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Comparing Allopathic and Herbal Treatments in Non-Alcoholic Fatty Liver Disease: A Novel Review of Emerging Phytopharmacological Interventions

Mamatha H S[1]*, Ashok Kumar B S[2], Disha N S[3], Mohammed Khalid[4], Bhargavi S[5]

[1,4]Department of Pharmaceutics, R. L. Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563103, Karnataka,India.

[2]Department of Pharmacognosy, R. L. Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563103, Karnataka, India.

[3]Department of Pharmaceutical Chemistry, R. L. Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563103, Karnataka, India.

[5]Department of Pharmacology, R. L. Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563103, Karnataka, India.

***Corresponding Author:**

Mamatha H S

Department of Pharmaceutics, R. L. Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563103, Karnataka,India

Email ID: shreemamatha6@gmail.com

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ABSTRACT

Objective: To contrast traditional allopathic treatments with newer herbal and phytopharmacological therapies for non-alcoholic fatty liver disease (NAFLD), a syndrome often occurring with metabolic syndrome, obesity, and type 2 diabetes.

Methodology: A narrative, PRISMA-guided PubMed, Web of Science, Scopus, and Google Scholar search (January 2015 – May 2025) was performed using "NAFLD," "NASH," "allopathic treatment," "herbal medicine," and "phytopharmacology." Peer-reviewed English human, animal, or in-vitro articles were included; non-NAFLD liver diseases and non-peer-reviewed articles were excluded. Two reviewers screened titles/abstracts independently, resolved disagreements by consensus, and extracted data on intervention type, dosage, duration, outcomes, mechanisms, and adverse effects.

Results: Allopathic drugs—pioglitazone, vitamin E, GLP-1 receptor agonists, and SGLT-2 inhibitors—enhance liver enzymes and histology but are hampered by single-pathway action and side effects. Herbal alternatives like Silybum marianum, berberine, curcumin, and Phyllanthus niruri have multitarget antioxidant, anti-inflammatory, and insulin-sensitizing actions with fewer adverse events, but are plagued by inconsistent standardization and limited high-quality trials.

Conclusion: A combination of lifestyle change, evidence-based drugs, and strictly proven phytopharmacological compounds could provide an enhanced NAFLD approach. Well-designed large clinical trials are necessary to prove the safety and efficacy of these herbal agents and allow them to be incorporated into regular care regimens.

1. INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most prevalent chronic liver condition in the West, Pooled meta-analyses estimate that it affects about 30 % of adults roughly nine hundred million people, corresponding to one-third of adults [1] NAFLD is a broad spectrum of liver disease from simple liver fat.NAFLD ranges from uncomplicated fat deposition in > 5 % of hepatocytes to its more severe form, non-alcoholic steatohepatitis (NASH), i.e., accumulation of more than 5% fat in hepatocytes, to severe non-alcoholic steatohepatitis (NASH) with hepatocellular ballooning, inflammation, and fibrosis that can lead ultimately to cirrhosis and hepatocellular carcinoma [2]. Significantly, NAFLD occurs without alcohol in excess (<20 g/day in females and <30 g/day in males), and after exclusion of other etiologies of liver diseases such as viral hepatitis, autoimmune hepatitis, or iron overload [3].The condition shows close association with metabolic syndrome characteristics—particularly obesity, type 2 diabetes mellitus, insulin resistance, and dyslipidemia—and thus the term hepatic

