

Fatty Liver Disease through the Lens of Unani Medicine: Conceptual Convergence with MASLD, Clinical Evidence and a Research Agenda

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ABSTRACT

Background: Fatty liver disease, now termed metabolic dysfunction-associated steatotic liver disease (MASLD), affects approximately one in three adults worldwide, and in India, drug options are narrow and largely restricted to advanced steatohepatitis. Unani (Greco-Arabic) medicine places the liver at the center of digestion and humoral balance; however, how its concepts relate to current pathophysiology has rarely been examined critically.

Objective: To summarize the Unani understanding of fatty liver, test its convergence with modern models of MASLD, appraise clinical evidence for Unani-associated interventions, and define the research gaps.

Methods: Narrative review of classical Unani sources (via translations and secondary reviews) and contemporary literature on MASLD and Unani or related traditional interventions, prioritizing randomized trials, meta-analyses, and guidelines.

Results: Classical texts do not describe MASLD as an entity; the closest correlates are Su-e-Mizaj Kabid Barid, Waram-e-Kabid Balghami, and Sudda-e-Kabid. Mapping these into current models suggests convergence on overnutrition and sedentary etiology, hepatic insulin resistance, dyslipidemia, impaired lipid export, and inflammatory injury, and on lifestyle-first treatment; the mapping is analogical and untested. Clinical evidence is strongest for *Nigella sativa* (Kalonji), with small randomized trials and meta-analyses showing lower transaminases and ultrasonographic steatosis, but marked heterogeneity; evidence for chicory, turmeric, licorice, and other single drugs is smaller or indirect, and for compound formulations rests on uncontrolled reports. Trials are short, small, ultrasound-based, rarely standardized, and not stratified by Mizaj. Some formulations contain toxic botanicals (e.g., *Aristolochia*).

Conclusions: Unani medicine offers a coherent, lifestyle-oriented framework with testable mechanistic claims. Progress requires case definitions anchored to MASLD criteria, validated Mizaj instruments, standardized and safety-screened preparations, and adequately powered trials with imaging or histological endpoints.

Keywords: *Unani medicine; metabolic dysfunction-associated steatotic liver disease; non-alcoholic fatty liver disease; Su-e-Mizaj; Nigella sativa; Cichorium intybus; traditional medicine; research gaps*

How to cite this article: Habibullah V^{1*} Mubasheera Begum² Nazia Tabasum² Sayeeda Begum³ Mohammed Waseem⁴ Aysha Farheen S F². Fatty Liver Disease through the Lens of Unani Medicine: Conceptual Convergence with MASLD, Clinical Evidence and a Research Agenda. *Int J Drug Deliv Technol.* 2026;16(86s):2101-2112. DOI: 10.25258/ijddt.16.86s.190.

INTRODUCTION

Fatty liver disease is the most common chronic liver condition worldwide (Wang et al., 2026). In 2023, a multisociety Delphi process replaced the term non-alcoholic fatty liver disease (NAFLD) with metabolic dysfunction-associated steatotic liver disease (MASLD), defined as hepatic steatosis accompanied by at least one of five cardiometabolic risk factors,

and renamed non-alcoholic steatohepatitis as metabolic dysfunction-associated steatohepatitis (MASH) (Ghany et al., 2023). A global meta-analysis estimated the prevalence of NAFLD at approximately 30%, rising over three decades. In India, the pooled adult prevalence was estimated at 38.6%, with 28.1% in average-risk and 52.8% in high-risk groups, and

roughly one in three children affected. The disease is not confined to the liver; it is entangled with obesity, type 2 diabetes, dyslipidemia, and cardiovascular risk, and a subset progresses through steatohepatitis and fibrosis to cirrhosis and hepatocellular carcinoma (Chen et al., 2026).

Management is anchored in sustained lifestyle change with weight loss, dietary modification, and exercise as first-line measures. Two drugs have recently gained regulatory approval in the United States for MASH with moderate-to-advanced fibrosis, resmetirom in 2024 and semaglutide in 2025, but neither addresses simple steatosis or early disease, and cost and access may limit their reach in many settings. Lifestyle programs are effective when weight loss is achieved, yet are difficult to sustain (Tacke et al., 2024). This therapeutic gap explains persistent patient and clinician interest in traditional systems of medicine.

Unani medicine, the Greco-Arabic humoral system practiced widely in South Asia, treats the liver as the principal organ governing digestion, nourishment, and the generation of humors (Hasan et al., 2025). Although no classical text describes fatty liver disease, Unani physicians have described hepatic conditions with overlapping features, and two recent reviews have mapped fatty liver disease onto Su-e-Mizaj Kabid Barid and related constructs (Mehraj et al., 2026). These reviews were largely descriptive. They do not ask whether the Unani constructs have mechanistic content that can be tested, they do not grade the clinical evidence, and they give little attention to safety, standardization, or trial design.

