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A Review on: Alkaloids as Potential Hepatoprotective Agents in the Treatment and Prevention of Liver Cirrhosis

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ABSTRACT

Liver cirrhosis is a chronic and progressive liver disorder characterized by fibrosis, nodular regeneration, and impaired hepatic function, resulting from long-term liver injury caused by viral infections, alcohol abuse, metabolic disorders, and toxins. Despite advances in medical therapy, effective pharmacological interventions remain limited. Alkaloids, a diverse group of naturally occurring nitrogen-containing compounds, have attracted significant attention for their hepatoprotective properties. Numerous plant-derived alkaloids such as berberine, matrine, and piperine have demonstrated antioxidant, anti-inflammatory, and antifibrotic activities in experimental models of liver injury. These compounds modulate oxidative stress pathways, inhibit hepatic stellate cell activation, regulate inflammatory cytokines, and improve liver enzyme profiles. This review summarizes the pharmacological mechanisms, therapeutic potential, and current research findings on alkaloids in the treatment and prevention of liver cirrhosis. Further clinical studies are necessary to establish their safety, efficacy, and future role in cirrhosis management.

Keywords : Alkaloids; Hepatoprotective activity; Liver cirrhosis; Antifibrotic; Antioxidant; Phytochemicals; Hepatic stellate cells; Chronic liver disease.

INTROCDUCTION

Liver cirrhosis is widely prevalent in both low-income and middle-income countries and in high-income countries, and is associated with high morbidity and mortality. Cirrhosis is a consequence of chronic liver inflammation that is followed by diffuse hepatic fibrosis, wherein the normal hepatic architecture is replaced by regenerative hepatic nodules, which eventually leads to liver failure. Chronic liver inflammation does not progress to cirrhosis in all patients, but when progression does occur, the rate at which it happens varies from weeks (in patients with complete biliary obstruction) to decades (in patients with longer-term causes, such as viral hepatitis C). The asymptomatic (initial) phase of cirrhosis can be followed by a relatively short symptomatic phase of months to years. The symptomatic phase, usually designated as decompensated cirrhosis, is associated with various complications that result in frequent hospital admission, impaired quality of life of patients and caregivers, and patient death in the absence of liver transplantation. Patients with cirrhosis without any symptoms are termed to have compensated cirrhosis. Complications such as ascites, variceal bleeding, hepatic encephalopathy, or non-obstructive jaundice, which can develop with cirrhosis of any origin, herald the onset of decompensated cirrhosis. In the presence of cirrhosis, superimposed hepatic injury (due to viral, drug-induced, or alcohol-associated hepatitis) or other complications, particularly bacterial infections, can lead to hepatic and extrahepatic organ failure—a condition known as acute-on-chronic liver failure—that is associated with high short-term mortality. Most deaths in patients with decompensated cirrhosis result from hepatic and extrahepatic organ failure. Deaths during the compensated stage are largely due to cardiovascular disease, malignancy, and renal disease. Cirrhosis appears to receive less public attention than other chronic diseases, such as congestive heart failure, chronic obstructive pulmonary disease, and chronic kidney disease, which is partly attributable to the stigmatisation of cirrhosis and the perception that the disease is largely related to alcohol consumption. Public awareness of the relevance of cirrhosis is still low and the disease is not commonly diagnosed during the development phase, which leads to missed opportunities to mitigate causative factors and prevent subsequent progression. Important research efforts over the past 20 years have improved our understanding of the pathogenesis, diagnosis, and treatment of the disease. In this Seminar, we summarise the current understanding of cirrhosis, and present a brief discussion on hepatocellular carcinoma.[1]

