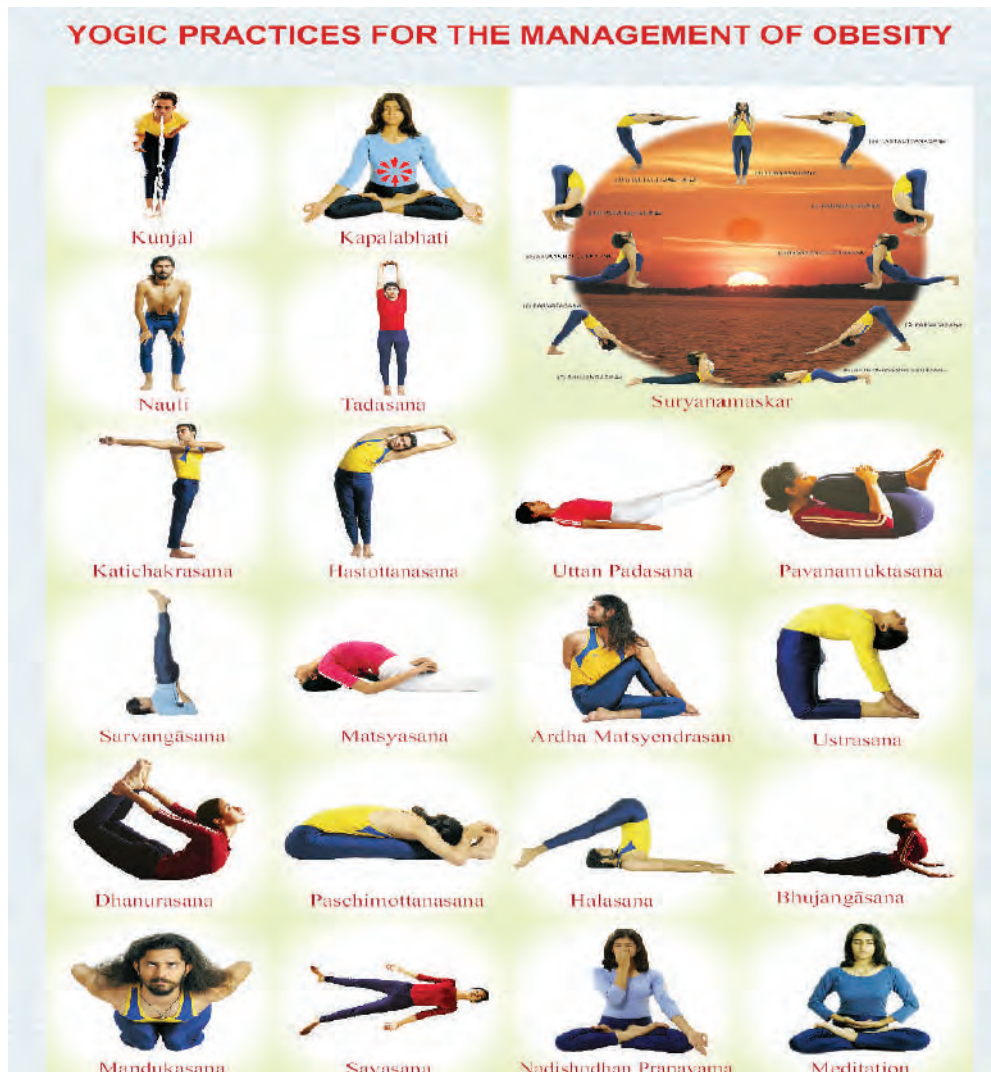
- The primary focus of diet therapy is to reduce overall calorie consumption.
- A calorie-deficient diet is advised, taking into consideration nutritional requirements.

