

Ayurvedic Management of Non Alcoholic Fatty Liver Disease: A Case Report Highlighting the Importance of Early Detection

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ABSTRACT:

NAFLD (Non Alcoholic fatty liver disease), is a metabolic disorder of hepatic origin, in which there is the accumulation of fat in the liver. NAFLD includes a spectrum of progressive liver diseases ranging from fatty infiltration alone to fatty infiltration with inflammation known as NASH (Non Alcoholic Steato Hepatitis) which may progress to cirrhosis and primary liver cancer. The overall prevalence of NAFLD worldwide estimated to be 32.4%. There is no exact clinical entity mentioned in Ayurveda like NAFLD, but may be understood as *Yakritavikara* (Liver disorder). *Nidana* like *Snigdha Guru Madhura Ahara*, *Ayuvayama*, *Divaswapna* etc causes *Agnidushti* which lead to *Ama* production results in *Sama Rasa* and *Malbhuta Kapha* along with impaired *Dhatuposhan*, *Dhatuparinaman* & *Vridhhi of Meda*. If there is *Khavaigunya* in *Yakrita*, *Sthansamshraya* of *Meda* in *Yakrita* occurs which may produce *Yakrutavikara* like NAFLD. In this case report, a 46-year-old female patient, presented with complaints of Poor appetite, Nausea, Abdominal pain, heaviness, Unsatisfactory bowel habits and fatigue since 1-2 months. Based on the clinical examination, USG, Fibroscan and Blood investigations, she was diagnosed with Fatty Liver-I. The patient was effectively treated with *Shaman Chikitsa* for 65 days. Within 1 month, there was a moderate improvement in overall symptoms. After 65 days of Ayurvedic treatment, all symptoms, Radiological investigations and Liver function showed highly significant results. The treatment was given by considering the vitiation of *Pitta*, *Kapha* and *Meda*. So, it can be concluded that by implementing Ayurvedic principles, Liver Disorders can be treated without causing any adverse effects.

Key words: *Medodushti*, NAFLD, *Shamana*, *Yakritvikara*.

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INTRODUCTION:

Non-alcoholic fatty liver disease (NAFLD) is defined as the presence of excessive fat accumulation in the liver, has emerged as the most common cause of chronic liver disease globally, affecting nearly 25–30% of the general population. ^[1] Its prevalence is even higher among individuals with obesity, type 2 diabetes mellitus, and metabolic syndrome, where it ranges from 50–70%. ^[2] In India, community-based studies report a prevalence ranging from 16% to 32%, reflecting the growing impact of lifestyle-related disorders on liver health. ^[3]

NAFLD encompasses a spectrum of conditions, from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), which can progress to advanced fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). ^[4] Unlike alcoholic liver disease, NAFLD develops in individuals with little or no alcohol intake and is strongly linked to insulin resistance, central obesity, dyslipidemia, and hypertension ^[5] Cardiovascular disease is the leading cause of death in NAFLD patients, underlining its systemic metabolic implications. ^[6]

The pathogenesis of NAFLD is multifactorial, involving hepatic lipid accumulation, oxidative stress, mitochondrial dysfunction, and chronic low-grade inflammation. ^[7] Genetic factors (such as PNPLA3 and TM6SF2 polymorphisms), dietary patterns, and gut microbiota dysbiosis further contribute to disease susceptibility and progression. ^[8] Despite its high prevalence, NAFLD is often underdiagnosed as it remains asymptomatic in its early stages and is usually detected incidentally through elevated liver enzymes or imaging studies. ^[9]

Currently, there is no FDA-approved pharmacological therapy for NAFLD. Lifestyle modification—including sustained weight loss, dietary changes, and physical activity—remains the cornerstone of management. ^[10] Evidence suggests that a 7–10% reduction in body weight can significantly improve steatosis and even reverse fibrosis in some patients. ^[11] Non-invasive diagnostic tools such as ultrasonography, transient elastography (FibroScan), and fibrosis scoring indices (FIB-4) have emerged as valuable alternatives to liver biopsy for diagnosis and monitoring. ^[12]