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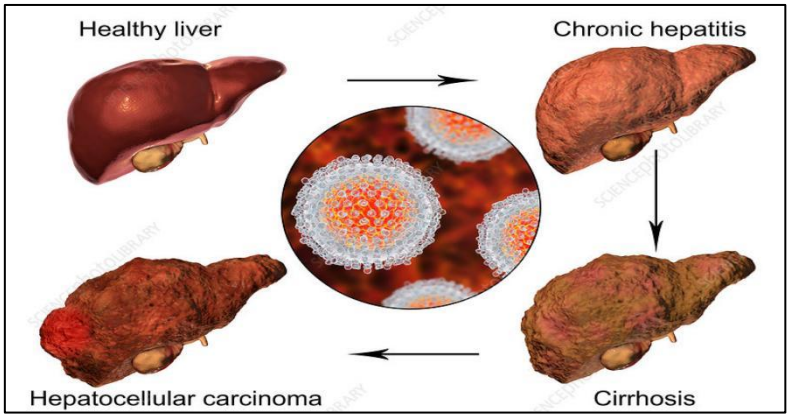


Fig- Liver Cirrhrosis

EPIDEMIOLOGY

Cirrhosis is an increasing cause of morbidity and mortality in more developed countries. It is the 14th most common cause of death in adults worldwide but the fourth in central Europe; it results in 1·03 million deaths per year worldwide,8 170 000 per year in Europe, and per year in the USA. Cirrhosis is the main indication for 5500 liver transplants each year in Europe. The main causes in more developed countries are infection with hepatitis C virus, alcohol misuse, and, increasingly, non-alcoholic liver disease; infection with hepatitis B virus is the most common cause in sub Saharan Africa and most parts of Asia. The prevalence of cirrhosis is difficult to assess and probably higher than reported, because the initial stages are asymptomatic so the disorder is undiagnosed. Prevalence was estimated at 0·3% in a French screening programme, and the annual incidence was per 100 000 people in studies in the UK and Sweden.[2]

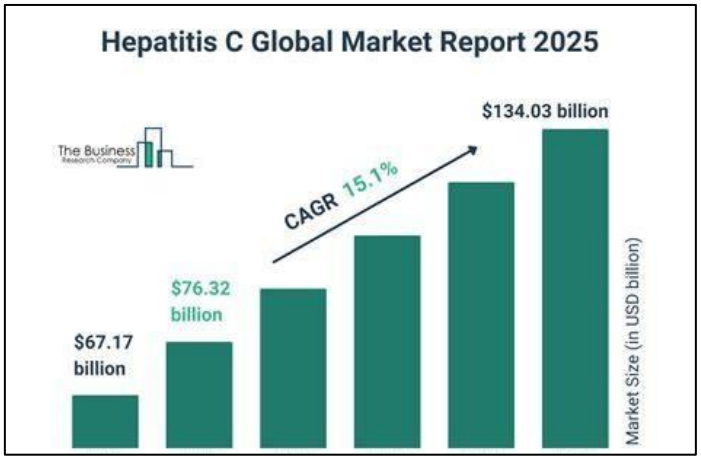


Fig- Global Market report of Liver Cirrhosis

PATHOPHYSIOLOGY

The transition from chronic liver disease to cirrhosis involves inflammation, activation of hepatic stellate cells with ensuing fibrogenesis, angiogenesis, and parenchymal extinction lesions caused by vascular occlusion. This process leads to pronounced hepatic microvascular changes, characterised by sinusoidal remodelling (extracellular matrix deposition from proliferating activated stellate cells resulting in capillarisation of hepatic sinusoids), formation of intra hepatic shunts (due to angiogenesis and loss of parenchymal cells), and hepatic endothelial dysfunction. The endothelial dysfunction is characterised by insufficient release of vasodilators, of which the most important is nitric oxide. Release of nitric oxide is inhibited by low activity of endothelial nitric oxide synthetase (as a result of insufficient protein-kinase-B-dependent phosphorylation, lack of cofactors, increased scavenging resulting from oxidative stress, and high concentrations of endogenous inhibitors of nitric oxide), with concomitant increased production of vasoconstrictors (mainly adrenergic stimulation and thromboxane A2, but also activation of the renin-angiotensin system, antidiuretic hormone, and endothelins). Increased hepatic resistance to portal blood flow is the primary factor increasing portal pressure in cirrhosis (figure). It results from the combination of structural disturbances associated with advanced liver disease