manifestation of metabolic syndrome [4]. NAFLD prevalence is much higher in patients with type 2 diabetes and obesity, 70% to 90% [5]. The majority of high prevalence rates have been in the Middle East and South America (approximately 30%), whereas increasing prevalence nearing 29.6% has been noted in Asia due to lifestyle and dietary modifications associated with increased urbanization [6]. NAFLD is likely underdiagnosed in primary care despite having a high prevalence and potential for serious complications [7]. Most patients are asymptomatic or present with non-specific symptoms such as fatigue, malaise, or right upper quadrant pain [8]. It is usually an incidental diagnosis following an abnormality in liver enzymes or imaging studies that shows increased hepatic echogenicity [9]. Histologic diagnosis via liver biopsy remains the gold standard for NASH vs. simple steatosis but is limited by invasiveness from being used broadly in practice [10]. Present understanding accounts for disease evolution with the "multiple-hit" hypothesis. The initial hit is lipid accumulation in the liver, which sensitizes hepatocytes. Follow-up "hits" are oxidative and endoplasmic-reticulum stress, inflammatory signaling, and gut-derived toxins , making the liver vulnerable to the following insults such as oxidative stress, endoplasmic reticulum stress, inflammatory cytokines, and gut-derived endotoxins that lead to progression to NASH and fibrosis. NAFLD significantly increases the risk of cardiovascular disease, extrahepatic malignancies, and liver-related death, particularly in patients with NASH [11,12]. In the year 2030, NAFLD is predicted to be the leading reason for liver transplantation worldwide [13].There are no approved pharmacologic treatments for NAFLD. Drug development has been hampered by earlier misconceptions of the disease as being benign or merely diabetes-related, and by clinical trial use of histological endpoints [14]. Furthermore, biochemical and pathophysiologic disparities between rodent models of NASH and human disease—particularly in insulin resistance, genetic predisposition, and fibrosis development—are translational impediments [15].In response to these obstacles, more attention is being devoted to alternative and adjunctive therapeutic modalities, particularly herbal and phytopharmacologic origin. Herbal therapy possesses emergent hepatoprotective, anti-inflammatory, and insulin-sensitizing properties, and is able to address multiple pathogenic targets along the NAFLD spectrum [16]. This review aims to provide a comparative summary of allopathic and herbal therapeutic approaches to treating NAFLD with particular focus on new phytopharmacologic agents with established efficacy.

FIG 1: Non-alcoholic fatty liver disease

2. METHODOLOGY

The review methodology adhered to PRISMA guidelines, with studies between 2015 and 2025. Literature was obtained from PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar with the keywords of "NAFLD," "allopathic treatment," "herbal therapy," "phytopharmacology," and "clinical trials." Only those articles that were peer-reviewed, written in English, and studying herbal or standard treatments for non-alcoholic fatty liver disease were included. Exclusion criteria were alcoholic liver disease studies, non-peer-reviewed articles, and non-English literature. Two reviewers screened and chose independently, and when there were disagreements, they settled by consensus. Information gathered was study type, interventions, sample size, outcomes, mechanisms, and adverse effects. Quality was determined using CONSORT for clinical trials, SYRCLE for animal trials, and AMSTAR 2 for reviews. The findings were narratively synthesized with a focus on phytocompounds such as silymarin, curcumin, berberine, and glycyrrhizin. Recent publications by Eslam (2020), Zhang (2021), and Liu (2024) were some of the references consulted to compare efficacy, safety, and mechanisms of both types of treatment.

3. RESULTS

PATHOPHYSIOLOGY:

The pathophysiology of Non-Alcoholic Fatty Liver Disease (NAFLD) is multi-factorial with intricate interactions between

