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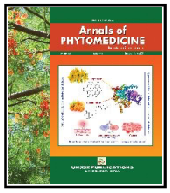

Review Article : Open Access

A scoping review of herbal and allopathic remedies: Insights into liver damage, diagnostic methods, and hepatoprotective strategies

B.S Ashok Kumar and N.S. Disha◆

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

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Abstract

Drug-induced liver injury (DILI) is a significant yet often underdiagnosed issue resulting from both pharmaceutical and herbal agents. While conventional allopathic drugs, including ursodeoxycholic acid, silymarin, and N-acetylcysteine, have established therapeutic roles in managing liver conditions through mechanisms such as cell membrane stabilization, antioxidant enhancement, and immune modulation, additional agents like glutathione, methionine, and ademetionine further contribute to hepatic detoxification and regeneration. Herbal remedies, including extracts from *Aerva javanica, Phyllanthus amarus,* and *Cichorium intybus*, also demonstrate hepatoprotective properties by reducing oxidative stress, inhibiting lipid peroxidation, and boosting natural liver defenses. However, concerns regarding the safety of herbal treatments arise when used unsupervised or combined with other medications. The liver's vulnerability to toxic agents, whether pharmaceutical or herbal, can lead to conditions ranging from mild enzyme elevation to acute liver failure. This review delves into the mechanisms underlying liver damage emphasizing oxidative stress, mitochondrial dysfunction, and immune-mediated response and explores various screening methods for assessing hepatoprotective efficacy, including *in vitro* assays (*e.g.*, hepatocyte cultures, cytotoxicity assays) and *in vivo* models (*e.g.*, carbon tetrachloride-induced liver injury). By evaluating both allopathic and herbal treatments, this review aims to clarify their mechanisms, screening techniques, and therapeutic applications in preventing and treating liver injury.

1. Introduction

The liver plays a crucial role in metabolic homeostasis, detoxification, and protein synthesis but is highly vulnerable to damage from various factors, including viral infections, toxins, alcohol, and drugs. Liver diseases such as hepatitis, cirrhosis, and hepatocellular carcinoma (HCC) often arise due to prolonged liver injury. Hepatotoxicity, resulting from both internal and external factors, can present as acute or chronic dysfunction. Although, the liver has a remarkable ability to regenerate, repeated or severe injury can lead to fibrosis and eventual liver failure (Mays *et al*., 2020)

Liver damage occurs through multiple mechanisms, including oxidative stress, inflammation, mitochondrial dysfunction, and apoptosis. Excessive production of reactive oxygen species (ROS) causes oxidative stress, leading to lipid peroxidation, cellular damage, and activation of inflammatory pathways. Chronic inflammation in the liver is a major driver of fibrosis and cirrhosis, exacerbating tissue injury. Activation of hepatic stellate cells (HSCs) in response to liver damage results in excessive deposition of extracellular matrix components, leading to fibrosis and impaired liver function (Chen *et al.,* 2023).

Corresponding author: Ms. N.S. Disha
Associate Professor, Department of Pharmaceutical Chemistry, R.L. Jalappa College of Pharmacy, SDUAHER Tamaka, Kolar-563103, Karnataka, India
E-mail: disha.s1215@gmail.com
Tel.: +91-9481800533

To combat liver damage, both allopathic and herbal treatments have been explored for their hepatoprotective effects. Allopathic medicine primarily relies on pharmaceutical drugs such as corticosteroids, antioxidants, and immune suppressants to manage liver diseases. While these treatments are effective, they often come with adverse effects and long-term complications. Certain allopathic drugs, such as acetaminophen and chemotherapy agents, are known for their hepatotoxicity, posing challenges in their therapeutic use.

Herbal medicines have gained attention for their potential hepatoprotective benefits with fewer side effects. Several medicinal plants, including *Silybum marianum* (milk thistle), *Andrographis paniculata*, and *Curcuma longa* (turmeric), have been studied for their liver-protective properties. Bioactive compounds such as silymarin, andrographolide, and curcumin exhibit strong antioxidant, anti-inflammatory, and antifibrotic effects, aiding in hepatocyte regeneration and reducing liver inflammation (Garg *et al.,* 2020; Singh *et al.,* 2022).

Despite the growing interest in herbal medicine, allopathic treatments remain the primary choice, particularly in acute and severe liver conditions. However, an integrative approach combining allopathic and herbal therapies may provide synergistic benefits. For instance, antioxidant-rich herbal medicines, when used alongside conventional drugs, could enhance liver protection by reducing oxidative stress and inflammation, key contributors to liver damage.

This review delves into the mechanisms of liver injury, screening methods for identifying hepatoprotective agents, and the therapeutic potential of both allopathic and herbal treatments. Understanding

these interventions may pave the way for more effective strategies in preventing and managing liver diseases.

2. Drug-induced liver injury (DILI)

2.1 Acetaminophen (Paracetamol)

Acetaminophen is one of the most common causes of acute drug-induced liver injury when taken in excessive amounts. It is metabolized in the liver, where it is usually detoxified by conjugation with glucuronide or sulfate. However, when taken in large doses, acetaminophen is metabolized by cytochrome P450 enzymes into a toxic intermediate, N-acetyl-p-benzoquinone imine (NAPQI). NAPQI depletes glutathione in the liver, leading to oxidative stress and hepatocyte damage. It may lead to acute liver failure if not promptly treated with NAC in cases of acute overdose (Hoofnagle and Bjornsson, 2022).

