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YAKRITODARA IN NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD): AN AYURVEDIC–MODERN CORRELATION AND INTEGRATIVE PERSPECTIVE

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD), now included under the broader term metabolic dysfunction-associated steatotic liver disease (MASLD), is a major global cause of chronic liver disease. It encompasses a spectrum ranging from simple hepatic steatosis to steatohepatitis, fibrosis, cirrhosis and hepatocellular carcinoma. Its development is strongly associated with obesity, visceral adiposity, insulin resistance, type 2 diabetes mellitus, dyslipidaemia and sedentary lifestyle. Altered lipid metabolism, lipotoxicity, oxidative stress, inflammation and gut–liver axis dysfunction further contribute to disease progression. The increasing burden of metabolic disorders in India makes NAFLD an important public health concern, including among individuals without overt obesity. Ayurveda does not describe NAFLD as a single disease entity corresponding directly to modern diagnostic criteria; however, concepts such as *Yakrit*, *Yakrit Vriddhi*, *Yakritodara*, *Medoroga*, *Sthaulya*, *Santarpana*, *Agnimandya* and *Āma* offer potentially relevant perspectives for understanding metabolic and hepatic disturbances. **Objective:** This review aimed to explore the possible correlation between NAFLD and *Yakritodara* by examining relevant Ayurvedic concepts of *Agni*, *Doṣa*, *Dhātu*, *Meda*, *Srotas* and *Āma*, while maintaining a clear distinction between classical

Ayurvedic descriptions and contemporary biomedical terminology. **Methods:** Classical Ayurvedic literature concerning *Yakrit*, *Yakritodara*, *Udara Roga*, *Medoroga*, *Sthaulya*, *Agni* and *Santarpana* was reviewed alongside contemporary literature from PubMed/MEDLINE and Scopus and relevant hepatology guidelines. **Results:** The review identified several areas of conceptual convergence. Excessive nutritional intake and physical inactivity may be interpreted through *Santarpana* and *Avyayama*, while *Kapha Prakopa* and *Meda Dhātu Dushti* provide a theoretical framework for excessive accumulation and disturbed lipid metabolism. *Agnimandya* may be considered in relation to impaired metabolic transformation, whereas *Āma* may represent a broader Ayurvedic concept for the pathological consequences of disturbed metabolism. In patients with hepatic enlargement, *Yakrit Vriddhi* and *Yakritodara* may provide a clinically relevant framework for interpretation. *Yakritodara* represents a broader description of hepatic enlargement and is not defined according to modern biochemical, imaging or histopathological criteria. **Conclusion:** *Yakritodara* may provide a useful Ayurvedic clinical perspective for understanding hepatomegaly associated with metabolic liver dysfunction, while *Santarpana*, *Agnimandya*, *Kapha–Meda Dushti*, *Āma* and *Srotodushti* offer complementary perspectives on its possible pathogenesis. Nevertheless, NAFLD should not be considered synonymous with *Yakritodara*. Future integrative research should combine standardized hepatic and metabolic investigations with systematic assessment of Ayurvedic parameters to determine whether these proposed correlations have reproducible clinical and biological significance.

KEYWORDS: Non-alcoholic fatty liver disease, metabolic dysfunction-associated steatotic liver disease, MASLD, MASH, *Yakritodara*, *Yakrit Vriddhi*, *Medoroga*, *Agnimandya*, Ayurveda, hepatomegaly, metabolic syndrome.

INTRODUCTION

Non-alcoholic fatty liver disease has become one of the major causes of chronic liver disease globally. Contemporary terminology has recently evolved from NAFLD to metabolic dysfunction-associated steatotic liver disease (MASLD). The change was introduced because the former terminology was based largely on exclusion of alcohol and did not adequately communicate the metabolic basis of the disorder. Under the new nomenclature, steatotic liver disease represents the overarching category, while MASLD identifies hepatic steatosis occurring in association with at least one cardiometabolic risk factor. MASH replaces the former term NASH.[1,2] The global burden of fatty liver disease is substantial. A recent meta-

analysis involving more than 78 million individuals from 38 countries estimated the global prevalence of NAFLD at approximately 30.2%. The prevalence was approximately 30.9% in Asia, demonstrating the particularly important burden in Asian populations.[3] India represents an important setting for MASLD research because metabolic disease is increasing rapidly. A systematic review and meta-analysis involving more than 23,000 Indian adults estimated a pooled NAFLD prevalence of 38.6%, with substantially higher prevalence among individuals with metabolic risk factors.[4] South Asian populations may also develop metabolic liver disease at comparatively lower levels of generalized obesity, making central adiposity, insulin resistance and metabolic phenotype particularly relevant.[5]