(accounting for about 70% of total hepatic vascular resistance) and of functional abnormalities leading to endothelial dysfunction and increased hepatic vascular tone; portal pressure could perhaps therefore be decreased by 30% if this functional abnormality were antagonised.[3]

The molecular mechanisms of these abnormalities are being delineated and represent new targets for therapy. Splanchnic vasodilation with an ensuing increase in the inflow of blood into the portal venous system contributes to aggravate the increase in portal pressure. Splanchnic vasodilation is an adaptive response to the changes in intrahepatic haemodynamics in cirrhosis; its mechanisms are directly opposite to those of the increased hepatic vascular tone. Because of this opposition, attempts to correct portal hypertension by acting on hepatic resistance or portal blood inflow should be ideally based on strategies acting as selectively as possible on the intrahepatic or the splanchnic circulation. In advanced cirrhosis, splanchnic vasodilation is so intense as to determine a hyper dynamic splanchnic and systemic circulation, which together with portal hypertension has a major role in the pathogenesis of ascites and hepatorenal syndrome. Systemic vasodilation further Increased hepatic resistance causes pulmonary ventilation/perfusion mismatch that in severe cases leads to hepatopulmonary syndrome and arterial hypoxaemia. Portopulmonary hypertension is characterised by pulmonary vaso constriction, which is thought to be due to endothelial dysfunction in the pulmonary circulation. Formation and increase in size of varices is driven by anatomical factors, increased portal pressure and collateral blood flow, and by angiogenesis dependent on vascular endothelial growth factor, all of which contribute to variceal bleeding. Dilation of gastric mucosal vessels leads to portal hypertensive gastropathy. In addition, the shunting of portal blood to the systemic circulation through the portosystemic collaterals is a major determinant of hepatic encephalopathy, of decreased first-pass effect of orally administered drugs, and of decreased reticulo endothelial system function. However, capillarisation of sinusoids and intrahepatic shunts are also important because these changes interfere with effective hepatocyte perfusion, which is a major determinant of liver failure.[4]