2.2 Isoniazid

It is a first-line antituberculosis agent. One of the side effects associated with it is liver toxicity, and this risk increases in individuals with existing liver diseases and those abusing alcohol. Isoniazid is metabolized by the liver, and its toxic metabolites result in liver cell injury and inflammation. Older individuals and slow acetylators may have an increased toxic metabolite burden, leading to a higher risk of liver injury, which presents as jaundice, dark-colored urine, and abdominal pain (Chalasani *et al.,* 2021)

2.3 Methotrexate

Methotrexate is commonly used to treat cancers, autoimmune diseases, and rheumatoid arthritis. It causes liver damage by accumulating in hepatic cells, where it inhibits folate metabolism. Prolonged use can result in liver fibrosis, steatosis, or cirrhosis. Methotrexate induces oxidative stress, inflammation, and mitotoxicity in hepatocytes. Therefore, regular liver monitoring is essential in patients undergoing long-term therapy (Teschke, 2021).

2.4 Amiodarone

It is prescribed for treating arrhythmias but has been found to be hepatotoxic. The exact mechanism of its toxicity is not completely understood, but it is believed to involve mitochondrial damage, oxidative stress, and immune responses. Hepatocellular injury caused by amiodarone can range from mild enzyme elevations to severe conditions such as cirrhosis (Fontana, 2022).

2.5 Statins (*e.g.*, atorvastatin, simvastatin)

Statins are widely used to lower cholesterol levels but may adversely affect liver enzymes in some individuals. The liver is the primary site of statin metabolism, and in rare cases, statins can lead to hepatocellular injury. The hepatotoxicity is believed to be due to mitochondrial dysfunction and oxidative stress. Severe cases may result in hepatocellular necrosis, jaundice, and acute liver failure (Andrade *et al.,* 2023).

2.6 Valproic acids

It is used for epilepsy and bipolar disorder but has been linked to liver damage, particularly in children under two years old and individuals with pre-existing liver disease. Its hepatotoxic effects are associated with mitochondrial dysfunction, increased oxidative stress, and hepatocyte apoptosis. The highest risk occurs within the first six months of treatment, necessitating close liver function monitoring (Bjornsson, 2020).

2.7 Fluoroquinolones (*e.g.*, ciprofloxacin, levofloxacin)

Fluoroquinolone antibiotics, such as ciprofloxacin and levofloxacin, have been linked to drug-induced liver injury. The mechanism likely involves oxidative stress and mitochondrial damage, leading to hepatocyte apoptosis. The severity of liver damage varies from mild enzyme elevations to hepatitis or fulminant hepatic failure. Though rare, fluoroquinolone-related hepatotoxicity may require discontinuation of the drug (Chen *et al*., 2020).

2.8 Rifampin

It is commonly used to treat tuberculosis, can cause hepatotoxicity, particularly when combined with other hepatotoxic drugs like isoniazid. It induces liver enzyme activity, leading to increased production of toxic metabolites. Symptoms of rifampin-induced liver injury include jaundice, fatigue, and elevated liver enzymes. Patients on rifampin therapy require regular liver function monitoring (Teschke and Danan, 2020).

Table 1: Allopathy drugs associated with liver damage

S.No.	Drug	Mechanism of liver damage	Clinical consequences
1.	Acetaminophen	NAPQI formation depletes glutathione, oxidative stress.	Acute liver failure, jaundice, hepatotoxicity
2.	Isoniazid	Toxic metabolites, oxidative stress, inflammation.	Hepatitis, liver failure
3.	Methotrexate	Inhibits folate metabolism, oxidative stress, liver fibrosis.	Hepatotoxicity, fibrosis, cirrhosis
4.	Amiodarone	Mitochondrial damage, oxidative stress	Hepatocellular injury, cirrhosis
5.	Statins	Oxidative stress, mitochondrial dysfunction	Elevated liver enzymes, rare cirrhosis
6.	Valproic acid	Mitochondrial dysfunction, oxidative stress	Liver failure, elevated liver enzymes
7.	Fluoroquinolone	Oxidative stress, mitochondrial damage	Hepatitis, jaundice
8.	Rifampin	Induces liver enzymes, increases toxic metabolites	Jaundice, hepatitis
9.	Ketoconazole	Mitochondrial dysfunction, oxidative stress.	Liver failure, elevated liver enzymes
10.	Aspirin	Prostaglandin inhibition, mitochondrial dysfunction.	Liver necrosis, jaundice

2.9 Antifungal Agents (*e.g.*, ketoconazole, itraconazole)

Antifungal medications, particularly ketoconazole, are associated with drug-induced liver toxicity. These drugs can elevate liver enzymes and cause mitochondrial damage, leading to oxidative stress and hepatocyte injury. The severity of liver damage varies from mild enzyme elevations to severe hepatic failure, making them contraindicated in patients with pre-existing liver disease (Chalasani *et al.,* 2021).

2.10 Aspirin

It isa widely used nonsteroidal anti-inflammatory drug (NSAID), can be hepatotoxic when taken in high doses or for extended periods. Its hepatotoxicity is linked to impaired prostaglandin synthesis, leading to mitochondrial dysfunction and liver necrosis. Patients with pre-existing liver disease or excessive alcohol use are at greater risk (Andrade *et al.,* 2023).

The serious nature of liver damage caused by drugs is well-documented and continuously monitored. While most drugs do not cause liver damage when used properly, some medications have a higher likelihood of leading to hepatotoxicity. Regular liver function tests and timely intervention can mitigate the risks of drug-induced liver injury (DILI) and improve patient outcomes (Table1).

3. Herbal drugs associated with liver damage

3.1 *Piper methysticum* G. Forst. (Kava)

Traditionally used as a sedative and anti-anxiety remedy, kava has been linked to severe liver injuries, including hepatitis, cirrhosis, and liver failure. The exact mechanism is not fully understood, but it is believed that kava inhibits liver enzymes, leading to hepatocyte damage. Regular or high-dose use increases the risk of liver damage, thus liver function should be monitored during kava consumption (Teschke *et al*., 2023).

