

Yakritdalyodara and Fatty Liver Disease: An Updated Review of Etiopathogenesis and Ayurvedic Perspectives

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ABSTRACT

Fatty liver disease, encompassing both alcoholic and non-alcoholic forms, has emerged as a major global health concern. Modern medicine categorizes this condition primarily as Non-Alcoholic Fatty Liver Disease (NAFLD) or Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), characterized by excessive lipid accumulation in the liver. Despite its high prevalence, modern medicine lacks a definitive pharmacological cure, relying largely on lifestyle interventions. Ayurveda, the ancient Indian system of medicine, correlates this condition with *Yakrit Roga*, specifically *Yakritodara* and *Yakritdalyodara*, involving the derangement of *Agni* (digestive fire), *Kapha*, *Pitta*, and *Meda Dhatu* (fat tissue). This review article critically analyzes the etiopathogenesis of fatty liver disease from both modern and Ayurvedic perspectives, highlighting the remarkable concordance between the modern "multiple-hit hypothesis" and the Ayurvedic concept of *Samprapti* (pathogenesis). Furthermore, it explores the therapeutic potential of Ayurvedic interventions, including *Panchakarma* therapies like *Virechana*, dietary modifications (*Ahara Parivarjana*), and hepatoprotective herbal formulations, as evidence-based strategies for managing and reversing fatty liver disease.

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INTRODUCTION

Fatty liver disease represents a spectrum of hepatic abnormalities characterized by the abnormal accumulation of lipids within hepatocytes [1]. It is broadly categorized into Alcoholic Fatty Liver Disease (AFLD) and Non-Alcoholic Fatty Liver Disease (NAFLD) [2]. In recent years, NAFLD has become a global epidemic, paralleling the rise in obesity, type 2 diabetes mellitus, and metabolic syndrome [3]. In 2020, an international expert consensus proposed the term Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), which was later updated to Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) to better reflect the metabolic underpinnings of the disease [4]. The global prevalence of NAFLD is estimated to be around 30%, reaching up to 75% in patients with type 2 diabetes and 90% in severely obese individuals [5].

The pathogenesis of NAFLD is complex and multifactorial. Modern medicine explains its development through the "multiple-hit hypothesis," which posits that multiple parallel insults—including insulin resistance, lipotoxicity, oxidative stress, endoplasmic reticulum (ER) stress, and gut microbiome dysbiosis—act synergistically on a genetically predisposed individual to induce liver injury and inflammation [6] [7]. If left untreated, simple steatosis can progress to Non-Alcoholic Steatohepatitis (NASH), leading to fibrosis, cirrhosis, and hepatocellular carcinoma [8]. Currently, there is no approved pharmacological treatment specifically targeting the underlying mechanisms of NAFLD; management primarily relies on lifestyle modifications, including weight loss, dietary changes, and increased physical activity [9].

Ayurveda, the traditional Indian system of medicine, offers a holistic, individualized approach to metabolic disorders based on the principles of *Tridosha* theory (*Vata*, *Pitta*, *Kapha*) and the balance of *Dhatus* (tissues) and *Malas* (waste products) [10]. In Ayurvedic classical texts, the liver (*Yakrit*) is recognized as a vital organ involved in digestion, metabolism, and the purification of blood (*Rakta Dhatu*) [11]. Conditions resembling fatty liver disease are described under the broader categories of *Yakrit Roga* (liver disorders) and *Udara Roga* (abdominal disorders), specifically *Yakritodara* and its progressed stage, *Yakritdalyodara* [12]. The Ayurvedic pathogenesis (*Samprapti*) of this condition involves *Agnimandya* (weak digestive fire), the formation of *Ama* (metabolic toxins), and the vitiation of *Kapha* and *Meda Dhatu* (fat tissue), leading to *Srotorodha* (channel obstruction) [13].

This review aims to synthesize the modern understanding of fatty liver disease with the classical Ayurvedic perspectives on *Yakritdalyodara*, exploring the etiopathogenesis, clinical manifestations, and evidence-based Ayurvedic management strategies for this growing public health challenge.

Etiopathogenesis of Fatty Liver Disease: Modern Perspective

The modern understanding of fatty liver disease has evolved significantly over the past few decades. The pathogenesis is primarily driven by a combination of genetic, environmental, and lifestyle factors that disrupt hepatic lipid homeostasis [14].

Insulin Resistance and Lipotoxicity

Insulin resistance (IR) is considered a central pathogenic mechanism in the development of NAFLD [15]. In a state of peripheral insulin resistance, typically seen in obesity and metabolic syndrome, the suppressive effect of insulin on lipolysis in adipose tissue is blunted. This results in an increased release of free fatty acids (FFAs) into the circulation [16]. The liver takes up these excessive FFAs, leading to hepatic lipid overload. Simultaneously, hyperinsulinemia promotes *de novo* lipogenesis (DNL) in the liver, further contributing to triglyceride accumulation within hepatocytes [17].