This review has four aims: (i) to summarize the Unani understanding of fatty liver in its own terms; (ii) to examine, critically and with explicit hypotheses, where that understanding converges with and diverges from current models of MASLD; (iii) to appraise the clinical evidence for Unani-associated interventions with attention to study design; and (iv) to propose a research agenda that could make Unani hypotheses falsifiable.

Methods

This is a narrative review reported with reference to the SANRA quality criteria. The work proceeded in three steps. First, classical Unani concepts of hepatic temperament (Mizaj), hepatic faculties (Quwa), obstruction (Sudda), humors (Akhlat), and treatment principles (Ilaj) were compiled from published translations of classical texts and secondary reviews. Second, PubMed/MEDLINE, Scopus, and Google Scholar were searched for literature published up to September 2026, combining terms for fatty liver (fatty liver, NAFLD, MASLD, MASH, steatohepatitis, steatosis) with terms for Unani and related traditional interventions (Unani, Tashahhum-

e-Kabid, Su-e-Mizaj, Ilaj, Nigella sativa, Cichorium intybus, Curcuma longa, Glycyrrhiza glabra, Artemisia absinthium, herbal medicine). Third, current guidelines and pivotal trials for MASLD were reviewed to anchor the modern side of the comparison. Randomized controlled trials, systematic reviews, and meta-analyses, and clinical practice guidelines were given priority; case reports and uncontrolled studies are reported but explicitly labeled as low-level evidence.

Fatty liver in contemporary medicine

Nomenclature and burden

Under the 2023 consensus, steatotic liver disease is an umbrella term. MASLD requires hepatic steatosis plus at least one cardiometabolic criterion; MetALD describes MASLD with higher alcohol intake (140–350 g/week for women and 210–420 g/week for men); and cryptogenic steatotic liver disease applies when no metabolic or other cause is found (Ghany et al., 2023a). The consensus emphasized non-stigmatizing language and positive diagnostic criteria for BPD. For Unani research, the practical consequence is that studies should enrol participants using explicit MASLD criteria rather than ultrasound appearance alone so that results can be interpreted alongside the wider literature.

Pathogenesis

The current understanding has moved from a “two-hit” model to a multiple-hit model. Insulin resistance in adipose tissue increases free fatty acid flux to the liver, while hyperinsulinemia and dietary sugars drive hepatic de novo lipogenesis; fatty acid oxidation and export of triglyceride as very-low-density lipoprotein (VLDL) fail to keep pace. Unesterified lipid species are lipotoxic, and together with oxidative and endoplasmic reticulum stress, adipokine imbalance, gut-derived endotoxins, and cytokines such as TNF- α , they promote hepatocellular injury, inflammation, and stellate cell activation, with genetic modifiers such as PNPLA3 shaping susceptibility (Buzzetti et al., 2016). This metabolic-inflammatory model is the natural counterpart against which Unani constructs should be compared.

Current management

European guidelines recommend stepwise case-finding with FIB-4 followed by elastography in people with cardiometabolic risk, lifestyle modification as the foundation of care, and incretin-based therapy where indicated for type 2 diabetes or obesity. Weight loss shows a graded relationship with histological improvement, with steatosis responding at lower thresholds and fibrosis requiring more substantial loss. Resmetirom, a thyroid hormone receptor- β agonist, achieved MASH resolution in approximately 26–30% of treated patients with F2–F3 fibrosis versus approximately 10% on placebo in

the MAESTRO-NASH trial (Tacke et al., 2024). In part 1 of the ESSENCE trial, semaglutide 2.4 mg weekly produced resolution of steatohepatitis without worsening fibrosis in approximately 63% of participants versus 34% on placebo at 72 weeks and improved fibrosis in approximately 37% versus 22% (Sanyal et al., 2025). No pharmacotherapy is currently recommended specifically for the cirrhotic stage. Therefore, the unmet need lies mainly at the earlier, larger end of the spectrum, where lifestyle changes are the only established option.

Fatty liver in the Unani framework

Is there an Unani “fatty liver”?

Classical texts do not describe an entity equivalent to MASLD. The conditions that most closely resemble it are Su-e-Mizaj Kabid Barid (a cold derangement of hepatic temperament), Waram-e-Kabid Balghami (phlegmatic inflammation of the liver), and Sudda-e-Kabid (hepatic obstruction). Contemporary Unani authors have adopted the term Ghair Khamri Tashahhum-e-Kabid for non-alcoholic fatty liver and have placed NAFLD under Su-e-Mizaj Kabid Barid on the basis of symptom overlap (Raza et al., 2026). It is important to note that these are correlations drawn by modern authors, not classical diagnoses, and different authors weigh the constructs differently.

