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# Fatty Liver Disease and Its Ayurvedic Management: A Scientific and Integrative Narrative Review

**Dachewar Archana[1], Danga SK[2]**

1. Professor, Dept of Kayachikitsa, Shri Ayurved Mahavidyalaya, Nagpur.
2. Professor, Dept of Kayachikitsa, Jupiter Ayurved Medical College, Nagpur.

**Abstract:**

Fatty liver disease now ranks among the most common chronic liver conditions seen worldwide. What was once called non-alcoholic fatty liver disease has since been reclassified as metabolic dysfunction-associated steatotic liver disease (MASLD) — a renaming that better captures its tight connection to obesity, insulin resistance, type 2 diabetes, dyslipidaemia, and other cardiometabolic disturbances. MASLD spans a wide spectrum, ranging from uncomplicated hepatic steatosis at one end to metabolic dysfunction-associated steatohepatitis, advancing fibrosis, cirrhosis, and hepatocellular carcinoma at the other. Its underlying pathogenesis is driven by multiple interacting processes: insulin resistance, a greater influx of free fatty acids into the liver, heightened de novo lipogenesis, mitochondrial dysfunction, oxidative stress, endoplasmic-reticulum stress, inflammatory signaling, and disruptions along the gut-liver axis.While Ayurvedic classics do not describe a disease entity that maps precisely onto MASLD, the condition can reasonably be interpreted through concepts such as Agnimandya, Ama, Medovaha Srotodushti, Meda Dhatu Vriddhi, Santarpanajanya Vyadhi, and the progressive involvement of Kapha, Pitta, Rakta, and Vata. In its earliest form, steatosis appears to align mainly with Kapha-Meda accumulation, while the inflammatory phase that follows tends to draw in Pitta, Rakta, and Ama. The fibrotic changes seen in more advanced disease can be understood as chronic Srotorodha, ongoing tissue damage, and secondary Vata involvement.An Ayurvedic approach to management should center on Nidana Parivarjana, restoring Agni, clearing Ama and excess Meda, correcting the underlying metabolic disturbances, and protecting liver tissue. Depending on the patient's constitution and how advanced the disease is, this may involve dietary changes, physical activity, weight loss, adequate sleep, and selected Deepana-Pachana, Lekhana, Medohara, Srotoshodhana, and Rasayana interventions. Early clinical studies of certain Ayurvedic treatments have produced encouraging results, though this evidence base is still held back by small sample sizes, brief follow-up periods, inconsistent formulations, and outcome measures that are not standardized. For this reason, Ayurvedic care is best applied alongside proper metabolic assessment, fibrosis risk stratification, and sustained long-term clinical monitoring.

**Keywords:** Fatty liver, MASLD, NAFLD, Agnimandya, Ama, Santarpanajanya Vyadhi, Yakrit.

**Corresponding Author:**

**Dr.Danga S.K.**

Professor, Dept of Kayachikitsa, Jupiter Ayurved Medical College, Nagpur.

Email: sunder147@gmail.com

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**Introduction:**

As a central hub of metabolism, the liver governs glucose balance, lipid processing, protein synthesis, bile production, detoxification, and immune regulation. When triglycerides build up excessively within liver cells, the result is hepatic steatosis — commonly known as fatty liver. When this steatosis appears alongside cardiometabolic dysfunction, current terminology labels the condition metabolic dysfunction-associated steatotic liver disease.

Under the revised international classification system, "steatotic liver disease" now serves as the umbrella term. A diagnosis of MASLD requires the presence of hepatic steatosis together with at least one cardiometabolic risk factor, once alcohol intake and other potential causes of steatosis have been ruled out. Metabolic dysfunction-associated steatohepatitis (MASH) has taken the place of the older label "non-alcoholic steatohepatitis" and describes steatosis accompanied by liver cell injury and inflammation.

Far from being confined to the liver alone, MASLD is closely intertwined with abdominal obesity, insulin resistance, type 2 diabetes, hypertension, dyslipidaemia, cardiovascular disease, chronic kidney disease, and obstructive sleep apnoea. Global estimates now suggest that roughly one in three adults may be living with metabolically driven steatotic liver disease. The picture in India is no different — a systematic review and meta-analysis put the pooled adult prevalence at approximately 38.6%, climbing even higher among people with diabetes, obesity, or other metabolic risk factors. This rising prevalence tracks closely with shifting dietary patterns, declining physical activity, greater consumption of refined carbohydrates and sugar-sweetened drinks, central obesity, poor sleep, and psychosocial stress. Recognizing its public-health weight, India has folded fatty liver prevention and management into its national non-communicable disease programme. Ayurveda brings a personalized, whole-systems lens to metabolic disorders of this kind. Rather than treating fatty liver as an isolated problem of fat deposition in one organ, Ayurvedic assessment looks at the status of Agni, the extent of Ama formation, the quality and quantity of Meda Dhatu, dietary and activity patterns, bowel function, individual constitution, coexisting conditions, and the functional state of the relevant Srotas. Building an evidence-informed bridge between modern hepatology and Ayurveda in this way could meaningfully strengthen comprehensive prevention and supportive care.