- The calorie deficit may be instituted through dietary substitutions or alternatives. Examples include choosing smaller portion sizes, eating more fruits and vegetables, consuming more whole-grain cereals, selecting leaner cuts of meat and skimmed dairy products.
- Adequate intake of micronutrients and fibre-rich foods such as pulses, nuts, chia seeds, flax seeds, whole grains including millets, vegetables and fruits helps to maintain levels of blood glucose, insulin, cholesterol as well as triglycerides. Use of healthy cooking methods like grilling, baking, steaming or sautéing with minimal oil instead of frying is recommended.
- A daily calorie deficit of 500-1000 kcal is commonly recommended which typically results in a weight loss of 0.5-1kg per week. Total calorie intake is 1200-1500 kcal/day for women, 1500-1800 kcal/day for men. These values may vary and should be adjusted to individual needs to avoid nutritional deficiencies. A reduction of half a kilogram body weight per week is considered to be safe. Approaches of rapid weight loss should be avoided. Consuming higher amounts of protein (15% energy from protein) may be important during typical energy deficient weight loss diets (i.e. 500 to 750 kilo calorie per day deficit) to preserve muscle mass. Nevertheless, the protective effect of higher protein diets on muscle mass is compromised if the energy deficit is more than 40% of daily energy needs and the dietary proteins are oxidised for energy production. Weight reducing diet should be nutrient rich and nutritionally balanced, with adequate intake of micro-nutrients and fibre rich foods.
- The Yogic diet, popularly known as Satvik diet is the most preferred diet in obese condition. Satvik diet contains more of fresh fruits and vegetables in its natural form, soup etc. Rajasik foods like fried food items, spicy foods, soft drinks and beverages, fast foods etc, should be limited.<sup>38</sup>
- Shift to healthy snacking such as fruits, vegetables and sprouts instead of cakes, biscuits and fried snacks.
- To have regular meals at fixed intervals.

## 2. Physical Activity Therapy

- A combination of dietary modification and increased physical activity or exercise is the most effective behavioural approach for the treatment of obesity. The most important role of exercise appears to be in the maintenance of weight loss.<sup>39</sup>
- At least 150 minutes aerobic physical activity (e.g., brisk walking) per week (equivalent to 30 minutes per day for 5 days of the week) for initial weight loss, increasing to round 200 to 300 minutes per week to maintain body weight and prevent weight regain is recommended.<sup>39</sup> Exercise intensity and duration should be increased gradually over a period of time.<sup>40</sup>
- Exercise for weight reduction goes beyond being simply physically active during the day, both in term of type and duration of activity or exercise.
- However, initiating type and duration of exercise and a gradual increase in physical activity needs to be undertaken with due consideration of the overall health condition, including systemic complications of the individual patient.
- Yoga practices can reduce weight and also improve stress, endocrinal imbalances and other factors associated with obesity. Yoga or physical exercises are suggested to be undertaken under the supervision of a trained therapist.

- Yogic practices include<sup>36</sup>:
  - Om chanting and Prayer
  - Shodhana Kriyas: Kapalabhati, Kunjal, Agnisara, Nauli
  - Suryanamaskar
  - Sukshma Vyayama
  - Yogasanas: Tadasana, Katichakrasana, UrdhwaHastottanasana, Pawanamuktasana, Sarvangasana, Matsyasana, Halasana, Bhujangasana, Dhanurasana, UttanPadasana, Paschimottanasana, Ardha Matsyendrasana, Ushtrasana, Mandukasana, Shavasana
  - Pranayama: Nadishodhana, Suryabhedhi Pranayama, Bhramari, Sitali, Bhastrika
  - Special Practice: Yoga Nidra
  - Dhyana (Meditation): Om Chanting, Om Meditation, and Anapana Meditation
  - Yama and Niyama: This will help to have a controlled behaviour and would help to pacify the wandering mind, and in turn help to have control over the eating and other habits of a person.
- Physical activity can be in the form of moderate to vigorous intensity aerobic activity, resistance training and muscle strengthening exercises.<sup>41</sup>



3. Behavioural therapy

- Cognitive behavioural therapy can change and reinforce new dietary and physical activity behaviours.
- Strategies include self-monitoring techniques (e.g., journaling, weighing, and measuring food and activity); stress management; stimulus control (e.g., using smaller plates, not eating in front of the television or in the car); social support; problem solving; and cognitive restructuring to help patients develop more positive and realistic thoughts about themselves.
- When recommending any behavioural lifestyle change, the patient should be asked to identify what, when, where, and how the behavioural change will be performed.<sup>42</sup>
- Encourage breast feeding as the child who gets proper breast feeding is less likely to develop obesity in the later age.

