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Phytomedicine and Liver Disorders: A Review on Hepatoprotective Properties of Medicinal Plants

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ABSTRACT

The liver is a vital organ involved in metabolism, detoxification, and storage. It's constantly exposed to toxins from the digestive tract. The liver metabolizes drugs and chemicals, generating reactive oxygen species (ROS).High ROS levels cause oxidative stress and liver damage. Antioxidant enzymes like superoxide dismutase and catalase protect liver cells. The liver's structure is highly sensitive to oxidative injury. Liver function is assessed using tests like serum bilirubin, ALT, AST, and ALP. Disorders like hepatitis, fatty liver, NAFLD, and acute liver failure can impair liver function. Hepatitis viruses (A-E) vary in transmission and severity. Jaundice is a common symptom of liver dysfunction. Early diagnosis and management are crucial for preventing liver failure. Medicinal plants have long been recognized for their potent antioxidant properties, which play a critical role in mitigating oxidative stress-associated liver pathologies. Historically, numerous herbs and plant-based formulations have been employed in traditional medical systems for the management of hepatic disorders, often demonstrating therapeutic efficacy with minimal adverse effects. In this review, an extensive literature survey was conducted using reputable scientific databases such as PubMed and Google Scholar to identify medicinal plants with documented hepatoprotective activity. The primary objective of this article is to provide a comprehensive and systematic evaluation of hepatoprotective medicinal plants, highlighting their bioactive phytoconstituents, underlying molecular mechanisms of action, botanical sources, and therapeutic potential in the prevention and treatment of various liver diseases.

Keywords: Detoxifications, Xenobiotics, Kupffer cells, Bilirubin, Hepatitis and hepatoprotection

1.1. INTRODUCTION

The liver, as the primary organ, governs various body functions including metabolism, secretion, storage, and detoxifications. Typically, impairment to the liver correlates with the disruption of several of these functions. The liver in continuously exposed to an elevated level of toxic agents, because the portal vein supplies blood to this organ after intestinal absorption [1]. The liver, because of its role in metabolism, is exposed to many kinds of xenobiotics and therapeutic agents [2].

Hepatitis is a common disease in the world especially in the developing countries [3]. The World Health Organization (WHO) determined that around 2.4 million deaths yearly are linked to liver disease, and that around 800 thousand of these deaths are attributable to cirrhosis. In 2013, Mexico had over 600,000 recorded deaths, with diabetes mellitus (14.25%), ischemic heart diseases (12.63%). Cerebrovascular diseases (5.29%) and liver diseases (4.79%) ranking as the primary cause according to studies by National Institute of Statistics and Geography [4].

1.2. LIVER AND ITS ROLE

Liver is one of the vital organs in our body and plays a very important role in regulating, maintaining and carry out several physiological functions such as metabolization of nutrients like fat, protein and carbohydrates, synthesizing of essential amino acids, excretion of waste metabolites, and detoxification of waste metabolites. Additionally, it also serves to metabolize and eliminate drugs and other xenobiotics from our body. During oxygen metabolism reactive oxygen species (ROS) are Generated and at certain level it performs various physiology functions.

Generation of ROS is a part of normal physiological process and is responsible for the manifestation of several cellular processes including various signal Transduction pathways. It also works as a defense mechanism invading Microbes and expression of many

genes which are responsible for growth and ageing [5].Superoxide dismutase, Catalase, and glutathione etc., are antioxidant Enzymes, which act as natural antioxidant defense systems and protect its own Cells against oxidative damages [6]. Both enzymatic and non-enzymatic systems Are necessary to deal with oxidative stress of cells. Generally, drugs, chemicals or xenobiotics first metabolize in liver and forms toxic intermediates. So, liver Is the main targeted site for damage or oxidative stress. Hepatic cells metabolize Most of the exogenous chemicals including xenobiotic compounds [7].

During The process of detoxification, reactive oxygen species (ROS) are generated which causes oxidative stress that leads to hepatic damage [8].

1.3. STRUCTURE AND HISTOLOGY

Liver helps in processing of absorbed nutrients and to minimize exposure of the body to toxins and foreign chemicals. Consequently, the liver may be exposed to large concentrations of exogenous substance and their metabolites [9].

Liver toxicity leads to generation of oxidative radical, where parenchymal hepatic cells get primarily affected. The mitochondrion microsomes and peroxisomes in parenchymal cells can produce ROS, regulating on PPAR, which is mainly related to the liver fatty acid oxidation gene expression. In addition, Kupffer cells, endothelial cells and hepatic stellate cells are more prone to oxidative stress molecules. A variety of cytokines like TNF-alpha can be produced in Kupffer cells due to oxidative stress that may increase inflammation and apoptosis [10].