The pathophysiology of MASLD is complex and involves insulin resistance, increased hepatic fatty-acid delivery, de novo lipogenesis, impaired fatty-acid oxidation, lipotoxicity, mitochondrial dysfunction, oxidative stress, hepatocellular injury, inflammatory signalling, fibrosis and gut–liver axis disturbances.[6-9]

Ayurveda provides a fundamentally different conceptual model. Instead of defining disease exclusively through anatomical or biochemical abnormalities, Ayurveda evaluates disturbances of *Doṣa*, *Agni*, *Dhātu*, *Srotas* and *Mala*, together with dietary and behavioural causative factors. Liver pathology is described through several scattered references rather than as a single unified disease chapter. The liver is identified as *Yakrit*, and pathological enlargement of the organ is described within the framework of *Yakritodara* and related conditions. Classical descriptions place *Yakritodara* closely with *Plihodara*, particularly because of similarities in etiological factors, symptomatology and management.[10-13]

Consequently, the relationship between MASLD and *Yakritodara* is clinically interesting but requires methodological caution. The purpose of this review is not to claim that ancient texts specifically described modern MASLD, but to determine whether the Ayurvedic constructs can provide a meaningful explanatory framework for metabolic fatty liver disease.

Ayurvedic Pharmacotherapy in Yakritodara: Classical management of *Yakritodara* and *Kapha–Meda* predominant pathologies incorporates phytotherapeutic agents known for *Yakrit-Rakshaka* (hepatoprotective), *Deepana-Pācana* (digestive-metabolic enhancing), and *Medohara-Lekhana* (anti-hyperlipidemic/scraping) properties. Single herbal remedies such as *Picrorhiza kurroa* (Katuki), *Phyllanthus niruri* (Bhumyamalaki), *Andrographis paniculata* (Kalmegh), and *Boerhavia diffusa* (Punarnava) target hepatic fat accumulation, mitigate

lipotoxicity, and reduce inflammatory responses. Furthermore, classical compound formulations such as *Arogyavardhini Vati* and *Patola Katurohinyadi Kashayam* are widely utilized to address *Dhātvāgni-mandya*, clear channel obstruction (*Srotorodha*), and regulate lipid transformation within hepatocytes.

Epidemiology

MASLD is now considered one of the most common chronic liver diseases worldwide. Its prevalence parallels the increasing prevalence of obesity, type 2 diabetes, dyslipidaemia and metabolic syndrome.[3,15] In India, the pooled prevalence of NAFLD among adults has been estimated at approximately 38.6%, although estimates vary according to population characteristics, diagnostic method and setting. Among high-risk populations, the prevalence is considerably greater.[4] Importantly, fatty liver is not restricted to individuals with severe obesity. South Asian individuals may demonstrate substantial visceral adiposity and insulin resistance despite relatively modest BMI. A South Asian meta-analysis found associations between NAFLD and diabetes mellitus, hypertension, dyslipidaemia, generalized obesity, central obesity and metabolic syndrome.[5]

From an Ayurvedic perspective, these epidemiological patterns can be conceptually understood in relation to excessive consumption of calorie-dense foods, particularly *Madhura*, *Snigdha* and *Guru* dietary patterns, along with sedentary behaviour, excessive sleep and inadequate physical activity. Such factors are considered conducive to *Kapha* aggravation, impairment of *Agni* and abnormal accumulation or vitiation of *Meda*. Consequently, the contemporary understanding of metabolic syndrome and the Ayurvedic concept of *Santarpanajanya Vyadhi* demonstrate certain conceptual similarities, particularly regarding overnutrition, reduced physical activity and metabolic imbalance. However, these parallels should be interpreted cautiously, as the two frameworks arise from distinct theoretical and physiological systems and should not be considered directly equivalent.