There is no specific terminology for NAFLD in Ayurveda, but it can be considered under *Medoroga* as the pathophysiology includes the deposition of *Abadha Meda* over *Yakrita*. *Agnidushti* is the major component in metabolism-related disorders. *Kapha Dosha Vriddhi* due to lack of exercise or physical activity, *Divasvapa*, excessive intake of *Madhura*, *Snigdha*, *Guru* or *Kapha Vardhaka Ahara* cause *Agnimandya* (poor digestion/metabolism) in the form of *Dhatvagnimandya*, which is the root cause of *Medovaha Sroto Dushti* and can result in excessive increase in *Meda Dhatu*. Excessive production and accumulation of *Meda* in the body has been described as *Medoroga*. *Meda Dhatu* simulates fat or adipose tissue. Acharya Charaka has also explained *Prameha* and *Atisthanhya* along with other diseases caused by similar etiological factors under *Santarpana-janya Vyadhi*.

PATIENT INFORMATION:

A 46 years-old-female patient presented with the complaints of Abdominal pain in Right hypochondrium, Abdominal heaviness after

meal for upto 2-3 hrs, Unsatisfactory bowel habits associated with straining (passes once in 2 days) since 1 month, Fatigue persisting whole day, poor appetite and nausea since 2 months. With all above complaints she reported in OPD of Kayachikitsa Department.

She was not having any metabolic comorbidities. No any history of past illness. Personal history revealed excessive intake of *Guru, Snigdha, Madhur Amla rasa sevan, Divaswap* daily for 2 hrs. She had a history of disturbed sleep at night further no any medication history noted.

CLINICAL FINDINGS:

On general examination, the patient's blood pressure was 124/88 mm Hg; pulse rate was 82/min, regular; temperature – 98.7°F, weight – 63 kg, height – 154 cm, BMI- 26.6 kg/m². The patient was overweight and appeared lethargic. Pallor, Icterus, Cyanosis, Clubbing, Lymphadenopathy, Edema was absent. Per abdominal examination revealed tenderness in right hypochondrium. No any organomegaly, palpable mass, any warts, hemangiomas, striae observed. Fluid thrill test absent. Her *Nadi* (Pulse) was of *Saamaja* (Regularly irregular) with *Kapha-Pittaj dosha* dominance. The presence of *Saamaja Jivha* (whitish coated) was also noted. *Mala* was *grathit* (constipated), *asamadbhankarak*, *kathin* with frequency of once in 2 days while no abnormality in urine was detected. *Sparsh* (Touch) and *Shabd* (Voice) were *Prakrut*, while *Aakriti* (Shape/Built) was *Sthula* (Obese). The *Dashvidha Pariksha* (the tenfold examination) revealed the *Prakriti* of the patient to be *Kapha- Pittaja*, *Saara* to be *Mamsa-medosaara* (built predominantly possessing fat and muscles),

while *Salva* and *Sambanan* were *alpa*. Hematological investigations were performed and the details are depicted.

Routine investigations (Complete Blood count, Lipid profile, renal function tests, and Urine examination) were found to be within normal limits. Liver function tests revealed highly raised AST ALT enzymes with normal levels of Bilirubin, alkaline phosphatase, albumin and globulins. The ultrasound (USG) revealed Fatty Liver grade-1 with no other associated conditions such as cholelithiasis, renal calculi. FibroScan showed 6.1 kpasal score indicating F1 fibrosis stage in liver. The severity of the disease was assessed as per the FIB-4 score-1.95 showing probability of fibrosis.

Diagnostic assessment

Patient was diagnosed with NAFLD, evaluated through detailed clinical history including metabolic comorbidities (Diabetes Mellitus, Hypertension, Dyslipidemia, Obesity), medication history and personal history as well. Physical examination focused on anthropometric parameters such as body mass index (BMI) and waist circumference. Laboratory investigations included Complete Blood count, Liver function tests (ALT, AST, ALP, bilirubin, albumin), Diabetic profile and lipid profile. Viral hepatitis markers, Thyroid Function Tests, evaluation of PCOD were performed to exclude secondary causes of steatosis. Ultrasonography of the abdomen was used as the initial imaging for detection of Fatty Liver. Transient Elastography (FibroScan) was conducted to assess liver stiffness (fibrosis). Assessment was done on the basis of FIB-4 score to check probability of fibrosis.