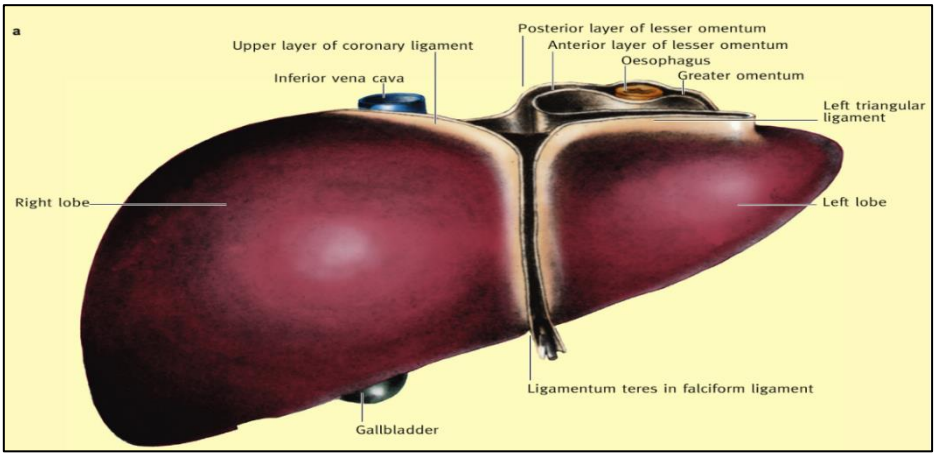

FIGURE 1: ANTERIOR VIEW OF LIVER

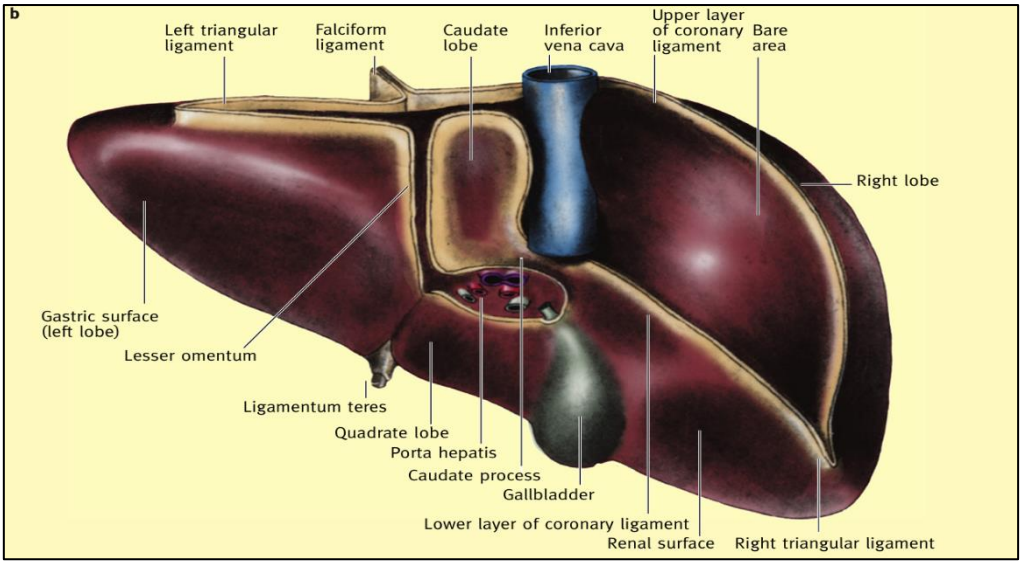

FIGURE 2: POSTERIOR VIEW OF LIVER [11]

1.4. FUNCTIONS OF LIVER

Homeostasis of various physiological processes are generally maintained by liver [12] and also performs various vital function in the body.

1. Vascular functions: The storage capacity of liver in liver sinusoids is 200 ml. Kupffer cells removes 90% of the bacteria and hepatocytes help in the synthesis of plasma protein.

2. Metabolic Functions: Liver cells have high metabolic rate.

a) Carbohydrate metabolism: Under the various effects of hormones, liver acts as a glucostat (glycogenesis, gluconeogenesis etc.). In liver glucose is converted into glycogen and stores inside the liver, and with the demand of energy this glycogen is again converted into glucose [13].

b) Metabolism of Protein: Liver cell synthesizes various essential amino acids and other plasma protein. Liver produces most of the blood clotting factors. It also produces some immune factors like phagocytes and in response to microbes these phagocytes start producing acute phase proteins which helps the body to fight against various infections. These proteins are also associated with tissue repair mechanism and activation of immune cells. It also helps in amino acid deamination and urea formation [14].

c) Metabolism of Fat: Fatty acids oxidation, formation of many cholesterols, lipoproteins, and phospholipids.

d) Detoxification: Liver functions to remove body wastes, drugs, and other foreign substances. Liver enzyme also converts some toxins into less toxic forms, which can be excreted out with urine. Bilirubin produced by red blood cells is due to breakdown of the hemoglobin and is also excreted into bile.

1.5. LIVER FUNCTION TEST

1.5.1. SERUM BILIRUBIN

Bilirubin is the catabolic product of haemoglobin produced within the reticuloendothelial system, released in unconjugated form which enters into the liver, converted to conjugated forms bilirubin Mono and diglucuronides by the enzyme UDP-glucuronyltransferase. Normal serum total bilirubin Varies from 2 to 21µmol/L. The indirect (unconjugated) bilirubin level is less than 12µmol/L and direct (conjugated) bilirubin less than 8µmol/L. The serum bilirubin levels more than 17µmol/L suggest Liver diseases and levels above 24µmol/L indicate abnormal laboratory liver tests.

Bilirubin is a breakdown product of hemoglobin. Produced in the reticuloendothelial system, it's released unconjugated.In the liver, UDP-glucuronyl transferase converts it to conjugated forms. Normal serum total bilirubin: 2-21 µmol/L. Unconjugated bilirubin < 12 µmol/L, conjugated bilirubin < 8 µmol/L.Levels > 17 µmol/L suggest liver disease, > 24 µmol/L abnormal liver tests. Jaundice occurs at 40 µmol/L. Unconjugated hyperbilirubinemia: Gilbert's syndrome, Crigler-Najjarsyndrome. Conjugated bilirubin elevated in viral hepatitis, liver damage. Biliary obstruction resolution shows bimodal decrease in conjugated bilirubin. Pregnancy lowers total and conjugated bilirubin. High bilirubin may protect against neurological damage from stroke.