insulin resistance, lipid metabolism, inflammation, oxidative stress, and hormonal imbalances (see fig 2), as is depicted in the image attached hereto. Insulin resistance is at the hub of NAFLD, resulting in increased lipolysis in adipose tissue, raising circulating FFAs[17]. These FFAs are absorbed by the liver and subjected to de novo lipogenesis (DNL), leading to hepatic triglyceride and free cholesterol buildup. At the same time, insulin resistance also inhibits mitochondrial β-oxidation, which further contributes to fat accumulation in hepatocytes.This lipid excess results in mitochondrial dysfunction and oxidative stress, inducing hepatic inflammation. Inflammation is further enhanced by adipokines like leptin (increased) and adiponectin (decreased). Furthermore, the reaction of the liver involves enhanced inflammatory cytokines production and Kupffer cell activation, enhancing the progression from simple steatosis to NASH(18,19).Liver insulin resistance also impairs glucose metabolism, enhancing gluconeogenesis and glycogenolysis, which elevate the level of blood glucose and fuel systemic glucotoxicity. Excessive secretion of liver-derived selenoprotein P (SeP) aggravates peripheral tissue insulin resistance [20, 21]. Other organs' hormones also have roles: GLP-1 derived from the gut possesses anti-inflammatory and metabolic control actions, whereas stomach-derived ghrelin (acylated and deacylated species) imbalances can modulate liver inflammation and fatty acid metabolism. Irisin from skeletal muscle and white adipose tissue (WAT) to brown adipose tissue (BAT) conversion impact energy expenditure as well as thermogenesis and may influence the progression of NAFLD [22]. These coupled mechanisms together promote the pathogenesis and progression of NAFLD, ranging from fatty liver to NASH, fibrosis, and eventually cirrhosis or hepatocellular carcinoma.

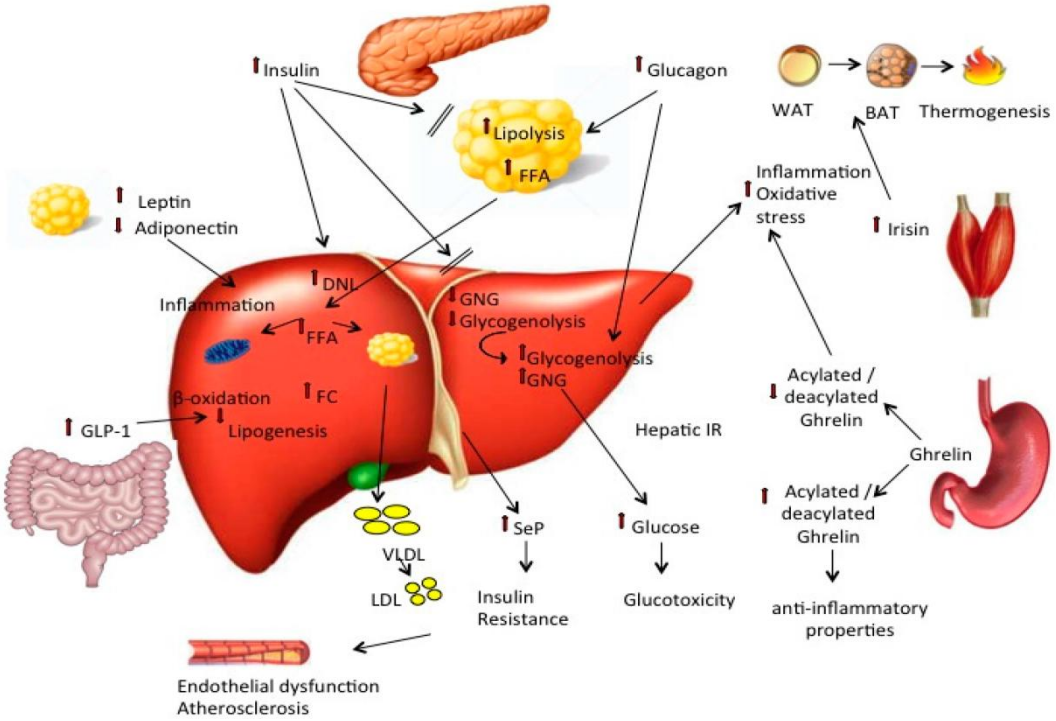

Fig2: Pathophysiology of NAFLD

MANAGEMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFID):

The treatment of Non-Alcoholic Fatty Liver Disease (NAFLD) today involves intensive lifestyle treatment for the reduction of liver fat, increasing insulin sensitivity, and halting or reversing disease advancement. It encompasses dietary changes, exercise, pharmacological treatment, and in some cases, bariatric surgery. While the prevalence of NAFLD has been on the rise worldwide, no pharmacologic treatment has been approved for its treatment; therefore, lifestyle changes are the foundation of therapy.