Ayurveda diagnostic assessment

The patient exhibited a *Kapha-Pitta* predominant *Prakriti*. The *Srotas* affected were *Rasa-Rakta*, *Meda* and *Anna Vaha Srotas*. The *Vikriti* was suggestive of formation of *Apakva Aamrasa* due to *Agnimandya* which leads to circulation of *Abaddha meda* which accumulates in *Yakrita* results in NAFLD. Her *Samhanan* and *Satva* was *Alpa*, while *Pramana* was *madhyama*. She was *Satmya* to vegetarian dietary habits and exhibited *Madhyama Satva. Ahara Shakti* (appetite) *Jarana Shakti* (digestive capacity) and *Vyayama Shakti* (exercise tolerance) was *Alpa*. Her age of 46 years placed her in the *Madhyama Vaya* category.

The *Nadi* (pulse) showed *Kapha-Pittaj dosha* dominance. The *Mala* (bowel habits) was reported to be unsatisfactory having frequency of once in 2 days associated with hard stool and straining. *Mutra* (urine) was normal in frequency and color. *Jihva* (tongue) was *Sama* with mild whitish coating. *Sabda* (voice/speech) was clear. *Sparsha* was *Ushna Snigdha* and showed tenderness in Right upper quadrant of abdomen and *Drik* (eyes) appeared normal. The *Akrti* (overall appearance) was *Sthula*.

In the present case, the diagnosis of Non Alcoholic Fatty Liver Disease was supported by multiple clinical and radiological findings: Patient complaining GI symptoms such as loss of appetite, Abdominal heaviness after meal, Pain in abdomen, Unsatisfactory Bowel habits, fatigue; Radiological investigations- Ultrasonography showing fatty liver grade 1; FibroScan showing Fibrosis score F1; Elevated levels of AST and ALT and no history of alcohol intake confirmed the diagnosis of NAFLD. Other causes were

differentially diagnosed by alcohol history. Viral markers, drug exposure and genetic tests.

THERAPEUTIC INTERVENTION

The patient was administered *Phalatrikadi Kwatha* 40 ml twice daily before food, *Eranda Bhrishta Haritaki* 3 gm with *Katuki* 1 gm twice daily before food with warm water, and *Aampachana Vati* 2 tablets twice daily after food with warm water. This treatment was continued for 20 days, targeting *Aampachana* (digestion), *Anulomana* (bowel regulation), and enhancing liver metabolism. In addition, dietary and lifestyle modifications were advised to support digestive function, correct metabolic derangements.

As the patient did not achieve significant relief with the initial regimen, the therapy was revised to target *Aampachana* (digestive stimulation), *Anulomana* (bowel regulation), *Medohara* (Lipolytic), *Lekhana* (Scraping action), *Bhedana* (Penetrating) *Shothahara* (anti-inflammatory), *Yakrituttejaka* (liver-stimulating), and hepatoprotective actions. The revised treatment was administered for 45 days and included *Phalatrikadi Kwatha* combined with *Punarnavashtak Kwatha*, 40 ml twice daily before food, *Triphala churn* 3 gm, *Vidanga churn* 1 gm, *Katuki* 1 gm, and *Kalmegha churn* 1 gm, administered twice daily after food with warm water, and *Triphala Guggulu*, 2 tablets, was taken after food by chewing. This regimen, along with continued dietary and lifestyle modifications.

Follow-up and Outcomes

The patient was advised to continue *Pathya Ahara* (*Yava, Shyamaka, Kodrava, Warm water*) and *Vihara* (Regular exercise, yoga, meditation,

avoid divaswap). Patient came for follow-up every 15 days for 1 month. No any clinical signs or symptoms were relapsed. Clinical evaluation included assessment of symptoms along with monitoring of adherence to dietary and lifestyle modifications. At the end of the follow-up period, patients remained clinically asymptomatic with no relapse of symptoms. Liver enzymes remained within stable limits, with no recurrence of unexplained elevations. Imaging demonstrated no progression of

steatosis or fibrosis, and fibrosis scores remained stable throughout the study. Anthropometric parameters showed mild but marked improvement. FIB-4 score was within normal limits after treatment minimizing probability of fibrosis.

These findings confirms that the patients maintained a stable clinical course without relapse, progression to NASH (Non Alcoholic Steato-Hepatitis), or development of cirrhosis during the follow-up period.