1.5.2. ALANINE TRANSMINASE

Alanine Transaminase (ALT) is primarily found in the liver. It's a cytoplasmic enzyme catalyzing transamination reactions. Normal range: 7-56 U/L. Elevated ALT indicates liver cell injury. Mild elevation (up to 300 U/L) is nonspecific. Marked elevation (>500 U/L) suggests viral hepatitis, ischemic liver injury, or toxin-induced damage. Elevated ALT levels indicate hepatocellular damage. Viral hepatitis (A, B, C, D, E) causes significant ALT increases. Hepatitis C elevations are higher than hepatitis A or B. Persistent ALT elevation (>6 months) indicates chronic hepatitis. Non-alcoholic steatohepatitis and obesity-related liver fat increase ALT. Elevated ALT is linked to metabolic syndrome and insulin resistance.ALT levels rise in asymptomatic pregnancies, especially during the 2ndtrimester.Symptomatic pregnancies (hyperemesis, preeclampsia, hemolysis) show higher ALT.ALT levels drop significantly postpartum. Coffee and caffeine consumption reduce elevated ALT risk. This applies to cases with excessive alcohol, viral hepatitis, iron overload, and glucose metabolism issues.ALT is a valuable marker for liver health and related metabolic conditions.

1.5.3. ASPARTATE AMINO TRANSFERASE (AST)

AST catalyze transamination reaction. AST exist two different isoenzyme forms which are genetically distinct, the mitochondrial and cytoplasmic form. AST is found in highest concentration in heart compared with other tissues of the body such as liver, skeletal muscle and kidney. Normal serum AST is 0 to 35U/L. Elevated mitochondrial AST seen in extensive tissue necrosis during myocardial infarction and also in chronic liver diseases like liver tissue degeneration and necrosis. About 80% of AST activity of the liver is contributed by the mitochondrial isoenzyme, whereas most of the circulating AST activity in normal people is derived from the cytosolic isoenzyme. However, the ratio of mitochondrial AST to total AST activity has diagnostic importance in identifying the liver cell necrotic type condition and alcoholic hepatitis. AST elevations often predominate in patients with cirrhosis and even in liver diseases that typically have an increased ALT. AST levels in symptomatic pregnant patient in hyperemesis gravidarum were 73U/L, in pre-eclampsia 66U/L, and 81U/L was observed in hemolysis with low platelet count and elevated liver enzymes.

1.5.4. ALKALINE PHOSPHATASE (ALP)

Alkaline phosphatase (ALP) is present in mucosal epithelia of small intestine, proximal convoluted tubule of kidney, bone, liver and placenta. It performs lipid transportation in the intestine and calcification in bone. The serum ALP activity is mainly from the liver with 50% contributed by bone. Normal serum ALP is 41 to 133U/L. In acute viral hepatitis, ALP usually remains normal or moderately increased. Elevation of ALP with prolonged itching is related with Hepatitis A presenting cholestasis. Tumors secrete ALP into plasma and there are tumor specific isoenzymes such as Regan, Nagao and Kasahara. Hepatic and bony metastasis can also cause elevated levels of ALP. Other diseases like infiltrative liver diseases, abscesses, granulomatous liver disease and amyloidosis may cause a rise in ALP. Mildly elevated levels of ALP may be seen in cirrhosis, hepatitis and congestive cardiac failure. Low levels of ALP occur in hypothyroidism, pernicious anemia, zinc deficiency and congenital hypophosphatasia [15].

1.6. LIVER DISEASE

Liver disease is a general term that refers to any condition affecting your liver. These conditions may develop for different reasons, but they can all damage your liver and affect its function. Liver disease accounts for approximately 2 million deaths per year worldwide and the drugs are also one potential cause of liver disease. Drug induced liver disease accounts for as much as 20% of acute liver failure in pediatric population and a similar percentage in adults with acute liver failure [16].

1.6.1. NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Nonalcoholic Fatty Liver Disease (NAFLD)causes abnormal liver enzymes. NAFLD's subset, Nonalcoholic Steatohepatitis (NASH), risks progressive liver damage. No single treatment is recommended for NASH. Pioglitazone and vitamin E don't improve fibrosis. Bariatric surgery may benefit morbidly obese NASH patients. NASH is the 2nd leading liver disease among US adults awaiting transplantation. Aggressive management of obesity, diabetes, and metabolic syndrome is crucial. Prevention strategies target high-risk NAFLD patients. Effective NASH treatments are needed. Randomized bariatric surgery trials are lacking. NAFLD is a growing health concern globally. Managing metabolic disorders is key to addressing NAFLD and NASH [17].

1.6.2. ACUTE LIVER FAILURE

Acute liver failure occurs when your liver rapidly loses its ability to function. More commonly, liver failure develops slowly over the course of years. But acute liver failure, develops in a matter of days. Acute liver failure can cause many complications, including excessive bleeding and increasing pressure in the brain. Another term for acute liver failure is fulminant hepatic failure. Acute liver failure is a medical emergency that requires hospitalization. Some causes of acute liver failure can be reversed with treatment. But in other situations, a liver transplant may be the only cure for acute liver failure.

1.6.3. HEPATITIS

Hepatitis is swelling and inflammation of the liver. The term is often used to refer to a viral infection of the liver. Hepatitis can be caused by immune cells in the body attacking the liver and causing autoimmune hepatitis, infections from viruses (such as Hepatitis A, B,C, D and E), bacteria or parasites, liver damage from alcohol, poisonous mushrooms or other poisons, medications such as an overdose of acetaminophen, which can be deadly, Liver disease can also be caused by inherited disorders such as cystic fibrosis or hemochromatosis, a condition that involves having too much iron in our body (the excess iron deposits in the liver). Other causes include Wilson's disease (excess copper deposits in the body). The symptoms of hepatitis include: Abdominal pain or distention, breast development in males, dark urine, pale or clay colored stools, fatigue, usually low-grade fever, general itching, jaundice (yellowing of the skin and eyes), loss of appetite, nausea, vomiting and weight loss.