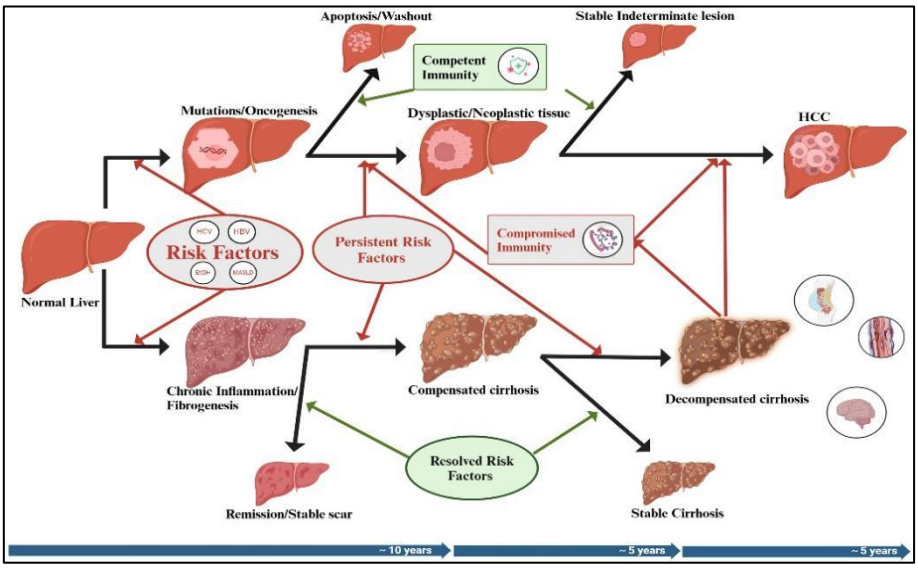


Fig- Pathophysiology of Liver Cirrhosis

MECHANISMS OF HEPATOPROTECTIVE ACTION OF ALKALOIDS

Alkaloids exert significant hepatoprotective effects through multiple interconnected molecular mechanisms that collectively target the major pathological processes involved in liver cirrhosis, including oxidative stress, chronic inflammation, hepatic stellate cell activation, fibrosis, apoptosis, and metabolic dysregulation. Because liver cirrhosis develops through sustained hepatocellular injury and progressive extracellular matrix deposition, compounds with multitarget actions such as alkaloids are of considerable therapeutic interest.

Oxidative stress is a central contributor to liver injury and fibrogenesis. Excessive generation of reactive oxygen species (ROS) during alcohol metabolism, viral infection, toxin exposure, or metabolic dysfunction leads to lipid peroxidation, mitochondrial damage, protein oxidation, and DNA injury in hepatocytes. Alkaloids counteract oxidative stress through both direct and indirect mechanisms. Many alkaloids possess intrinsic free radical–scavenging properties that neutralize superoxide anions, hydroxyl radicals, and hydrogen peroxide, thereby reducing lipid peroxidation markers such as malondialdehyde. More importantly, several alkaloids enhance endogenous antioxidant defense systems by upregulating enzymes including superoxide dismutase, catalase, glutathione peroxidase, and increasing intracellular reduced glutathione levels. A key molecular mechanism underlying this effect

involves activation of the nuclear factor erythroid 2–related factor 2 (Nrf2) signaling pathway. Upon activation, Nrf2 translocates to the nucleus and binds to antioxidant response elements (ARE), promoting transcription of cytoprotective genes such as heme oxygenase-1 and NAD(P)H quinone dehydrogenase 1. For example, berberine derived from Berberis vulgaris has been shown to activate the Nrf2 pathway, thereby strengthening the cellular antioxidant network and protecting hepatocytes from oxidative injury.[5]

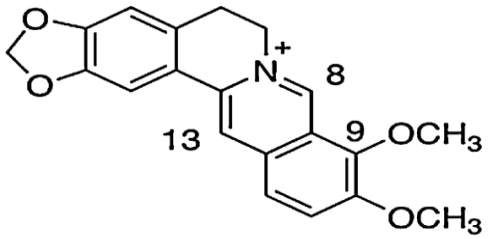
Chronic inflammation represents another fundamental mechanism driving the progression from liver injury to fibrosis and cirrhosis. Persistent activation of Kupffer cells and infiltrating immune cells results in excessive production of pro-inflammatory cytokines such as tumor necrosis factor-alpha, interleukin-1β, and interleukin-6. These mediators further amplify hepatocellular damage and stimulate hepatic stellate cell activation. Alkaloids suppress inflammatory responses primarily through inhibition of the nuclear factor-kappa B (NF-κB) signaling pathway. By preventing phosphorylation and nuclear translocation of NF-κB, alkaloids reduce transcription of genes encoding pro-inflammatory cytokines, chemokines, and enzymes such as cyclooxygenase-2 and inducible nitric oxide synthase. In addition, modulation of mitogen-activated protein kinase (MAPK) pathways, including p38, JNK, and ERK, contributes to the anti-inflammatory activity of alkaloids. Matrine, isolated from Sophora flavescens, has demonstrated significant suppression of inflammatory mediators in experimental models of hepatic fibrosis, highlighting the importance of inflammation-targeted therapy in cirrhosis prevention.[5]

ALKALOID'S USED IN LIVER CIRRHOSIS

Alkaloids are naturally occurring nitrogen-containing organic compounds, primarily derived from plants, but also found in fungi, bacteria, and animals. They usually exhibit significant pharmacological activity and often possess heterocyclic ring structures containing nitrogen atoms. Many medicinal plants used in traditional systems such as Ayurveda and Traditional Chinese Medicine contain alkaloids that show protective effects against liver damage.[6]