Hepatic physiology, temperament and faculties

In Unani physiology, the liver is a vital organ (Uzw-e-Raeesa), the seat of the natural faculty (Quwwat-e-Tabaiyya), and the site where digested food is converted to blood and humors. Its normal temperament is hot and moist. Four intrinsic faculties govern hepatic function: Quwwat-e-Jazibah (attraction of nutrients), Quwwat-e-Hazimah (digestion or concoction), Quwwat-e-Masikah (retention), and Quwwat-e-Dafiah (expulsion). Weakness of the attractive and retentive faculties compromises nourishment; impaired digestive faculty produces corrupted matter (Fasad); and weakness of the expulsive faculty leaves the residue of food (Saqaal-e-Ghiza) lodged in the organ, deranging its temperament and leading to accumulation of morbid matter (Mawad-e-Fasida) (Rasool et al., 2025). In the Unani scheme of digestion, the stage that occurs in the liver follows gastric digestion; therefore, failure in earlier stages naturally burdens the liver.

Etiological concepts

The classical causes of cold hepatic derangement include eating before the previous meal is digested, drinking cold water immediately after exercise, bathing or intercourse, sedentary habits with excessive rest and sleep, and heavy, viscous, or fatty diets. Obesity (Saman-e-Mufrit, with bodily congestion or Imtila) is said to diminish innate heat (Hararat-e-Ghariziya) and overload the liver, promoting fatty deposition. Accumulation of cold,

moist, and viscous Balgham, particularly when digestion is weak, obstructs the pores and channels of the liver (Masamat, Mujari, Uruq) and impedes the flow of humors and vital spirit, a state called Sudda. Recent reviews have described an excess of Balgham together with Sauda in NAFLD (Hasan et al., 2025a). Figure 1 sets this classical sequence next to the contemporary multiple-hit model. The horizontal links in this figure represent the correspondences we are proposing; however, none of them have been demonstrated.

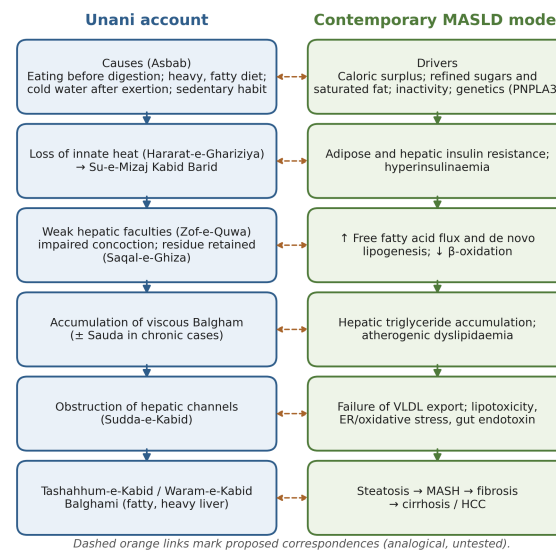


Figure 1. Parallel accounts of fatty liver pathogenesis. Left, the Unani sequence from causes (Asbab) through cold hepatic derangement, weakened faculties, Balgham accumulation, and Sudda to Tashahhum-e-Kabid; right, the contemporary MASLD model from metabolic drivers to steatosis, MASH, and fibrosis. Dashed lines indicate proposed, untested correspondences. HCC, hepatocellular carcinoma; MASH, metabolic dysfunction-associated steatohepatitis; VLDL, very-low-density lipoprotein.

Clinical assessment in the classical tradition

Classical diagnosis relies on history, symptoms and inspection of the pulse (Nabz), urine (Baul) and stool (Baraz). For a cold hepatic temperament, Jurjani describes a slow, weak pulse and pale, watery urine, and heaviness or pain in the right hypochondrium, poor appetite, dyspepsia and lethargy are reported as accompanying features. These features are non-specific and, to our knowledge, have not been validated against imaging, elastography or biochemical markers. Any Unani study of fatty liver should therefore establish the diagnosis with contemporary tools and treat Unani clinical signs as exploratory variables.

Principles of treatment

Treatment aims to restore temperament (Tadeel-e-Mizaj), dissolve obstruction (Taftteh-e-Sudda), evacuate morbid matter according to its site, and strengthen the liver and its faculties (Taqwiyat-e-Kabid). Therapy is organized in three modes: dietotherapy (Ilaj bil Ghiza), regimenal therapy (Ilaj bit Tadbeer), and pharmacotherapy (Ilaj bil Dawa). Dietary advice favors light, easily digestible foods and discourages oily, spicy, fried, and heavy preparations as well as overeating and prolonged fasting. Regimenal therapy includes exercise (Riyazat), with Galen and later authorities recommending brisk walking to reduce body mass and maintain hepatic health. Drug therapy follows the principle of opposites (Ilaj bil Zid) using deobstruents (Mufattihat), diuretics (Mudirrat), purgatives (Mushilat), resolvents (Muhallilat), warming agents (Musakhkhinat), and liver tonics, with the route of evacuation chosen by the site of pathology (Raza et al., 2026).