3.2 *Symphytum officinale* L. (Comfrey)

It is used in traditional medicine for wound healing and inflammation, comfrey contains pyrrolizidine alkaloids (PAs), which are hepatotoxic. These compounds can inhibit DNA replication, causing hepatocyte death, fibrosis, and cirrhosis. Prolonged or high-dose ingestion of comfrey has led to severe liver damage, prompting some countries to ban its internal use (Birt and Pottenger, 2021).

3.3 *Larrea tridentata* (DC.) Coville (Chaparral)

It is Known for its antibacterial and anti-inflammatory properties, chaparral has been associated with acute hepatitis and liver failure. The herb contains nordihydroguaiaretic acid (NDGA), which induces oxidative stress and damages liver mitochondria. Several cases of liver failure have been reported, leading to restrictions on its use in some countries (Teschke *et al*., 2022).

3.4 *Camellia sinensis* L. (Green tea extract)

While green tea is generally considered healthful, concentrated extracts can exhibit hepatotoxic effects when consumed excessively. The polyphenols, especially epigallocatechin gallate (EGCG), can induce oxidative stress in liver cells. High doses have been linked to acute hepatitis and liver failure, particularly in individuals with pre-existing liver conditions or those taking other hepatotoxic medications (Couteau *et al*., 2020).

3.5 *Cimicifuga racemosa* (L.) Nutt. (Black cohosh)

Commonly used to alleviate menopausal symptoms, black cohosh has been associated with hepatotoxicity, though incidents are rare. The exact mechanism remains unclear, but it may involve alterations in liver metabolism or interactions with other drugs, leading to hepatitis. Cases of acute liver failure have been reported; caution is advised for individuals with existing liver diseases (Teschke and Eickhoff, 2021) .

3.6 *Mentha pulegium* L. (Pennyroyal)

Historically used for menstrual and digestive issues, pennyroyal oil is now recognized for its severe hepatotoxicity, including acute liver failure and death. The primary toxic compound, pulegone, is metabolized into reactive metabolites that damage liver cells. Due to its high toxicity, internal use of pennyroyal is strongly discouraged (Joshi *et al.,* 2022).

3.7 *Borago officinalis* L. (Borage)

Employed for its anti-inflammatory and skin-healing properties, borage contains pyrrolizidine alkaloids, similar to comfrey, leading to potential liver damage. These compounds have been linked to veno-occlusive disease and cirrhosis. To avoid liver toxicity, prolonged or high-dose use of borage oil or leaves should be avoided (Lintner and Berthold, 2023).

3.8 *Aloe barbadensis* L. (Aloe vera)

While aloe vera is beneficial for external applications and as a laxative, internal use in high doses or over extended periods has been associated with liver toxicity. Compounds such as anthraquinones may induce oxidative stress, damaging liver cells. Cases of hepatitis and liver enzyme abnormalities have been reported with chronic consumption; large intakes should be avoided, especially in individuals with pre-existing liver conditions (Niaz *et al*., 2024).

3.9 *Hypericum perforatum* L. (St. Johns Wort)

Popular for treating depression and anxiety, St. John's Wort can induce liver enzymes through the cytochrome P450 system, affecting the metabolism of other drugs and potentially leading to hepatotoxicity. Although, liver damage is infrequent, the herb has been associated with liver enzyme abnormalities and should be used cautiously, particularly in patients on multiple medications (Izzo and Ernst, 2021).

3.10 *Silybum marianum* (L.) Gaertn. (Milk thistle)

Its primarily known for its hepatoprotective properties, excessive or uncontrolled use of milk thistle has been linked to adverse effects, including hepatotoxicity. The active ingredient, silymarin, is generally safe in moderate doses but may impact liver function at high doses or if contaminated with toxic substances. Isolated cases of liver toxicity have been reported, especially with contaminated products or large quantities (Teschke and Saller, 2025).

Herbal medicines, despite their natural origin, can cause severe liver damage if not used appropriately. The herbs listed above highlight the potential for hepatotoxicity through direct liver cell damage or alterations in liver metabolism. Monitoring liver function is essential when using herbal supplements, particularly in individuals with pre-existing liver conditions or those taking concomitant medications (Table 2).

Table 2: Herbal drugs associated with liver damage

S.No.	Herbal drug	Scientific name	Common uses	Hepatotoxic components	Hepatotoxic effects	Note
1.	Kava	*Piper methysticum* G. Forst.	Sedative, antianxiety	Unknown	Hepatitis, cirrhosis, liver failure	Regular use or high doses increase risk; liver function monitoring recommended
2.	Comfrey	*Symphytum officinale* L.	Wound healing, anti-inflammatory	Pyrrolizidine alkaloids (PAs)	Hepatocyte death, liver fibrosis, cirrhosis	Internal use banned in some countries due to hepatotoxicity
3.	Chaparral	*Larrea tridentata* (DC.) Coville	Antibacterial, anti-inflammatory	Nordihydroguaiaretic acid (NDGA)	Oxidative stress, liver mitochondria damage, acute hepatitis, liver failure	Use restricted in some countries
4.	Green Tea Extract	*Camellia sinensis* L. Kuntze	Antioxidant, weight loss aid	Polyphenols (e.g., EGCG)	Oxidative stress, acute hepatitis, liver failure	High doses are associated with hepatotoxicity; avoid in pre-existing liver conditions
5.	Black Cohosh	*Cimicifuga racemosa* (L.) Nutt.	Menopause symptom relief	Unknown	Hepatic enzyme activity increase, hepatitis	Rare incidence of hepatotoxicity; use with caution in liver disease.
6.	Pennyroyal	*Mentha pulegium* L.	Menstrual and digestive ailments	Pulegone	Acute liver failure, hepatocyte damage	Internal use is extremely dangerous; should not be used.
7.	Borage	*Borago officinalis* L.	Anti-inflammatory, skin healing	Pyrrolizidine alkaloids (PAs)	Veno-occlusive disease, cirrhosis	Prolonged or high-dose use should be avoided.
8.	Aloe Vera	*Aloe barbadensis* Mill.	Laxative, skin treatment	Anthraquinones	Hepatitis, liver enzyme abnormalities	Avoid high doses or chronic use, especially in pre-existing liver conditions.
9.	St. John's Wort	*Hypericum perforatum* L.	Depression, anxiety	Unknown (via cytochrome P450 interaction)	Liver enzyme abnormalities, altered drug metabolism	Use with caution in patients on multiple medications.
10.	Milk Thistle	*Silybum marianum* (L.) Gaertn.	Liver protection, detoxification	Contamination or excessive silymarin	Isolated hepatotoxicity cases	Typically safe; hepatotoxicity linked to contamination or high doses.