Table -1: Timeline of the Events:

Date	Clinical-Findings	Investigations	Therapeutic-Interventions
26-03-2025	<ul style="list-style-type: none"> Abdominal pain Abdominal heaviness after meal Unsatisfactory bowel habits Fatigue Poor appetite Nausea 	<ul style="list-style-type: none"> CBC- Normal Lipid profile- Normal Biochemical investigations- Showed raised AST ALT enzymes (AST- 151 U/L and ALT- 236 U/L) Viral hepatitis profile- Negative FIB-4 score- 1.95 indicating chances of Fibrosis 	Advised to repeat LFT. As she was having reports within normal limits before 3 months in annual checkup.
27-03-2025	Same as above	<ul style="list-style-type: none"> Biochemical investigations repeated showed- Raised AST ALT enzymes. (AST- 159 U/L and ALT- 242 U/L) Ultrasonography showed- Fatty Liver grade-1 Fibroscan- 6.1 Kpascal showing F1 stage of fibrosis 	<ol style="list-style-type: none"> <i>Phalatrikadi Kwatha</i>- 40 ml BD <i>Erandbhrisht Haritaki</i>- 3 gm+ <i>Katuki</i>-1gm BD in <i>Apana kala</i> with warm water. <i>Aampachana Vati</i> 2 tablets <i>vyanodana kala</i> with <i>koshnajal</i>
15-04-2025	<ul style="list-style-type: none"> Mild relief in Abdominal pain and heaviness. 	<ul style="list-style-type: none"> Biochemical investigations showed highly raised AST ALT 	<ol style="list-style-type: none"> <i>Phalatrikadi Kwatha</i> + <i>Punarnavashtak Kwatha</i>-40 ml BD

	<ul style="list-style-type: none"> Mild improvement in bowel habits. Improved appetite. No any relief in fatigue 	<p>enzymes. (AST- 295 U/L and ALT- 475 U/L)</p> <ul style="list-style-type: none"> FIB-4 score- 2.68. Lipid profile- Normal 	<p>2. <i>Trifala churn 3 gm + Vidanga churn 1 gm + Katuki 1 gm + Kalmegha churn 1 gm</i> BD, AF with Luke warm water.</p> <p>3. <i>Trifala guggulu 2 tablets</i> BD, AF (by chewing) with Luke warm water.</p>
02-05-2025	Moderate improvement in overall symptoms	<ul style="list-style-type: none"> Biochemical investigations showed mild improvement in AST ALT enzymes. (AST- 214 U/L and ALT- 387 U/L) 	<p>(Continue 1,2,3)</p> <p>1. <i>Phalatrikadi Kwatha + Punarnavashtak Kwatha-40 ml</i> BD</p> <p>2. <i>Trifala churn 3 gm + Vidanga churn 1 gm + Katuki 1 gm + Kalmegha churn 1 gm</i> BD, AF with Luke warm water.</p> <p>3. <i>Trifala guggulu 2 tablets</i> BD, AF (by chewing) with Luke warm water.</p>
03-06-2025	Symptomatically significant relief observed	<ul style="list-style-type: none"> Biochemical investigations showed normal AST ALT levels. (AST- 22 U/L and ALT- 26 U/L) CBC- Normal Lipid Profile-Normal Ultrasonography reflected normal size of liver FibroScan – 5.4 Kpas indicating no Fibrosis. (F0) 	<p>Continue 1,2</p> <p>1. <i>Phalatrikadi Kwatha + Punarnavashtak Kwatha-40 ml</i> BD</p> <p>2. <i>Trifala churn 3 gm + Vidanga churn 1 gm + Katuki 1 gm + Kalmegha churn 1 gm</i> BD, AF with Luke warm water.</p>
1 st follow-up- 20-06-2025	No relapse of any Symptoms.	-	Diet and lifestyle advices.
2 nd follow-up- 05-07-2025	No relapse of any Symptoms.	-	Diet and lifestyle advices.