**Contemporary Scientific Understanding of Fatty Liver:**

**Disease spectrum:**

The presence of hepatic fat alone is termed steatosis. In a proportion of patients, steatosis is accompanied by inflammation and hepatocellular ballooning, resulting in MASH. Persistent injury may activate hepatic stellate cells and increase extracellular-matrix deposition, producing fibrosis. Progressive fibrosis can eventually result in cirrhosis, portal hypertension, hepatic decompensation and hepatocellular carcinoma. Fibrosis stage is the most important histological predictor of liver-related and overall mortality. Mortality risk increases progressively from the absence of fibrosis to advanced fibrosis and cirrhosis. Therefore, clinical evaluation should not stop after reporting “Grade I” or “Grade II fatty liver” on ultrasonography; assessment of fibrosis risk is essential.

**Major risk factors:**

Central obesity, type 2 diabetes, insulin resistance, elevated triglycerides, low HDL cholesterol, hypertension, and metabolic syndrome stand out as the leading risk factors for this condition. A number of other contributors also play a role, including polycystic ovarian syndrome, hypothyroidism, obstructive sleep apnoea, a sedentary lifestyle, insufficient sleep, heavy intake of refined carbohydrates, fructose-laden drinks, ultra-processed foods, and certain medications.

Notably, MASLD isn't confined to people with a high body mass index — it can develop in those with a normal BMI as well. This so-called "lean MASLD" carries particular relevance for Asian populations, where visceral fat accumulation and insulin resistance tend to emerge at comparatively lower body weights. A normal weight, in other words, offers no guarantee against significant steatosis or fibrosis. Alcohol intake needs to be assessed carefully, since metabolic dysfunction and alcohol-related liver damage can occur together rather than as mutually exclusive processes. Depending on the clinical picture, other secondary causes of hepatic steatosis — viral hepatitis, autoimmune

liver disease, Wilson disease, drug-induced liver injury, and severe malnutrition among them — also warrant consideration.

**Insulin Resistance and the Influx of Free Fatty Acids**

Under normal conditions, insulin keeps lipolysis in adipose tissue in check. Once insulin resistance sets in, this braking effect weakens, allowing more free fatty acids to spill into the bloodstream. From there, these fatty acids travel to the liver, where they are re-esterified into triglycerides. At the same time, both hyperinsulinaemia and excess carbohydrate consumption push the liver toward de novo lipogenesis, acting through transcription factors like sterol regulatory element-binding protein-1c and carbohydrate-responsive element-binding protein. The net effect is that the liver keeps manufacturing new fatty acids even while hepatocytes are already awash in lipid excess.

**Lipotoxicity and Mitochondrial Dysfunction**

Storing triglycerides may initially serve a protective purpose, converting potentially harmful free fatty acids into a form that is comparatively inert. As the disease advances, however, the bigger driver becomes the buildup of toxic lipid intermediates — diacylglycerols, ceramides, free cholesterol, and saturated fatty acids. These lipotoxic compounds interfere with mitochondrial function, raise reactive oxygen species, hamper ATP production, and place hepatocytes under considerable stress. The oxidative damage that follows harms cellular proteins, membranes, and even mitochondrial DNA, compounding the underlying metabolic disturbance further.

**Inflammation and Fibrosis**

Injured hepatocytes release danger signals that switch on Kupffer cells, inflammasomes, and broader inflammatory pathways. Cytokines such as tumour necrosis factor-alpha and various interleukins add to hepatocellular injury while drawing in more inflammatory cells. Sustained inflammation, in turn, activates hepatic stellate cells, converting them into collagen-producing, myofibroblast-like cells. As extracellular matrix continues to accumulate, the liver's normal architecture becomes distorted, giving rise to fibrosis. Taken together, the interplay between insulin resistance, lipotoxicity, mitochondrial stress, inflammation, and fibrogenesis explains why MASLD is best understood as a multisystem metabolic disorder rather than a case of simple, passive fat storage.