1. BERBERINE:

Berberine is a natural isoquinoline alkaloid obtained from medicinal plants such as: Berberis vulgaris Coptis chinensis Hydrastis canadensis Tt has been traditionally used in Chinese and Ayurvedic medicine for infections, metabolic disorders, and liver diseases. Although not officially approved for treating cirrhosis, it is being studied for its hepatoprotective and anti-fibrotic properties.[7]



Berberine

Chemical Nature :-

- Class: Isoquinoline alkaloid Subtype: Protoberberine alkaloid Molecular formula: $C_{20}H_{18}NO_4^+$ Structure: Quaternary ammonium salt Color: Yellow crystalline powder Taste: Bitter

Pathophysiology of Liver Cirrhosis :-

Cirrhosis is the **end-stage chronic liver disease**, characterized by: Chronic inflammation Activation of hepatic stellate cell Excess collagen deposition Fibrosis and regenerative nodules Portal hypertension Liver dysfunction Major causes include alcohol, viral hepatitis (B, C), NAFLD/NASH, and autoimmune disorders, Inhibits hepatic stellate cell activation Downregulates TGF-β signaling Reduces collagen types I and III Decreases extracellular matrix deposition.[8]

2. PIPERINE:

Piperine is a natural alkaloid compound responsible for the pungency of black pepper. It is obtained mainly from: Piper nigrum Piper longum Traditionally used in Ayurveda and herbal medicine, piperine has anti-inflammatory, antioxidant, and bioavailability-enhancing properties.[9]

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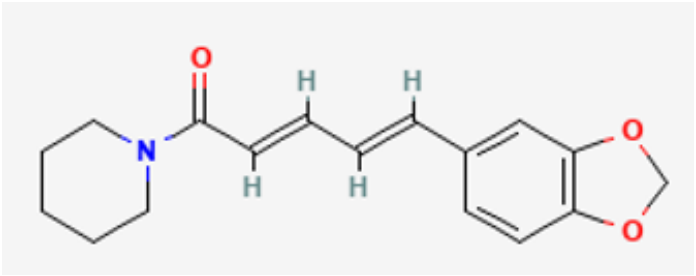
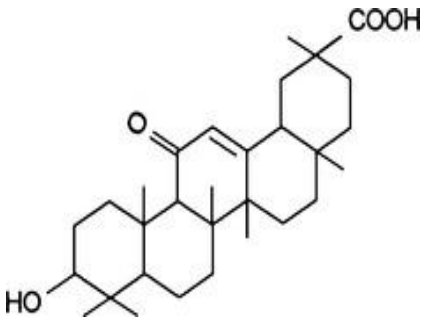


Chemical Nature Class: Alkaloid Chemical type: Piperidine alkaloid Molecular formula: $C_{17}H_{19}NO_3$ Structure: Contains a piperidine ring linked to a methylenedioxy phenyl group Appearance: Yellow crystalline solid Taste: Pungent.[8]

Pathophysiology of Liver Cirrhosis : Cirrhosis involves: Chronic liver injury Hepatic stellate cell activation Collagen deposition Fibrosis and nodular regeneration Portal hypertension Impaired liver function Major causes: Alcoholic liver disease Viral hepatitis (B, C) NAFLD/NASH Autoimmune diseases. Good oral absorption Extensively metabolized in liver Inhibits drug-metabolizing enzymes Excreted via bile and urine In cirrhosis: Reduced liver metabolism Risk of increased drug levels Potential accumulation.[9]

3. GLYCYRRHIZA GLABRA (LICORICE):

Licoriceis a medicinal plant whose root has been used in traditional systems such as Ayurveda and Chinese medicine for liver disorders, cough, and gastric problems. The main active compound responsible for liver-related effects is: Licorice extracts (especially glycyrrhizin-containing preparations) are used in some countries for chronic liver diseases, particularly viral hepatitis.[10]

