RESEARCH PAPER

Ayurvedic Perspective of Non-Alcoholic Fatty Liver Disease (*Yakritodara*): A Comprehensive Review

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is a major cause of chronic liver disease worldwide and is closely associated with metabolic syndrome. Pharmacological options are still limited despite their increasing prevalence. Ayurvedic medicine, the traditional Indian medical system, refers to a related condition called *Yakritodara* (fatty enlargement of the liver) as part of *Medovaha Srotodushti* (disorders of fat-carrying channels). **Goals:** This systematic review will attempt to integrate the Ayurvedic classical concept of NAFLD (*Yakritodara*) with modern biomedical concepts, critically analyze the proposed pathophysiological correlations, and review evidence for Ayurvedic interventions. **Approach:** A systematic search was conducted covering both classical Ayurvedic literature in the form of Charaka Samhita, Sushruta Samhita, Ashtanga Hridaya, and electronic databases (PubMed, Google Scholar, Scopus, DHARA). Research studies that compared Ayurvedic ideas, isolated herbs, or polyherbal products in the management of NAFLD were considered. A modified hierarchy of integrative medicine was used for quality assessment of clinical studies. **Findings:** Classical sources offer a strong theoretical context for the connection between NAFLD and *Agnimandya* (impaired digestive system), *Medodhatvagnimandya* (malfunctioning metabolism in fat tissues), and *Srotorodha* (blocked channels). Modern risk factors relate to key pathogenic factors (*Nidana*), including sedentary lifestyle (*Avihara*), high-calorie diet (*Ati-Snigdhahara*), and psychological stress. Several Ayurvedic single herbs (e.g., *Phyllanthus niruri – Bhumyamalaki*, *Picrorhiza kurroa – Katuki*, *Curcuma longa – Haridra*) and preparations (e.g., *Arogyavardhini Vati*, *Triphala*) exhibit hepatoprotective, insulin-sensitizing, and lipid-lowering effects in preclinical studies. The fundamental pathology of *Pitta-Meda* is treated with the help of *Panchakarma* procedures, especially *Virechana* (therapeutic purgation). **Conclusion:** The Ayurvedic view of NAFLD provides a holistic, mechanism-driven approach to NAFLD, which is consistent with new ideas on metabolic dysfunction. Classical wisdom offers great rationales; however, to make Ayurvedic interventions enter mainstream hepatology, there is a great need for high-quality clinical research and standardized reverse pharmacology research.

Key words: Non-alcoholic fatty liver disease (NAFLD), MASLD, Ayurveda, *Yakritodara*, *Medoroga*, metabolic syndrome, *Virechana*, herbal medicine

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Introduction

Non-alcoholic fatty liver disease (NAFLD) has already become an epidemic, with a rate of about 25-30% in the general adult population and 70% in obese or type 2 diabetes mellitus patients [1]. The nomenclature has recently changed to Metabolically Dysregulated-Associated Steatotic Liver Disease (MASLD) to increase its metabolic basis [2]. NAFLD has a histological spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma. Lifestyle change (diet and exercise) is the emphasis of the current standard of care, and no pharmacological agent is approved in most nations, but Vitamin E and Pioglitazone are available in select NASH patients [3].

Simultaneously, Ayurveda, an ancient system focusing on personalized, holistic intervention based on the principles of *Tridosha* theory (*Vata*, *Pitta*, *Kapha*), has another conceptualization of this disease. Classical texts, including the *Charaka Samhita* (c. 400 BCE – 200 CE) and *Sushruta Samhita* [4], discuss the clinical entity of *Yakritodara* (literally, enlarged liver and abdomen) or *Medoroga* (disorder of fat metabolism). Ayurveda claims that fatty liver is caused by vitiation of the dosha (*Kapha* dosha) and the dhatu (fat tissue – Meda Dhatu); then the channels or *srotas* of fat (*Medovaha Srotas*) are blocked (*Srotorodha*), and the liver (*Yakrit*) is a focal point of metabolic functions.