Mechanistic convergence: where the frameworks meet, and where they do not

Points of convergence

Three areas of convergence stand out. The first is etiology: the classical emphasis on eating before digestion, heavy and viscous diets, inactivity, and obesity corresponds closely to the modern risk profile of overnutrition, high intake of refined carbohydrates and saturated fat, physical inactivity, and adiposity (Ahmed & Mohammed, 2025). The second is the metabolic core. The picture of a liver that is “cooled,” sluggish, and overloaded, with weak expulsion of residue, is compatible in general terms with hepatic insulin resistance, reduced fatty acid oxidation, and impaired lipid export. At the same time, “Balgham” as a cold, moist, viscous humor associated with obesity resembles a surplus-energy, adipose-expanded, dyslipidemic state (Matsuzaka & Shimano, 2011). The third is therapeutic principle. Ilaj bil Ghiza and Riyazat align with the diet-and-exercise foundation of current guidelines, and the emphasis on liver-directed drugs with antioxidant, anti-inflammatory, and lipid-modulating activity parallels modern pharmacological targets. Table 1 and Figure 2 set out a working map.

Table 1. Working map between Unani constructs for fatty liver, candidate modern correlates, and testable readouts. The correlates are hypotheses for testing, not established equivalences.

Unani construct	Classical description	Candidate modern correlate	Testable readout
<i>Su-e-Mizaj Kabid Barid</i> (cold-moist hepatic derangement) (Hasan et al., 2025a)	Normally hot-moist liver cooled by irregular eating, cold water after exertion, inactivity	Metabolic slowing; hepatic insulin resistance, reduced fatty acid oxidation, sedentary energy surplus	HOMA-IR, fasting insulin, CAP or MRI-PDFF, activity level
<i>Fasad-e-Hazm</i> (faulty digestion or concoction) (IJAABR, 2025)	Eating before digestion of prior meal; heavy diet; <i>Saqal-e-Ghiza</i> retained	Overnutrition, fructose-rich and processed diet, de novo lipogenesis, gut dysbiosis and endotoxemia	Dietary intake, fasting TG, microbiome and endotoxin markers
<i>Ghalba-e-Balgham / Waram-e-Kabid Balghami</i> (excess phlegm) (Sosin et al., 2025)	Cold, moist, viscous humor accumulating when digestion is weak	Adipose expansion, dyslipidemia, low-grade inflammation	Waist circumference, BMI, TG/HDL-C, hs-CRP
<i>Sudda-e-Kabid</i> (hepatic obstruction) (Firdaus & Ali, 2016)	Viscous <i>Balgham</i> lodges in <i>Masamat</i> , <i>Mujari</i> and <i>Uruq</i> , obstructing humors and spirit	Impaired VLDL export, sinusoidal and biliary changes, progression toward fibrosis	ALP, GGT, liver stiffness (kPa), FIB-4
<i>Zof-e-Quwa</i> (weak faculties: <i>Jazibah</i> , <i>Hazimah</i> , <i>Masikah</i> , <i>Dafiah</i>) (Siddiqui & Azmi, 2022)	Failure to attract, digest, retain or expel nutrient and residue	Imbalance of hepatic fatty acid uptake, synthesis, oxidation and export	Exploratory lipidomic or tracer studies

Abbreviations: ALP, alkaline phosphatase; CAP, controlled attenuation parameter; ELF, enhanced liver fibrosis; GGT, gamma-glutamyl transferase; HOMA-IR, homeostasis model assessment of insulin resistance; MRI-PDFF, MRI proton density fat fraction; TG, triglycerides; VLDL, very-low-density lipoprotein.

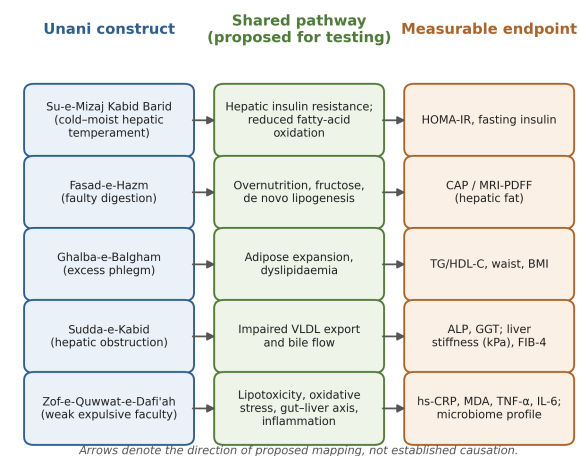


Figure 2. Conceptual convergence map. Left: Unani constructs; center: shared pathways proposed for testing; right: measurable endpoints. Arrows indicate the direction of the proposed mapping, not the established causation.