4. Screening for hepatoprotectivity

Research into hepatoprotectivity involves three testing approaches which evaluate compounds that protect against or cure liver damage from toxins or oxidative stress or inflammation by using cell-based assays combined with animal and human tests.

4.1 *In vitro* screening (Cell-based assays)

The evaluation of hepatoprotective potential occurs through experiments that use HepG2 and Huh7 and primary hepatocytes cell cultures. The assessment of cell viability uses hepatotoxins such as acetaminophen and CCl_2 and TBHP in cytotoxicity assays that monitor MTT, trypan blue exclusion and LDH release. Scientists measure oxidative stress markers through the analysis of ROS with DCFDA probes combined with studies of antioxidant enzymes including SOD, catalase and glutathione peroxidase. The evaluation of molecular pathways (technically known as oxidative stress, apoptosis, and inflammation) uses RT-PCR along with Western Blotting according to (Wu *et al*., 2022; Green *et al.,* 2021;Sharma *et al.,* 2020).

4.2 *In vivo* screening (Animal models)

Rodent experiments evaluate the protective impact of substances on liver health by testing under normal biological functions. The evaluation of acute liver injury models investigates ALT and AST level changes in addition to assessing liver tissue modifications after hepatotoxin exposure such as CCl_2 or paracetamol. Chronically inflamed animals receive repeated hepatotoxin treatments to monitor cirrhosis development through tests of liver enzymes and glutathione and superoxide dismutase together with tissue marker analyses of TGF-β and collagen I. Scientists quantify fibrosis by performing sirius red and masson's trichrome staining as explained in the research by Patel *et al.* (2021); Kim *et al*. (2020)

Hepatoprotectives act through different mechanisms at the molecular level. They exhibit antioxidant action by scavenging free radicals and reducing oxidative stress, most responsible for liver damage. They are anti-inflammatory by inhibiting cytokines such as TNF-α and pathways such as NF-kB, thus reducing inflammation in the liver. Agents that induce liver regeneration stimulate growth factors such as HGF and EGF, promoting the healing of hepatocytes. Some compounds also increase detoxification by upregulating phase I and II enzymes (*i.e*., cytochrome P450 and glutathione S-transferases), which play their roles in liver defense (Lin *et al*., 2022; Lee *et al*., 2021; Wang *et al*., 2020).

4.3 Clinical screening (Human studies)

Clinical trials confirm the ability of the active compounds to protect the liver in humans. The monitoring of liver enzymes such as ALT, AST, and bilirubin constitutes the primary focus of pre-and post-treatment analysis against the trial agent. Liver biopsy has also been considered for histological changes observed; however, there is an increasing reliance on various non-invasive imaging techniques like elastography and MRI to evaluate liver stiffness and fibrosis. These methods are thought to close the gap between preclinical findings and therapeutic application (Zhao *et al*., 2022; Nelson *et al.,* 2021; Moore *et al.,* 2020).

4.4 Safety and toxicology screening

Studies on safety and toxicology are necessary to establish safety profiles of individual compounds. Acute and chronic toxicological evaluations determine the maximum tolerated dose, as well as the possible irreversible effects on liver function. In addition, hepatoprotective agents are screened for possible drug interactions that might hinder or alter the intended effect of the drug on liver metabolism with other agents. Therefore, these evaluations are essential for the clinical translation of potential hepatoprotective agents (Thomas *et al*., 2021; Kumar *et al.,* 2021).

Hepatoprotective screening methods, including *in vitro, in vivo*, and clinical tests, are important for evaluating compounds for efficacy and safety in protecting the liver from damage. Going through these approaches yields a comprehensive understanding of the mechanism by which these hepatoprotective agents work and their therapeutic potential, making it easier for clinical use.

5. Mechanisms of liver damage

The combination of alcohol abuse, infection, toxic substances, and metabolic problems leads to liver damage, progressing through oxidative stress, inflammation, apoptosis, and necrosis. These processes may ultimately result in fibrosis or cirrhosis.

5.1 Alcohol consumption

The metabolic process that breaks down ethanol using alcohol dehydrogenase (ADH) and cytochrome P450 2E1 (CYP2E1) creates acetaldehyde, which both stresses cell oxygen supplies and activates inflammatory responses. Long-term alcohol consumption leads to fatty liver, mitochondrial dysfunction, hepatocyte death, and eventual fibrosis development (Zhou *et al.,* 2021).