HEPATITIS A

Hepatitis A is an inflammation (irritation and swelling) of the liver from the hepatitis A virus. The hepatitis A virus is found mostly in the stools and blood of an infected person about 15 - 45 days before symptoms occur and during the first week of illness. Symptoms will usually show up 2 - 6 weeks after being exposed to the hepatitis A virus. They are usually mild, but may last for up to several months, especially in adults. Dark urine, fatigue, itching, loss of appetite, low-grade fever, nausea, vomiting, anorexia, malaise, pale or clay-colored stools and yellow skin.

HEPATITIS B

Hepatitis B is an irritation and swelling (inflammation) of the liver due to infection with the hepatitis B virus (HBV). Hepatitis B spread by contact with the blood or body fluids (such as semen, vaginal fluids, and saliva) of a person who has the virus. Symptoms of hepatitis B may not appear for up to 6 months after the time of infection. Early symptoms include loss of appetite, fatigue, low fever, muscle and joint aches, nausea and vomiting, jaundice, dark urine right upper quadrant pain and hepatomegaly.

HEPATITIS C

Hepatitis C is a viral disease that leads to swelling (inflammation) of the liver. Hepatitis C infection is caused by the hepatitis C virus (HCV). Hepatitis C spreads by contact with the blood of someone who has hepatitis C. The following symptoms may occur with hepatitis C infection like pain in the right upper abdomen, abdominal swelling due to fluid (ascites), clay-colored or pale stools, dark urine, fatigue, fever, itching, jaundice, loss of appetite, nausea and vomiting.

DELTA AGENT (HEPATITIS D)

Delta agent is a type of virus called hepatitis D. It causes symptoms only in people who also have a hepatitis B infection. Hepatitis D virus (HDV) is found only in people who carry the hepatitis B virus. HDV may make a recent (acute) hepatitis B infection or an existing longterm (chronic) hepatitis B liver disease which is worse. It can even cause symptoms in people who carry hepatitis B virus but who never had symptoms. Risk factors include abusing intravenous (IV) or injection drugs, being infected while pregnant (the mother can pass the virus to the baby), carrying the hepatitis B virus, men having sexual intercourse with other men, receiving many blood transfusions. Symptoms may include abdominal pain, darkcolored urine, fatigue, jaundice, joint pain, loss of appetite, nausea and vomiting.

HEPATITIS E

Hepatitis E is inflammation of the liver caused by infection with hepatitis E virus. It is one of five known human hepatitis viruses. A,B,C,D and E. Hepatitis E Virus is a positive –sense, single stranded non enveloped RNA icosahedral virus. HEV is predominantly transmitted by faecal contamination of drinking water as a result of poor sanitation.

1.7. FATTY LIVER

Fatty liver disease is the buildup of extra fat in liver cells. It is the earliest stage of alcoholrelated liver disease. There are usually no symptoms. If symptoms do occur, they may include fatigue, weakness, and weight loss. Almost all heavy drinkers have fatty liver disease. However, if they stop drinking, fatty liver disease will usually go away.

1.8. JAUNDICE

In jaundice, the skin and sclera of the eyes look yellow. Jaundice occurs when there is too much bilirubin (a yellow pigment) in the blood (a condition called hyperbilirubinemia). Bilirubin is formed when hemoglobin (the part of red blood cells that carries oxygen) is broken down as part of the normal process of recycling old or damaged red blood cells. These symptoms may include nausea, vomiting, abdominal pain, and small spider like blood vessels that are visible in the skin (spider angiomas). Men may have enlarged breasts, shrunken testes, and pubic hair that grows as it does in women [18].

2. ROLEOF MEDICINAL PLANTS IN HEPATOPROTECTIVE ACTIVITY

In the traditional system of medicine, medicinal plants play the major role in cure of various diseases [19].In large number of peoples of world population,still herbal medicines are popular remedies for ill health. Asian countries like India, China, Indonesia, and Japan herbal medicines are used to treat and prevention of various diseases [20]. Leading pharmaceutical companies have helped to popularize herbal therapies for liver diseases, which have been used for a long time in India. Even though a number of herbal

remedies are becoming increasingly popular, both generally and specifically for liver illnesses, they are still not suitable treatments for liver ailments. There are several issues that limit the possibility of this happening: (i) the herbal drugs are not standardized; (ii) the active ingredient(s) or principles are not identified; (iii) there are not enough randomized controlled clinical trials (RCTs); and (iv) there is not a toxicological review [21]. The extraction of pharmaceuticals from their natural sources has been an essential component of the drug discovery process [22].

Natural treatments might expedite the liver's healing process. Drugs made from herbs or their extracts are very important to human health. The CCl_4, D-Galactosamine, paracetamol, and ethanol-induced hepatotoxicity model is a commonly employed method for investigating the hepatoprotective properties of pharmaceuticals and hazardous substances [23].

2.1. Example of some drug with hepatotoxicity effect

Table 1: Example of some drug with hepatotoxicity effect [24]

Drugs	Implication
Flucanazole	It leads to hepatitis; it increases the transaminase level,fulminant hepatic failure, and cholestasis
Acetamenophene	It makes the cytochrome P-450-2E1 produce a toxicmetabolite NAPQI that causes hepatic necrosis
Amoxicillin	It moderates or brings about an increase in SGPT andSGOT levels, hepatic failure such as jaundice, acute cytolytic hepatitis, and hepatic cholestasis.
Diclofinac	It elevates AST and ALT levels, jaundice, fulminanthepatitis, and liver necrosis
Rifampicin	It leads to hepatitis, hyperbilirubinemia, and cholestasis
Ciprofloxacin	Elevation of SGOT alkaline phosphatase and SGPTlevels occurs from cholestatic jaundice
Oral contraceptives	Benign neoplasm, hepatic vein occlusion, and jaundice,but rarely neoplasm of the liver
Chlorepromazine	It leads to infectious hepatitis with obstructive jaundiceas a biomarker
Isoniazid	It elevates the serum transaminase level, severe and fatal hepatitis.
Erythromycin	It increases SGPT and SGOT concentration, and it also brings about hepatocellular hepatitis that are sometimes associated with it.