Where the analogy breaks

Convergence should not be overstated. First, Unani constructs are qualitative and humoral, whereas

modern pathophysiology is quantitative and molecular; a mapping such as Balgham to adipose expansion is a heuristic and could be adjusted after the fact to fit almost any finding (Miraj & Kiani, 2016). Second, MASLD is heterogeneous. Lean MASLD, a genetically driven disease and one dominated by type 2 diabetes, may not all fit a cold-moist derangement, and a framework that assigns every fatty liver to Su-e-Mizaj Kabid Barid would be unfalsifiable (Habib, 2024). Third, some classical dietary and drug recommendations are framed by temperament rather than metabolic effects and need to be reconciled with current nutritional and toxicological evidence before being carried into trials. Fourth, current Unani reviews are largely secondary syntheses of classical texts and do not themselves generate empirical evidence on whether the proposed correlates hold.

Testable hypotheses

Convergence becomes scientifically useful only if it yields predictions that could be incorrect. We suggest four. We suggest the following four hypotheses: **H1:** In adults meeting MASLD criteria, a Barid-Ratab Mizaj phenotype, measured with a validated instrument, will be associated with higher HOMA-IR and hepatic fat fraction than other phenotypes after adjustment for BMI and age. **H2:** Response to a standardized program of Ilaj bil Ghiza plus Riyazat will differ by Mizaj phenotype, independently of weight change. **H3:** Mufattih and Mudirr formulations will alter markers of the gut-liver axis and lipid export (for example, VLDL-triglyceride, bile acid profile, microbiome composition) in a manner that mediates their effect on hepatic fat. **H4:** Clinical markers of Sudda (heaviness, appetite, urine character) will track elastography or FIB-4 more closely than chance. If H1 and H2 fail, Mizaj-based stratification would lose its empirical rationale for this condition, which would itself be an informative result.

Therapeutic evidence

Dietotherapy and regimenal therapy (Ilaj bil Ghiza and Ilaj bit Tadbeer)

In the literature retrieved for this review, we did not identify controlled trials that tested classical regimenal procedures such as cupping, venesection, bathing, or massage specifically in MASLD, and no trial that evaluated Unani dietotherapy as a packaged intervention against a standard lifestyle program. This is a notable gap because these are the components of Unani practice that map most directly onto guideline-endorsed care. Their plausibility rests on modern evidence for diet, exercise, and weight loss: lifestyle-induced weight reduction improves steatosis and, at higher magnitudes, steatohepatitis and fibrosis. A pragmatic Unani lifestyle intervention

(light, easily digestible diet; avoidance of overeating and meal stacking; regular brisk walking) is thus likely to be beneficial, and the research question is whether the Unani formulation adds adherence, acceptability, or effect beyond conventional counselling.

Pharmacotherapy (Ilaj bil Dawa): single drugs

Nigella sativa (Kalonji). This is the best-studied Unani-associated single drug in fatty liver disease. In a placebo-controlled trial in Pakistan of 70 patients with NAFLD, 1 g twice daily for 12 weeks lowered ALT from 78.1 to 52.6 IU/L and AST from 65.5 to 44.6 IU/L, and 57% of treated patients had a normal ultrasound grade at follow-up, with concurrent reductions in body weight and BMI. A double-blind trial of standardized seed oil (2.5 mL every 12 hours for 12 weeks) in 120 patients reported reductions in aminotransferases, triglycerides, LDL-cholesterol, and steatosis grade, with a rise in HDL-cholesterol. A smaller trial of 44 patients over 8 weeks reported lower transaminases, inflammatory markers (hs-CRP, TNF- α , IL-6), lipids, and fasting glucose, without a significant change in GGT. A meta-analysis of eight randomized trials, of which four were in NAFLD, found a significant reduction in AST (weighted mean difference -8.11 IU/L) but a borderline, non-significant change in ALT (-7.26 IU/L, $p = 0.051$), with very high heterogeneity (I^2 96–98%). A second meta-analysis of four NAFLD trials (224 patients) reported improvements in aminotransferases, LDL-cholesterol, triglycerides, and HDL-cholesterol without a significant effect on BMI. Taken together the signal is consistent in direction, but the trials are small, short, largely ultrasound-based, drawn from a few countries, and use different preparations (powder, capsule, oil) (Hormati et al., 2019).