Recommended Diet and Lifestyle- Unani Medicine’s Perspective:

Table 7: Dos and Don’ts

Dos	Don’ts (Disease aggravating factors)
<ul style="list-style-type: none"><li>• Intake of <i>Aghdhiya Yābisa</i> (dryness-producing diets)<sup>7,8</sup></li><li>• Intake of <i>Aghdhiya Hirrifa</i> (spicy diets)<sup>7,8</sup></li><li>• Intake of <i>Aghdhiya Māliha</i> (Salty diets)<sup>7,8</sup> if not hypertensive</li><li>• Intake of Lemon, piper, garlic, onion<sup>7,8</sup></li><li>• Intake of <i>Sirka</i> (vinegar) on empty stomach<sup>7,8</sup></li><li>• Intake of hot water<sup>7,8</sup></li><li>• Intake of reduced quantity of food<sup>7,8</sup></li><li>• Use of low-calorie diet<sup>9,10</sup></li><li>• Food prepared with <i>Sirka</i> (vinegar)<sup>9,10</sup></li><li>• Bread prepared with husk of <i>Jav</i> (<i>Hordeum vulgare</i> L.)<sup>7</sup></li></ul>	<ul style="list-style-type: none"><li>• Meat<sup>8</sup></li><li>• Milk<sup>8</sup></li><li>• Sweet dishes<sup>8</sup></li><li>• Sedentary Lifestyle<sup>8,9</sup></li><li>• Alcohol Consumption</li><li>• Smoking</li><li>• Excess sleep and day-time sleep<sup>8</sup></li><li>• Overeating<sup>8,9</sup></li><li>• Luxurious lifestyle<sup>9</sup></li></ul>

OPD level management –

In patients with Class I obesity (*Siman Mufrit*), two or more of the following forms of medications may be given along with diet restriction:

Table 8: Single Drugs and Compound Formulations at Level 1

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration and Frequency	<i>Badraqa</i> (Vehicle)
1.	<i>Luk Maghsul</i> (Lac) <sup>7,8</sup>	Powder	7 gm	Empty stomach	15 days to 1 month	Water/ vinegar
2.	<i>Sindrūs</i> ( <i>Vateria indica</i> L.) <sup>7,8</sup>	Powder	3.5 gm	After meal	15 days to 1 month	<i>Sikanjbeen</i> <sup>36</sup>
3.	<i>Sufuf-i-Mohazzil</i> <sup>7,8, 43</sup>	Powder	5 - 10 gm	Morning empty stomach	15 days to 1 month	water
4.	<i>'Araq -i-Zira</i> <sup>44</sup>	Liquid	20-40 ml	Morning empty stomach	15 days to 1 month	Not required

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration and Frequency	Badraqa (Vehicle)
5.	<i>Itirifal Saghīr</i> <sup>7,8</sup>	Semi-Solid	10-25 gm	Bed time	15 days to 1 month	water
6.	<i>Jawārish Kamūnī</i> <sup>7,8,43</sup>	Semi-Solid	10-15 gm in two divided doses	After meal	15 days to 1 month	water

**Note:** Out of the drugs mentioned above, any one or a combination of two or more may be prescribed by the physician. *'Ilāj bi'l Tadbīr* (Regimenal Therapy) described above may be recommended as per assessment of physician about the condition of the patient and status of obesity. The duration of the treatment may vary from patient to patient. The physician should decide the dosage and duration of the therapy based on the clinical findings and response to the therapy.

**Follow Up:-** 15 days or as recommended by the physician

#### Review should include:

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

#### Referral Criteria

The following patients may be referred to higher centers for better management.

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

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

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

### Investigations

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

### Management

- Same as level 1 and/or treatments mentioned at this level.
- Patients may be kept on fasting for early few days.
- **Recommended and Restricted Diet and Lifestyle: Same as level 1**

**Table 9: Single drugs and Compound Formulations at Level 2**

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)	Precaution/contraindication
1.	<i>Bekh-i-Khatmī</i> ( <i>Althaea officinalis</i> ), <i>Bekh-i- Qisaul himār</i> ( <i>Pueraria tuberosa</i> DC.), <i>Bekh-i-Jaoshīr</i> ( <i>Ferula galbaniflua</i> ) in equal parts <sup>8</sup>	Powder	3.5 gms	After meal	15 days to 1 month	water	Nothing specific (NS)
2.	<i>Sindrūs</i> ( <i>Vateria indica</i> L.), <i>Luk Maghsūl</i> (Lac), <i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.) in equal part <sup>8</sup>	Powder	4.5 gms	Morning	15 days to 1 month	water	NS
3.	<i>Luk Maghsūl</i> (Lac)-7gms, <i>Zīra Siyāh</i> ( <i>Carum carvi</i> L.), <i>Nankhwah</i> ( <i>Trachyspermum ammi</i> L.Spragne)-14 gms each <sup>8</sup>	Powder	7 gms	Morning	15 days to 1 month	<i>Sikanjbeen</i> <sup>26</sup> - 24 ml	NS