1. **Dietary Interventions and Weight Loss:**

Diet is responsible for NAFLD development and progression. Patients usually have a diet rich in fructose, red meat, saturated fats, and processed foods, but low in fiber, omega-3 fatty acids, and essential micronutrients [23]. Excessive intake of calories, particularly from fructose-containing drinks and refined carbs, leads to de novo lipogenesis (DNL), insulin resistance, and liver fat accretion.

Weight loss is still the best method to decrease hepatic fat and to correct liver histology. A weight reduction of at least 7% has been demonstrated to result in marked improvements in steatosis, hepatocellular ballooning, inflammation, as well as even fibrosis in a proportion of patients with NASH [24]. A single randomized controlled trial (RCT) showed that intensive

dietary treatment by a multidisciplinary team over 48 weeks led to histological response of NASH if ≥7% weight loss was attained [25]. Even small weight loss (3–5%) can reduce liver fat content, but ≥10% is usually required for reversal of fibrosis [26].

Caloric restriction is the main key to weight loss, independent of macronutrient composition. Some patterns of diet may have extra benefits, though. The Mediterranean diet, which is high in monounsaturated fats, whole grains, vegetables, fruits, and fish, has been linked to reductions in liver fat and enhanced insulin sensitivity—even in the absence of substantial weight loss[27].

2. **Physical Activity & sedentary behaviour:**

Physical exercise is a central part of NAFLD treatment. Aerobic and resistance training alone decrease intrahepatic fat, visceral fat and inflammatory markers even without weight loss [28]. Exercise increases hepatic insulin sensitivity and reduces oxidative stress and systemic inflammation, reducing the risk of progression to NASH and fibrosis. Studies indicate that 150–200 minutes a week of moderate-intensity aerobic exercise can decrease the content of liver fat considerably. At least 15–30 minutes of physical activity per day has been observed to decrease mortality due to all causes and cancer[29]Nevertheless, research shows that almost 50% of patients with NAFLD are inactive physically, and approximately one-third have no regular physical activity [30].

Decreasing sedentary behavior is also critical. Accumulating sitting time was found to be linked with metabolic syndrome, elevated insulin resistance, and liver fat deposition. Although data regarding sedentary time in the context of NAFLD patients are scarce, reducing screen time and encouraging regular breaks from sitting are crucial suggestions [31].

3. **Bariatric Surgery:**

Bariatric surgery, although not a first-line therapy for NAFLD or NASH, has also demonstrated impressive histologic improvements in very obese patients. Operations like Roux-en-Y gastric bypass and sleeve gastrectomy can lead to huge weight loss, which subsequently cures steatosis, decreases inflammation, and can even reverse severe fibrosis and early-stage cirrhosis [32]

These advantages are largely mediated by decreased visceral fat mass and pro-inflammatory mediators including TNF-α and IL-6, enhancing insulin resistance and hepatic inflammation [33]. Although not specifically indicated for NAFLD, the presence of NASH is not a contraindication in obese patients needing bariatric intervention [34].

4. Allopathic (pharmacological) treatments for Non-Alcoholic Fatty Liver Disease (NAFLD):

To date, no drug is formally approved for the treatment of NAFLD or NASH. Pharmacological therapy is warranted for patients with biopsy-documented NASH or at increased risk of progression of fibrosis.

Pioglitazone (Thiazolidinedione):

Pioglitazone, a peroxisome proliferator-activated receptor gamma (PPAR-γ) agonist, is usually prescribed for type 2 diabetes mellitus but also has been shown to benefit in the treatment of NASH (Non-Alcoholic Steatohepatitis) in diabetic and non-diabetic . Pioglitazone exerts its effect by activating PPAR-γ receptors, resulting in enhanced insulin sensitivity, decreased hepatic lipogenesis, and diminished pro-inflammatory cytokine production. Pioglitazone enhances insulin sensitivity, lowers liver fat, inflammation, and ballooning and in some instances, fibrosis. However, its clinical use is limited due to side effects including weight gain, fluid retention, and potential long-term risks such as bladder cancer[35].