Cichorium intybus (Kasni) and turmeric. Chicory is a cornerstone of Unani hepatic formulations, such as Arq Kasni and Sikanjabeen Bazoori. In a four-arm randomized trial of 92 patients with NAFLD, chicory seed infusion (9 g/day), turmeric (3 g/day), their combination, or placebo for 12 weeks, chicory alone and in combination reduced BMI and waist circumference, the combination lowered ALP, and turmeric raised HDL-cholesterol and lowered TG/HDL and LDL/HDL ratios; effects on transaminases were not a headline finding. Mechanistic support for chicory comes from cell and animal work in which seed extract modulated expression of SREBP-1c and PPAR α in steatotic hepatocytes and diabetic rats, but this remains preclinical. For turmeric, meta-analyses disagree on details: one pooled 16 trials (1,028 participants) found improved ultrasonographic severity (RR 3.52) and steatosis resolution (RR 3.96) with reductions in AST and ALT, whereas another pooled 15 trials (835

participants) found a reduction in AST but not ALT, high heterogeneity, no added benefit of piperine, and a negative relationship between trial duration or relative dose and effect (Maleki et al., 2023).

Glycyrrhiza glabra (Asl-us-Soos, licorice), Artemisia absinthium (Afsanteen), cinnamon (Darchini), and Rosa damascena (Gul-e-Surkh). In a double-blind trial of 60 women with NAFLD, licorice root (1,000 mg/day for 12 weeks) added to lifestyle advice improved ALT, insulin, insulin resistance, malondialdehyde, and ultrasonographic steatosis compared with placebo. A randomized single-blind study of Afsanteen reported that it relieved the subjective and objective features of fatty liver without adverse effects, although we accessed it only through a secondary summary. Randomized trials of cinnamon and Rosa damascena in NAFLD reported improvements in liver enzymes and, for cinnamon, in lipids, insulin resistance, and hs-CRP. Experimental data, chiefly in rodents, suggest that *Solanum nigrum* (Mako) and saffron may reduce hepatic inflammation, oxidative stress, and lipogenesis; however, these do not yet justify clinical claims (Rostamizadeh et al., 2022).

A benchmark botanical. Silymarin, although not a classical Unani drug, provides a useful yardstick for what a well-studied hepatoprotective botanical can achieve. A meta-analysis of 26 randomized trials (2,375 patients) reported reductions in ALT, AST, lipids, and insulin, and improved histological steatosis (OR 3.25, 95% CI 1.80–5.87), while acknowledging heterogeneity. Unani single drugs have not yet reached this level of evidence.

Compound formulations and clinical reports

Compound formulations are the mainstay of Unani prescribing, and their evidence base is thin. In an open-label study of 23 patients with central obesity and raised liver enzymes, Jawarish Bisbasa (7 g/day for 8 weeks) lowered SGOT, SGPT, and ALP relative to baseline (AZHAR et al., 2023). A case report described improvement in FibroScan findings in a 60-year-old woman with FibroScan-confirmed steatohepatitis treated with Qurs-e-Zarishk, Arq Mako, and Arq Kasni (Hussain et al., 2025). A clinical case series reported lower shear wave elastography values in NAFLD patients treated with Unani regimens. These reports are hypothesis-generating. They cannot separate treatment effect from lifestyle change, regression to the mean, or natural fluctuation. Table 2 summarizes the clinical evidence, and Figure 3 shows how thin it still is: no single trial of a Unani-associated drug in fatty liver has enrolled more than 120 participants.

Table 2. Summary of clinical evidence for Unani-associated interventions in fatty liver disease (NAFLD/MASLD) discussed in this review.

Intervention (Unani name)	Design and population	Dose and duration	Principal findings	Main limitations
<i>Nigella sativa</i> powder (Kalonji)	Placebo-controlled RCT, n = 70, NAFLD, Pakistan	1 g twice daily, 12 wk	Lower ALT, AST, weight, BMI; 57% normal ultrasound grade	Single center; ultrasound endpoint
<i>N. sativa</i> seed oil	Double-blind RCT, n = 120, NAFLD, Iran	2.5 mL every 12 h, 12 wk	Lower ALT, AST, TG, LDL-C, steatosis grade; higher HDL-C	Short duration; ultrasound endpoint
<i>N. sativa</i> oil capsule	RCT, n = 44, NAFLD, Iran	1 g/day, 8 wk	Lower ALT, AST, hs-CRP, TNF- α , IL-6, lipids, glucose; GGT unchanged	Small; short
<i>N. sativa</i> (pooled)	Meta-analyses of RCTs	Various, 6–12 wk	AST reduced; ALT borderline (8 RCTs); improved liver enzymes and lipids (4 NAFLD RCTs)	Very high heterogeneity; few NAFLD trials
<i>Cichorium intybus</i> seed (Kasni) $\pm$ turmeric	Four-arm RCT, n = 92, NAFLD, Iran	Chicory 9 g/day, turmeric 3 g/day, 12 wk	Lower BMI, waist; lower ALP (combination); better lipid ratios	No clear transaminase effect; short
<i>Curcuma longa</i> (turmeric)	Meta-analyses, 15–16 RCTs	Various	Steatosis resolution and AST improved; ALT effect inconsistent	Heterogeneity; formulation and dose differences
<i>Glycyrrhiza glabra</i> (Asl-us-Soos)	Double-blind RCT, n = 60 women, NAFLD	1 g/day, 12 wk, plus diet	Lower ALT, insulin, HOMA-IR, MDA; less ultrasonographic steatosis	Women only; safety monitoring needed
<i>Artemisia absinthium</i> (Afsanteen)	Single-blind RCT	Not extracted	Reported symptomatic and objective improvement	Accessed via secondary summary; single-blind
Jawarish Bisbasa (compound)	Open-label, n = 23, central obesity with raised enzymes	7 g/day, 8 wk	Lower SGOT, SGPT, ALP vs baseline	No control; NAFLD not confirmed
Qurs-e-Zarishk, Arq Mako, Arq Kasni; Unani regimens	Case report and case series	Various	Improved elastography findings	Uncontrolled; anecdotal