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)	Precaution/contraindication
4.	<i>Sufuf-i-Mohazzil</i> 43	Powder	5 - 10 gm	Morning empty stomach	15 days to 1 month	water	NS
5.	<i>Dawā al Luk</i> <sup>7,8,10</sup> 43	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water	Diabetes Mellitus Type I&II
6.	<i>Jawārish Falāfilī</i> 8,43	Semi-Solid	5-10 gms	After meal	15 days to 1 month	water	Diabetes Mellitus Type I&II

### **‘Ilāj bi’l Tadbīr (Regimenal Therapy)**

#### **Riyāḍat (Exercise)**

- *Riyāḍat Kathīra* (exercise for a prolonged duration)<sup>7,10</sup>
- *Riyāḍat Hathītha* (rigorous and rapid exercise)<sup>7,10</sup>
- *Riyāḍat Shāqqa* (Heavy physical exercise)<sup>8</sup>

#### **Dalk (Massage)**

- *Dalk Şulb* (massage with firm/ strong pressure) with *Roghan Shibit* and *Roghan Qust*<sup>8</sup>
- *Dalk Kathīr* (prolonged massage) with *Roghan Shibit* and *Roghan Qust*.<sup>8</sup>

#### **Ḥammām (Therapeutic Bath)**

- *Ḥammām Yābis* (dry bath) on empty stomach<sup>7,8,9,10</sup>
- *Ḥammām Ḥārr* (hot bath)<sup>7,8,9,10</sup>

#### **Laṭūkh (Epithem)**

- *Laṭūkh* (Epithem) with *Natrūn* (Sodium carbonate) in *Ḥammām* (Therapeutic Bath)<sup>8</sup>

**Follow Up:** 15 days or as recommended by the physician

#### **Referral Criteria**

The following patients may be referred to higher centers for better management.

- Same as level 1 with:
- Patients not responding to above mentioned management and needs further management in the form of ‘*Ilāj bi’l Tadbīr* (Regimenal Therapy) procedures
- Patients with a BMI  $\geq 40$  kg/m<sup>2</sup> (or  $\geq 35$  kg/m<sup>2</sup> with obesity related morbidities) who have not achieved sufficient weight loss to address health goals following behavioral treatment, with or without pharmacotherapy, may be referred to an obesity medicine specialist/ a bariatric surgeon.<sup>45</sup>

**At Level 3** (Unani hospitals attached with teaching institution, District Level/Integrated/ State Unani Hospitals, Tertiary care allopathic hospitals having Unani facilities), multiple departments/facilities for diagnosis and interventions. Must provide additional facilities like dieticians, counselling, and physiotherapy unit.

## Clinical Diagnosis

- Same as level 1 & 2.
- The case referred from Level 1 or 2, or a fresh case reporting directly should be evaluated thoroughly for any complications. 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

## Investigations

- Same as level 1 & 2 and Investigations mentioned at this level

## Management

- Same as level-1 & 2 and/or treatment mentioned at this level
- **Recommended Diet and Lifestyle:** Same as level 1

In patients with Class III Obesity (*Siman Mufrit*), two or more of the following forms of medications may be given along with recommended diet and lifestyle:

**Table 10: Single drugs and Compound Formulations at Level 3**

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)	Precaution/contraindication
1.	<i>Zīra Siyāh</i> ( <i>Carum carvi</i> L.) <sup>8</sup>	Powder	3-5 gms	Morning empty stomach	40 days	water	NS
2.	<i>Mur Makkī</i> ( <i>Commiphora myrrha</i> Engl.) <sup>7</sup>	Powder	3-5 gms	Morning empty stomach	15 days to 1 month	vinegar	NS
3.	<i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.) <sup>7</sup>	Powder	3-5 gms	Morning empty stomach	15 days to 1 month	water	NS
4.	<i>Tukhm-i-Karafs</i> ( <i>Apium graveolens</i> L.) <sup>7</sup>	Powder	3-5 gms	Morning empty stomach	15 days to 1 month	water	NS
5.	<i>Luk Maghsūl</i> (Lac)-28 gms, <i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.)-3.5gm, <i>Zīra Siyāh</i> ( <i>Carum carvi</i> L.), <i>Nānkhwāh</i> ( <i>Trachyspermum ammi</i> L. Spragne), <i>Sudāb</i> ( <i>Ruta graveolens</i> L.), <i>Bādiyān</i> ( <i>Foeniculum vulgare</i> Mill.)-14 gms each, <i>Būra Armanī</i> ( <i>Armenian earth</i> )-3.5gms <sup>8</sup>	Powder	4.5 gms	Morning	15 days to 1 month	'Araq -i-Zeera Siyah	NS