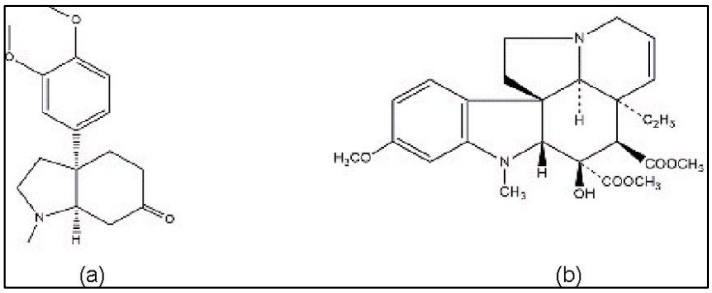


Chemical Constituents Main active components: **Glycyrrhizin** Triterpenoid saponin Sweet compound (50× sweeter than sugar) Hydrolyzed to glycyrrhetinic acid in the intestine **Flavonoids** Liquiritin Isoliquiritigenin Glabridin.[10]

Pathophysiology of Liver Cirrhosis Cirrhosis is characterized by: Chronic inflammation Hepatic stellate cell activation Excess collagen deposition Fibrosis and nodular regeneration Portal hypertension Liver dysfunction Common causes: Alcohol Viral hepatitis (B, C) NAFLD/NASH Autoimmune disease Use in Cirrhosis – Clinical Considerations May be beneficial in: Early fibrosis Viral hepatitis-related liver disease Compensated cirrhosis Should be avoided or used cautiously in: Decompensated cirrhosis Severe ascites Uncontrolled hypertension Low potassium levels.[11]

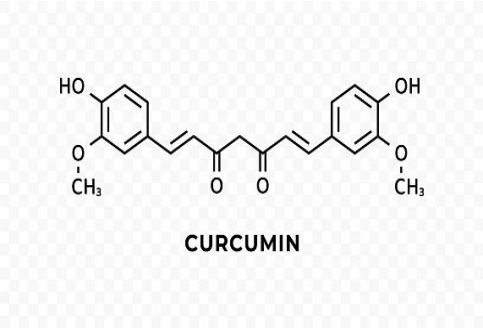
4. CATHARANTHUS ROSEUS (VINCA):

“Vinka” usually refers to the plant Catharanthus roseus (Madagascar periwinkle), which contains important anticancer drugs known as vinca alkaloids. Major active alkaloids include: Vincristine Vinblastin.[12]



Chemical Nature Class: Indole alkaloids Derived from: Catharanthus roseus Structure: Complex dimeric indole–indoline alkaloids Highly cytotoxic (inhibit cell division) Vinca alkaloids: Bind to tubulin Inhibit microtubule formation Arrest cell division at metaphase Cause apoptosis (cell death They are mainly used to treat cancers such as:Leukemia Lymphoma Breast cancer Testicular cancer. [13]

5. CURCUMA LONGA (TURMERIC):



Introduction Turmeric is a medicinal plant widely used in Ayurveda and traditional medicine for inflammatory and liver disorders. The main active compound responsible for its pharmacological effects is: Curcumin Turmeric and curcumin are studied for their anti-inflammatory, antioxidant, and anti-fibrotic properties, which may help slow liver damage. However, they are not approved treatments for liver cirrhosis.[14]

Chemical Nature Curcumin Class: Polyphenol (diarylheptanoid) Chemical formula: $C_{21}H_{20}O_6$ Color: Bright yellow pigment Solubility: Poorly water-soluble Bioavailability: Low (rapid metabolism and elimination) Other curcuminoids: Demethoxycurcumin Bisdemethoxycurcumin Because of low bioavailability, curcumin is often combined with bioenhancers like piperine.

Pathophysiology of Liver Cirrhosis (Brief) Cirrhosis is characterized by: Chronic inflammation Oxidative stress Hepatic stellate cell activation Excess collagen deposition Fibrosis and regenerative nodules Portal hypertension[14].