2.2. Methodology

A comprehensive literature search was conducted to identify medicinal plants and their phytoconstituents with hepatoprotective potential. The search strategy included the use of specific keywords such as “liver protective plants,” “hepatoprotective plants,” “phytoconstituents against liver disease,” and “phytocompounds against liver disease,” cross-referenced with “liver diseases” and “hepatoprotective activity”. Additionally, combinations of the terms “hepatoprotective” OR “liver protective” were paired with classes of bioactive compounds including alkaloids, terpenoids, glycosides, flavonoids, lignans, and polyphenolic compounds.The search was conducted across multiple scientific platforms and databases, including PubMed, Google Scholar, ResearchGate, Web of Science, NOPR, ScienceDirect, and other relevant sources such as academic books and peer-reviewed printed journals. The literature retrieval was limited to publications available in the English language and covered a time frame from January 2010 to January 2025.Articles were screened for relevance based on the presence of experimental evidence supporting hepatoprotective activity, particularly those involving *in vivo* animal models or *in vitro* cellular assays. References cited within the initially identified articles were also reviewed to extract additional relevant studies and experimental data.

2.3. MEDICINAL PLANTS USED IN HEPATOPROTECTIVE ACTIVITY

2.3.1. *Silybum marianum* (milk thistle)

Silybum marianum has been used for hepatoprotection for 2000 years. Silymarin protects the liver against toxins like carbon tetrachloride and acetaminophen. Its hepatoprotective effects are due to antioxidant activity and free radical scavenging. Silymarin increases glutathione concentration and stabilizes hepatocellular membranes. It stimulates DNA polymerase, increasing ribosomal

RNA synthesis and liver cell reconstruction. Silymarin decreases liver enlargement by inhibiting 5-lipoxygenase and leukotriene production. It inhibits lipid peroxidation and cellular damage in hepatocytes. Silymarin alters membrane lipids, including cholesterol and phospholipids. It influences lipoprotein uptake and secretion in the liver. Silymarin may diminish triglyceride synthesis and activate fatty acid beta-oxidation. Its effects on triglyceride metabolism in the liver are still being studied. Silymarin's antioxidant and hepatoprotective properties make it a valuable herbal remedy. It has been used to treat liver diseases, including hepatitis and cirrhosis. Silymarin's benefits extend to protecting against liver damage from toxins. It has also been shown to improve liver function in patients with liver disease. Silymarin's antioxidant properties help protect the liver from oxidative stress. Its anti-inflammatory properties also help reduce liver inflammation. Overall, silymarin is a promising herbal remedy for liver health [25].

2.3.2. ***Allium sativum***

A study found that fermented garlic extracts produced by lactic acid bacteria have liver-protecting potential. The extracts suppressed acetaminophen-induced cell death in hepatocytes by inhibiting MAPK phosphorylation. They also down-regulated p53, involved in liver autophagy, and reduced oxidative stress. Garlic compounds shielded the liver from ethanol-induced peroxidation. Alcohol administration increased ROS/RNS generation, depleted liver antioxidant status, and decreased glutathione concentrations. Garlic bulb isolate had no effect on alloxan-induced plasma advancements in liver enzymes and urinary metabolic indicators. The isolate did not alter plasma levels of urea, creatinine, albumin, AST, ALT, and ALP. Garlic and ascorbic acid mixtures have hepatoprotective action toward cadmium damage. Cadmium administered mice had increased malignant growth, reversed by ascorbic acid and garlic extract. Garlic's organosulfur compounds, like diallyldisulfide and diallyltrisulfides, have antioxidant and detoxifying properties. These compounds contribute to garlic's hepatoprotective properties. Garlic extracts have been shown to reduce oxidative stress and inflammation in the liver. They also have been found to improve liver function and reduce liver damage. The liver-protecting potential of garlic extracts makes them a promising natural remedy. Further research is needed to fully understand the mechanisms behind garlic's hepatoprotective effects. Garlic's antioxidant and detoxifying properties make it a valuable addition to a healthy diet. Overall, garlic has shown significant potential in protecting the liver from damage [26].

2.3.3. *Solanum nigrum*

Solanum nigrum extracts were studied for hepatoprotective activity in rats with CCl_4-induced liver damage. Aqueous and methanolic extracts (250-500 mg/kg) showed hepatoprotective effects. Serum AST, ALT, ALP, and bilirubin levels decreased significantly in treated rats. Histopathological lesions were mild in treated rats compared to untreated ones.Ethanol extract of *S. nigrum* also showed remarkable hepatoprotective activity. Biochemical parameters (AST, ALT, ALP, and bilirubin) were used to evaluate hepatoprotective activity. Histopathological changes in liver samples were compared between treated and control groups. *S. nigrum* extracts demonstrated potential in protecting against CCl_4-induced liver damage. The study suggests *S. nigrum* as a potential natural remedy for liver protection [27].

2.3.4. *Foeniculum vulgare*

The essential oil of *Foeniculum vulgare (F. vulgare)* seeds was studied. Its hepatoprotective effects against carbon tetrachloride-induced acute hepatotoxicity in rats were evaluated. Oral administration of the essential oil was found to be effective. It reduced serum levels of AST, ALT, ALP, and bilirubin. The reduction in serum enzymes indicated liver protection. The essential oil's constituents, d-limonene and β-myrcene, may have contributed to the liver protection. These compounds may have antioxidant and anti-inflammatory effects. The study suggests that *F. vulgare* essential oil has hepatoprotective properties. It may be useful in preventing liver damage. Further research is needed to confirm its efficacy. The study provides evidence for the potential benefits of *F. vulgare* essential oil. Its hepatoprotective effects are promising. The oil may be a useful natural remedy for liver protection [28].

