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A Review of Medicinal Plant with Hepatoprotective Effect

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ABSTRACT

Liver diseases have become a major global health challenge and may be triggered by several toxic chemicals, which include chemotherapeutic agents, thioacetamide, carbon tetrachloride, certain antibiotics, excessive alcohol consumption, and pathogenic microbes. Hence, safeguarding a healthy liver is vital for good health and well-being. Despite advances in pharmacology, the demerits associated with synthetic drugs have outshone the merits. Treatment of liver diseases based on modern medical principles is becoming ineffective and also associated with adverse effects of long-term use, in addition to prohibitive costs in developing countries. Thus, exploring medicinal plants which are easily available and cheap and do not involve strenuous pharmaceutical production processes appears to have gained worldwide attention as alternative therapeutic agents for the diseases. Phytochemical compounds with hepatoprotective effects were also discussed, and finally, the future work in the field was also highlighted.

Keywords: hepatotoxicity, liver diseases, medicinal plant, pharmacology

INTRODUCTION

Human liver is the most essential visceral organ in body concerned with synthesis, excretion, metabolism and detoxification of diverse exogenous and endogenous substances such as drugs. Because of these multi-dimensional functions, it is prone to many diseases. Death of hepatocytes is the main feature of liver diseases and liver disease is a worldwide major health issue. Causative agents include diseases that interfere with liver functions; chemicals (ethanol, CCl_4, thioacetamide, D-galactosamine; environmental toxins) and drugs such as paracetamol.

The amount of liver damage brought on by hepatotoxin-like substance intake or inhalation is rising globally, and the standard medications used to treat this condition are typically inadequate and have major side effects. According to the United States Acute Liver Failure Study Group, almost 50% of acute liver damage is caused by drugs. Around 13% of hospitalisations are due to drug-induced liver damage from paracetamol overdose, or PILD.

Only 0.6% of the 0.08% of UK residents who are hospitalised each year for paracetamol intoxication experience acute liver injury (Jones, 2002). Although less frequent than in the UK, paracetamol overdose is a serious clinical issue in the USA, Denmark, and Australia. It is also the leading cause of acute liver injury in the USA (Ostapowicz et al., 2002). About 0.01% of Americans and 0.02% of Australians are hospitalised with paracetamol poisoning each year (Jones, 2002). Thus, the second most frequent reason for liver transplants globally is paracetamol poisoning. A number of previously approved medications were taken off the market for the common cause of liver injury.

Since carbon tetrachloride (CCl4) is a hepatotoxin, it is frequently utilised to cause liver injury in animal models for research This drug has been effectively used on mice , birds , and rats One of the primary processes behind CCl4 hepatotoxicity is the induction of oxidative stress Supplementing with antioxidants may therefore be beneficial in treating its hepatotoxic effects.

DILI occurs due to the formation of **toxic metabolites** during hepatic biotransformation, often mediated by cytochrome P450 enzymes. These reactive intermediates can bind to cellular proteins, induce **oxidative stress**, trigger **immune-mediated reactions**, and lead to **mitochondrial dysfunction**, all contributing to hepatocellular injury.

Hepatotoxicity refers to the damage or injury caused to the liver by harmful substances, such as drugs, chemicals, or toxins. It can lead to liver dysfunction, inflammation, liver failure. **Hepatoprotective** refers to substances or agents that help protect the liver

from damage or injury.

These agents can prevent liver cell damage, promote healing, or reduce inflammation in the liver. Some diseases associated with hepatotoxicity are: Alcoholic Liver Disease (ALD), Non- Alcoholic Fatty Liver Disease (NAFLD), Drug-Induced Liver Injury (DILI), Viral Hepatitis, Autoimmune Hepatitis, Hemochromatosis, Wilson's Disease etc.