SUMMARY

Liver cirrhosis is a chronic, progressive liver disease characterized by continuous inflammation, hepatocyte damage, fibrosis, and distortion of normal liver architecture. Over time, these pathological changes impair liver function and may lead to liver failure, portal hypertension, and increased mortality. Cirrhosis commonly arises from long-standing liver injury caused by chronic viral infections such as Hepatitis B and Hepatitis C, excessive alcohol consumption, non-alcoholic fatty liver disease (NAFLD), autoimmune liver disorders, and prolonged exposure to hepatotoxic substances. Current treatment strategies mainly focus on managing symptoms and slowing disease progression, with limited capacity to reverse established fibrosis. This limitation has driven interest in identifying novel hepatoprotective agents from natural sources.

Alkaloids are naturally occurring nitrogen-containing compounds predominantly found in plants, though they may also be derived from microorganisms and marine organisms. They exhibit a wide range of pharmacological activities, including antioxidant, anti-inflammatory, antiviral, antifibrotic, and immunomodulatory effects. These properties make alkaloids promising candidates for the treatment and prevention of liver cirrhosis.

The hepatoprotective mechanisms of alkaloids are multifaceted. They reduce oxidative stress by scavenging reactive oxygen species and enhancing endogenous antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase. Alkaloids also

suppress inflammatory responses by downregulating pro-inflammatory cytokines like TNF-α and IL-6 and inhibiting signaling pathways such as NF-κB. In addition, several alkaloids inhibit hepatic stellate cell activation, a key process in liver fibrosis, by modulating fibrogenic mediators including transforming growth factor-β (TGF-β). Some alkaloids further demonstrate antiviral activity against hepatitis viruses, addressing a primary cause of cirrhosis.

CONCLUSION

Liver cirrhosis remains a major global health burden characterized by progressive fibrosis, chronic inflammation, oxidative stress, and irreversible architectural distortion of the liver. Despite advances in modern medicine, current therapeutic options are limited and primarily focus on managing complications rather than reversing the underlying pathology. In this context, natural compounds—particularly alkaloids—have emerged as promising candidates for hepatoprotection and antifibrotic therapy. This review highlights the significant potential of alkaloids derived from medicinal plants in the treatment and prevention of liver cirrhosis. Numerous experimental studies have demonstrated that alkaloids exert hepatoprotective effects through multiple mechanisms, including antioxidant activity, inhibition of inflammatory cytokines, suppression of hepatic stellate cell activation, modulation of fibrogenic signaling pathways, and prevention of apoptosis and necrosis of hepatocytes. Compounds such as berberine, matrine, piperine, and other plant-derived alkaloids have shown encouraging results in both in vitro and in vivo models of liver injury. The multitarget nature of alkaloids makes them particularly attractive as therapeutic agents for complex diseases like cirrhosis, which involves interconnected molecular pathways. Furthermore, their natural origin and long history of use in traditional medicine provide a strong foundation for further pharmacological exploration. However, despite promising preclinical evidence, there remains a significant gap in large-scale clinical trials to validate their safety, efficacy, optimal dosage, and long-term effects in human subjects. Standardization of plant extracts, identification of active constituents, pharmacokinetic profiling, and toxicity assessments are essential steps before clinical translation. In conclusion, alkaloids represent a promising and valuable class of bioactive compounds with significant hepatoprotective and antifibrotic potential. Future research focusing on mechanistic studies, well-designed clinical trials, and formulation development may pave the way for integrating alkaloid-based therapies into mainstream management strategies for liver cirrhosis, ultimately improving patient outcomes and reducing the global burden of chronic liver disease.