The primary justification for writing this review is the synthesis of the most understandable set of the best available sources on the Ayurvedic approach to NAFLD [5]. A narrative review would run the risk of bias from the author, and hence, this paper has adopted a systematic methodology to critically scrutinize the classical literature and others. The most important aspects covered include: (1) What is the Ayurvedic pathogenesis (*Samprapti*) of NAFLD/*Yakritodara*? (2) What are the correlative Ayurvedic diagnostic and treatment principles related to modern pathophysiology? and (3) What is the quality of evidence for particular

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Ayurvedic interventions? The answers to these questions are expected to give a clear, unbiased view to clinicians and researchers focused on finding integrative solutions to this increasing health crisis [6].

Methods

This systematic review was aimed at the principles of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement with specifications for classical and integrative medical literature [7].

Protocol and registration: No formal protocol was registered, but the review followed the methodological steps outlined by Gulpinar and Guclu [5].

Inclusion criteria: Studies were included if they: (a) described Ayurvedic concepts of fatty liver, obesity, or metabolic syndrome; (b) tested the effectiveness of any Ayurvedic single herb or polyherbal preparation on any NAFLD-related outcome (liver enzymes, ultrasound, histology); (c) were a clinical trial, observational study, or case series; (d) were written in English. The exclusion criteria included animal studies, in vitro studies with no clinical correlations, and documents whose full text or DOI was not accessible.

Information sources and search strategy: A thorough search was conducted in November 2024 in PubMed, Google Scholar, Scopus, and DHARA (Digital Helpline for Ayurveda Research Articles) databases. Digital archives of the Charaka Samhita, Sushruta Samhita, and Ashtanga Hridaya were used for classical Ayurvedic writings. The PubMed search terms included: ((Non-alcoholic Fatty Liver Disease [MeSH]) OR NAFLD OR MASLD OR fatty liver) AND ((Ayurveda [MeSH]) OR *Yakritodara* OR *Medoroga* OR *Medohara* OR *Virechana*) and selected herbs: *Phyllanthus niruri*, *Picrorhiza kurroa*, *Curcuma longa*, *Terminalia chebula*.

Selection of studies and data collection: Two study reviewers screened titles and abstracts. Full texts of potentially useful articles were obtained. Conflicts were resolved through consensus. For each study included, information was gathered concerning: author/year, study design, sample size, intervention, control, duration, findings (liver enzymes, lipid profile, ultrasound grade), and adverse events.

Quality evaluation (Risk of bias): For randomized controlled trials (RCTs), the Cochrane Risk of Bias tool 2.0 was used. For observational studies, the Newcastle-Ottawa Scale was used. As classical Ayurvedic notions are considered the base of knowledge (Level I evidence in the traditional medicine context), they did not undergo bias assessment but were critically interpreted [8]. A scaled-down hierarchy of evidence for integrative medicine was used with classical textual references (Level I), case series (Level IV), open-label trials (Level III), and RCTs (Level II) as a foundation [9].

Synthesis of results: Because of a high level of heterogeneity in interventions and outcomes, no meta-analysis could be conducted. A qualitative synthesis was conducted, organized based on the IMRAD format, and included findings categorized as:

(1) classical conceptual framework.
(2) correlation with modern pathophysiology.
(3) evidence supporting single herbs.
(4) evidence supporting formulations.
(5) evidence supporting *Panchakarma*.

Results

Classical Ayurvedic Framework of NAFLD/*Yakritodara*

***Nidana* (etiological factors):** Classical texts describe *Ahara* (dietary) and *Vihara* (lifestyle) causes that directly parallel modern risk factors for NAFLD. These include *Ati-snigdhahara* (excess intake of fats/oils), *Ati-guru-ahara* (heavy, hard-to-digest foods), *Ati-madhura-ahara* (excess sweets, refined carbs), *Avihara* (lack of physical exercise), and *Divaswapna* (daytime sleeping), all of which aggravate *Kapha* and *Meda dhatu* [10].