Vitamin E (α-Tocopherol)

Vitamin E, which is an antioxidant, is also effective in non-diabetic patients with biopsy-proven NASH. Its mechanism involves scavenging ROS, inhibiting lipid peroxidation, and reducing hepatic oxidative stress. Vitamin E in the PIVENS trial dramatically improved liver histology, such as steatosis and lobular inflammation, over placebo. Nevertheless, there are lingering concerns regarding long-term use based on potential associations with prostate cancer risk in men (reported in the SELECT trial) and a possible rise in all-cause mortality at doses above 400 IU/day.

GLP-1 Receptor Agonists (such as Liraglutide, Semaglutide):

GLP-1 receptor agonists like liraglutide and semaglutide are incretin-based drugs initially licensed for type 2 diabetes. They stimulate glucose-dependent insulin secretion, slow gastric emptying, suppress appetite, and induce weight loss, all of which play a role in the improvement of hepatic steatosis. The LEAN trial demonstrated that liraglutide resulted in resolution of NASH in 39% versus 9% on placebo, and fibrosis progression reduction [36]. Semaglutide demonstrated encouraging histologic remission in NASH resolution in phase II trials, though its long-term effectiveness on fibrosis is under investigation. Most side effects consist of gastrointestinal complaints such as nausea, vomiting, and diarrhea.

SGLT-2 inhibitors (e.g., Empagliflozin, Dapagliflozin):

SGLT-2 inhibitors enhance glycemic control through increased urinary excretion of glucose. They decrease fat in the liver by enhancing insulin sensitivity, decreasing body weight, and improving lipid metabolism. Empagliflozin and dapagliflozin have been demonstrated in clinical trials such as E-LIFT and DEAN trials to decrease liver fat content, ALT level, and body weight in NAFLD patients with type 2 diabetes [37, 38]. Although promising, the drugs are not yet approved for NAFLD/NASH use. Side effects are urinary tract infection, genital infection, and potential volume depletion in geriatric patients.

Obeticholic Acid (FXR Agonist):

Obeticholic acid (OCA) is a farnesoid X receptor (FXR) agonist that modulates bile acid production, decreases hepatic steatosis, inflammation, and induces antifibrotic signaling. The REGENERATE trial showed OCA improved fibrosis severity with no worsening of NASH in advanced disease patients [39]. Though side effects like severe pruritus (up to 50% in certain groups), increased LDL cholesterol, and long-term cardiovascular outcomes concerns restrict its use as routine therapy, it is now being reviewed by the FDA for the treatment of NASH-related fibrosis.

Statins (e.g., Atorvastatin, Rosuvastatin):

Although not indicated for the direct treatment of NAFLD, statins are instrumental in controlling cardiovascular risk, the most common cause of mortality in NAFLD patients. Research has shown that statins can also lower aminotransferases, inflammation within the liver, and even liver-related morbidity in NAFLD. Statins are safe to be used in patients with NAFLD and should not be withdrawn on account of minor elevations in liver enzymes. Common side effects are myopathy, elevated liver enzymes, and infrequently rhabdomyolysis, particularly when used in combination with other hepatotoxic agents.

Metformin:

Metformin, a first-line medication for type 2 diabetes, increases insulin sensitivity by reducing hepatic gluconeogenesis and enhancing peripheral glucose delivery. While it optimizes metabolic values, most clinical trials, including the TONIC study, have not demonstrated substantial histological improvement in NASH. Metformin is thus not at present approved for the treatment of NAFLD in non-diabetics. However, it is still useful in the treatment of concomitant diabetes or metabolic syndrome. Side effects are gastrointestinal distress and an uncommon risk of lactic acidosis in patients with renal insufficiency [40].