REFERENCE

1. French SW. Ethanol and hepatocellular injury. Clin Lab Med Stockert RJ. The asialoglycoprotein receptor: relationships between structure, function, and expression.
2. Savill JS, Wyllie AH, Henson JE, Walport MJ, Henson PM, Haslett C. Macrophage phagocytosis of aging neutrophils in inflammation. Programmed cell death in a neutrophil leads to its recognition by macrophages.
3. L.B. Seeff, K.L. Lindsay, B.R. Bacon, T.F. Kresina, J.H. Hoofnagle, Hepatology 34 (2001) 595–603.
4. Chalasani NP, Maddur H, Russo MW, Wong RJ, Reddy KR; Practice Parameters Committee of the American College of Gastroenterology. ACG Clinical Guideline: Diagnosis and Management of Idiosyncratic Drug-Induced Liver Injury. *Am J Gastroenterol* 2021; 116: 878-898.
5. Tillmann HL, Rockey DC. Signatures in drug-induced liver injury. *Curr Opin Gastroenterol* 2020; 36: 199-205 [*RCA*] [PMID: 32205565.
6. Chow HC, So TH, Choi HCW, Lam KO. Literature Review of Traditional Chinese Medicine Herbs-Induced Liver Injury From an Oncological Perspective With RUCAM. *Integr Cancer Ther* 2019; 18: 1534735419869479 [*RCA*] [PMID: 31405304.
7. Zheng Y., Wang J., Zhao T., Duan X., Wang L., Zheng Y., Yang S. (2025). *Role of alkaloid compounds in regulating chronic liver diseases.* Journal of Chronic Hepatic Disorders. Alkaloids shown to regulate liver fibrosis, inflammation, apoptosis, and metabolic homeostasis in chronic liver disease models.
8. Zhang M., Guo C., Li Z., Cai X., Wen X., Lv F., Lin C., Ji L. (2024). *Mulberry twig alkaloids improve progression of metabolic-associated fatty liver disease by modulating PGC1α/PPARα and KEAP1/NRF2 pathways in mice. Pharmaceuticals*, 17(10), 1287. Demonstrated hepatoprotective effects of plant alkaloids against liver steatosis and fibrosis in an obesity model.
9. Wang Q., Zhang M., Meng M. et al. (2025). *Integration of bile acid metabolomics and gut microbiome to study anti-liver fibrosis effects of total alkaloids of Corydalis saxicola Bunting. Chinese Medicine*, 20, 106. Highlights anti-fibrotic actions of total alkaloids via metabolomics profiling and microbiome modulation.
10. Wang Qiu, Jin Y., Fan F., Feng X., Yin X., Wang X. (2025). *Alkaloids in Tibetan Medicine Corydalis conspersa Maxim. and their hepatoprotective effect against acute liver injury. Molecules*, 30(10), 2127. Reports isolation of multiple alkaloids with protective effects against CCl_4-induced liver damage.
11. Sun Yanhong, Tan H., Wang F., Hu J., Duan X., Bai W., Wu J., Bai J., Hu J. (2025). *Inhibitory effects of alkaloids on OATP1B1 and hepatoprotective potential based on structure-activity relationships. Chemical Research in Toxicology*, 38(2), 281–295. Discusses alkaloids' interactions with hepatic transporters and protective outcomes in liver injury models.
12. Li X-L., Sun Y-P., Wang M., Wang Z-B., Kuang H-X. (2024). *Alkaloids in Chelidonium majus L.: phytochemistry, pharmacology, and toxicology. Frontiers in Pharmacology*, 15, 1440979. Comprehensive review of alkaloids in a medicinal herb with implications for liver-related effects.

13. Sun Y., Tan H., Wang F., Hu J., Duan X., Bai W., Wu J., Bai J., Hu J. (2025). Inhibitory effects of alkaloids on liver transporter OATP1B1 and hepatoprotective effects based on structure-activity relationships. *Chemical Research in Toxicology*, 38(2), 281–295. Discusses how multiple alkaloids reduce transporter-mediated liver toxicity and protect against hepatotoxic injury.
14. Tilg H., & Moschen AR. (2010). Evolution of inflammation in nonalcoholic fatty liver disease: The multiple parallel hits hypothesis. *Hepatology*, 52(5), 1836–1846.

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