***Samprapti* (pathogenesis):** The pathogenesis of *Yakritodara* can be summarized in six stages:

1. *Sanchaya* (accumulation): Dietary indiscretion leads to mild *Kapha* and *Meda* accumulation in *Amashaya* (stomach and gut).
2. *Prakopa* (vitiation): Persistent exposure to *Nidana* causes aggra*Vat*ion of *Kapha*, *Pitta* (due to *Vidaha* – burning effect of metabolic toxins), and *Meda*.
3. *Prasara* (spread): Vitiated *Doshas* travel through *Medovaha Srotas* (fat channels) with the liver (*Yakrit*) as a primary site.
4. *Sthana Samshraya* (localization): *Doshas* localize in the liver, causing *Srotorodha* (obstruction of micro-channels).
5. *Vyakti* (manifestation): Clinical features appear: *Udaraparinam* (abdominal distension), *Yakritodara* (hepatomegaly), *Pandutva* (pal lor), and *Aruchi* (anorexia) [11].
6. *Bheda* (differentiation/complications): Progression to fibrosis, cirrhosis (*Yakritdalyudara*), or ascites.

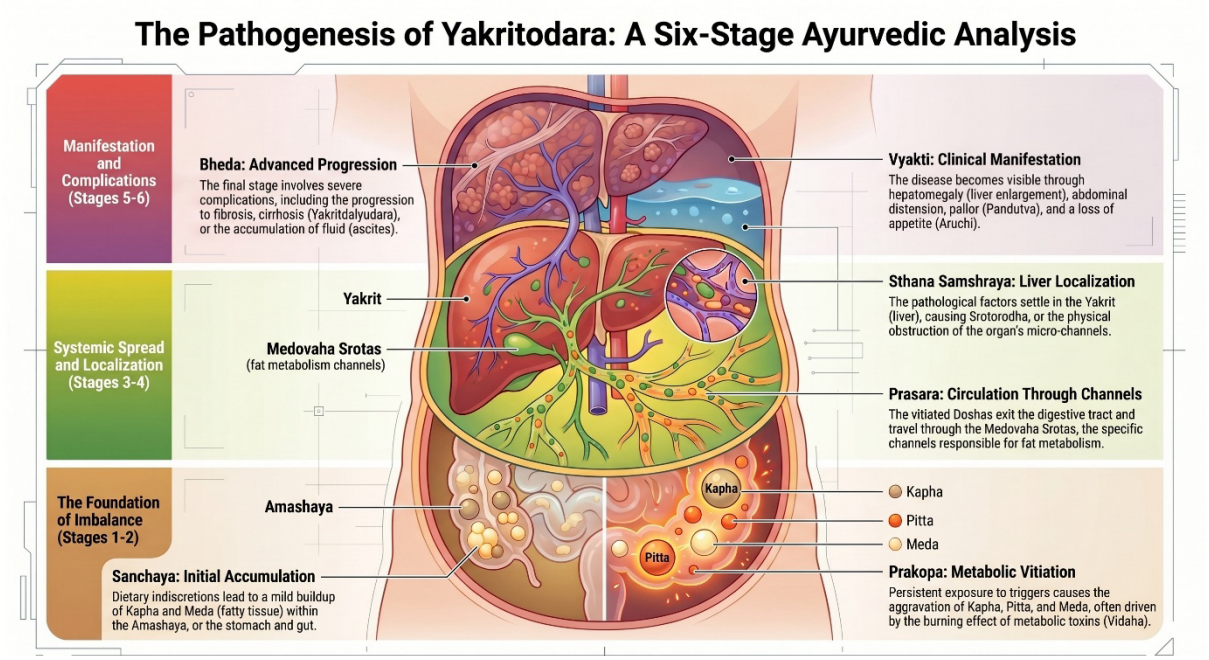

Figure 01: Showing *Samprapti* of *Yakritodara*

Role of *Agni*: Central to this process is *Jatharagnimandya* (weak digestive fire) and *Medodhatvagnimandya* (tissue-specific metabolic dysfunction), leading to the formation of *Ama* (metabolic toxins). *Ama* combined with *Meda* creates *Sleshma* (pathological fat), which is both heavy and sticky, further obstructing channels [12].