Normal Functions of *Yakrit* in Ayurveda

The *Yakrit* holds an important position in Ayurvedic anatomy and physiology and is closely associated with the formation and maintenance of *Rakta Dhātu*. Classical texts describe *Yakrit* and *Pliha* as important sites related to blood physiology, suggesting their role in broader systemic functions. According to Ayurveda, tissue nourishment and transformation begin with the digestion of *Āhāra* by *Jatharāgni*, followed by the formation of *Āhāra Rasa* and its sequential transformation into different *Dhātu*. Disturbance of this process may affect

Rasa, *Rakta* and *Meda Dhātu* and consequently influence the functional state of the *Yakrit*. From the contemporary perspective, the liver is a central metabolic organ involved in carbohydrate, lipid and protein metabolism, cholesterol homeostasis, bile production, detoxification, hormone metabolism and nutrient storage. Its extensive role in maintaining systemic metabolic homeostasis provides a useful basis for exploring the Ayurvedic understanding of *Yakrit* in metabolic disorders such as NAFLD. In particular, the concepts of *Agni*, *Rakta Dhātu* and *Meda Dhātu* may offer complementary perspectives for understanding the relationship between impaired metabolism and hepatic dysfunction. However, these conceptual parallels should be interpreted cautiously, as Ayurvedic and modern physiological frameworks are fundamentally different.

Yakritodara in Classical Ayurveda

Udara Roga is described as an important group of disorders in classical Ayurvedic literature, with different types being distinguished according to their etiological and pathological characteristics. *Yakritodara* broadly refers to abdominal enlargement associated with pathological enlargement of the *Yakrit* (liver) and is closely described alongside *Plihodara*. In the Charaka Saṃhitā, the causes, clinical features and management of *Plihodara* and *Yakritodara* are discussed together, forming the broader concept of *Yakrit-Plihodara*.[10] Classical descriptions indicate features such as abdominal enlargement, heaviness, impaired digestion, anorexia, abdominal discomfort, weakness, altered bowel function and thirst, along with other systemic manifestations associated with *Doṣa* disturbance. Particular emphasis is given to enlargement in the anatomical region corresponding to the liver, especially the right hypochondrium.[10-13] Importantly, *Yakritodara* should not be interpreted as a direct histopathological equivalent of any single modern liver disease. Rather, it may be understood as a clinical-pathological syndrome centred on hepatic enlargement, providing a potentially useful Ayurvedic framework for interpreting hepatomegaly in conditions such as NAFLD.

Modern Etiology and Pathophysiology of NAFLD

Non-alcoholic fatty liver disease (NAFLD), recently incorporated within the broader terminology of metabolic dysfunction-associated steatotic liver disease (MASLD), is a multifactorial metabolic disorder characterized by excessive accumulation of hepatic fat in the absence of significant alcohol consumption or other predominant causes of hepatic steatosis. The development and progression of NAFLD are strongly associated with obesity, visceral adiposity, insulin resistance, type 2 diabetes mellitus, dyslipidaemia, hypertension,

metabolic syndrome, sedentary lifestyle and excessive caloric intake. In addition to these established metabolic risk factors, genetic and epigenetic susceptibility, dietary composition, adipose tissue dysfunction, sleep disturbances and alterations in the gut–liver axis may influence individual susceptibility and disease progression.[1,2,6-9]

The pathogenesis of NAFLD is complex and involves multiple interrelated metabolic and inflammatory mechanisms. Insulin resistance promotes increased lipolysis in adipose tissue, resulting in an increased influx of free fatty acids into the liver. Concurrently, hepatic de novo lipogenesis is increased, whereas fatty-acid oxidation and hepatic lipid export may become relatively inadequate. The imbalance between lipid acquisition, synthesis, oxidation and export favours accumulation of triglycerides and other lipid species within hepatocytes, resulting in hepatic steatosis.[6,7] Although triglyceride storage may initially represent an adaptive mechanism for relatively safe lipid sequestration, accumulation of potentially toxic lipid intermediates may subsequently result in lipotoxicity, mitochondrial dysfunction, endoplasmic-reticulum stress and oxidative stress.