5.2 Viral infections

Hepatitis B, C, and D viruses cause T lymphocyte- and TNF-alpha-mediated viral damage, leading to cirrhosis and hepatocellular carcinoma (HCC). Hepatitis A virus (HAV) follows the non-enveloped, positive-sense, single-stranded RNA virus transmission path, spreading mainly through the fecal-oral route *via* contaminated food, water, and human contact. HAV infection results in acute hepatitis, whereas hepatitis B and C viruses lead to chronic liver disease. Although, most individuals recover from HAV without complications, rare cases of acute liver failure occur. Prevention strategies for HAV include proper sanitation, safe food preparation, and vaccination, which provide lasting immunity to vulnerable populations (Tsai *et al.,* 2022).

5.3 Toxins and drugs

Hepatocytes undergo cell death due to oxidative stress and mitochondrial damage caused by acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics, and environmental toxins such as carbon tetrachloride (CCl_2). Acute liver failure results from acetaminophen overdose when its toxic metabolite, N-acetyl-p-benzoquinone imine (NAPQI), depletes glutathione stores, leading to hepatocyte necrosis (Ramachandran *et al.,* 2021).

5.4 Metabolic disorders

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is characterized by excessive fat accumulation in hepatocytes and is closely linked to obesity, type 2 diabetes, insulin resistance, and metabolic syndrome. MASLD pathogenesis results from increased de novo lipogenesis, impaired fatty acid oxidation, and dysfunctional lipid export mechanisms, leading to hepatic steatosis. The progression to metabolic dysfunction-associated steatohepatitis (MASH) involves hepatocellular damage, inflammation, and fibrosis, primarily driven by oxidative stress, mitochondrial dysfunction, and pro-inflammatory cytokine release. Prolonged liver inflammation and fibrosis can progress to cirrhosis, increasing the risk of hepatocellular carcinoma. Understanding these complex pathways is essential for developing targeted treatment strategies for MASLD (Mantzoros *et al.,* 2025).

5.5 Biological mechanisms of liver injury

Excessive reactive oxygen species (ROS) originating from alcohol consumption, viral infections, and metabolic disorders create destructive conditions that damage lipids, proteins, and DNA, leading to lipid peroxidation (Singh *et al.,* 2023).The liver responds to injury by releasing cytokines and chemokines, which activate hepatic stellate cells, leading to hepatocyte damage, extracellular matrix (ECM) deposition, fibrosis, and cirrhosis (Aruoma *et al.,* 2023). Apoptosis occurs in response to oxidative stress and mitochondrial dysfunction, involving cytochrome c release and caspase activation, ultimately contributing to liver tissue damage (Friedman *et al.,* 2022). Acute liver failure triggered by acetaminophen overdose results in necrosis, uncontrolled cell death, membrane rupture, and inflammation, which exacerbate liver damage (Kilaru *et al.,* 2024).

6. Allopathic hepatoprotective drugs

6.1 Ursodeoxycholic acid (UDCA)

Ursodeoxycholic acid (UDCA) is a bile acid commonly used for cholestatic liver diseases. It protects hepatocytes from bile acid damage by membrane stabilization, promotion of bile flow, and reduction in toxic bile acid production. UDCA is effective in primary biliary cirrhosis and non-alcoholic fatty liver disease (NAFLD) due

to its antiapoptotic action against hepatocytes, preventing programmed cell death and improving liver function (Li *et al.,* 2022).

6.2 Silymarin

Silymarin, a flavonoid complex derived from Silybum marianum, displays hepatoprotective properties *via* antioxidant, anti-inflammatory, and membrane-stabilizing actions. It scavenges free radicals, reducing oxidative stress that contributes to liver cell injury caused by toxins. Silymarin is used for cirrhosis, hepatitis, and alcoholic liver disease and promotes liver regeneration by stimulating protein synthesis for cellular repair and growth (Tajmohammadi *et al.,* 2021).

6.3 N-Acetylcysteine (NAC)

N-Acetylcysteine (NAC), a cysteine derivative, is primarily used as an antidote to replenish depleted hepatic glutathione stores in acetaminophen overdose. Glutathione detoxifies reactive metabolites, thereby protecting hepatocytes. NAC also has anti-inflammatory and antioxidant properties and has been explored for treating chronic liver conditions such as NAFLD and chronic viral hepatitis (Kumar *et al.,* 2023)

6.4 Pentoxifylline exerts

Pentoxifylline exerts hepatoprotective effects through anti-inflammatory and antifibrotic mechanisms. It enhances hepatic blood flow, reduces fibrosis, and inhibits pro-inflammatory cytokines. It is used in liver cirrhosis with portal hypertension and has been shown to lower liver enzyme levels and improve liver function in chronic liver diseases (Singh *et al*., 2020).

6.5 Liv.52

Liv.52, a polyherbal formulation by the Himalaya Drug Company, contains extracts from plants such as Cichorium intybus, Cassia occidentalis, and Solanum nigrum. It demonstrates hepatoprotective, antioxidant, and anti-inflammatory effects by stabilizing hepatocyte membranes, promoting liver regeneration, and enhancing bile secretion. Liv.52 is used as a supportive treatment for chronic liver diseases, including cirrhosis and fatty liver, and has been reported to reduce liver enzyme levels while improving liver function (Sharma *et al*., 2021).

6.6 Betaine

Betaine, a metabolite of glycine, acts as a hepatoprotective agent in treating NAFLD. It facilitates triglyceride transport out of the liver, reducing hepatic fat accumulation. Additionally, betaine donates methyl groups, supporting hepatic protein methylation and detoxification processes, which significantly reduce liver fat and improve liver function markers in NAFLD patients (Sun *et al*., 2023).