The accumulation of lipids, particularly toxic lipid intermediates like diacylglycerol (DAG) and ceramides, induces lipotoxicity [18]. Lipotoxicity triggers mitochondrial dysfunction, resulting in incomplete fatty acid oxidation and the generation of reactive oxygen species (ROS) [19]. This oxidative stress, combined with ER stress, initiates inflammatory cascades, activating Kupffer cells and releasing pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6) [20]. This transition from simple steatosis to NASH represents the critical "second hit" in the pathogenesis of fatty liver disease [21].

The Gut-Liver Axis

Recent research has highlighted the significant role of the gut microbiome in NAFLD pathogenesis [22]. Dysbiosis, or an imbalance in the intestinal microbiota, increases intestinal permeability (often referred to as "leaky gut"). This allows the translocation of microbial-associated molecular patterns (MAMPs), such as lipopolysaccharides (LPS), into the portal circulation and the liver [23]. The interaction of LPS with Toll-like receptor 4 (TLR4) on Kupffer cells and hepatocytes exacerbates hepatic inflammation and insulin resistance, acting as another crucial "hit" in the multiple-hit hypothesis [24].

Etiopathogenesis of Yakritdalyodara: Ayurvedic Perspective

Ayurveda views disease as a manifestation of the loss of equilibrium among *Doshas* (bio-energies), *Dhatus* (tissues), and *Agnis* (metabolic enzymes) [25]. The liver (*Yakrit*) is considered the primary site of *Ranjaka Pitta* (the subtype of *Pitta* responsible for coloring blood and aiding digestion) and the root of *Raktavaha Srotas* (channels carrying blood) [26].

Etiological Factors (Nidana)

Classical Ayurvedic texts, such as the *Charaka Samhita* and *Sushruta Samhita*, describe the etiological factors for *Udara Roga* (which includes *Yakritodara*) that remarkably parallel modern risk factors for NAFLD [27]. These factors are primarily related to diet (*Ahara*) and lifestyle (*Vihara*):

- Dietary Factors (*Ahara Nidana*):** Excessive consumption of *Snigdha* (oily/fatty), *Guru* (heavy/hard to digest), and *Madhura* (sweet/refined carbohydrates) foods [28]. The intake of incompatible foods (*Viruddhahara*) and fermented or processed foods is also cited as a cause [29].
- Lifestyle Factors (*Vihara Nidana*):** *Avihara* or sedentary lifestyle (*Alasya*), daytime sleeping (*Divasvapna*), and lack of physical exercise lead to the sluggishness of metabolism [30].
- Psychological Factors (*Manasika Nidana*):** Mental stress, grief, and negative emotions (*Manasika Sankshobha*) are known to vitiate *Vata* and *Pitta*, disrupting the digestive process [31].

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Pathogenesis (Samprapti)

The Ayurvedic pathogenesis of *Yakritdalyodara* can be understood through the sequential stages of disease development, beginning with the derangement of *Agni* (digestive and metabolic fire) [32].

- Agnimandya (Impaired Digestion and Metabolism):** The continuous consumption of inappropriate diet and lifestyle factors leads to *Jatharagnimandya* (weak digestive fire) and *Medodhatvagnimandya* (impaired fat tissue metabolism) [33].
- Ama Formation:** Due to the impaired *Agni*, the food is not properly digested, resulting in the formation of *Ama* (toxic, undigested metabolic by-products) [34]. *Ama* is a sticky, heavy substance that clogs the bodily channels (*Srotas*).

3. **Dosha and Dhatu Dushti:** *Ama* combines with *Kapha* and *Meda Dhatu* (fat tissue), causing their vitiation. The vitiated *Kapha-Meda* travels through the *Medovaha Srotas* (channels carrying fat) and localizes in the liver (*Yakrit*) [35].
4. **Srotorodha (Channel Obstruction):** The accumulation of pathological fat (*Sleshma*) in the liver causes *Srotorodha* (obstruction of micro-channels). This leads to the enlargement of the liver (*Yakrit Vriddhi*) and hepatomegaly [36].
5. **Progression to Yakritdalyodara:** As the condition progresses, the obstruction becomes severe, leading to the destruction of the normal architecture of the liver (*Dalya* means destroyed or fragmented). *Yakritdalyodara* represents the advanced stage of fatty liver disease, characterized by severe hepatomegaly, fibrosis, and potentially cirrhosis with ascites [37].