**The Gut-Liver Axis**

Because the liver receives blood directly from the gastrointestinal tract via the portal circulation, disturbances there can have direct hepatic consequences. Intestinal dysbiosis, altered bile-acid handling, and heightened gut permeability may all expose the liver to bacterial byproducts and inflammatory metabolites, fueling hepatic inflammation, insulin resistance, and fibrogenesis. That said, using probiotics or other microbiome-targeted products as routine clinical treatment still lacks sufficiently strong evidence, and such measures should not be substituted for established lifestyle and metabolic management.

**Clinical Features and Diagnostic Evaluation:**

Most people with early-stage MASLD remain entirely asymptomatic, with the condition often picked up incidentally through an ultrasound scan or abnormal liver enzyme results. Some patients do report fatigue, a reduced tolerance for exercise, dyspepsia, a sense of abdominal heaviness, or mild discomfort under the right ribs, and hepatomegaly is occasionally found on examination. While serum ALT and AST levels may be mildly raised, normal enzyme values by no means rule out MASH or advanced fibrosis. A typical laboratory workup covers liver enzymes, bilirubin, albumin, complete blood count, fasting glucose, HbA1c, lipid profile, renal function, and testing to exclude other causes of liver disease.

Ultrasonography remains a widely used first step for detecting steatosis, though it has limited sensitivity for mild fat accumulation and cannot reliably gauge either inflammation or the stage of fibrosis. More detailed information can come from vibration-controlled transient elastography, MRI-based techniques, and serum fibrosis markers.

For an initial fibrosis risk assessment, clinicians commonly rely on the FIB-4 index, calculated from age, AST, ALT, and platelet count. In most adults, a FIB-4 score under 1.3 points to a relatively low likelihood of advanced fibrosis, though a higher threshold applies for those over 65. Patients whose score comes back elevated or indeterminate typically need a secondary workup — transient elastography, enhanced liver fibrosis testing, or referral to a specialist. Liver biopsy still stands as the reference standard for distinguishing simple steatosis from steatohepatitis and for accurately staging

fibrosis, but given its invasive nature, it's reserved for select cases where the diagnosis or treatment decision remains genuinely unclear.

**Ayurvedic Understanding and Correlation:**

**Role of Yakrit in Ayurveda;**

In Ayurveda, *Yakrit* is regarded as an important organ associated with *Rakta Dhatu* and *Raktavaha Srotas*. *Charaka Samhita* describes the liver and spleen as roots of the *Raktavaha Srotas*. The liver also has a close functional relationship with digestion, transformation of nutrients, blood formation and *Pitta*.However, fatty liver should not automatically be labelled as *Yakritodara*. *Yakritodara* primarily describes clinically appreciable enlargement and abdominal manifestations involving the liver. A patient with ultrasonographic steatosis without hepatomegaly or abdominal enlargement does not necessarily fulfil that description.A more appropriate Ayurvedic understanding may be developed from the combined concepts of:

- *Agnimandya* and impaired metabolic transformation;
- *Ama* formation;
- *Santarpanajanya Vyadhi*;
- *Meda Dhatu Vriddhi* or *Medodushti*;
- *Medovaha Srotodushti*;
- *Kapha–Meda Avarana* and *Srotorodha*;
- secondary involvement of *Pitta*, *Rakta* and *Vata* during inflammation and fibrosis.

**Santarpanajanya nature of the disease:**

Excessive consumption of sweet, oily, heavy and unctuous food, frequent eating, lack of exercise and daytime sleep are described as causes of excessive nourishment and *Kapha–Meda* aggravation. These factors closely resemble modern lifestyle-related determinants of obesity, insulin resistance and hepatic steatosis.In susceptible individuals, chronic overnutrition can produce *Agnimandya*. Inadequately transformed nutritional material contributes to *Ama* and abnormal formation or accumulation of *Meda Dhatu*. When the capacity of *Medovaha Srotas* to transport and utilise lipid-related nutrients is impaired, pathological accumulation may occur in different tissues and organs, including the liver. Thus, early fatty liver can be interpreted as a *Santarpanajanya*, *Kapha–Meda-pradhana* metabolic disorder originating from impaired *Agni* and *Medovaha Srotas* function.

**Dosha-wise interpretation of disease progression:**

**Kapha and Meda predominance:**

*Kapha* and *Meda* share properties such as heaviness, unctuousness, stability and sluggishness. Excessive hepatic lipid accumulation, obesity, lethargy, heaviness, dyslipidaemia and sluggish metabolism may therefore be correlated with increased *Kapha* and pathological *Meda*.