5.Herbal medicine in NAFLD management:

Rationale for Exploring Herbal Remedies

NAFLD is a multifactorial metabolic disease featuring liver fat, inflammation, and fibrosis. Pharmacotherapies conventionally focus on single pathways, which might be insufficient in treating the multi-factorial pathophysiology of NAFLD. Herbal drugs, being rich in multiple classes of bioactive compounds, provide a multi-targeted strategy potentially altering multiple pathological mechanisms at once. Additionally, they generally carry fewer side effects, hence posing as promising alternatives or adjuncts to conventional treatments.

Historical/Traditional Use of Herbs in Liver Disorders

Ancient health traditions, like Traditional Chinese Medicine (TCM) and Ayurveda, have traditionally used herbs for liver conditions. Silybum marianum (milk thistle), for example, has been used for centuries to cure liver conditions because it is hepatoprotective. Likewise, Curcuma longa (turmeric) and Phyllanthus niruri have been commonly used for centuries to deal with liver conditions, highlighting the millennia-old utilization of herbal remedies for liver care

Benefits of Herbal Therapy: Multi-Target Action and Less Side Effects

Herbal medicines typically have more than one active constituent that are able to act synergistically to produce therapeutic effects. This multi-targeted strategy is especially useful in those complicated diseases such as NAFLD, wherein several pathways are dysregulated. Moreover, used judiciously, herbal therapies have a good safety profile, with fewer rates of adverse effects than some conventional drugs

Herbal Medicines in NAFLD Management:

Herb Name (Scientific Name)	Location	Part Used	Chemical Constituents	Mechmanism of Action	Clinical evidence	Standardizatio n & Regulatory Issues
Silybum marianum	Mediterranea n	Seeds	Silymarin complex	Antioxidant, anti-inflammatory,	Systematic review (2023,	Variable silymarin %

(Milk Thistle)			(silybin, silydianin, silychristin)	antifibrotic; membrane-stabilizing, NF-κB modulation [41,42]	n = 8 RCTs) showed ↓ ALT/AST & NAS. 48-week RCT (600 mg/day) improved steatosis & ballooning.	across products; EU herbal monograph exists but no FDA approval. Large RCTs still lacking; QC & dose standardisation needed
Berberine (from Berberis spp.)	Asia, Europe, N. America	Roots / Stem bark	Isoquinoline alkaloid berberine	AMPK activation → ↑ insulin sensitivity, ↓ lipogenesis, anti-inflammatory[43,44]	Meta-analysis 2024 (12 RCTs) showed ↓ ALT/AST, TG, LDL-C; Typical dose 0.5-1.5 g/day for 12–24 weeks.	Dietary supplement status; poor oral bioavailability; High batchto batch variability; limited longterm safety data.
Curcuma longa (Turmeric)	South Asia	Rhizome	Curcuminoids (curcumin, DMC, BDMC)	NF-κB & PPAR-γ modulation, antioxidant, ↓ hepatic lipid accumulation[45, 46]	Meta-analysis 2025 (7 RCTs) ↓ ALT/AST. 2023 RCT (500 mg curcumin+5 mg piperine) ↓ CAP & bodyfat %.	Low systemic bioavailability; numerous enhanced formulations. GRAS by FDA but supplement quality varies; standard curcuminoid content required.
Phyllanthus niruri	South America, India	Whole plant	Lignans, flavonoids, tannins	Antioxidant, anti-inflammatory, controls lipid metabolism[47,48]	2023 RCT (n = 80, 12 mo) improved fibrosis score but not CAP. Preclinical studies show hepatoprotection in HFD models.	No pharmacopeial standards; variability in phytochemical content. Heavymetal contamination reported; limited clinical evidence; not approved by major regulators.
Geniposide (Gardenia jasminoides)	East Asia	Fruit	Iridoid glycoside geniposide	Anti-oxidant & anti-inflammatory, NF-κB blockadenprotection of hepatocytes [49,50]	Preclinical studies (2021-2025) show ↓ steatosis & fibrosis in HFD mice. No human	Content varies with harvest & processing; potential genipin toxicity. Requires rigorous QC; lacks clinical

					RCTs to date.	data for regulatory approval.