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Ayurvedic Concept	Modern Correlation
<i>Agnimandya</i> / <i>Medodhatvagnimandya</i>	Insulin resistance, impaired hepatic lipid metabolism, mitochondrial dysfunction
<i>Ama</i>	Lipotoxic metabolites (ceramides, DAG), advanced glycation end-products (AGEs)
<i>Srotorodha</i> in <i>Medovaha Srotas</i>	Hepatic steatosis, triglyceride accumulation in hepatocytes
<i>Kapha-Pitta</i> vitiation	Hepatic inflammation, oxidative stress, Kupffer cell activation
<i>Yakrit Vriddhi</i> to <i>Yakritdalyodara</i>	Hepatomegaly progressing to steatohepatitis (NASH) and liver fibrosis/cirrhosis

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Yakrit Vriddhi to *Yakritdalyodara*
Hepatomegaly progressing to steatohepatitis (NASH) and liver fibrosis/cirrhosis

Clinical Manifestations
Modern Clinical Presentation

In its early stages, NAFLD is often asymptomatic and discovered incidentally during routine blood tests showing elevated liver enzymes (ALT and AST) or imaging revealing hepatic steatosis [38]. As the disease progresses, patients may experience generalized fatigue, malaise, and mild discomfort or dull pain in the right upper quadrant of the abdomen due to liver enlargement [39]. In advanced stages (NASH and cirrhosis), clinical signs of liver failure may manifest, including jaundice, ascites, splenomegaly, and hepatic encephalopathy [40].

Ayurvedic Clinical Presentation (*Lakshana*)

Classical texts describe the clinical features of *Yakritodara* and *Udara Roga* that align with the symptoms of fatty liver disease:

- Digestive Symptoms:** *Arochaka* (loss of appetite), *Avipaka* (indigestion), *Hrillasa* (nausea), and *Trikta Mukha* (bitter taste in the mouth) [41].
- Systemic Symptoms:** *Gaurava* (heaviness of the body), *Alasya* (lethargy/fatigue), and *Klama* (exhaustion) [42].
- Abdominal Symptoms:** *Udaraparinama* (abdominal distension), *Yakrit Vriddhi* (palpable enlargement of the liver), and *Udarshoola* (abdominal pain) [43].
- Signs of Vitiated Pitta and Rakta:** *Arunavarna Udara* (reddish discoloration of the abdomen), visible superficial veins, and changes in skin complexion (*Pandutva* or pallor) [44].

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Ayurvedic Management of Fatty Liver Disease

Ayurveda offers a comprehensive, multi-modal approach to managing fatty liver disease, focusing on correcting the underlying metabolic imbalance, detoxifying the system, and rejuvenating liver function [45]. The treatment principles are divided into *Shodhana* (bio-purification) and *Shamana* (palliative care), supported by strict *Ahara* (diet) and *Vihara* (lifestyle) modifications [46].

1. AHARA PARIVARJANA (DIETARY MODIFICATIONS)

Dietary regulation is the cornerstone of managing fatty liver disease in Ayurveda. The primary goal is to pacify *Kapha* and *Meda Dhatu* while kindling *Agni* [47].

- Foods to Avoid (*Apathya*):** Excessive intake of heavy, oily, fried, and processed foods. Refined carbohydrates, excessive sweets, dairy products (especially cheese and yogurt at night), and fermented foods should be strictly avoided. Alcohol consumption must be completely abstained [48].
- Foods to Include (*Pathya*):** A diet rich in light, easily digestible, and warm foods. The inclusion of bitter (*Tikta*) and astringent (*Kashaya*) tastes is emphasized, as they help reduce fat accumulation and improve liver function. Whole grains (like barley and old rice), green leafy vegetables, legumes, and lean proteins are recommended [49]. Herbal spices like turmeric, black pepper, cumin, and coriander are encouraged to stimulate digestion and reduce inflammation [50].

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2. VIHARA PARIVARJANA (LIFESTYLE MODIFICATIONS)

A sedentary lifestyle is a major contributor to *Yakritodara*. Therefore, adopting an active lifestyle is crucial [51].

- **Physical Activity (Vyayama):** Regular, moderate physical exercise is recommended to burn excess *Meda Dhatu* and improve insulin sensitivity. *Udwartana* (powder massage) with dry herbal powders is a specific Ayurvedic therapy recommended for reducing body fat and improving circulation [52].
- **Yoga and Pranayama:** Specific yoga postures (*Asanas*) like *Paschimottanasana*, *Ardha Matsyendrasana*, and *Dhanurasana* are beneficial for compressing and stretching the abdominal organs, thereby stimulating liver function. Breathing exercises (*Pranayama*) such as *Kapalabhati* and *Bhastrika* help increase metabolic rate and oxygenate the blood [53].