**Pitta and Rakta involvement:**

The liver is functionally associated with *Ranjaka Pitta* and *Rakta*. When simple steatosis progresses to hepatocellular stress and inflammation, *Pitta* and *Rakta Dushti* may become more prominent. Clinically, this stage may be accompanied by increased liver enzymes, inflammatory activity, burning sensation, irritability, altered digestion or other *Pitta*-dominant manifestations, although these features are not present in every patient.

**Vata involvement:**

Long-standing inflammation, tissue degeneration and fibrosis may involve *Vata*, particularly when there is dryness, structural alteration, irregular metabolism, pain, weakness or progressive loss of normal organ function. This may be viewed as secondary *Vata Prakopa* resulting from chronic *Srotorodha*, tissue injury and depletion of healthy *Dhatus*. This stage-wise correlation is an interpretative model rather than a literal translation of histopathological categories into *Doshas*.

**Probable Samprapti:**

Chronic intake of *Guru*, *Snigdha*, *Madhura*, excessively processed or calorie-dense food, together with physical inactivity, daytime sleep and irregular eating, may aggravate *Kapha* and *Meda*. These factors impair *Jatharagni* and subsequently disturb *Dhatvagni*, particularly *Medodhatvagni*.Impaired transformation promotes *Ama* and inadequately processed lipid components. Accumulated *Kapha*, *Meda* and *Ama* obstruct the channels of metabolism and circulation. The liver, owing to its role in metabolic transformation and relationship with *Rakta* and *Pitta*, becomes a major site of pathological accumulation. Continued metabolic stress leads to *Pitta–Rakta* involvement and

inflammatory injury. Persistent *Srotorodha* and tissue damage may subsequently aggravate *Vata*, contributing to progressive structural changes and fibrosis.

**Samprapti Ghataka:**

**Dosha:** Predominantly *Kapha*, with associated *Pitta* and secondary *Vata* involvement.
**Dushya:** *Meda*, *Rasa* and *Rakta*; later involvement of other *Dhatus.*
**Agni:** *Jatharagni Mandya* and *Medodhatvagni Mandya.*
**Ama:** Commonly involved, particularly in patients with indigestion, heaviness and metabolic toxicity.
**Srotas:** *Annavaha*, *Rasavaha*, *Raktavaha* and *Medovaha Srotas.*
**Srotodushti:** *Sanga*, abnormal accumulation and disturbed transformation.
**Udbhava Sthana:** *Amashaya* and gastrointestinal-metabolic system.
**Adhisthana:** *Yakrit*, with systemic metabolic involvement.
**Rogamarga:** *Abhyantara Rogamarga.*
**Nature:** Predominantly *Santarpanajanya* and *Chirakari.*

**Principles of Ayurvedic Management:**

Ayurvedic treatment should be individualised according to *Prakriti*, *Vikriti*, strength of the patient, digestive capacity, obesity, diabetes, fibrosis risk, bowel pattern and associated diseases. The principal objectives are:

1. Removal of causative dietary and lifestyle factors;
2. Improvement of *Agni*;
3. Digestion or reduction of *Ama*;
4. Correction of *Kapha–Meda* accumulation;
5. Restoration of *Srotas* function;
6. Regulation of glucose and lipid metabolism;
7. Protection of liver tissue;
8. Prevention of progression to inflammation and fibrosis.

**Nidana Parivarjana:**

Avoidance of causative factors is the foundation of management. Treatment is unlikely to be successful when calorie excess, refined carbohydrates, alcohol, physical inactivity and irregular sleep continue unchanged.Patients should reduce or avoid sugar-sweetened beverages, sweets, deep-fried foods, bakery products, refined flour, frequent snacking, excessive saturated fat, ultra-processed foods and large late-night meals. Alcohol should preferably be avoided, particularly in patients with elevated liver enzymes, MASH, fibrosis or concurrent medication use. Daytime sleep, prolonged sitting and eating before digestion of the previous meal should be discouraged. Correction of these factors corresponds closely with contemporary strategies for reducing caloric excess and improving insulin sensitivity.

**Ahara Chikitsa:**

Dietary intervention should aim to create a sustainable energy deficit in overweight patients while preserving adequate protein, micronutrients and dietary fibre. Extreme fasting, crash diets and nutritionally deficient regimens should be avoided. Foods that may be selected according to digestive capacity include barley, millets, green gram, vegetables, leafy vegetables, pulses, fibre-rich grains and preparations with bitter, pungent and astringent predominance. Lightly prepared meals are preferable to heavily fried or oily foods.