6.7 Thioctic acid

Thioctic acid, also known as alpha-lipoic acid (ALA), is a potent antioxidant involved in cellular energy production. ALA protects hepatocytes from oxidative stress-related damage and promotes liver regeneration. It is particularly beneficial in various liver diseases, including chronic liver disease and alcohol-related liver injury, as it facilitates glutathione regeneration and enhances detoxification capacity (Gorinstein *et al*., 2022).

Table 3: Allopathy hepatoprotective drugs

S.No.	Drug	Chemical constituents	Mechanism of action	Clinical use
1.	Ursodeoxycholic acid	Bile acid derivative	Reduces bile acid toxicity, stabilizes hepatocyte membranes, promotes bile flow	Primary biliary cirrhosis, NAFLD
2.	Silymarin	Flavonolignans	Antioxidant, anti-inflammatory, membrane-stabilizing, hepatocyte regeneration	Cirrhosis, alcoholic liver disease, hepatitis
3.	N-Acetylcysteine	Amino acid derivative	Antioxidant, replenishes glutathione, detoxifies reactive metabolites	Acetaminophen overdose, chronic liver diseases
4.	Pentoxifylline	Methylxanthine derivative	Anti-inflammatory, antifibrotic, improves liver circulation, reduces fibrosis	Liver fibrosis, cirrhosis
5.	Liv.52	Polyherbal (*Cichorium intybus*, *Cassia occidentalis*, *Solanum nigrum*)	Antioxidant, anti-inflammatory, promotes liver regeneration, stabilizes liver cells	Hepatitis, cirrhosis, fatty liver
6.	Betaine	Amino acid derivative	Reduces liver fat accumulation, supports methylation, improves liver function	Non-alcoholic fatty liver disease (NAFLD)
7.	Thioctic Acid (ALA)	Organic acid	Antioxidant, regenerates glutathione, reduces oxidative stress	Chronic liver disease, alcohol-induced liver injury

7. Herbal hepatoprotective drugs

7.1 *Aerva javanica* (Burm.f.) Juss. ex Schult. (Amaranthaceae)

It is rich in glycosides, flavonoids, saponins, terpenes, and tannins, which contribute to its hepatoprotective effects. An aqueous methanolic extract of *A.javanica* was found to lower the levels of liver enzymes (AST, ALT, ALP) and, in animal models, inhibited liver damage resulting from oxidative stress. These free radical-scavenging properties of the plant are most significant for its protective mechanism against liver toxicity by paracetamol-induced damage.

The extract has been proven to improve antioxidant activity and reduce lipid peroxidation in liver tissue, thus maintaining normal liver function (Khan *et al.*, 2021).

7.2 *Alhagi maurorum* Medik (Leguminosae)

The species *Alhagi maurorum* Medik. consists of tannins, flavonoids, and saponins, which have proved their efficacy as significant antioxidants and anti-inflammatories. Hepatoprotective ability was evaluated using extracts obtained through methanol. In animal models, *A.maurorum* gave a promising result in lowering liver enzyme levels (AST, ALT) during liver toxicity induced by paracetamol and CCl_2 . The antioxidant activities of the plant are likely responsible for its protective action against oxidative damage, and the flavonoids are thought to facilitate a reduction in liver inflammation and promote liver regeneration (Rehman *et al.,* 2022).

7.3 *Clitoria ternatea* L. (Fabaceae)

Alkaloids, flavonoids, tannins, and saponins are some of the components found in *Clitoria ternatea* L, which are suggested to contribute to its hepatoprotective impact. The ethanolic extracts of this plant were utilized to study its hepatoprotective activity, resulting in significant reductions in liver enzyme levels (AST and ALT) in animal models of acetaminophen-induced liver damage. The plant's antioxidant properties prevent damage to hepatocytes by trapping free radicals and inhibiting oxidative damage. Flavonoids aid hepatocyte regeneration and decrease liver inflammation (Rai *et al*., 2023).

7.4 *Phyllanthus amarus* Schumach. & Thonn. (Phyllanthaceae)

It is understood to contain lignans, flavonoids, and phenolic compounds, which are responsible for its hepatoprotective properties. Upon ethanolic extraction, it was found to exhibit hepatoprotective activity by reducing liver enzyme levels and improving liver function. In experimental models, *P. amarus* was found to potentiate protection against acetaminophen-induced liver injury through antioxidative action and anti-inflammatory activity. It has also been reported to improve liver function by inducing regeneration of damaged hepatocytes (Puri *et al.,* 2020).

7.5 *Acacia modesta* Wall. (Leguminosae)

The chemical constituents of*Acacia modesta* Wallinclude tannins and saponins, compounds with antioxidant actions that help protect the liver from injury. The methanolic extract of *A. modesta* showed hepatoprotective activity by reducing hepatotoxicity caused by CCl_2 and paracetamol in experimental models, as evidenced by reduced liver enzyme levels (AST, ALT, ALP). It helps decrease oxidative stress and liver inflammation, thus proving to be highly effective for liver diseases (Murugaiyah *et al.*, 2008).

7.6 *Cuminum cyminum* L. *(*Apiaceae)

It is commonly known as cumin, *Cuminum cyminum* L. contains active phytochemicals like alkaloids, flavonoids, and terpenoids that are hepatoprotective. An aqueous ethanolic extract of this plant was evaluated for its protective property against liver toxicity induced by Nimesulide. The profile of this plant also helps in reducing levels of liver enzymes and improving the histopathology of liver tissue against oxidative stress and inflammation. This is mainly due to the antioxidant property of the plant.