Persistent metabolic stress promotes hepatocellular injury and activation of inflammatory pathways. Alterations in adipokine secretion, Kupffer-cell activation, cytokine signalling, oxidative stress and disturbances of the gut–liver axis contribute to the transition from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH).[6-9] Continued hepatocellular injury and inflammation may activate hepatic stellate cells, promote extracellular-matrix deposition and result in progressive hepatic fibrosis. Advanced fibrosis may ultimately progress to cirrhosis, hepatic decompensation and hepatocellular carcinoma in susceptible individuals.

Importantly, NAFLD should be regarded as a systemic metabolic disorder rather than an isolated disease of the liver. Cardiometabolic comorbidities, particularly obesity, type 2 diabetes mellitus, hypertension and dyslipidaemia, have important implications for disease progression and long-term cardiovascular and hepatic outcomes.[1,2,14]

Ayurvedic Etiology and Pathophysiological Concepts

Ayurveda does not describe NAFLD as a discrete disease entity corresponding directly to the contemporary biomedical definition. However, several classical concepts provide a theoretical framework for understanding the dietary, metabolic and hepatic disturbances observed in NAFLD. Relevant concepts include *Santarpana*, *Agnimandya*, *Āma*, *Kapha*

Prakopa, *Meda Dhātu Dushti*, *Meda Vriddhi*, *Srotodushti*, *Medoroga*, *Sthaulya* and pathological involvement of the *Yakrit*. The classical descriptions of *Yakritodara* are particularly relevant when hepatic enlargement accompanies the metabolic disorder.

From the Ayurvedic perspective, excessive consumption of *Guru*, *Snigdha* and *Madhura* dietary substances, excessive food intake, frequent overeating, sedentary behaviour and inadequate physical activity may contribute to *Santarpana*. Persistent *Santarpana* may lead to aggravation of *Kapha* and excessive accumulation or vitiation of *Meda Dhātu*. When such excessive nutritional load is accompanied by impaired digestive and metabolic activity, *Agnimandya* may develop. Impaired transformation of nutrients may subsequently contribute to the formation of *Āma* and disturbance of normal physiological channels or *Srotas*. Continued progression of these disturbances may ultimately involve the *Yakrit*.

The concept of *Agni* is particularly important in Ayurvedic interpretation of metabolic disorders. *Agni* represents the physiological processes responsible for digestion, transformation and utilization of nutrients. *Agnimandya* denotes impaired digestive or metabolic capacity and is considered an important pathological factor in several disorders associated with improper tissue nourishment and accumulation. In the context of NAFLD, this concept may provide a broad theoretical framework for understanding disturbances in nutrient transformation and metabolic homeostasis. Nevertheless, *Agni* should not be equated directly with insulin signalling, mitochondrial metabolism or any single biochemical pathway. It represents a broader Ayurvedic physiological construct encompassing processes of transformation and metabolism.

The concept of *Meda Dhātu* is also relevant because of its relationship with nourishment, storage and lipid-associated metabolic processes. Excessive nutritional intake combined with inadequate physical activity may lead to *Meda Vriddhi* and *Meda Dhātu Dushti*. From an integrative perspective, this may be considered relevant to the metabolic phenotype characterized by obesity, central adiposity and dyslipidaemia. However, *Meda Dhātu Dushti* should not be restricted to visible obesity, as hepatic steatosis may also occur in individuals with normal body weight. Therefore, Ayurvedic assessment should consider the overall metabolic phenotype rather than using body weight alone as a surrogate for *Meda* pathology.

The involvement of *Kapha* may be considered particularly relevant during the accumulation-dominant stage of metabolic disease. The classical attributes of *Kapha*, including *Guru*,

Snigdha and *Sthira*, provide a conceptual framework for understanding excessive accumulation, heaviness and reduced metabolic mobility. Thus, an early metabolic phenotype may be interpreted through *Kapha–Meda Dushti* associated with *Agnimandya*. *Pitta* may also be considered relevant because of its association with transformation and metabolic activity. During progression toward inflammatory hepatic injury, *Pitta Prakopa* may therefore be hypothesized to contribute to inflammatory manifestations. In advanced disease, further *Vāta* involvement may be hypothesized in association with progressive structural alteration and tissue pathology. This stage-wise interpretation remains theoretical and requires validation through appropriately designed clinical research.