S. No.	Drug	Dosage Form	Dose (per day)	Time of Administration	Duration	Badraqa (Vehicle)	Precaution/contraindication
6.	<i>Luk Maghsūl</i> (Lac) soaked in decoction of <i>Rewand Khatāi</i> ( <i>Rheum emodi</i> Wall.) followed by drying. This process is repeated seven times and the resultant product is powdered again. <sup>8</sup>	Powder	5-7 gm	After meal	15 days to 1 month	water	NS
7.	<i>Luk Maghsūl</i> (Lac), <i>Nānkhwāh</i> ( <i>Trachyspermum ammi</i> L.Spragne), <i>Tukhm-i-Sudāb</i> ( <i>Ruta graveolens</i> L.), <i>Zīra Siyāh</i> ( <i>Carum carvi</i> L.)- 1 part each, <i>Marzanjosh</i> ( <i>Origanum vulgare</i> L.), <i>Būra Armanī</i> (Armenian earth)- 1/4 part each <sup>8</sup>	Powder	4.5 gms	Morning	15 days to 1 month	water	NS
8.	<i>Sufūf-i-Mohazzil</i> <sup>36</sup>	Powder	5 - 10 gm	Morning empty stomach	15 days to 1 month	water	NS
9.	<i>Dawā al- Kurkum</i> <sup>7,8,43</sup>	Semi-Solid	2-3 gm in two divided doses	After meal	15 days to 1 month	water	NS
10.	<i>Itrīfal Saghīr</i> <sup>7,8,9,10</sup>	Semi-Solid	10-25 gm in two divided doses	Bed time	15 days to 1 month	water	NS
11.	<i>Jawārish Kamūnī</i> <sup>7,8,9,10</sup>	Semi-Solid	10-15 gm in two divided doses	After meal	15 days to 1 month	water	Diabetes Mellitus Type I&II
12.	<i>Jawārish Falāfil</i> <sup>8,43</sup>	Semi-Solid	5-10 gms	After meal	15 days to 1 month	water	Diabetes Mellitus Type I&II

### ‘Ilāj bi’l Tadbīr (Regimenal Therapy)

#### Riyāḍat (Exercise)

- *Riyāḍat Kathīra* (exercise for a prolonged duration)<sup>7,8,9,10</sup>

#### Dalk (Massage)

- *Dalk* (massage) with *Roghan Shibit* and *Roghan Qust*<sup>7,8,9,10</sup>

### ***Ḥammām* (Therapeutic Bath)**

- *Ḥammām Muḥallil* (Bath causing resolution of fluids) <sup>7,8,9,10</sup>

### **Recommended diet and lifestyle:**

- Same as Level 1& 2

### **Restricted diet and lifestyle:**

- Same as Level 1& 2

### **Follow up**

- 15 days or as recommended by the physician

### **Referral Criteria<sup>38</sup>**

- Same as mentioned earlier at Level 2, with:
- 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. 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>.
3. 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).
4. 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>.
5. 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>.
6. 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.
7. Ibn Sīnā, 1417 H., *al-Qānūn fi'l-Tibb*, Vol. IV, Jamia Hamdard, New Delhi, p.437.
8. Khān M A, 1906, *Iksir-i A'zam*, Vol. IV, Matba' Nāmī Munshī Naval Kishor, Lucknow, pp. 575-578.
9. Ibn Hubal, 1364 H., *Kitāb al-Mukhtārāt fi'l-Tibb*, Vol. IV, Dā'ira al-Ma'ārif al-Usmāniyya, Hyderabad, pp. 154-156.
10. Kabiruddin M. *Tarjama-i-Kabīr*, Vol.IV. Hyderabad, Hikmat Book Depot; n.d.p.72.
11. 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/>.
12. 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>.
13. Burrige K, Christensen SM, Golden A, Ingersoll AB, Tondt J, Bays HE. Obesity history, physical exam, laboratory, body composition, and energy expenditure: an Obesity Medicine Association (OMA) Clinical Practice Statement (CPS) 2022. *Obes Pillars*. 2022 Jan 10;1:100007. doi: 10.1016/j.obpill.2021.100007. PMID: 37990700; PMCID: PMC10661987. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10661987/>.
14. 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/>.
15. 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/>.
16. Kalra S, Kapoor N, Verma M, Shaikh S, Das S, Jacob J, Sahay R. Defining and diagnosing obesity in India: a call for advocacy and action. *J Obes*. 2023;2023:4178121. Available from: <https://www.hindawi.com/journals/job/2023/4178121>.
17. 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/>.
18. 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>