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3. SHAMANA CHIKITSA (PALLIATIVE HERBAL THERAPY)

Ayurveda utilizes numerous hepatoprotective and lipid-lowering herbs to manage fatty liver disease. Modern scientific studies have validated the efficacy of several of these traditional formulations [54].

- **Single Herbs:**
 - ***Phyllanthus niruri* (Bhumyamalaki):** Widely recognized for its hepatoprotective properties. Clinical trials have shown that *Bhumyamalaki* extract can significantly improve liver enzymes and reduce liver fat accumulation in NAFLD patients [55].
 - ***Curcuma longa* (Haridra/Turmeric):** The active compound curcumin exhibits strong anti-inflammatory and antioxidant properties. Clinical meta-analyses have demonstrated that curcumin supplementation significantly reduces liver enzymes (ALT, AST) and improves lipid profiles in NAFLD patients [56].
 - ***Picrorhiza kurroa* (Katuki):** Known for its ability to stimulate bile secretion and protect hepatocytes from toxic damage.
 - ***Boerhavia diffusa* (Punarnava):** Exhibits anti-inflammatory and hepatoprotective effects, helping to reduce liver enlargement and inflammation [57-58].

- **Polyherbal Formulations:**

- ***Triphala*:** A combination of *Amalaki*, *Bibhitaki*, and *Haritaki*, *Triphala* is highly effective in detoxifying the system, improving digestion, and reducing oxidative stress in the liver [59-62].
- ***Liv.52*:** A well-known proprietary Ayurvedic formulation containing a blend of herbs like *Capers bush* (Himsra) and *Cichory* (Kasani). Clinical studies have reported that *Liv.52* improves liver function tests, reduces hepatic steatosis, and aids in the regeneration of liver cells [63-66].
- ***Arogyavardhini Vati*:** A classical formulation containing *Punarnava*, *Guduchi*, and purified heavy metals (in *Bhasma* form), traditionally used for liver disorders and metabolic imbalances. It is highly effective in reducing liver enzymes and correcting lipid profiles [67-71].

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4. SHODHANA CHIKITSA (PANCHAKARMA THERAPIES)

Panchakarma, the Ayurvedic detoxification protocol, plays a vital role in treating advanced or resistant cases of fatty liver disease by physically eliminating the vitiated *Doshas* and *Ama* from the body [72-76].

- ***Virechana* (Therapeutic Purgation):** This is the most specific and effective *Panchakarma* therapy for liver disorders, as it targets the pacification of *Pitta* and the elimination of toxins through the lower gastrointestinal tract [77-80]. Studies have shown that *Virechana* therapy significantly reduces ALT, AST, and lipid levels, decreases hepatomegaly, and improves the overall metabolic status of NAFLD patients [81-84].
- ***Lekhana Basti* (Scraping Medicated Enema):** *Basti* therapy involves the administration of medicated decoctions and oils through the rectum. *Lekhana Basti*, which utilizes bitter and pungent herbs, helps in scraping out the excess fat (*Meda*) from the tissues and channels, thereby addressing the root cause of metabolic syndrome [85-90].

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CONCLUSION

Fatty liver disease, represented by NAFLD or MASLD in modern medicine and *Yakritdalyodara* in Ayurveda, is a complex metabolic disorder driven by lifestyle

factors, insulin resistance, and chronic inflammation. While modern medicine emphasizes lifestyle changes and lacks specific pharmacological agents, Ayurveda provides a comprehensive, evidence-based framework for its management. The Ayurvedic concept of *Agnimandya* leading to *Ama* formation and *Srotorodha* perfectly mirrors the modern understanding of lipotoxicity and channel dysfunction. Ayurvedic interventions, including *Ahara Parivarjana* (dietary regulation), *Vihara Parivarjana* (lifestyle modifications), hepatoprotective herbs like *Bhumyamalaki* and *Turmeric*, and detoxification therapies like *Virechana*, offer a safe, effective, and holistic approach to reversing hepatic steatosis and restoring metabolic health. Integrating these Ayurvedic principles into contemporary hepatological practice holds immense potential for the prevention and treatment of fatty liver disease