The Ayurvedic concept of *Āma* may provide another conceptual perspective on the pathological consequences of impaired metabolism. In contemporary NAFLD, hepatic steatosis is accompanied in some patients by accumulation of potentially harmful lipid intermediates capable of inducing lipotoxicity, oxidative stress, mitochondrial dysfunction and inflammatory signalling.[6-8] Conceptually, the Ayurvedic sequence of *Agnimandya* leading to *Āma* and the modern sequence of metabolic dysfunction leading to lipotoxicity both recognize that abnormal metabolic processing may result in pathological consequences. However, *Āma* should not be interpreted as being chemically identical to any specific lipid intermediate, oxidative product or inflammatory mediator.

The concept of *Srotodushti* may additionally be relevant to the contemporary understanding of the gut–liver axis. Modern studies demonstrate that alterations in intestinal microbiota, intestinal permeability and microbial metabolites may influence hepatic lipid metabolism, inflammation and disease progression.[8,9] Ayurveda describes *Srotas* as pathways involved in the transportation and distribution of physiological substances, with abnormalities in their function being described as *Srotodushti*. Although this provides a potentially useful conceptual framework for discussing systemic and gut–liver disturbances, *Srotas* cannot currently be considered equivalent to the intestinal microbiome, intestinal barrier or any specific anatomical structure.

Modern Pathogenesis of NAFLD

Non-alcoholic fatty liver disease (NAFLD) is a complex metabolic disorder resulting from the interaction of genetic susceptibility, environmental influences, dietary factors, adipose tissue dysfunction and metabolic abnormalities. The pathogenesis is multifactorial and involves insulin resistance, altered lipid metabolism, oxidative stress, mitochondrial

dysfunction, hepatocellular injury and chronic inflammation. Insulin resistance is considered a major driver of hepatic steatosis. It promotes increased lipolysis in adipose tissue and consequently increases the delivery of free fatty acids to the liver. At the same time, hepatic de novo lipogenesis is increased, while fatty-acid oxidation and hepatic lipid export may become insufficient to maintain normal lipid homeostasis.[6,7]

The resulting imbalance in hepatic lipid metabolism leads to accumulation of triglycerides and other lipid species within hepatocytes. Although triglyceride accumulation may initially represent a relatively protective mechanism for storing excess fatty acids, accumulation of potentially toxic lipid intermediates may subsequently result in lipotoxicity. Lipotoxicity induces oxidative stress, mitochondrial dysfunction and endoplasmic-reticulum stress, leading to hepatocellular injury. Activation of Kupffer cells and other innate and adaptive immune pathways further promotes inflammatory responses and contributes to progression from simple steatosis to non-alcoholic steatohepatitis (NASH). Persistent inflammation and hepatocellular injury may activate hepatic stellate cells, resulting in extracellular-matrix deposition and progressive hepatic fibrosis. Advanced fibrosis may ultimately progress to cirrhosis and hepatocellular carcinoma (HCC) in susceptible individuals.

Thus, insulin resistance plays a central role in the development of hepatic steatosis, whereas lipotoxicity, oxidative stress and immune-mediated mechanisms are particularly important in the transition from simple steatosis to steatohepatitis and progressive fibrosis.

Ayurvedic Pathogenesis of the *Yakritodara*

NAFLD does not have a direct one-to-one equivalent as a disease entity in classical Ayurvedic literature. Therefore, its Ayurvedic interpretation requires correlation of its etiological factors, metabolic manifestations and hepatic involvement with relevant classical concepts. The concepts of *Santarpana*, *Kapha Prakopa*, *Agnimandya*, *Āma*, *Meda Dhātu Dushti*, *Meda Vriddhi*, *Srotodushti* and *Yakrit* involvement provide a potential conceptual framework for this purpose.

Excessive consumption of *Guru*, *Snigdha* and *Madhura* dietary substances, excessive caloric intake, irregular dietary practices, inadequate physical activity and sedentary behaviour may collectively contribute to *Santarpana*. Persistent *Santarpana* may promote *Kapha Prakopa* and excessive accumulation or vitiation of *Meda Dhātu*. When the nutritional load exceeds the body's capacity for appropriate transformation and utilization, impairment of *Agni* or

Agnimandya may be considered. Subsequent abnormal metabolic processing may be interpreted through the concept of *Āma*, followed by disturbance of physiological channels or *Srotas*. Continued progression may result in *Yakrit* involvement and, when hepatic enlargement is present, may be considered in relation to the clinical phenotype of *Yakritodara*.