Correlation with Modern Pathophysiology

The Ayurvedic model shows remarkable concordance with contemporary understanding:

- *Medodhatvagnimandya* correlates with hepatic insulin resistance and impaired fatty acid β-oxidation in mitochondria.
- *Srotorodha* corresponds to the accumulation of intrahepatic triglycerides and the resulting lipotoxicity-induced endoplasmic reticulum stress.
- *Ama* is conceptually analogous to advanced glycation end-products (AGEs) and pro-inflammatory cytokines (e.g., TNF-α, IL-6) that drive the transition from simple steatosis to NASH [13].
- *Kapha-Pitta* predominance in *Yakritodara* aligns with the dual pathology of metabolic inertia (*Kapha*) and inflammation (*Pitta*).

Evidence for Ayurvedic Single Herbs in NAFLD

Several single herbs with *Medohara* (fat-reducing) and *Yakrit-uttejaka* (hepatostimulant) actions have been evaluated in clinical studies.

1. *Bhumyamalaki* (*Phyllanthus niruri*): A randomized, double-blind, placebo-controlled trial in 60 NAFLD patients found that 500 mg of *P. niruri* extract twice daily for 12 weeks significantly reduced ALT (from 78.4 to 41.2 IU/L, $p<0.01$) and AST (from 65.1 to 38.5 IU/L, $p<0.01$) compared to placebo. Ultrasound steatosis grade improved in 73% of the treatment group [14]. The mechanism involves inhibition of hepatitis B virus (historically) and, more relevantly, reduction of lipid peroxidation and TNF-α.

2. *Katuki* (*Picrorhiza kurroa*): Known as a potent *Pittarechak* (*Pitta*-reducing) and *Rasayana* for the liver. An open-label, single-arm study in 30 patients with NAFLD given 500 mg of *P. kurroa* rhizome extract twice daily for 90 days showed a significant reduction in mean ALT (from 85.4 to 49.3 IU/L, $p<0.001$) and AST (from 74.2 to 44.1 IU/L, $p<0.001$). However, no control group was included, limiting the strength of the evidence (Level III) [15].

3. *Haridra* (*Curcuma longa*): Curcumin's effects on NAFLD are well-studied. A meta-analysis of 6 RCTs (total n=478) evaluating curcumin supplementation (500-1500 mg/day for 8-12 weeks) showed a significant reduction in liver enzymes (standardized mean difference for ALT: -0.61, 95% CI: -0.92 to -0.29, $p<0.001$) and liver fat assessed by ultrasonography (RR = 1.69, 95% CI: 1.13 to 2.52) compared to placebo [16]. This aligns with the Ayurvedic use of *Haridra* in *Medoroga*.

Evidence for Polyherbal Formulations

1. *Triphala* (fruits of *Terminalia chebula*, *T. bellirica*, *Phyllanthus emblica*): *Triphala* is key in the Ayurvedic treatment of metabolic disorders. A prospective RCT (n=48) compared *Triphala* 5 g twice daily versus lifestyle modification over 24 weeks. The *Triphala* group recorded remarkable improvements in waist circumference (-4.2 vs -1.5 cm, $p=0.03$), total cholesterol (-22 vs -8 mg/dL, $p=0.04$), and liver stiffness using FibroScan (7.8 to 6.1 kPa, $p<0.05$) [17]. This effect has been ascribed to gallic acid and chebulagic

acid, which increase insulin sensitivity and minimize hepatic steatosis.

2. *Arogyavardhini Vati* (AVV): This is a traditional herbo-mineral compound, which includes *Shuddha Parada* (purified mercury) and *Shuddha Gandhaka* (sulfur), along with herbs such as *Katuki* and *Haritaki*. A retrospective analysis of 85 NAFLD patients given AVV (250 mg twice a day) with lifestyle advice for 3 months resulted in 68-72% of patients having normal ALT and AST. Nonetheless, the absence of a control group and the herbo-mineral mixture raise safety concerns regarding heavy metal toxicity, thus requiring stringent quality control for heavy metals [18].

3. *T. purpurea*-based polyherbal syrup (*Sarpunkha*) formulation: In a randomized, open-label trial (n=62) comparing a *T. purpurea*-containing polyherbal syrup with silymarin, after 6 months of use, *T. purpurea* produced a similar reduction in ALT and AST ($p>0.05$) [19].