7.7 *Conyza bonariensis* L. (Asteraceae)

It contain quercetin, the abundant flavonoid in *Conyza bonariensis* L, has well-established antioxidant and anti-inflammatory effects. Further, liver protection against hepatotoxic damage induced by paracetamol is evident in the ethanolic extract of this plant, which reduces enzyme levels in serum (AST, ALT). The antioxidant properties of *C. bonariensis* reduce both oxidative stress and hepatocyte injury, classifying this species as hepatoprotective (Gholamreza *et al*., 2015).

7.8 *Malva sylvestris* L. (Malvaceae)

Itcontains mucilage, flavonoids, and tannins. Aqueous extracts from the plant were tested for protection against liver damage due to paracetamol. This plant demonstrated the ability to lower liver enzyme levels and protect against liver injury induced by paracetamol exposure. Its antioxidant activity helped neutralize free radicals and reduced oxidative stress in liver cells, mediating its hepatoprotective property (Barros *et al.,* 2010).

7.9 *Dicranopteris linearis* (Burm.f.) (Pteridaceae)

The flavonoid and phenolic content of *Dicranopteris linearis* (Burm.f.) is very high. The extracts from this plant, particularly methanol extracts, were shown to lower liver enzymes and inhibit liver injury induced by CCl_4, and paracetamol. This hepatoprotective activity is attributed to its potent antioxidant properties, which protect the liver from oxidative damage by scavenging reactive oxygen species (Tiwari *et al.,* 2013).

7.10 *Bauhinia hookeri* F. Muell. (Fabaceae)

Hepatoprotective activity of *Bauhinia hookeri* F. Muell. is attributed to the presence of polyphenols and flavonoids. The ethanol extract of the plant has been shown to reduce liver enzyme levels and mitigate oxidative stress and damage caused by various hepatotoxins. This suggests its protective role against liver injury from multiple sources. Antioxidant action protects hepatocytes and enhances liver regeneration (Chouhan *et al.*, 2016).

7.11 *Taraxacum officinale* (Asteraceae)

Commonly referred to as dandelion, *Taraxacum officinale* is rich in triterpenoids, flavonoids, and sterols, which exhibit hepatoprotective properties. Extracts from the plant have been investigated for their potential to improve liver enzyme levels in animal models and provide protection against damage induced by CCl_2 and ethanol toxicity. The hepatoprotective action is primarily attributed to its ability to augment antioxidant defenses and lower oxidative stress (Ghanem *et al.*, 2017).

7.12 *Cichorium intybus* L. (Asteraceae)

Chicory contains flavonoids and alkaloids, which contribute to its hepatoprotective properties. This activity was demonstrated using an ethanolic extract of the plant that protected the liver from CCl_2 -induced injury. *Cichorium intybus* also decreased liver enzyme levels and promoted liver regeneration. Notably, enhanced effects were observed when it was combined with *T. officinale*, resulting in synergistically improved liver function (Hossain *et al.*, 2015).

7.13 *Canscora decussata* (Roxb.) Schult. (Gentianaceae)

The hepatoprotective activity of *Canscora decussata* (Roxb.) Schult. is attributed to several flavonoids. Extracts of the plant were found to protect against liver damage induced by CCl_2 and paracetamol by reducing liver enzyme levels and improving liver function. The antioxidant properties of *C. decussata* help combat oxidative damage and support liver health (Saroja *et al.,* 2009).

7.14 *Dalbergia spinosa* Roxb. (Fabaceae)

Flavonoids and alkaloids in *Dalbergia spinosa* Roxb. extracts have demonstrated hepatoprotective effects. The methanolic and aqueous extracts of the plant reduced liver enzymes and bilirubin levels elevated by acetaminophen-induced liver damage. The plant's antioxidant properties contribute to liver healing and reduce inflammation, making it effective in managing liver disorders (Gharib *et al.,* 2014).

7.15 *Convolvulus arvensis* L. (Convolvulaceae)

It contain Quercetin, a flavonoid with hepatoprotective properties, is present in *Convolvulus arvensis* L. Ethanol extracts of this plant demonstrated a dose-dependent reduction in liver enzyme and bilirubin levels in paracetamol-induced liver toxicity. The hepatoprotective ability is mainly attributed to its antioxidant properties, which neutralize free radicals and prevent hepatic damage (Sahu *et al.,* 2016).

7.16 *Momordica charantia* (Cucurbitaceae)

It's commonly known as bitter melon, *Momordica charantia* is rich in alkaloids, flavonoids, and triterpenoids. Methanolic and aqueous extracts of this plant have shown protective effects against liver injury induced by acetaminophen and cadmium. The protective action involves reducing liver enzyme levels, improving liver function, and enhancing antioxidant properties (Tiwari *et al*., 2014).

7.17 *Mentha arvensis* L. (Lamiaceae)

Mentha arvensis L. contains flavonoids, tannins, and terpenoids, which are thought to exert hepatoprotective effects. Ethanol, chloroform, and aqueous extracts of this plant have reduced liver enzyme levels and increased antioxidant enzyme activities within the liver, preventing CCl, -induced damage by neutralizing oxidative stress (Jyothilekshmi *et al.*, 2020).