The following practical principles may be adopted:

- Emphasise vegetables, legumes, whole grains and appropriately selected fruits;
- Prefer freshly prepared food over packaged and ultra-processed products;
- Reduce refined rice or flour portions where excessive;
- Restrict sweets, sweetened drinks and concentrated fruit juices;
- Use oils and ghee in controlled quantities rather than complete elimination;
- Avoid repeated reheating of oils;
- Ensure adequate protein through pulses, legumes, dairy where suitable, eggs or other sources according to dietary preference;
- Take dinner earlier and keep it lighter than the principal daytime meal;
- Maintain a consistent meal schedule and avoid continuous grazing.

**Vihara and physical activity:**

Regular physical activity directly reduces liver fat and improves insulin sensitivity, even when major weight reduction does not occur. Aerobic activity and resistance training are both beneficial. A practical target is at least 150 minutes per week of moderate-intensity activity, progressively adjusted to the person's age, fitness and comorbidities. Brisk walking, cycling, swimming,

strength training and appropriately supervised Yoga may be incorporated. Prolonged sitting should be interrupted every 30–60 minutes with brief movement. Useful Yoga practices may include *Tadasana*, *Trikonasana*, *Ardha Matsyendrasana*, *Bhujangasana*, *Dhanurasana*, *Pavanamuktasana*, *Mandukasana* and selected *Surya Namaskara* sequences. Practices should be modified in patients with severe obesity, hypertension, hernia, musculoskeletal disease, pregnancy or advanced liver disease. Breathing exercises and relaxation practices may improve stress regulation and support adherence to dietary and physical-activity programmes. However, Yoga should complement rather than replace aerobic and resistance exercise.

**Deepana and Pachana:**

Where *Agnimandya* and *Ama* are evident, appropriately selected *Deepana–Pachana* measures may be used before stronger *Shodhana* or *Medohara* treatment. The selection should depend on *Dosha*, acidity, bowel pattern, comorbidities and tolerance. Drugs traditionally considered for this purpose include *Shunthi*, *Pippali*, *Maricha*, *Chitraka*, *Musta*, *Ajmoda* and *Hingu*. These are not suitable for every patient. Strong pungent drugs may worsen hyperacidity, gastritis or *Pitta*-dominant symptoms and should therefore be prescribed judiciously.

**Rukshana, Lekhana and Medohara approach:**

In *Kapha–Meda*-dominant patients with good strength, *Rukshana*, *Lekhana* and *Medohara* principles may help counter excessive heaviness, unctuousness and metabolic stagnation. These principles can be implemented through diet, physical activity, dry powder massage in suitable patients and selected medicines. *Udvartana* may be used as an adjunct in obesity management, although it should not be presented as a direct treatment for hepatic fibrosis. Its potential value lies in encouraging activity, reducing subjective heaviness and supporting a broader weight-management programme.

**Shodhana therapy:**

*Shodhana* must not be routinely advised solely on the basis of an ultrasonography report. It requires assessment of *Dosha*, *Bala*, *Agni*, age, comorbidities and fibrosis status. *Mild Virechana* may be considered in selected *Kapha–Pitta*, *Meda* and *Rakta*-associated presentations under qualified supervision. Its Ayurvedic rationale includes regulation of *Pitta*, correction of the gastrointestinal–hepatic pathway and removal of accumulated *Dosha* through the lower route. *Vamana* is not universally indicated and may be unsuitable in elderly, weak, hypertensive, cardiovascular or advanced liver-disease patients. *Basti* may be considered when obesity, metabolic dysfunction or secondary *Vata* involvement is prominent, but direct clinical evidence for fibrosis reversal remains inadequate. Panchakarma procedures should never be undertaken as unsupervised "liver detoxification." In advanced fibrosis, cirrhosis, thrombocytopenia, ascites, jaundice or significant comorbidity, strong purification procedures may be hazardous and require specialist assessment.

**Ayurvedic drugs:**

**Sharapunkha:**

*Sharapunkha* (*Tephrosia purpurea*) is traditionally used in disorders involving the liver and spleen. It is described as possessing properties relevant to *Kapha*, *Pitta*, swelling and abdominal disorders. A randomised placebo-controlled clinical study of Sharapunkhadi powder, containing *Sharapunkha*, *Bhumyamalaki* and *Katuki*, reported greater improvement when the formulation was combined with lifestyle modification than with lifestyle measures alone. The study was relatively small and relied substantially on ultrasonographic grading; therefore, the findings require confirmation through larger trials with validated fibrosis outcomes.