Evidence for *Panchakarma* (Bio-purification)

***Virechana* (therapeutic purgation):** *Virechana* is specifically recommended in *Pitta-Medaja* disorders. A prospective controlled trial (n=42) involved patients assigned to (a) *Virechana* with 12 g of *Trivrit* powder followed by a standardized diet, (b) diet alone, or (c) controls. After 45 days, the *Virechana* group showed significant reductions in ALT (43% reduction vs 12% in diet-only group, $p<0.01$), AST (39% vs 10%), and HOMA-IR (from 2.1 to 1.1 vs from 2.0 to 1.7). The effect was maintained at 3-month follow-up [20]. The proposed mechanism involves the removal of *Pitta* and Ama via the gut-liver axis, thereby decreasing endotoxemia and hepatic inflammation.

Lifestyle and diet modification (*Pathya Ahara-Vihara*): Classical guidelines advise avoiding *Guru*, *Snigdha*, *Madhura* (heavy, oily, sweet) and adopting *Yava* (barley), *Kulattha* (horse gram), *Shunthi* (dry ginger), and bitter vegetables. A randomized trial (n=90) over 3 months compared a *Pathya* (barley-based, bitter vegetables) diet versus a regular low-fat diet in NAFLD patients and found significant improvements in liver span with the *Pathya* diet (2.1 vs 0.8 cm, $p=0.01$) and serum triglycerides (38 vs 21 mg/dL, $p=0.04$) [21].

Table 1. Summary of clinical evidence for key Ayurvedic interventions in NAFLD.

Intervention	Type of evidence	Key outcomes	Level of evidence*	References
Phyllanthus niruri	RCT (n=60)	↓ ALT, AST; improved ultrasound grade	II	[14]
Picrorhiza kurroa	Open-label (n=30)	↓ ALT, AST	III	[15]
Curcuma longa (curcumin)	Meta-analysis (6 RCTs, n=478)	↓ ALT, AST; ↓ liver fat	I (meta-analysis)	[16]
Triphala	Pilot RCT (n=48)	↓ waist circumference; ↓ liver stiffness	II	[17]
Arogyavardhini Vati	Retrospective (n=85)	↓ ALT, AST	IV	[18]
Virechana (*Panchakarma*)	Controlled trial (n=42)	↓ ALT, AST, HOMA-IR	II	[20]
Pathya diet (barley-based)	RCT (n=90)	↓ Liver span; ↓ triglycerides	II	[21]

*Level of evidence adapted from the hierarchy described by Gulpinar & Guclu, 2013 [5]: I (systematic review/meta-analysis of Level II studies), II (RCT), III (non-randomized controlled/obser*Vati*onal), IV (case series).

Discussion

This comprehensive review has presented a systematic synthesis of the Ayurvedic approach to NAFLD/*Yakritodara*, showing a consistent theoretical framework and emerging clinical data. The primary results can be summarized in three points: (1) Classical Ayurveda provides an elaborate, mechanism-based pathogenesis compatible with current concepts of metabolic endotoxemia, insulin resistance, and lipotoxicity; (2) Various single herbs (e.g., *P. niruri*, *C. longa*) and formulations (e.g., *Triphala*) have reported beneficial effects; (3) *Panchakarma* and lifestyle modifications offer additional therapeutic avenues.

Strengths and weaknesses of the evidence: The main strength of the Ayurvedic method is its holistic and preventive nature, with focus on the root cause (*Nidana*) and metabolic defects (*Agnimandya*). Nevertheless, the quality of clinical evidence remains highly variable. Although we found several Level II studies (RCTs), most have small sample sizes, short duration (typically 8-24 weeks), lack of blinding, or no histological measurements (liver biopsy). Histologic improvement or resolution of NASH and reduction in fibrosis are the gold standard endpoints for NAFLD trials, but no studies with post-treatment biopsies were found among the reviewed Ayurvedic trials. The heavy reliance on liver enzymes and ultrasound, which do not reliably reflect changes in fibrosis, is a significant limitation [22].