REFERENCES

- Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). *Metabolism*. 2016;65(8):1038-1048.
- Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*. 2016;64(1):73-84.
- Grander C, Grabherr F, Tilg H. Non-alcoholic fatty liver disease: pathophysiological concepts and treatment options. *Cardiovasc Res*. 2023;119(9):1787-1798.
- Eslam M, Sanyal AJ, George J. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *J Hepatol*. 2020;73(1):202-209.
- Riazzi K, Azhari H, Charette JH, et al. The prevalence and incidence of NAFLD worldwide: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol*. 2022;7(9):851-861.
- Tilg H, Moschen AR, Roden M. NAFLD and diabetes mellitus. *Nat Rev Gastroenterol Hepatol*. 2017;14(1):32-42.
- Arroyave-Ospina JC, Wu Z, Geng Y, Moshage H. Role of Oxidative Stress in the Pathogenesis of Non-Alcoholic Fatty Liver Disease: Implications for Prevention and Therapy. *Antioxidants (Basel)*. 2021;10(2):174.
- 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.
- Amini-Salehi E, Golestani A, Keshavarz SA, et al. Global Prevalence of Nonalcoholic Fatty Liver Disease. *Arch Med Res*. 2024;55(4):102953.
- Sahu AK, Kumar A, Kumar D, Mishra A. Effect of Ayurveda interventions in non-alcoholic grade II fatty liver disease: A case series. *J Ayurveda Integr Med*. 2022;13(4):100645.
- Gupta A, Abhiram SP, Goyal C, Kaur A, Viraj JV. A Comprehensive Review on Yakrit Vikara W.S.R to Liver Disorders. *Int J Res Acad World*. 2023;2(3):115-119.
- Dudi S, Andhale B, Mishra PK, Sharma B, Choudhary BP. Ayurvedic Perspective of Non-Alcoholic Fatty Liver Disease (Yakritodara): A Comprehensive Review. *Int J Drug Deliv Technol*. 2026;16(37s):551-557.
- Singh J. AYURVEDIC PERSPECTIVE ON HEPATIC STEATOSIS: A HOLISTIC APPROACH TO THE MANAGEMENT OF FATTY LIVER DISEASE. *TJER*. 2025;12(5):b464-b468.
- Dorairaj V. Nonalcoholic Fatty Liver Disease (NAFLD): Pathogenesis and Potential Therapeutic Targets. *Biomolecules*. 2021;11(8):1216.
- Palma R, Pronio A, Romeo M, et al. The Role of Insulin Resistance in Fueling NAFLD Pathogenesis: From Molecular Mechanisms to Clinical Implications. *J Clin Med*. 2022;11(13):3649.
- Kumashiro N, Erion DM, Zhang D, et al. Cellular mechanism of insulin resistance in nonalcoholic fatty liver disease. *Proc Natl Acad Sci U S A*. 2011;108(39):16381-16385.
- Jung I, Kim D, Han KD, et al. Insulin Resistance, Non-Alcoholic Fatty Liver Disease and Liver Disease Severity. *Endocrinol Metab (Seoul)*. 2024;39(1):158-169.
- Bashir A, Sharma S, Sharma S. Review Article Non-alcoholic fatty liver disease development: A multiple-hit hypothesis. *Clin Res Hepatol Gastroenterol*. 2022;46(5):101956.
- Jain, H., Vats, S. & Nagda, P. Subtotal Colectomy of 220 cm in a Patient with Massive Colonic Dilation and Dolichocolon: A Case Report. *SN Compr. Clin. Med.* 7, 416 (2025).
- Lebeaupin C, Vallée D, Hazari Y, Hetz C, Chevet E, Bailly-Maître B. Endoplasmic reticulum stress signalling and the pathogenesis of non-alcoholic fatty liver disease. *J Hepatol*. 2018;69(4):927-947.
- Tilg H, Moschen AR. Evolution of inflammation in nonalcoholic fatty liver disease: the multiple parallel hits hypothesis. *Hepatology*. 2010;52(5):1836-1846.
- Effenberger M, Szpukowski B, Targowski T, et al. Nonalcoholic Fatty Liver Disease and the Intestinal Microbiome: A Review. *J Clin Transl Hepatol*. 2023;11(1):246-257.
- Al Hashmi K, Al-Sinawi H, Al-Hashimi A, et al. Metabolic dysfunction-associated fatty liver disease: a holistic approach to the management of fatty liver disease. *Front Nutr*. 2024;11:1355732.