**Bhumyamalaki:**

*Bhumyamalaki*, generally identified with *Phyllanthus* species, is widely used in traditional management of hepatic and *Pitta–Rakta*-related disorders. Preclinical research suggests antioxidant, anti-inflammatory and hepatoprotective actions. However, clinical findings are not uniformly positive. A one-year randomised, double-blind, placebo-controlled trial of *Phyllanthus niruri* in fatty liver did not demonstrate a significant reduction in controlled attenuation parameter or liver-enzyme levels. This finding illustrates that traditional hepatoprotective use and preclinical activity do not automatically establish clinical effectiveness in MASLD.

**Katuki:**

*Katuki* (*Picrorhiza kurroa*) is traditionally considered bitter, *Pitta–Kapha Shamaka* and useful in impaired digestion, hepatic disorders and metabolic accumulation. Experimental studies suggest effects on oxidative stress,

inflammation and lipid metabolism. Nevertheless, robust long-term human trials in MASLD are limited. Excessive or inappropriate use may produce gastrointestinal intolerance or purgation.

**Kalmegha:**

*Kalmegha* (*Andrographis paniculata*) is bitter and traditionally used in disorders associated with *Pitta*, digestion and liver dysfunction. Experimental evidence suggests anti-inflammatory and antioxidant activity, but high-quality clinical evidence for meaningful fibrosis improvement is insufficient.

**Guduchi:**

*Guduchi* (*Tinospora cordifolia*) is described as *Deepana*, *Rasayana* and *Tridoshaghna* in appropriate contexts. It may be considered where metabolic inflammation and reduced tissue resilience coexist. However, cases of herb-associated liver injury have been reported with certain herbal products, and species authentication, manufacturing quality and individual susceptibility are important. It should not be presumed that every preparation marketed as "natural" is automatically safe.

**Punarnava:**

*Punarnava* (*Boerhavia diffusa*) may be selected when swelling, fluid retention or *Kapha*-dominant features are present. Its role in uncomplicated hepatic steatosis is supportive rather than disease-specific, and it should not replace assessment for advanced liver or renal disease.

**Haritaki and Triphala:**

*Haritaki* and *Triphala* may support bowel regulation, *Anulomana*, digestion and metabolic correction in selected patients. Preclinical findings suggest possible effects on lipid and glucose metabolism, but direct evidence for reversing clinically significant hepatic fibrosis remains inadequate.

**Haridra:**

*Haridra* or turmeric contains curcuminoids with antioxidant and anti-inflammatory properties. Several small randomised trials have reported improvements in hepatic steatosis, lipid parameters or liver enzymes with curcumin preparations. Nevertheless, heterogeneity in formulation, bioavailability, dosage and study quality prevents routine generalisation. Concentrated curcumin products, especially enhanced-bioavailability formulations, may interact with medicines and have occasionally been associated with liver injury.

**Monitoring and Integrative Clinical Management:**

An integrative management plan should document baseline body weight, waist circumference, blood pressure, glucose status, HbA1c, lipid profile, liver enzymes, platelet count and fibrosis risk. Ultrasonographic improvement alone should not be treated as sufficient evidence of disease resolution.

Follow-up may include:

- Body weight and waist circumference;
- Dietary and physical-activity adherence;
- Fasting glucose and HbA1c;
- Triglycerides and other lipid parameters;
- AST, ALT, GGT, bilirubin and albumin;
- Platelet count and repeated FIB-4 assessment;
- Transient elastography in selected patients;
- Assessment of alcohol intake and medication or supplement use.

Patients with diabetes, multiple metabolic risk factors or previously elevated fibrosis markers require more frequent reassessment. Patients with suspected advanced fibrosis, cirrhosis, persistent elevation of liver enzymes, thrombocytopenia, jaundice, ascites or splenomegaly should be referred to a hepatologist. Current hepatology guidance recommends sequential use of non-invasive tests and specialist-directed therapy for patients at significant risk of MASH with moderate or advanced fibrosis.

**Discussion:**

Rather than a simple case of triglycerides piling up in one organ, fatty liver disease functions as a metabolic condition with effects reaching well beyond the liver itself. Insulin resistance and dysfunction within adipose tissue drive more fatty acids toward the liver, while hyperinsulinaemia and excess carbohydrate exposure push the liver into de novo lipogenesis. Whether simple steatosis eventually progresses into MASH and fibrosis comes down to the combined influence of lipotoxicity, oxidative stress, mitochondrial dysfunction, and inflammatory signalling.