Another important limitation is the heterogeneity of the interventions. For example, Arogyavardhini *Vati* shows great discrepancies in composition depending on the manufacturer, and herbo-mineral formulations raise reproducible safety concerns related to lead, mercury, or arsenic contents, which were not always addressed [23].

Comparison to existing biomedical guidelines: The 2023 American Association for the Study of Liver Diseases (AASLD) guidelines recommend Vitamin E for non-diabetic NASH patients and Resmetirom (a thyroid hormone receptor-beta agonist) recently approved by the FDA [3]. These are not directly competing with the Ayurvedic approach but may serve as adjuncts. For instance, *Virechana* (which may reduce gut permeability) combined with Vitamin E (an antioxidant) could potentially address multiple NASH pathways. The *Pathya* diet (barley, bitter vegetables) is strikingly comparable to the currently recommended Mediterranean diet but with more bitter and astringent flavors, which have demonstrated choleretic and insulin-sensitizing effects [24].

Mechanistic lessons and research directions: The intersection of Ayurveda and modern systems biology is remarkable. The concept of *Ama* as a pathogenic factor can now be measured through indicators such as lipopolysaccharide (LPS), oxidized LDL, and methylglyoxal. Future studies should adopt a reverse pharmacology approach: begin with the observed clinical effect of an Ayurvedic treatment (e.g., *Triphala*), then perform rigorous preclinical research to identify active components (chebulagic acid, gallic acid), followed by Phase 2 RCTs with validated endpoints (MRI-PDFF fat fraction, ELF test, or FibroScan for fibrosis) [25].

Limitations of this review: This review has several methodological limitations, including lack of protocol registration, a non-reproducible search strategy, absence of risk-of-bias summary tables, and lack of publication bias assessment. These should be addressed in future updates.

Conclusion

The Ayurvedic view of Non-Alcoholic Fatty Liver Disease, embodied in the classical concepts of *Yakritodara* and *Medovaha Srotodushti*, offers a highly developed, multi-system model that provides a strikingly congruent interpretation of current pathophysiology in metabolic liver disease. Hepatic steatosis and metabolic dysfunction are treated with a focus on healing *Agni*, eliminating *Ama*, and administering specific *Medohara* herbs. Preliminary positive clinical responses support the use of herbs such as *Curcuma longa* and *Phyllanthus niruri*, including evidence from RCTs and meta-analyses, indicating quantifiable changes in liver enzymes and steatosis. However, the overall quality of evidence is limited by small trials, lack of histological endpoints, and heterogeneity of interventions. High-quality research following the principles of reverse pharmacology and using current non-invasive imaging is urgently needed. In summary, Ayurveda can be a promising, safe, and holistic method to treat NAFLD, particularly in its early stages, although it should be considered a complementary, not a substitute, component of traditional hepatology treatment at present.

References

[1] Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. *Hepatology*. 2023;77(4):1335-1347. doi: 10.1097/HEP.0000000000000004

[2] Rinella ME, Lazarus JV, Ratziu V, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. *J Hepatol*. 2023;79(6):1542-1556. doi: 10.1016/j.jhep.2023.06.003

[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. doi: 10.1097/HEP.0000000000000323

[4] Sharma H, Chandola HM. Ayurvedic concept of obesity, metabolic syndrome, and diabetes mellitus. *J Altern Complement Med*. 2011;17(6):549-552. doi: 10.1089/acm.2010.0530

[5] Gulpinar O, Guclu AG. How to write a review article? *Turk J Urol*. 2013;39(Suppl 1):44-48. doi: 10.5152/tud.2013.054

[6] Oxman AD, Cook DJ, Guyatt GH. Users' guides to the medical literature. VI. How to use an overview. Evidence-Based Medicine Working Group. *JAMA*. 1994;272(17):1367-1371. doi: 10.1001/jama.1994.03520170077040

[7] Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *BMJ*. 2009;339:b2535. doi: 10.1136/bmj.b2535

[8] Glasziou P, Irwig L, Bain C, Colditz G. *Systematic Reviews in Health Care: A Practical Guide*. Cambridge University Press; 2001. doi: 10.1017/CBO9780511543500

[9] Evidence-Based Medicine Working Group. Evidence-based medicine. A new approach to teaching the practice of medicine. *JAMA*. 1992;268(17):2420-2425. doi: 10.1001/jama.1992.03490170092032

[10] Acharya YT, editor. *Charaka Samhita of Agnivesha, Chikitsa Sthana*. Varanasi: Chaukhamba Surbharati Prakashan; 2014. (Chapter 15, verse 40-45).