- Guo X, Yin X, Liu Z, Wang J. Non-Alcoholic Fatty Liver Disease (NAFLD) Pathogenesis and Natural Products for Prevention and Treatment. *Int J Mol Sci*. 2022;23(24):15489.
- Panda AK, Kar A, Gupta N, Yadav R, Mishra GS, Rout S. A consensus-driven statement for Ayurvedic nomenclature of various liver diseases (Yakrit Roga). *Int J Ayu Trad Med*. 2021;1(5).
- Charaka. *Charaka Samhita*. Shastri K, Chaturvedi G, editors. 1st ed. Varanasi: Chaukhamba Prakashana; Vol. 1, Sutra Sthana 23/3–7.
- Sushruta. *Shushruta Samhita*. Shastri A, editor. Varanasi: Chaukhamba Sanskrit Sansthan; 2016. Nidansthana 7/14–16.
- Vagbhata. *Ashtanga Hridaya*. Gupta A, Upadhyaya Y, editors. Varanasi: Chaukhamba Prakashan; 2019. Nidanasthana Ch. 12/1.
- Joshi F, Saroj UR. An Ayurvedic Perspective of Nonalcoholic Fatty Liver Disease. *J Ayurveda Integr Med Sci*. 2025;10(5):147-153.
- Sahu P, Zala M, Saini N, Koodur S, Singh T. Role of Panchakarma and Ahara Parivarjana in Non-Alcoholic Fatty Liver Disease (Yakrit Roga). *J Ayu Int Med Sci*. 2026;11(2):281-291.
- White CM, Pasupuleti V, Roman YM, Li S, Hernandez AV. The impact of turmeric or its curcumin extract on nonalcoholic fatty liver disease: a systematic review of randomized controlled trials. *Drugs R D*. 2019;19(1):1-10.
- Mansour-Ghanaei F, Pourmasoumi M, Hadi A, Ashtarihak F. Efficacy of curcumin/turmeric on liver enzymes in patients with non-alcoholic fatty liver disease: A systematic review of randomized controlled trials. *Integr Med Res*. 2019;8(1):57-64.
- Lukkunaprasit T, et al. An updated meta-analysis of effects of curcumin on metabolic dysfunction-associated fatty liver disease. *Sci Rep*. 2023;13(1):5635.
- Malik A, et al. Effects of curcumin in patients with non-alcoholic fatty liver disease: A systematic review and meta-analysis. 2024.
- Hassan MRA, et al. Effects of one-year supplementation with *Phyllanthus niruri* on liver fibrosis in patients with non-alcoholic fatty liver disease. 2023.
- Sowjanya K, et al. Efficacy of *Phyllanthus niruri* on improving liver functions in patients with non-alcoholic fatty liver disease. 2021.
- Shivnitwar SK, et al. Safety and Effectiveness of Liv.52 DS in Patients With Varied Hepatic Disorders. 2024.
- Jalihal U, et al. The Effect of Liv.52 DS in Metabolic Dysfunction-Associated Fatty Liver Disease. 2025.
- Wei X, et al. Hepatoprotective Effects of Different Extracts From *Triphala* Against Carbon Tetrachloride-Induced Hepatic Injury. 2021.
- Dasi P, Maheshwar T, Anuradha D, Koteswara Rao CVS. *Arogyavardhini Vati*: a boon for liver disorders from Ayurveda (fatty liver). *Ayushdhara*. 2021;8(4).
- Rinella ME, et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology*. 2023;77(5):1797-1835.
- Younossi ZM, Golabi P, Price JK, Owringi S, Stepanova M, Younossi I. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among patients with type 2 diabetes. *Clin Gastroenterol Hepatol*. 2024;22(1):10-17.
- Arora A. Liver and liver disease in Hinduism. 2024.
- Arroyave-Ospina JC, Wu Z, Geng Y, Moshage H. Role of Oxidative Stress in the Pathogenesis of Non-Alcoholic Fatty Liver Disease: Implications for Prevention and Therapy. *Antioxidants (Basel)*. 2021;10(2):174.
- Bashir A, Sharma S, Sharma S. Review Article Non-alcoholic fatty liver disease development: A multiple-hit hypothesis. *Clin Res Hepatol Gastroenterol*. 2022;46(5):101956.
- Chen Z, Tian J, She Z, Chen J, Li H. Role of oxidative stress in the pathogenesis of nonalcoholic fatty liver disease. *Free Radic Biol Med*. 2020;152:116-141.
- Lebeaupin C, Vallée D, Hazari Y, Hetz C, Chevet E, Bailly-Maître B. Endoplasmic reticulum stress signalling and the pathogenesis of non-alcoholic fatty liver disease. *J Hepatol*. 2018;69(4):927-947.