Clinical Correlation of NAFLD with *Yakritodara*

NAFLD is often asymptomatic, particularly in its early stages, although some patients may experience nonspecific symptoms such as fatigue, generalized weakness or vague right-upper-quadrant discomfort. Hepatomegaly may occur in patients with hepatic steatosis but is not essential for the diagnosis. The clinical correlation with *Yakritodara* becomes more relevant when NAFLD is accompanied by hepatic enlargement and abdominal or digestive manifestations such as abdominal heaviness or distension, reduced appetite, indigestion, altered bowel habits and fatigue, particularly in the presence of obesity or other metabolic risk factors. However, this relationship requires cautious interpretation because NAFLD may occur without clinically detectable hepatomegaly, while *Yakritodara* may result from causes other than metabolic fatty liver disease. Therefore, *Yakritodara* should not be regarded as synonymous with NAFLD; rather, NAFLD presenting with hepatomegaly may be considered a clinical context in which *Yakritodara* provides a potentially relevant Ayurvedic interpretative correlate.

Modern Diagnostic Assessment

Diagnosis of NAFLD should not be based solely on elevated liver enzymes or the presence of hepatomegaly. Contemporary evaluation involves identification of hepatic steatosis together with assessment of metabolic risk factors and the probability of progressive hepatic fibrosis. Common biochemical investigations include alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), alkaline phosphatase, bilirubin, fasting blood glucose, glycated haemoglobin (HbA1c), lipid profile, complete blood count, serum albumin and platelet count.

Imaging plays an important role in detecting hepatic steatosis and assessing liver structure. Ultrasonography is widely used as an initial imaging modality because of its accessibility and non-invasive nature. Transient elastography can provide information regarding liver stiffness and, when combined with controlled attenuation parameter (CAP), can assist in assessing hepatic steatosis and fibrosis. Magnetic resonance-based techniques may provide more

accurate quantitative assessment of hepatic fat and are particularly useful in research and selected clinical settings.

Non-invasive fibrosis assessment is essential because fibrosis is one of the most important determinants of long-term hepatic outcomes. Commonly used approaches include the fibrosis-4 index (FIB-4), NAFLD fibrosis score and transient elastography. Contemporary clinical guidelines emphasize systematic fibrosis risk stratification, particularly among individuals with obesity, type 2 diabetes mellitus, metabolic risk factors, abnormal liver enzymes or imaging evidence of hepatic steatosis.[14]

Ayurvedic Assessment

From an Ayurvedic perspective, assessment may be individualized according to *Prakṛti*, *Vikṛti*, *Agni*, *Koṣṭha*, *Mala*, *Āhāra*, *Vihāra* and *Doṣa* predominance. Particular attention may be given to features suggestive of *Meda Dushti*, *Kapha* aggravation and impairment of metabolic function. Clinical assessment of the abdomen, including evaluation of *Yakrit* size, consistency and tenderness where appropriate, may provide additional information.

An integrative diagnostic approach may therefore combine objective hepatological investigations with systematic Ayurvedic phenotyping. Such an approach may facilitate future research into whether specific Ayurvedic phenotypes are associated with particular metabolic, inflammatory or fibrotic profiles in patients with NAFLD.

Modern Management of NAFLD[15]

Lifestyle modification remains the foundation of management for NAFLD. Therapeutic strategies should address both hepatic disease and the associated cardiometabolic abnormalities. Weight reduction is particularly important in individuals who are overweight or obese. Evidence indicates a dose-dependent relationship between weight loss and improvement in hepatic steatosis, steatohepatitis and, in appropriately selected patients, hepatic fibrosis.[14-16]

Regular physical activity, including aerobic and resistance exercise, improves insulin sensitivity and metabolic health and can reduce hepatic steatosis even when substantial weight loss is not achieved. Dietary management should focus on reducing excessive intake of refined carbohydrates, sugar-sweetened beverages, saturated fats and energy-dense ultra-processed foods. A sustainable dietary pattern that can be maintained over the long term is

generally preferable to highly restrictive diets.