4. DISCUSSION

COMPARISON OF ALLOPATHIC AND HERBAL THERAPIES IN THE MANAGEMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

NAFLD management is still an important clinical challenge as it possesses a multifactorial pathophysiology involving insulin resistance, oxidative stress, lipotoxicity, and inflammation. Although lifestyle modification is still the mainstay of therapy, both allopathic and herbal therapies have been investigated as add-on therapies. All methods possess individualized mechanisms, advantages, and disadvantages.

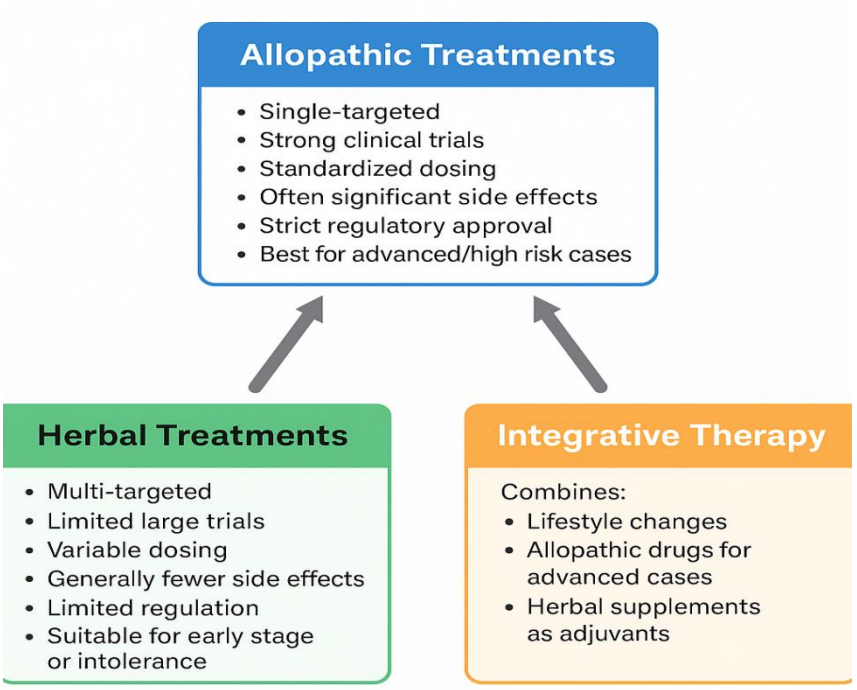

Fig3: comparing allopathic and herbal treatments alongside integrative therapy

Allopathic Treatments: Mechanism and Limitations

Allopathic (traditional) pharmacologic treatments for NAFLD generally address individual mechanisms like insulin resistance, oxidative stress, and inflammation. Drugs like pioglitazone enhance insulin sensitivity and NASH histology but also have risks such as weight gain, edema, and possible carcinogenicity (e.g., bladder cancer) (PIVENS trial). Likewise, vitamin E greatly enhances histologic scores in non-diabetic NASH patients by lowering oxidative stress, but it has been linked to higher all-cause mortality as well as increased risks of prostate cancer with high doses.Other agents including GLP-1 receptor agonists (e.g., liraglutide, semaglutide) and SGLT-2 inhibitors (e.g., empagliflozin, dapagliflozin) have been promising in weight loss and resolution of liver fat, but they are not yet approved for the treatment of NAFLD and their long-term hepatic effects are under investigation[51-53]. Agents like obeticholic acid, which act through activation of FXR, reduce fibrosis but could potentially exacerbate lipid profiles and induce pruritusSpecifically, metformin and statins, while being useful in the management of both metabolic syndrome and cardiovascular risk, are of limited use on liver histology and cannot be used as a first-line treatment for NAFLD.