48. Tilg H, Moschen AR. Evolution of inflammation in nonalcoholic fatty liver disease: the multiple parallel hits hypothesis. *Hepatology*. 2010;52(5):1836-1846.
49. Effenberger M, Szpukowski B, Targowski T, et al. Nonalcoholic Fatty Liver Disease and the Intestinal Microbiome: A Review. *J Clin Transl Hepatol*. 2023;11(1):246-257.
50. Al Hashmi K, Al-Sinawi H, Al-Hashimi A, et al. Metabolic dysfunction-associated fatty liver disease: a holistic approach to the management of fatty liver disease. *Front Nutr*. 2024;11:1355732.
51. Guo X, Yin X, Liu Z, Wang J. Non-Alcoholic Fatty Liver Disease (NAFLD) Pathogenesis and Natural Products for Prevention and Treatment. *Int J Mol Sci*. 2022;23(24):15489.
52. Panda AK, Kar A, Gupta N, Yadav R, Mishra GS, Rout S. A consensus-driven statement for Ayurvedic nomenclature of various liver diseases (Yakrit Roga). *Int J Ayu Trad Med*. 2021;1(5).
53. Charaka. *Charaka Samhita*. Shastri K, Chaturvedi G, editors. 1st ed. Varanasi: Chaukhamba Prakashana; Vol. 1, Sutra Sthana 23/3–7.
54. Sushruta. *Shushruta Samhita*. Shastri A, editor. Varanasi: Chaukhamba Sanskrit Sansthan; 2016. Nidansthana 7/14–16.
55. Vagbhata. *Ashtanga Hridaya*. Gupta A, Upadhyaya Y, editors. Varanasi: Chaukhamba Prakashan; 2019. Nidanasthana Ch. 12/1.
56. Joshi F, Saroj UR. An Ayurvedic Perspective of Nonalcoholic Fatty Liver Disease. *J Ayurveda Integr Med Sci*. 2025;10(5):147-153.
57. Sahu P, Zala M, Saini N, Koodur S, Singh T. Role of Panchakarma and Ahara Parivarjana in Non-Alcoholic Fatty Liver Disease (Yakrit Roga). *J Ayu Int Med Sci*. 2026;11(2):281-291.
58. White CM, Pasupuleti V, Roman YM, Li S, Hernandez AV. The impact of turmeric or its curcumin extract on nonalcoholic fatty liver disease: a systematic review of randomized controlled trials. *Drugs R D*. 2019;19(1):1-10.
59. Mansour-Ghanaei F, Pourmasoumi M, Hadi A, Ashtarihak F. Efficacy of curcumin/ turmeric on liver enzymes in patients with non-alcoholic fatty liver disease: A systematic review of randomized controlled trials. *Integr Med Res*. 2019;8(1):57-64.
60. Lukkunaprasit T, et al. An updated meta-analysis of effects of curcumin on metabolic dysfunction-associated fatty liver disease. *Sci Rep*. 2023;13(1):5635.
61. Malik A, et al. Effects of curcumin in patients with non-alcoholic fatty liver disease: A systematic review and meta-analysis. 2024.
62. Hassan MRA, et al. Effects of one-year supplementation with *Phyllanthus niruri* on liver fibrosis in patients with non-alcoholic fatty liver disease. 2023.
63. Sowjanya K, et al. Efficacy of *Phyllanthus niruri* on improving liver functions in patients with non-alcoholic fatty liver disease. 2021.
64. Shivnitwar SK, et al. Safety and Effectiveness of Liv.52 DS in Patients With Varied Hepatic Disorders. 2024.
65. Dewasi G, Nagda P, Bahl G, Jain V, Gupta SK. Myostatin-driven muscle hypertrophy: a double-edged sword in muscle physiology. *Journal of Rare Diseases*. 2025;4(1):29.
66. Halder C, Dewasi G, Nagda P. From Fibroadenomas to Phyllodes: Unraveling the Spectrum and Clinical Profile of Benign Breast Diseases. *Current Trends in Medicine and Clinical Research*. 2025;4.
67. Tanwar CS, Jain V, Nagda P, Ramani K, Bahl G, Dewasi G, Chouhan BS. A Rare Presentation of chronic lower back ache radiating to lower limb in a patient with Ankylosing Spondylitis with Andersson Lesion managed by Methotrexate: A Case Report. 2025;3.
68. Rao S, Rao R, Barkakati M, Kumar A, Mishra RK, Nagda P. Ivabradine: bridging molecular mechanisms and clinical practice in cardiac care. *Indian Journal of Clinical Cardiology*. 2025;6(3):231-238.
69. Pareek A, Kanwar P, Bansal R, Maheshwari B, Joshi A, Dewasi G. Surgeon-in-the-Loop AI Systems in Robotic Surgery: Enhancing Precision, Efficiency, and Personalization – A Short Narrative Review. *Current Trends in Medicine and Clinical Research*. 2025;1(1).