2.3.5. *Spinacia oleraceae*

Spinacia oleracea L. (S. oleracea) leaves' alcoholic extract (SE) showed hepatoprotective effects against carbon tetrachlorodide (CCl_4)-induced hepatosuppression. SE pre-treatment restored serum and liver parameters to near-normal levels. The extract was administered at 100 and 200 mg/kg/day for 7 days. Serum marker enzymes, bilirubin, and protein levels were evaluated. Hepatic antioxidants, cytochrome P-450 enzyme, and lipid peroxidation were also assessed. SE significantly restored these parameters. The hepatoprotective potential of *S. oleracea* may involve blocking P-450 mediated CCl_4 bioactivation. It may also involve selective inhibition of reactive oxygen species (ROS). *S. oleracea's* antioxidant properties contribute to its liver-protective effects. The extract's ability to restore liver function suggests its potential as a therapeutic agent. Further research is needed to fully understand *S. oleracea's* hepatoprotective mechanisms. *S. oleracea* may prove to be a valuable natural remedy for liver protection. Its antioxidant properties make it a promising candidate for further study. Overall, *S. oleracea's* hepatoprotective effects are attributed to its antioxidant and ROS-inhibiting properties. This natural extract may offer a safe and effective way to protect the liver [29].

2.3.6. *Curcuma longa*

Curcumin, an antioxidant in *Curcuma longa*, enhances apoptosis of damaged hepatocytes. This mechanism may down-regulate inflammatory effects and fibrogenesis in the liver. Ethanolic extract of *Curcuma longa* showed significant hepatoprotective effects. The extract was administered orally at 250 mg/kg and 500 mg/kg, with dose-dependent effects. Main constituents include curcumin, tumerone, atlantone, and zingiberene. Hepatoprotective effects may be due to antioxidant and free radical scavenging mechanisms. Curcumin also indirectly augments glutathione levels, aiding hepatic detoxification. Volatile oils and curcumin exhibit potent anti-inflammatory effects. *Curcuma longa's* hepatoprotective effects make it a valuable natural remedy. Its antioxidant and anti-inflammatory properties contribute to its liver-protective effects [30].

2.3.7. *Tinospora cordifolia*

The study evaluated the hepatoprotective activity of *Tinospora cordifolia* extracts. Ethanolic extracts of the leaf, stem, and root were tested. The extracts were administered orally to Wistar albino rats at 200mg/kg body weight. Silymarin was used as a reference standard. The ethanolic extracts showed significant hepatoprotective effects. They reduced serum enzymes ALT, AST, ALP, and total bilirubin. Aqueous and pet ether extracts also showed hepatoprotective effects. The plant's bioactive compounds are likely responsible for its hepatoprotective activity. Flavonoids and alkaloids are among the bioactive compounds present. The results justify the use of *Tinospora cordifolia* as a hepatoprotective agent. The plant has been used in traditional medicine for its medicinal properties. The study confirms its hepatoprotective potential [31].

2.3.8. *Zingiber officinale*

The ethanolic extract of *Zingiber officinale (Z. officinale)* showed antihepatotoxic effects. It was tested against carbon tetrachloride (CCl_4) and paracetamol-induced hepatotoxicity in rats. Administering ginger extract before CCl_4 reduced serum enzymes (SGPT and SGOT) more effectively. The extract also reduced plasma SOD levels at 200mg/kg. At 300mg/kg, it reduced SOD, hepatic MDA, and serum AST. Plasma proteins were increased at the higher dose. The results suggest that *Z. officinale* extract has hepatoprotective properties. It supports the traditional use of ginger in medicine. The extract's antihepatotoxic effects are promising. Further research is needed to confirm its efficacy. *Z. officinale* may be a useful natural remedy for liver protection. Its hepatoprotective effects may be attributed to its antioxidant properties. The study provides evidence for the potential benefits of ginger extract [32].

2.3.9. *Pterocarpus marsupium*

Pterocarpus marsupium Roxb. leaves ethanolic extract (EEPM) was evaluated for hepatoprotective activity against paracetamol-induced liver damage (PILD) in rats. Rats were divided into five groups and treated with EEPM or silymarin for seven days. On the eighth day, a single dose of paracetamol was administered. EEPM (400 mg/kg/day) significantly decreased liver damage markers. It reduced ALT, AST, ALP, LDH, total cholesterol, bilirubin, triglyceride, and TBARS levels. EEPM also increased antioxidant levels, including SOD, GSH, and CAT. Its hepatoprotective activity was comparable to silymarin. The extract prevented paracetamol-induced oxidative stress and altered biochemical markers. EEPM's therapeutic efficacy against liver damage was indicated. The study suggests *Pterocarpus marsupium* as a potential natural remedy for liver protection. Its antioxidant and hepatoprotective properties contribute to its liver-protective effects. EEPM may offer a safe and effective way to protect the liver. Further research is needed to fully understand its mechanisms. Overall, EEPM shows promise as a hepatoprotective agent. Its natural origin and potential efficacy make it a valuable candidate for liver protection [33].

2.3.10. *Rhodiola imbricata*

Rhodiola imbricata, a perennial herb, has traditional medicinal usage. It biosynthesizes phytochemicals like flavonoids and phenyl glycosides. This study investigated the hepatoprotective activity of *R. imbricata* rhizome acetone extract. The extract was tested against paracetamol-induced liver toxicity in Wistar rats. Rats were administered with 200 and 400 mg/kg doses of the extract. Silymarin was used as a standard reference. The extract showed elevated liver antioxidants and improved biochemical parameters. It protected hepatic cells from damage and normalized hematological parameters. The extract's hepatoprotective activity was comparable to silymarin. HPLC analysis revealed important phenolic compounds responsible for the activity. This study suggests *R. imbricata* as a natural source of hepatoprotective agents. The herb's traditional medicinal usage is validated by its hepatoprotective properties. *R. imbricata's* antioxidant and hepatoprotective activities make it a valuable natural remedy. Further research is needed to fully understand its mechanisms. The study's findings have implications for the development of natural hepatoprotective agents. *R. imbricata's* potential as a liver protectant is significant. Its natural origin and efficacy make it a valuable candidate for liver protection. Overall, *R. imbricata* shows promise as a hepatoprotective agent [34].