Management of associated metabolic disorders is an essential component of NAFLD care. Appropriate management of type 2 diabetes mellitus, hypertension, dyslipidaemia, obesity and cardiovascular disease should be integrated into the overall therapeutic strategy. Contemporary guidelines emphasize that NAFLD management should not focus exclusively on the liver because cardiovascular and metabolic complications constitute major determinants of morbidity and mortality.

Ayurvedic Therapeutic Perspective[16]

The Ayurvedic therapeutic approach to the NAFLD phenotype should primarily focus on correction of causative factors, restoration of appropriate metabolic function and management of *Kapha–Meda* imbalance. Treatment should be individualized according to *Doṣa*, *Dūṣya*, *Agni*, disease stage, strength of the patient and associated comorbidities.

Nidāna Parivarjana

Avoidance of causative dietary and behavioural factors represents the fundamental therapeutic principle. Reduction of excessive *Guru*, *Snigdha* and *Madhura* dietary intake, avoidance of excessive caloric consumption and correction of sedentary behaviour may be particularly relevant in patients exhibiting *Santarpana* and *Meda Dushti*.

Langhana

Appropriately individualized *Langhana* may be considered in patients with excessive nutritional load and *Kapha–Meda* predominance. In contemporary terms, this may conceptually correspond to reduction of excessive energy intake. However, aggressive caloric restriction should be avoided, particularly in individuals with malnutrition, advanced liver disease or other contraindications.

Deepana–Pācana

Deepana and *Pācana* principles may be considered when clinical features suggest *Agnimandya*. Their conceptual objective is to support appropriate digestive and metabolic processes. However, specific therapeutic recommendations should be based on individualized Ayurvedic assessment and supported by appropriate safety evidence.

***Medohara* and *Lekhana* Principles**

In patients with *Meda Vriddhi* and obesity-associated metabolic dysfunction, *Medohara* and

Lekhana principles may be considered as part of a comprehensive therapeutic strategy. These principles may conceptually correspond to reduction of excessive adiposity and correction of metabolic imbalance. Their clinical efficacy in NAFLD should, however, be established through appropriately designed clinical trials.

Virechana

Classical Ayurvedic literature describes *Virechana* among therapeutic approaches for selected *Pitta*-associated and abdominal disorders. Its application in patients with liver disease requires careful patient selection and supervision. At present, evidence regarding the efficacy and safety of *Virechana* specifically for NAFLD remains insufficient to consider it a replacement for established hepatological management.

Vyayama

Vyayama has particular relevance because physical inactivity is a major risk factor for modern metabolic disease. Regular, individualized physical activity may therefore represent one of the strongest areas of convergence between Ayurvedic principles and contemporary lifestyle medicine.

Ayurvedic Pharmacotherapy and Herbal Interventions in NAFLD / Yakritodara[17]

In classical Ayurvedic therapeutics, managing conditions mapped to *Yakritodara* or *Santarpana*-induced *Kapha–Meda* imbalances relies on single herbal drugs (*Eka Moolika*) and compound classical formulations (*Yoga*) that exhibit *Deepana* (digestive fire stimulation), *Pācana* (digestion of *Āma*), *Yakrit-Rakshaka* (hepatoprotection), *Pittasaraka* (cholagogue), and *Lekhana* (lipid-scraping) properties.

Single Medicinal Plants (*Eka Dravya*)[18]

- **Katuki (*Picrorhiza kurroa*):** Widely recognized for its *Tikta* (bitter) taste and *Bhedana* (purgative/cholagogue) action, Katuki stimulates bile flow, enhances lipid clearance, and reduces *Kapha–Pitta* accumulation in the liver. Modern pre-clinical studies demonstrate its active iridoid glycosides (kutkin, picroside) exert significant hepatoprotective, antioxidant, and anti-steatotic effects.
- **Bhumyamalaki (*Phyllanthus niruri / Phyllanthus amarus*):** Traditionally used in *Yakrit Roga*, Bhumyamalaki possesses *Kashaya* (astringent) and *Tikta* (bitter) properties that help restore liver enzyme levels (ALT/AST), alleviate metabolic *Āma*, and prevent hepatic lipid peroxidation.