RCT, randomized controlled trial. The findings are as reported in the cited studies; however, the effect sizes are not directly comparable across rows.

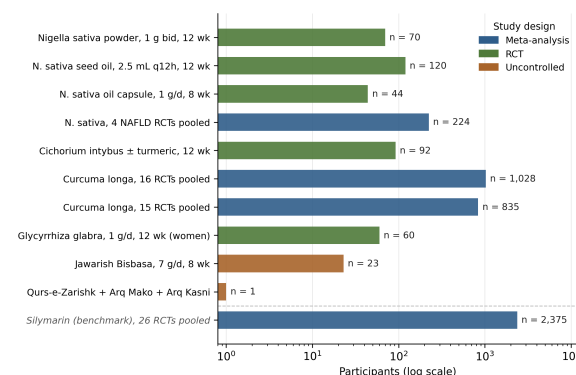


Figure 3. Evidence map of Unani-associated interventions in fatty liver, by study design and number of participants (log scale). Pooled analyses are shown as the total number of participants across trials. Silymarin, which is not a classical Unani drug, is included below the dashed line as a benchmark. Data are taken from the studies cited in the text and Table 2.

Safety and standardization

Hepatoprotective claims are meaningless if the preparations are unsafe. Herbal and dietary supplements are a recognized cause of drug-induced liver injury. Several botanicals listed in recent Unani reviews of fatty liver warrant explicit safety screening. *Aristolochia* species (Zarawand), which appear in classical compound formulations and in the fatty-liver literature, contain aristolochic acids, which

are associated with nephropathy and urothelial cancer; trial protocols should exclude or substitute them. *Artemisia absinthium* contains thujone, a neurotoxic constituent at high doses; licorice can cause hypertension and hypokalemia with prolonged use through mineralocorticoid-like effects; and long-term purgative use may cause fluid and electrolyte disturbance. Interactions with metformin, statins, antihypertensives, and incretin therapies have not been studied. Preparations in the trials above vary from whole powder to standardized oil to aqueous infusion, which limits comparability. For Unani compound formulations, pharmacopeial standardization, chemical fingerprinting, and testing for adulterants and heavy metals should be prerequisites for clinical testing.

Research gaps and a proposed agenda

Table 3 consolidates the gaps identified above and proposes concrete responses, and Figure 4 arranges them as a phased program because some steps (a case definition, a validated Mizaj tool, standardized drugs) must be in place before any trial result can be interpreted. Two of these gaps deserve further emphasis. First, Mizaj is central to Unani diagnosis and prescription, yet few validated instruments exist. Questionnaires for Mizaj and Su-e-Mizaj have been developed and shown to have acceptable content validity and reliability in small samples, but with limited construct and criterion validity, and expert agreement on Mizaj identification is imperfect. Without a measurable Mizaj phenotype, H1 and H2 cannot be tested. Second, the quality of trials must be improved. Reporting should follow CONSORT and its herbal extension, and outcomes should move beyond ultrasound grade and transaminases to CAP or MRI-PDFF for steatosis, elastography for fibrosis, and histology in a subset of patients.

Table 3. Priority research gaps and proposed actions

Gap	Why it matters	Proposed action
Case definition	No classical entity equals MASLD; authors map constructs differently	Delphi consensus among Unani physicians; enrol by MASLD criteria; define operational criteria for <i>Su-e-Mizaj Kabid Barid</i>
Mizaj measurement	Few validated tools; imperfect inter-rater agreement	Validate instruments in MASLD cohorts; report inter-rater reliability; pre-specify Mizaj