Herbal Therapies: Broad Spectrum and Fewer Side Effects

Herbal drugs provide multi-targeted approaches, usually useful in a disease like NAFLD that encompasses multiple dysregulated pathways. For instance, Silybum marianum-derived silymarin has antioxidant, anti-inflammatory, and anti-fibrotic activities and also affects NF-κB, a key inflammatory pathway [54,55]. Berberine from Berberis species activates AMPK, thus improving the sensitivity of insulin and decreasing hepatic lipogenesis[56,57].Similarly, curcumin from

Curcuma longa and Phyllanthus niruri have multifaceted actions that involve reduction of oxidative stress, inflammation control, and modulation of lipid metabolism[58,59]. Such agents generally have reduced side effects and improved patient tolerance. Additionally, the extensive history of their use in traditional medicine (e.g., Ayurveda, Traditional Chinese Medicine) attests to their safety profile when utilized in their traditional context.But difficulties with herbal medicine are variability of composition, non-standardization, few large-scale RCTs, and drug-herb interaction or contamination concerns.

Allopathic medication provides single-entity, evidence-based interventions with dose control and extensive safety assessment. Their specificity and predictability make them indispensable in severe or high-risk conditions. Yet, these medications tend to target isolated mechanisms and produce significant side effects, precluding long-term administration. Conversely, herbal medications have multi-target effects, normally improved tolerability, and lower side effects. Their intricate bioactive compounds can target several disease pathways at once, providing whole-system benefits. This potential is dampened, however, by the lack of large-scale clinical trials, standardization, and regulatory approval for herbal therapies.In practice, an integrative approach that incorporates lifestyle changes—like diet and exercise—along with pharmacotherapy and standardized herbal supplements is the most balanced and holistic approach. Herbal treatments can be useful adjuvants in early-stage NAFLD or in intolerant patients to conventional medications.To close present gaps, research in the future will need to concentrate on rigorous randomized controlled trials comparing herbal medicine's effectiveness and safety, seeking to be comparable to allopathic standards. This will provide insight into their place—whether as adjuncts or possible substitutes—among clinical practices. Ultimately, this integrative methodology, based on solid evidence, has the greatest promise to maximize patient outcomes while reducing adverse effects.

5. CONCLUSION

Non-Alcoholic Fatty Liver Disease (NAFLD) is an emergent worldwide health concern with its multifactorial pathogenesis and relationship with metabolic syndrome. Though it has a very high prevalence and potential risk of end-stage complications like cirrhosis and hepatocellular carcinoma, so far there has been no pharmacological therapy approved by regulatory authorities. Treatment currently is predominantly based on lifestyle change, with nutritional therapy and exercise being the pillars of management. Allopathic pharmacotherapies such as pioglitazone, vitamin E, GLP-1 receptor agonists, and SGLT-2 inhibitors provide targeted therapeutic benefits in insulin sensitivity, liver fat, and inflammation but are constrained by side effects, expense, and limited efficacy against NAFLD's multi-factorial pathways.Herbal treatments, on the other hand, offer a multi-targeted and potentially safer option. Phytochemicals like silymarin, berberine, curcumin, and geniposide have antioxidative, anti-inflammatory, insulin-sensitizing, and anti-fibrotic activities. These agents, which have been used in traditional systems of medicine for years, act on multiple pathological mechanisms simultaneously and have good safety profiles. Despite this, uncertainty about their standardization, quality control, and insufficient robust clinical trial data persists. An integrative model of therapy uniting evidence-based allopathic treatments with established, standardized herbal therapies presents an exciting path toward holistic management of NAFLD. Herbal medicine can be especially indicated for early NAFLD or as supportive therapy in drug-intolerant patients. Yet their introduction into mainstream practice requires additional large-scale, randomized clinical trials to affirm efficacy and safety. Finally, a tailored, multi-modal treatment based on lifestyle change, pharmacotherapy, and safer phytotherapy offers the best hope to combat the heterogeneous and worsening course of NAFLD.

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