Ayurvedic concepts such as Agnimandya, Ama, Meda Dhatu Vriddhi, Medovaha Srotodushti, and

Santarpanajanya Vyadhi offer a coherent way to frame the metabolic background of this disease. A stage-oriented interpretive model emerges when Kapha is linked to heaviness and pathological accumulation, Pitta-Rakta to hepatic transformation and inflammation, and Vata to chronic structural degeneration in the later stages. Even so, this correlation demands careful handling — MASLD is defined through measurable cardiometabolic and hepatic criteria, whereas an Ayurvedic diagnosis rests on Dosha, Dushya, Agni, Ama, constitution, and clinical presentation. Two patients showing an identical ultrasonographic grade may carry entirely different Dosha patterns, digestive capacity, and comorbidities, and consequently may need distinctly different Ayurvedic strategies.

Where Ayurveda and modern hepatology find their strongest common ground is lifestyle correction. Both traditions recognize the damage done by overnutrition, physical inactivity, poor sleep, and progressive weight gain. Concepts like Nidana Parivarjana, Pathya Ahara, regular physical activity, and restoring proper metabolic function line up naturally with modern strategies of calorie control, cutting back refined carbohydrates, structured exercise, and cardiometabolic risk management.

That said, the published clinical evidence behind specific Ayurvedic interventions remains at an early stage. The Sharapunkhadi trial hints that a selected polyherbal therapy may add benefit on top of lifestyle changes alone, though its modest sample size, short duration, and reliance on ultrasonography limit what can be concluded about MASH resolution or fibrosis regression. By contrast, the negative one-year trial of Phyllanthus niruri serves as a useful reminder of why neutral findings need to be published just as much as positive ones. Many herbal studies rely on liver enzymes, subjective symptoms, or ultrasonographic grading as their outcome measures — informative, but insufficient to reliably confirm improvement in inflammation or fibrosis. Future trials would do better to incorporate controlled attenuation parameter, liver stiffness measurement, validated serum fibrosis markers, MRI-based fat quantification, or histological endpoints wherever ethically feasible. Ayurvedic research also needs sharper botanical authentication, tighter chemical standardization, greater batch-to-batch consistency, more rigorous adverse-event reporting, and closer scrutiny of interactions with antidiabetic, lipid-lowering, antihypertensive, and anticoagulant medications. Trials should further work to separate the effect of the herbal intervention itself from the effects of concurrent calorie restriction, exercise, and weight loss.

Once advanced fibrosis is present, the management picture changes fundamentally. These patients require formal hepatology evaluation, ongoing surveillance for complications, and cautious selection of any Ayurvedic procedure — aggressive purgation, fasting, or unsupervised herbal combinations can be genuinely unsafe in the presence of cirrhosis, malnutrition, thrombocytopenia, or impaired hepatic synthetic function. The most defensible role for Ayurveda, then, is as an individualized, closely monitored, complementary system — one that reinforces dietary adherence, physical activity, digestive regulation, and metabolic correction, while steering clear of unsupported claims about guaranteed "detoxification" or fibrosis reversal.

**Conclusion**

Metabolic dysfunction-associated steatotic liver disease is a progressive metabolic disorder tied closely to obesity, diabetes, dyslipidaemia, and cardiovascular risk, with a pathology built on insulin resistance, lipid excess, lipotoxicity, oxidative stress, inflammation, and fibrosis. Seen through an Ayurvedic lens, the disease can be understood as a predominantly Santarpanajanya, Kapha-Meda-dominant disorder rooted in Agnimandya, Ama, and Medovaha Srotodushti, with Pitta-Rakta and eventually Vata becoming progressively involved. Management should center on Nidana Parivarjana, dietary correction, regular physical activity, gradual weight loss, and individualized measures aimed at restoring Agni and correcting Meda metabolism.

Certain Ayurvedic medicines and procedures may offer supportive value, but the current evidence base isn't strong enough to single out any one medicine as a universally effective treatment. Safe integration calls for authenticated medicines, qualified clinical supervision, metabolic control, non-invasive fibrosis assessment, and timely referral of high-risk patients to hepatology services. A collaborative, evidence-based approach remains the most rational path toward preventing disease progression and safeguarding long-term metabolic and hepatic health.