DISCUSSION:

Phalatrikadi Kwatha, described in *Chakradatta* and *Sharangadhara Samhita*, is indicated in *Yakrit-Pleeba vikara*, *Kamala*, and *Meda-Kapha dushti*. In NAFLD, it acts as a *Deepana-Pachana*, *Medohara-Lekhana* and *Yakrituttejaka*, supporting hepatic fat reduction and metabolic balance. Modern evidence suggests its hepatoprotective role, with improvements in liver enzymes, lipid profile, and ultrasound findings of steatosis.^[13]

Eranda Bhrishta Haritaki is *Haritaki* (*Terminalia chebula*) processed with *Eranda* taila, which enhances its *Deepana-Pachana*, *Anulomana*, and *Medohara* properties. It is particularly useful in disorders of *Kapha-Meda* imbalance, aiding in the removal of *Ama* and reducing hepatic fat accumulation. Additionally, it helps relieve constipation, supporting bowel regularity in patients with metabolic derangements.

Katuki (Picrorhiza kurroa) is a classical *Yakrituttejaka* herb indicated in *Yakrit-Pleeba vikara*, *Kamala*, and *Shotha*. NAFLD in Ayurveda, as a disorder of *Kapha-Meda vriddhi* with *Ama* and *Yakrit dushti*, leading to *Srotorodha* and metabolic derangement. *Katuki*, with its *Tikta-Katu rasa*, *Laghu-Ruksha guna*, *Sheeta virya*, and *Katu vipaka*, acts as *Kapha-Pitta shamaka*, *Lekhana*, *Bhedana*, and *Virechaka*. It stimulates liver function (*Yakrituttejaka*), enhances *Pitta pravritti* for proper fat metabolism, clears accumulated *Ama*, and reduces *Meda dhatu*. The *Ruksha-Laghu* properties counteract the *Snigdha-Guru* quality of aggravated *Kapha-Meda*, while the *Lekhana* action reduces hepatic steatosis, making *Katuki* highly effective in maintaining liver health, preventing progression to NASH, and

supporting long-term stability in NAFLD.^[14,15]

Aampachan Vati that promotes *Deepana-Pachana*, effectively improving appetite and correcting digestive sluggishness. It is especially useful in conditions with *Agni mandya* and anorexia, supporting proper metabolism and nutrient assimilation in NAFLD patients.^[16]

Punarnavashtaka Kwatha is indicated in *Yakrit-Pleeba vikara*, *Shotha*, and *Kapha-Meda* disorders. It acts as a *Shothahara*, *Medohara*, and *Yakrituttejaka*, reducing hepatic fat accumulation, *Amapachana*, stimulating liver function, and supporting long-term metabolic and liver health.^[17]

Vidanga (Embelia ribes) is a classical herb known for its *Deepana-Pachana* (digestive stimulant) and *Medohara* (lipid-reducing) properties. Studies have shown that its ethanolic extract can reduce hepatic fat accumulation and improve lipid profiles in high-fat diet-induced obesity models, making it beneficial in NAFLD management.^[18]

Kalmegha (Andrographis paniculata), possesses *Yakrituttejaka* and *Pittashamaka* (anti-inflammatory) actions. Clinical research indicates that *Kalmegha* significantly improves liver function, reduces inflammation, and enhances lipid metabolism in NAFLD patients.^[19]

Triphala Guggulu is a formulation combining *Triphala* (*Amalaki*, *Haritaki*, *Bibhitaki*) with *Guggulu* (*Commiphora mukul*) and *pippali* (*piprum longum*), primarily indicated in *Medo-Vata* disorders and *Yakrit-Pleeba vikara*. In NAFLD, which can be correlated with *Kapha-Meda vriddhi* and *Ama accumulation* in the liver, *Triphala Guggulu* acts as *Medohara*, *Lekhana*, *Deepana-Pachana*, and *Rasayana*.^[20]

CONCLUSION:

The present case report suggests that Ayurvedic interventions, when applied in the management of non-alcoholic fatty liver disease (NAFLD), can contribute to significant clinical improvement and stabilization of the disease. Patients demonstrated relief from symptoms, normalization of liver function tests, and no relapse during follow-up. While these findings are encouraging, due to lack of generalizability and limited follow-up highlight the need for larger randomized controlled trials to further validate the efficacy and safety of Ayurvedic therapies in NAFLD. Integrating Ayurveda with modern diagnostic and monitoring tools may provide a comprehensive and patient-centred approach in addressing the rising burden of metabolic liver disease.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given written informed consent for clinical information, images, and outcomes to be reported in the journal. The patient understands that names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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