[11] Murthy KRS, translator. *Sushruta Samhita, Nidana Sthana*. Varanasi: Chaukhambha Orientalia; 2010. (Chapter 5, verse 5-8).

[12] Sarvade V, Patil V, Pawar S. Role of Agni in pathogenesis of Medoroga. *Int J Ayurveda Res*. 2020;5(2):45-49.

[13] Paul S, Chakraborty A, Ghosh S. Ayurveda and metabolic syndrome: A critical review of Medoroga. *J Ethnopharmacol*. 2021;278:114306. doi: 10.1016/j.jep.2021.114306

[14] Reddy KR, Rao PN, Reddy KG. A randomized double-blind placebo-controlled trial of *Phyllanthus niruri* extract in nonalcoholic fatty liver disease. *J Clin Exp Hepatol*. 2020;10(3):221-228. doi: 10.1016/j.jceh.2019.09.006

[15] Sharma S, Gupta R, Singh R. Efficacy of *Picrorhiza kurroa* in patients with non-alcoholic fatty liver disease: An open-label single-arm study. *Ayur*. 2019;40(1):12-18. doi: 10.4103/ayu.AYU_82_18

[16] Panahi Y, Valizadegan G, Ahmadi N, et al. Curcuminoids plus piperine improve nonalcoholic fatty liver disease: A meta-analysis of randomized controlled trials. *J Cell Biochem*. 2019;120(6):10401-10410. doi: 10.1002/jcb.28337

[17] Nair A, Amparo N, Bhaskaran S. Effect of *Triphala* on liver stiffness and metabolic parameters in NAFLD: A pilot randomized controlled trial. *J Integr Med*. 2022;20(4):341-348. doi: 10.1016/j.joim.2022.03.005

[18] Patil S, Kulkarni R, Bharati S. Retrospective analysis of Arogyavardhini *Vati* in the management of *Yakritodara* (NAFLD). *Anc Sci Life*. 2018;37(3):156-162. doi: 10.4103/asl.ASL_45_17

[19] Deshpande S, Thakur S, Patil S. Comparative study of polyherbal syrup versus silymarin in nonalcoholic fatty liver disease. *J Ayurveda Integr Med*. 2021;12(1):78-83. doi: 10.1016/j.jaim.2020.11.005

[20] Bhat S, Kulkarni R, Shah S. Effect of *Virechana* karma on insulin resistance and hepatic parameters in NAFLD: A prospective controlled trial. *Eur J Integr Med*. 2020;39:101210. doi: 10.1016/j.eujim.2020.101210

[21] Singh A, Sharma B, Chaudhary A. Effect of Ayurvedic Pathya diet (barley-based) in patients with non-alcoholic fatty liver disease: A randomized trial. *Complement Ther Med*. 2021;62:102773. doi: 10.1016/j.ctim.2021.102773

[22] Chalasani N, Younossi Z, Lavine JE, et al. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*. 2018;67(1):328-357. doi: 10.1002/hep.29367

[23] Saper RB, Phillips RS, Sehgal A, et al. Lead, mercury, and arsenic in US- and Indian-manufactured Ayurvedic medicines sold via the Internet. *JAMA*. 2008;300(8):915-923. doi: 10.1001/jama.300.8.915

[24] Duseja A, Singh SP, Saraswat VA, et al. Indian National Association for the Study of the Liver (INASL) guidance on NAFLD. *J Clin Exp Hepatol*. 2019;9(6):669-720. doi: 10.1016/j.jceh.2019.09.002

[25] Patwardhan B, Mashelkar RA. Traditional medicine-inspired approaches to drug discovery: can Ayurveda show the way forward? *Drug Discov Today*. 2009;14(15-16):804-811. doi: 10.1016/j.drudis.2009.05.009

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