Table 4: Herbal hepatoprotective drugs

S.No.	Plant	Family	Chemical constituents	Mechanism of action	Type of extraction
1.	*Aerva javanica* (Burm.f.) Juss. ex Schult.	Amaranthaceae	Glycosides, flavonoids, saponins, terpenes	Antioxidant, anti-inflammatory, reduces liver enzymes	Aqueous methanol
2.	*Alhagi maurorum* Medik.	Leguminosae	Tannins, flavonoids, saponins	Reduces liver enzymes, antioxidant, regenerates liver tissue	Aqueous methanol
3.	*Clitoria ternatea* L.	Fabaceae	Alkaloids, flavonoids, tannins, saponins	Antioxidant, anti-inflammatory, protects liver from toxins	Ethanol extract
4.	*Phyllanthus amarus* Schumach. & Thonn.	Phyllanthaceae	Lignans, flavonoids, phenols	Antioxidant, anti-inflammatory, protects liver from acetaminophen	Aqueous extract
5.	*Acacia modesta* Wall.	Leguminosae	Tannins, saponins	Reduces liver enzymes, antioxidant	Methanolic extract
6.	*Cuminum cyminum* L.	Apiaceae	Alkaloids, flavonoids, terpenoids	Reduces liver enzymes, antioxidant, protects against Nimesulide	Aqueous ethanolic extract
7.	*Conyza bonariensis* (L.) Cronquist	Asteraceae	Flavonoids (quercetin)	Antioxidant, reduces liver enzyme levels	Ethanolic extract
8.	*Malva sylvestris* L.	Malvaceae	Mucilage, flavonoids, tannins	Antioxidant, reduces liver enzymes	Aqueous extract
9.	*Dicranopteris linearis* (Burm.f.)	Pteridaceae	Flavonoids, phenols	Antioxidant, protects liver from CCl, reduces liver enzymes	Aqueous, methanolic extract
10.	*Bauhinia hookeri* F. Muell.	Fabaceae	Polyphenols, flavonoids	Reduces liver enzymes, protects against toxins	Ethanol extract
11.	*Taraxacum officinale*	Asteraceae	Triterpenoids, flavonoids, sterols	Antioxidant, reduces liver enzymes, protects from CCl_2, ethanol	Aqueous extract
12.	*Cichorium intybus* L.	Asteraceae	Flavonoids, alkaloids	Protects liver from CCl -induced damage	Water extract

13.	*Canscora decussata* (Roxb.) Schult.	Gentianaceae	Flavonoids	Antioxidant, reduces liver enzymes	Aqueous, methanolic extract
14.	*Dalbergia spinosa* Roxb.	Fabaceae	Flavonoids, alkaloids	Reduces liver enzymes, bilirubin, antioxidant activity	Methanolic extract
15.	*Convolvulus arvensis* L.	Convolvulaceae	Quercetin	Antioxidant, reduces liver enzymes, bilirubin levels	Ethanol extract
16.	*Momordica charantia* L.	Cucurbitaceae	Alkaloids, flavonoids, triterpenoids	Protects liver from acet aminophen and cadmium -induced damage	Methanolic, aqueous extract
17.	*Mentha arvensis* L.	Lamiaceae	Flavonoids, tannins, terpenoids	Reduces liver enzymes, enhances antioxidant activity	Ethanol, chloroform, aqueous
18.	*Nyctanthes arbor-tristis* L.	Oleaceae	Alkaloids, flavonoids, triterpenoids	Antioxidant, anti-infl-ammatory, and regene rative effects, which help reduce oxidative stress, inflammation	Ethanol, chloroform, aqueous

7.18 *Nyctanthes arbor-tristis* L. (Nalpamaram)

It is commonly known as Night Jasmine, *Nyctanthes arbor-tristis* L. is a medicinal plant widely used in traditional medicine, particularly in Ayurveda. It is known for its hepatoprotective, anti-inflammatory, and antioxidant properties, making it useful in treating liver diseases, detoxifying the body, and managing inflammation. *Nalpamaram* is often included in polyherbal formulations for liver protection, especially in alcohol-induced hepatotoxicity. Its bioactive compounds help reduce oxidative stress and support liver regeneration. Additionally, this plant has been reported to have analgesic, antipyretic, and immunomodulatory effects (Jyothilekshmi *et al.,* 2020).

8. Allopathy vs. herbal treatment

Individual health responses as well as the severity of illness determine whether patients should pursue allopathic or herbal protection for their liver function. Acute liver conditions mostly require allopathic treatment because such approaches gain clinical approval from regulatory bodies thus becoming standard for critical conditions. The herbal medicines *P. amarus* and *S. marianum* serve as safe prolonged treatments that detoxify the liver and fight inflammation while reinvigorating tissue health. These compounds demonstrate improved safety profile compared to allopathic medicines while yielding beneficial effects on liver functions. A complete healthcare strategy involves allopathic pharmaceuticals for emergency treatments followed by herbal treatments for long-term liver overall health. Medical expertise becomes crucial for checking herb-drug reactions which enables better therapeutic effects.

9. Conclusion

Organic medications along with allopathic medicines display hepatoprotective effects but have different strengths and weaknesses. The clinically safe and broadly used allopathic drugs ursodeoxycholic acid, silymarin, and N-Acetylcysteine demonstrate tested effectiveness against acute and chronic liver diseases which makes them better choices in medical settings because of their established properties. Research into *A. javanica* and *P. amarus* and *C. intybus* demonstrates their potential as therapeutic agents since these plants possess antioxidant effects and anti-inflammatory properties and can support natural tissue regeneration. Additional clinical trials based on stringent protocols need to demonstrate these treatments' safety level and prevent unwanted side effects. A future synergy between herbal medicine and allopathic care through treatment combination models could build better hepatoprotective medicine options. Progressive nanotechnology together with advanced drug delivery platforms improves the availability of herbal compounds by dealing with absorption problems along with metabolic limitations. Genetic and metabolomic analyses integrated into personalized medicine would help scientists develop specific treatment plans that best protect liver health. Hepatoprotection's future rests on integrating evidence-based herbal research with innovative pharmaceutical technologies and precision medicine to create safer and more effective liver therapeutics.

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Conflict of interest

The author declares no conflicts of interest relevant to this article.

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