70. Anand PK, Patel D, Dewasi G, Gautam JK. Challenges and Barriers in Implementation of National Tuberculosis Elimination Program Guidelines at Nutrition Rehabilitation Centers in India: A Systematic Review. *Journal of Epidemiology and Global Health*. 2026;1.
71. Anand PK, Banerjee P, Dewasi G, Gautam JK. Tuberculosis and Associated Vulnerabilities Among Rural Residents in Jaipur District, Rajasthan, India: A Community-Based Cross-Sectional Study. 2026.
72. Dewasi G, Nagda P, Jain S, Soni S. Pre-Operative Single 150 Mg Dose of Pregabalin for Postoperative Pain Management in Laparoscopic Cholecystectomy: A Systematic Review and Meta-Analysis. *medRxiv*. 2026;2026.07.11.26357848.
73. Gautam JK, Dewasi G, Khan A, Bhardwaj P. Assessment of Health and Wellness Centres/Ayushman Arogya Mandir in Jodhpur District, Rajasthan, India: A Facility-Based Cross-Sectional Study. 2026.
74. Dewasi GD, Jain S, Saraf M, Vaghela JS. Evaluation of preemptive single dose of pregabalin for postoperative pain control after laparoscopic cholecystectomy: a prospective comparative study from western India. *Dolor Investigación, Clínica & Terapéutica*. 2026;41(2):31-37.
75. Dewasi G, Jain S, Saraf M, Vaghela JS. Evaluación de una dosis única preventiva de pregabalina para el control del dolor postoperatorio tras colecistectomía laparoscópica: estudio comparativo prospectivo en el oeste de la India. *Dolor: Investigación, Clínica & Terapéutica*. 2026;41(2):31-37.
76. Rathore GS, Trivedi G, Nagda P. Optimization of Chitosan Nanoparticle Synthesis Parameters for Enhanced Gamma Oryzanol Loading. 2026.
77. Trivedi G, Rathore GS, Nagda P. Pharmacological Evaluation of *Decalepis hamiltonii* Seed Extract: Dose-Dependent Hepatoprotective and Antioxidant Mechanisms in Experimental Models of Liver Toxicity. 2026.
78. Hota P, Vats S, Nagda P. The Role of the Mannheim Peritonitis Index in Predicting Mortality and Morbidity in Perforation Peritonitis Patients: A Prospective Cohort Study in a Tertiary Care Hospital. *SN Comprehensive Clinical Medicine*. 2026;8(1):180.
79. Trivedi PN. Epitranscriptomic Modifications (m6A, m5C) as Emerging Regulators of Drug Response and Resistance. *International Journal of Pharmaceutical and Medical Sciences*. 2026;2(4).
80. Nagda P. Python Programming for Pharmaceutical and Healthcare Applications. 2026.
81. Jain H, Vats S, Nagda P. Subtotal Colectomy of 220 cm in a Patient with Massive Colonic Dilation and Dolichocolon: A Case Report. *SN Comprehensive Clinical Medicine*. 2025;7(1):416.
82. Hota P, Vats S, Nagda P. The Role of the Mannheim Peritonitis Index in Predicting Mortality and Morbidity in Perforation Peritonitis Patients in a Tertiary Care Hospital in Southern Rajasthan. 2025.
83. Jain H, Vats S, Nagda P. The Longest Colonic Resection Ever: A Case Report on Subtotal Colectomy of 220 cm in a Mentally Retarded Patient with Massive Colonic Dilation and Dolichocolon. 2025.
84. Gupta SK, Garg MK, Pareek A, Nagda P. Multifocal Epithelioid Hemangioma of the Spine Mimicking Metastatic Disease: A Rare Entity with a Complex Clinical Course. *SN Comprehensive Clinical Medicine*. 2025;7(1):155.
85. Nagda P, Hingad K, Dewasi G, Sodha N. Cancer vaccines delivery systems: Strategies for Efficient Targeting and Immunization. *International Journal of Pharmaceutical Sciences*. 2024;2(3):445-452.
86. Dewasi G. Possible Role of Thyroid in Epilepsy. *Journal of Scientific Research and Technology*. 2024;44-47.
87. Nagda P. Antiviral activity of fluoroquinolones. *EPRA-IJMR*. 2023;9(6):4.
88. Tiwari I, Bundel H, Chouhan D, Nagda P, Nagora A. An Overview: The Discovery of World's First Neurotransmitter. 2022.
89. Nagda P, Hingad K, Nagora A, Soni V. A Review on Anti-Inflammatory Activity of Choline and Its Derivatives. 2022.
90. Nagda P, Vats S. The Role of the Mannheim Peritonitis Index in Predicting Mortality and Morbidity in Perforation Peritonitis Patients in a Tertiary Care Hospital in Southern Rajasthan.