2.3.11. *Taraxacum officinale*

The liver's protection against oxidative stress and harm is crucial. Medicinal plants, like *Taraxacum officinale* (dandelion), offer hepatoprotective effects. This review evaluates dandelion's hepatoprotective effects and mechanisms. Dandelion extracts show hepatoprotective effects against chemicals due to antioxidant and anti-inflammatory activities. Its benefits include anti-inflammatory effects, prebiotic effects, and inhibiting lipopolysaccharides release. Dandelion reduces liver inflammation and lipid accumulation, enhancing liver functions. Emerging evidence supports dandelion's hepatoprotective effects. Large human clinical studies are necessary to confirm its benefits. Dandelion's traditional use for liver issues is validated by its antioxidant properties. Its hepatoprotective effects make it a valuable natural remedy. Further research is needed to fully understand its mechanisms. Dandelion's potential as a liver protectant is significant. Its natural origin and efficacy make it a valuable candidate for liver protection. Overall, dandelion shows promise as a hepatoprotective agent. Its benefits extend beyond liver protection, offering anti-inflammatory and prebiotic effects. Dandelion's versatility makes it a valuable addition to natural medicine. As research continues, dandelion's potential as a natural remedy will become clearer [35].

2.3.12. *Artemisia absinthium*

This study evaluated the hepatoprotective activity of *Artemisia absinthium* L. aqueous extract (AEAA) in mice. AEAA was administered orally at 50-200 mg/kg/day. Liver injury was induced chemically or immunologically. Results showed AEAA significantly prevented increase in serum hepatic enzymes. It reduced lipid peroxidation and restored antioxidant enzymes. AEAA also suppressed pro-inflammatory mediators TNF-α and IL-1. Histopathology showed AEAA attenuated hepatocellular necrosis and reduced inflammatory cells infiltration. Phytochemical analysis revealed sesquiterpene lactones, flavonoids, phenolic acids, and tannins in AEAA. The study supports AEAA's traditional use in treating liver disorders. AEAA's antioxidative and immunomodulatory activity protects against acute liver injury. The extract's hepatoprotective effects are dose-dependent. AEAA's ability to reduce lipid peroxidation and restore antioxidant enzymes is notable. The study's findings have implications for the development of natural hepatoprotective agents. AEAA's potential as a liver protectant is significant. Its natural origin and efficacy make it a valuable candidate for liver protection. Overall, AEAA shows promise as a hepatoprotective agent. Further research is needed to fully understand its mechanisms [36].

2.3.13. *Cichorium intybus*

This study evaluated the hepatoprotective activity of *Cichorium intybus (C. intybus)* hydroalcoholic extract against carbon tetrachloride (CCl_4)-induced liver injury in rats. The extract (200-500 mg/kg) showed significant protective effects against CCl_4-induced hepatotoxicity. CCl4 treatment increased serum markers AST, ALT, ALP, and total bilirubin, while *C. intybus* extract suppressed these increases, indicating improved liver function. The study confirms the hepatoprotective activity of *C. intybus* hydroalcoholic extract [37].

2.3.14. *Croton bonplandianum*

The hepatoprotective activity of hydro-methanolic extract of *Croton bonplandianum*leaves extract showed the highest hepatoprotective activity by ameliorating haloalkane induced liver injury in the murine model. Silymarin was used as a standard drug. The increased level of biochemical activity was significantly restored by hydro-methanolic extract of *Croton bonplandianum*treatment. A molecular docking study revealed that the compound α-amyrin present in the leaves extract of *Croton bonplandianum*has better capability to ameliorate hepatocellular damages than the positive control Silymarin [38].

2.3.15. *Abutilon indicum*

This study evaluated the hepatoprotective activity of *Abutilon indicum* (AI) leaf extracts. Hydroalcoholic and ethyl acetate extracts were prepared and administered to rats. Liver damage was induced using CCl_4. Biochemical parameters were analyzed, including AST, ALT, ALP, and total protein. Histopathological studies were also conducted. Results showed that both extracts possessed hepatoprotective activity. However, the ethyl acetate extract showed greater hepatoprotection. It effectively protected the liver against CCl_4-induced toxicity. The study suggests that *Abutilon indicum* leaf extracts may be useful in preventing liver damage. Further studies are needed to confirm its hepatoprotective effects. The ethyl acetate extract's greater efficacy makes it a promising natural remedy [39].

2.3.16. *Terminalia arjuna*

Terminalia arjuna's arjunolic acid (AA) was studied for its hepatoprotective effects against Non-Alcoholic Fatty Liver Disease (NAFLD). AA was isolated from the heartwood of *T. arjuna* and its structure confirmed. In vitro studies using HepG2 cells showed

AA reduced triglyceride accumulation and lipotoxicity. In vivo studies using HFD-fed rats demonstrated AA's efficacy in reducing transaminases, phosphatase, and GGT levels. AA up regulated PPARα and FXRα expressions while down regulating PPARγ. Liver histology showed reduced steatosis and inflammation. These findings suggest AA as a promising lead for treating NAFLD, warranting further research [40].