19. 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>
20. 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/>
21. Thaker VV. Genetic and epigenetic causes of obesity. *Adolesc Med State Art Rev*. 2017 Fall;28(2):379-405. PMID: 30416642; PMCID: PMC6226269.
22. 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/>
23. 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/>
24. 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>.
25. 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/>
26. 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/>
27. 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>.
28. 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/>
29. 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>.
30. 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.
31. 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](https://www.researchgate.net/publication/236934339_Metabolic_alterations_following_visceral_fat_removal_and_expansion_Beyond_anatomic_location).
32. 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/>
33. Branscum P, Sharma M. After-School Based Obesity Prevention Interventions: A Comprehensive Review of the Literature. *International Journal of Environmental Research and Public Health*. 2012; 9(4):1438-1457. <https://doi.org/10.3390/ijerph9041438>
34. Smith AB, Jones CD. Evaluation and Management of Obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. *Harrison's Principles of Internal Medicine*. 14th ed. New York: McGraw-Hill, Health Professions Division; 1998. P.11243
35. Tiwari A, Daley SF, Balasundaram P. Obesity in pediatric patients. [Updated 2023 Mar 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK570626/>

36. Morarji Desai Institute of Yoga. Yogic management of obesity. Dolphin Printo-Graphics; p. 4-7. Available from: <https://yoga.ayush.gov.in/Publications/gallery/PUBLICATION/Obesity.pdf>
37. Smith AB, Jones CD. Treatment of Obesity. In: Smith AB, Jones CD. Evaluation and Management of Obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. Harrison's Principles of Internal Medicine. 14th ed. New York: McGraw-Hill, Health Professions Division; 1998.p.11249
38. 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.
39. Yurista S, Eder R, Feeley M, et al. A closer look at ACC/AHA and ESC guidelines for managing obesity and overweight in adults. JACC Adv. 2023 Sep;2(7). Available from: <https://www.jacc.org/doi/10.1016/j.jacadv.2023.100570>
40. DGI\_07th\_May\_2024\_fin. [Internet]. 2024. Available from: [https://main.icmr.nic.in/sites/default/files/upload\\_documents/DGI\\_07th\\_May\\_2024\\_fin.pdf](https://main.icmr.nic.in/sites/default/files/upload_documents/DGI_07th_May_2024_fin.pdf)
41. Kim BY, Choi DH, Jung CH, Kang SK, Mok JO, Kim CH. Obesity and physical activity. J Obes Metab Syndr. 2017 Mar;26(1):15-22. doi: 10.7570/jomes.2017.26.1.15. Epub 2017 Mar 30. PMID: 31089489; PMCID: PMC6484923.
42. Smith AB, Jones CD. Treatment of obesity. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, editors. Harrison's Principles of Internal Medicine. 14th ed. New York: McGraw-Hill, Health Professions Division; 1998. p. 11255.
43. Anonymous. National Formulary of Unani Medicine (NFUM), Vol. I 1<sup>st</sup> Edn. Department of AYUSH, Ministry of Health & Family Welfare, Govt. of India, New Delhi, 2006, p. 239,100, 88,89,99,221.
44. Anonymous. National Formulary of Unani Medicine (NFUM), Vol. V, Department of AYUSH, Ministry of Health & Family Welfare, Govt. of India, New Delhi, 2008, p. 138.
45. Papadakis MA, McPhee SJ, Rabow MW, McQuaid KR (2022). Current Medical Diagnosis & Treatment (CMDT), 61<sup>st</sup> edition, McGraw Hill. San Francisco, pp. 1268-1274.



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