- **Kalmegh (*Andrographis paniculata*):** Functioning as a potent *Deepana* and *Pācana* agent, its active constituent (andrographolide) mitigates lipotoxicity, downregulates inflammatory signaling pathways, and reduces hepatic fat accumulation.
- **Punarnava (*Boerhavia diffusa*):** Possessing *Sothahara* (anti-inflammatory/anti-edematous) and *Mutrala* (diuretic) properties, Punarnava aids in resolving micro-inflammation and cellular congestion within hepatocyte beds.
- **Guduchi (*Tinospora cordifolia*):** Serves as an *Immuno-modulator* and *Rasayana* (rejuvenator) that targets underlying insulin resistance, lowers systemic metabolic oxidative stress, and regulates dyslipidemia.

Polyherbal Formulations (*Classical Formulations*).[19 20]

Formulation	Core Ingredients	Mechanism / Classical Rationale
Arogyavardhini Vati	Katuki, Shuddha Guggulu, Shuddha Shilajit, Abhraka Bhasma, Tamra Bhasma, Triphala	Acts as a primary *Medohara* (lipid-lowering) and *Yakrit-Uttejaka* (liver-stimulating) formulation. It corrects *Agnimandya* at the cellular (*Dhātvāgni*) level and clears *Srotorodha* (channel obstruction).
Patola Katurohinyadi Kashayam	Patola, Katuki, Guduchi, Chandana, Patha	Normalizes *Pitta-Kapha* dyscrasias, promotes *Pittasarak* (bile clearance), reduces hepatic inflammation, and clears circulating metabolic toxins (*Āma*).
Punarnavadi Mandoor	Punarnava, Trivrit, Mandoor Bhasma, Haritaki, Amalaki, Bibhitaki	Indicated in metabolic hepatomegaly with underlying micro-edema, anemia, or tissue sluggishness; helps restore microvascular hepatic microcirculation.
Rohitakarishta / Vasaguduchyadi Kwath	Rohitaka (*Tecomella undulata*), Guduchi, Vasak	Classical interventions specifically indicated for *Yakrit-Plihadalyudara* (hepatic and splenic enlargements) to reduce organ volume and abdominal heaviness.

Clinical Considerations & Integrative Safety[21]

While these Ayurvedic agents demonstrate multi-target pharmacological activity against steatosis, lipotoxicity, and oxidative stress, their administration in MASLD/NAFLD requires strict individualized clinical assessment. Herbo-mineral compounds (such as *Arogyavardhini Vati*) should be used under supervised dosage and duration, with monitoring of liver function tests (LFTs) and lipid profiles to ensure therapeutic safety and efficacy.

CONCLUSION

NAFLD is a multifactorial metabolic liver disorder resulting from complex interactions among insulin resistance, visceral adiposity, altered hepatic lipid metabolism, lipotoxicity,

oxidative stress, inflammation and genetic and environmental factors. Although NAFLD is not described as a discrete disease entity in classical Ayurvedic literature, several Ayurvedic concepts may provide complementary theoretical perspectives for understanding its etiological and pathological dimensions. *Santarpana*, *Avyayama*, *Kapha Prakopa*, *Agnimandya* and *Meda Dhātu Dushti* may provide a conceptual framework for the metabolic and lifestyle-related components of the disease, while *Āma* and *Srotodushti* may provide broader interpretative models for pathological metabolic processing and systemic communication. When hepatic enlargement is present, *Yakrit Vriddhi* and *Yakritodara* may offer a clinically relevant Ayurvedic framework for interpretation. However, *Yakritodara* should not be considered synonymous with NAFLD. The most defensible integrative interpretation is that NAFLD with hepatomegaly may be clinically correlated with the *Yakritodara* phenotype, while its metabolic pathogenesis may be interpreted through a combination of *Santarpana*, *Agnimandya*, *Kapha–Meda Dushti* and *Srotodushti*. These correlations should be considered hypothesis-generating and require validation through clinical, biochemical, imaging and translational research.

An integrated approach combining standardized Ayurvedic assessment with contemporary hepatological investigations may provide a valuable framework for future research into the prevention and management of NAFLD. Such integration should preserve the conceptual integrity of both systems while avoiding unsupported equivalence between classical Ayurvedic constructs and specific biomedical mechanisms.

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