**References:**

1. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *Hepatology*. 2023;78(6):1966–1986.
2. Tacke F, Horn P, Wong VW, et al. EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease. *Journal of Hepatology*. 2024;81(3):492–542.
3. Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology*. 2023;77(5):1797–1835.
4. Riazi K, Azhari H, Charette JH, et al. The prevalence and incidence of NAFLD worldwide: A systematic review and meta-analysis. *Lancet Gastroenterology & Hepatology*. 2022;7(9):851–861.
5. Shalimar, Elhence A, Bansal B, Gupta H, Anand A, Singh TP, Goel A. Prevalence of non-alcoholic fatty liver disease in India: A systematic review and meta-analysis. *Journal of Clinical and Experimental Hepatology*. 2022;12(3):818–829.
6. Dulai PS, Singh S, Patel J, et al. Increased risk of mortality by fibrosis stage in nonalcoholic fatty liver disease: Systematic review and meta-analysis. *Hepatology*. 2017;65(5):1557–1565.
7. Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease. *Metabolism*. 2016;65(8):1038–1048.
8. Friedman SL, Neuschwander-Tetri BA, Rinella M, Sanyal AJ. Mechanisms of NAFLD development and therapeutic strategies. *Nature Medicine*. 2018;24(7):908–922.
9. Promrat K, Kleiner DE, Niemeier HM, et al. Randomized controlled trial testing the effects of weight loss on nonalcoholic steatohepatitis. *Hepatology*. 2010;51(1):121–129.
10. Vilar-Gomez E, Martinez-Perez Y, Calzadilla-Bertot L, et al. Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. *Gastroenterology*. 2015;149(2):367–378.e5.
11. Hallsworth K, Fattakhova G, Hollingsworth KG, et al. Resistance exercise reduces liver fat and its mediators in non-alcoholic fatty liver disease independent of weight loss. *Gut*. 2011;60(9):1278–1283.
12. Johnson NA, Sachinwalla T, Walton DW, et al. Aerobic exercise training reduces hepatic and visceral lipids in obese individuals without weight loss. *Hepatology*. 2009;50(4):1105–1112.
13. Ryan MC, Itsiopoulos C, Thodis T, et al. The Mediterranean diet improves hepatic steatosis and insulin sensitivity in individuals with non-alcoholic fatty liver disease. *Journal of Hepatology*. 2013;59(1):138–143.
14. Remya E, Goyal M, Varsakiya J. Efficacy of Sharapunkhadi powder and lifestyle modification in the management of nonalcoholic fatty liver disease: A randomized placebo-controlled clinical trial. *AYU*. 2020;41(2):98–106.
15. Abu Hassan MR, Hj Md Said R, Zainuddin Z, et al. Effects of one-year supplementation with *Phyllanthus niruri* on fibrosis score and metabolic markers in patients with non-alcoholic fatty liver disease: A randomized, double-blind, placebo-controlled trial. *Heliyon*. 2023;9(6):e16652.
16. Rahmani S, Asgary S, Askari G, et al. Treatment of non-alcoholic fatty liver disease with curcumin: A randomized placebo-controlled trial. *Phytotherapy Research*. 2016;30(9):1540–1548.
17. Panahi Y, Valizadegan G, Ahamdi N, et al. Curcuminoids plus piperine improve nonalcoholic fatty liver disease: A clinical trial. *Journal of Cellular Biochemistry*. 2019;120(9):15989–15996.
18. Philips CA, Paramaguru R, Joy AK, Antony KL, Augustine P. Clinical outcomes, histopathological patterns and chemical analysis of Ayurveda and herbal medicine-associated liver injury. *Journal of Clinical and Translational Hepatology*. 2020;8(3):263–274.
19. Ministry of Health and Family Welfare, Government of India. *Operational Guidelines for Integration of Non-Alcoholic*

*Fatty Liver Disease into the National Programme for Prevention and Control of Non-Communicable Diseases*. New Delhi: Government of India.

20. Agnivesha. *Charaka Samhita*, revised by Charaka and Dridhabala, Vimana Sthana, Srotovimana Adhyaya. Varanasi: Chaukhambha Publications.
21. Agnivesha. *Charaka Samhita*, Sutra Sthana, Ashtauninditiya Adhyaya. Varanasi: Chaukhambha Publications.
22. Agnivesha. *Charaka Samhita*, Chikitsa Sthana, Grahani Chikitsa Adhyaya. Varanasi: Chaukhambha Publications.
23. Sushruta. *Sushruta Samhita*, with Nibandhasangraha commentary of Dalhana. Varanasi: Chaukhambha Sanskrit Sansthan.
24. Vagbhata. *Ashtanga Hridaya*, with commentaries of Arunadatta and Hemadri. Varanasi: Chaukhambha Sanskrit Series.
25. Government of India, Ministry of AYUSH. *The Ayurvedic Pharmacopoeia of India*. New Delhi: Pharmacopoeia Commission for Indian Medicine and Homoeopathy.