		subgroups
Trial design and endpoints	Small, short, often unblinded, ultrasound-based trials	Adequately powered RCTs of at least 24 weeks; CAP or MRI-PDFF and elastography; histology subset; lifestyle and active comparators; CONSORT with herbal extension
Standardization	Variable powders, oils, infusions and compounds	Pharmacopeial specification, chemical fingerprinting, batch documentation, heavy-metal and adulterant testing
Safety	Aristolochia, thujone, glycyrrhizin; drug-induced liver injury; interactions	Exclude or substitute hazardous botanicals; monitor liver, renal, blood pressure and potassium; formal pharmacovigilance; study herb–drug interactions
Mechanism	Convergence map is untested	Lipidomic, metabolomic and microbiome studies; network pharmacology; preclinical high-fat-diet models with classical formulations
Regimenal therapy	No trials of <i>Riyazat</i> , <i>Hammam</i> or other <i>Tadbeer</i> components in MASLD	Pragmatic trials of Unani lifestyle packages against standard counselling; measure adherence and acceptability
Integration and outcomes	Unknown place relative to approved	Comparative-effectiveness and add-on designs; long-term follow-

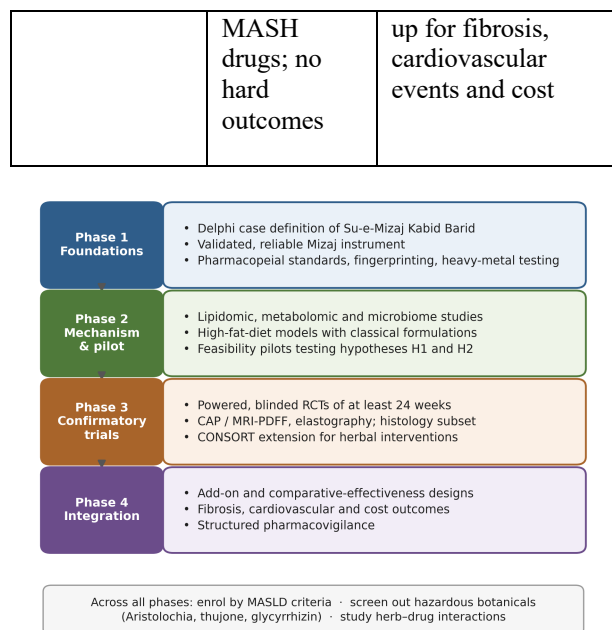


Figure 4. Proposed phased research roadmap for Unani approaches to MASLD. Each phase builds on the outputs of the one before: safety screening, MASLD enrolment criteria, and herb–drug interaction studies apply throughout. CAP, controlled attenuation parameter; MRI-PDF, magnetic resonance imaging proton density fat fraction; RCT, randomized controlled trial.

Limitations of this review

This is a narrative rather than a systematic review; therefore, selection bias is possible, and the literature search, though structured, was not exhaustive or pre-registered. Classical Unani statements were taken from published translations and secondary reviews rather than primary Arabic or Persian manuscripts, and translations vary. Several clinical studies were accessed as abstracts or through secondary summaries, and effect sizes are reported as presented by the original authors without independent re-analysis. Evidence from Iranian and Pakistani trials of traditional preparations may not transfer directly to Indian Unani practice. Finally, the convergence map proposed here is a hypothesis-generating framework and should not be read as evidence that Unani constructs are mechanistically validated.

Conclusions

Unani medicine offers a coherent, lifestyle-oriented framework for a condition that current medicine manages mainly through lifestyle changes. Its etiological emphasis on diet, digestion, inactivity, and obesity, and its therapeutic emphasis on diet, exercise, and liver-directed botanicals, converge with contemporary understanding of MASLD at the level of principle. Mechanistic claims that go beyond this, particularly the role of Mizaj and Sudda, have not

been tested. Clinical evidence is most encouraging, though still modest and heterogeneous, for *Nigella sativa*, with weaker support for chicory, turmeric, licorice, and other single drugs, and minimal controlled evidence for compound formulations and regimenal therapy. Safety and standardization deserve the same priority as efficacy. If Unani researchers adopt MASLD criteria, validated Mizaj instruments, standardized and safety-screened preparations, and adequately powered trials with imaging endpoints, the tradition's hypotheses can be tested on equal terms and either supported or set aside.

Declarations

Funding: This review received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest: The authors declare no conflicts of interest.

Ethics approval: Not applicable; this review used only published literature.

Author contributions (CRediT): Habibullah V: Conceptualization, Methodology, Investigation, Visualization, Writing – original draft, Project administration. Mubasheera Begum: Conceptualization, Validation, Supervision, Writing – review & editing. Nazia Tabasum: Investigation, Data curation, Writing – review & editing. Sayeeda Begum: Investigation, Validation, Writing – review & editing. Mohammed Waseem: Investigation, Resources, Writing – review & editing. Aysha Farheen S F: Data curation, Visualization, Writing – original draft. All authors read and approved the final manuscript and agree to be accountable for its content.

Use of generative AI: An AI-based tool (Claude, Anthropic) was used only for language correction and editing of the manuscript; the authors reviewed all content and took full responsibility for the work.

Data availability: No new datasets were generated or analyzed in this study.

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