2.3.17. *Psidium guajava*

Psidium guajava, a traditional medicinal plant, was studied for its hepatoprotective activity. The plant's ethanolic extract and phospholipid complex were evaluated against paracetamol-induced liver damage in rats. The study aimed to verify the plant's traditional use in treating hepatitis. Results showed significant hepatoprotective effects, with decreased serum levels of SGOT, SGPT, ALP, and bilirubin. Histopathology verified the hepatoprotective effect. The phospholipid complex showed better activity than the plain extract. The complex's activity was comparable to standard silymarin. The study supports the traditional use of *P. guajava* for treating hepatitis. The plant's hepatoprotective activity was confirmed. The phospholipid complex's enhanced activity suggests its potential as a natural hepatoprotective agent. Further research is needed to fully understand the plant's mechanisms. *P. guajava's* traditional use in medicine is validated by its hepatoprotective effects. The plant's potential as a natural remedy for liver damage is significant. Its natural origin and efficacy make it a valuable candidate for liver protection. Overall, *P. guajava* shows promise as a hepatoprotective agent. Its phospholipid complex may offer enhanced protection against liver damage [41].

2.3.18. *Tamarindus indica*

The study evaluated the hepatoprotective potential of *Tamarindus indica* leaves ethyl acetate fraction (EFTI). Pregnant rats were divided into groups and administered EFTI, aluminum chloride, or vitamin E. The goal was to assess EFTI's protective effects against prenatal aluminum chloride exposure. Aluminum chloride can cause oxidative stress and liver damage. EFTI was administered at 400 and 800 mg/kg doses. Results showed that EFTI significantly improved liver tissue levels of malondialdehyde, caspase-3, and tumor necrosis factor-alpha. EFTI also reduced liver enzymes, including aspartate aminotransferase and alkaline phosphatase. Histopathological changes were also improved. The study concluded that EFTI has hepatoprotective effects during prenatal aluminum chloride exposure. EFTI's protective effects are mediated by anti-lipid peroxidative, antiapoptotic, and anti-inflammatory activities. The findings suggest that EFTI may be useful in preventing liver damage caused by aluminum chloride exposure. Further research is needed to confirm these findings. The study provides evidence for the hepatoprotective potential of *Tamarindus indica* leaves. EFTI may be a useful natural remedy for liver protection. The study's results have implications for the prevention of liver damage. EFTI's protective effects may also extend to other oxidative stress-related conditions [42].

2.3.19. *Bambusa bambos*

The young shoots of *Bambusa bambos* are used in Ayurvedic medicine. They contain bioactive compounds like cholin, betain, and cyanogenetic glucosides. This study evaluated the hepatoprotective activity of the methanolic shoot extract. Carbon tetrachloride (CCl_4) was used to induce hepatotoxicity in rats. Rats were divided into groups and administered CCl_4, silymarin, or methanolic shoot extract. The extract attenuated the increase in liver enzymes (AST, ALT, ALP). Total bilirubin levels were also reduced. The study suggests that *B. bambos* methanolic shoot extract may be useful in preventing liver damage. The extract showed hepatoprotective potential against CCl_4-induced hepatotoxicity. The bioactive compounds in the extract may be responsible for its hepatoprotective effects. Further research is needed to fully understand the extract's mechanisms. The study supports the traditional use of *B. bambos* in Ayurvedic medicine. The extract may be a useful natural remedy for liver damage [43].

2.3.20. *Carica papaya*

This study investigated the hepatoprotective effect of papaya (*Carica papaya* L.) ethanol extract on acetaminophen-induced liver damage in rats. 25 rats were divided into five groups, including a normal group, negative control, and three treatment groups with different doses of papaya extract. The extract was administered for 7 days, followed by acetaminophen induction. SGOT and SGPT levels were measured, showing significant differences between the negative control and treatment groups. The most effective dose was found to be 13.9 mg/200g body weight, which decreased SGOT and SGPT levels to near-normal values [44].

3. CONCLUSION:

Natural products derived from medicinal plants are increasingly recognized for their favorable safety profiles compared to synthetic pharmaceutical agents. Liver disorders represent a major global health concern, affecting populations across both developed and developing nations. In India, a vast repository of traditional medicinal knowledge has long incorporated plant-based therapies for the treatment of hepatic ailments. This review aims to consolidate and critically evaluate current evidence on the hepatoprotective potential of select medicinal plants commonly used in the Indian traditional system of medicine. The literature review highlights

promising hepatoprotective activity attributed to species such as *Silybum marianum, Pterocarpus marsupium, Cichorium intybus, Solanum nigrum, Croton bonplandianum, Tinospora cordifolia, Artemisia absinthium, Terminalia arjuna , Rhodiola imbricata , and Zingiber officinale.* These plants, along with their bioactive phytoconstituents, have demonstrated significant efficacy in countering hepatic damage induced by various hepatotoxins in experimental models. The current scientific evidence suggests that these phytochemicals exert hepatoprotective effects primarily through their potent antioxidant activity, which helps mitigate oxidative stress and subsequent hepatocellular injury. Given their lower incidence of side effects compared to conventional synthetic drugs, these plants present strong candidates for further exploration. Future research should prioritize the rigorous evaluation of these medicinal plants through well-designed preclinical and clinical studies to validate their efficacy and safety in human subjects. In particular, *in silico* approaches-including ADME (Absorption, Distribution, Metabolism, and Excretion) profiling, molecular docking studies, and network pharmacology analyses-can elucidate mechanisms of action, predict pharmacokinetic behavior, and support rational drug development strategies. Moreover, the potential for synergistic effects between plant-derived compounds and conventional drugs opens a promising avenue for combination therapies. Such integrative approaches could enhance therapeutic efficacy and minimize adverse effects in liver disease management. In conclusion, medicinal plants from traditional Indian medicine, supported by modern pharmacological validation, may offer novel, effective, and safer alternatives or adjuncts in the treatment of liver disorders. Their incorporation into mainstream medicine could significantly improve outcomes for patients suffering from hepatic diseases.

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