Metabolic dysfunction and histological abnormalities such neoplasms, vascular lesions, granulomas, steatosis, cholestasis, hepatitis, and zonal necrosis are characteristics of hepatotoxicity, or liver damage, which accounts for 5% of all injuries and is hence common. As a result, over a million deaths are reported annually, with hepatocellular cancer or liver deformity being the indirect cause. We are protected from a variety of internal and external illnesses by conventional or synthetic medications. Unfortunately, the medications used to treat liver problems are inadequate since they might have major long-term negative effects and do not completely protect the organ.

Understanding the function of complementary and alternative medicine (CAM) as a treatment option for liver disease is therefore essential. Because of its abundance of polyphenols, CAM is known to have good therapeutic efficacy with few negative side effects . Therefore, foods high in plant polyphenols may help reduce the risk of chronic illnesses.

Hepatotoxicity refers to liver damage caused by chemical substances, including drugs and toxins. The clinical manifestations of hepatotoxicity commonly include:

Fever: which may result from the inflammatory response associated with liver injury.

Abdominal pain: often localized in the upper right quadrant due to liver inflammation or swelling.

Severe Itching: Severe itching (pruritus), which can result from the accumulation of bile acids in the skin due to impaired liver function.

Jaundice: **Jaundice**, characterized by yellowing of the skin and eyes, occurs due to the buildup of bilirubin, a byproduct of red blood cell breakdown that the liver normally processes.

Nausea: Nausea, a nonspecific symptom often associated with liver dysfunction.

Although the liver attempts to protect itself by increasing the activity of intracellular antioxidant enzymes, the response is often insufficient to fully neutralize the oxidative damage. Meanwhile, some alkylation reactions may follow a non-toxic pathway without causing major cellular dysfunction, but they still contribute to overall stress on the liver. Importantly, the image also highlights the **hepto-curative pathway**, where IGFRF seed extract plays a therapeutic role.

It helps in mitigating the effects of oxidative stress by reducing lipid peroxidation and preventing mitochondrial and membrane dysfunction. As a result, IGFRF seed extract offers protective effects, reducing liver inflammation and halting the progression of fibrosis and cirrhosis, thereby supporting liver healing and function.

Herbs Reported for Hepatoprotective Activity:

Name of the Plant	Plant parts used	Extract used	Dose (mg/kg)	Hepatotoxicity inducing agents	Biochemical and histopathological parameter studied	Ref
Phyllants Muellari anus	Leaves	Aqueos	400 mg/kg (p.o.)	Acetaminophen 300mg/kg b.w. i.p.	ALP, ALT, AST, ALB,TB,CAT,SO D, GSH-Px, GSH	7
Canna indica	Aerial parts	Methanol	100 and 200 mg/kg (p.o.)	CCl4 1ml/kg b.w. i.p.	SGPT, SGOT, TB, CAT, GSH, LPO	8
Ficus cordata	Roots	Methanol/ ethyl acetate	400 mg/kg (p.o.)	CCl4 1ml/kg b.w. i.p.	LDH	9
	Leaves	Methanol	500	Alloxan 200 ml/kg b.w.	AST, LDLC, ALT, HDL STG,	10

Dodonae a viscosa			mg/kg (p.o.)		and TC	
Eclipta prostrata	Fresh leaves	Methanol	10, 80 mg/kg (p.o.)	CCl4	ALT, AST, and serum bilirubin	11
Alocasia Macrorrh izos	Leaves	Ethanol and aqueous	200 mg/kg (p.o.)	CCl4	Serum ALT and AST	13
Leptadea Pyrotech nica	Whole plant	Methanol petroleum ether, chloroform, acetone, and aqueous	150 ml/kg (p.o.)	PCM	SGPT, TB, ALP, and SGOT	14
Tylophora	Leaves	Methanol	200 and 300 mg/kg (p.o.)	CCl4	SGPT, ALP, SGOT, and bilirubin content	15
Clausena lansium	Stem bark	Methanol	100 and 200 mg/kg (p.o.)	CCl4	Reduction in phenobarbitone, sleeping time and serum liver protein, serum AST, ALT, and ATP.	16
Cucurba maxima	Aerial parts	Methanol	250 and 500 mg/kg (p.o.)	CCl4	SGPT, SGOT, ALP, TP, and TB	17
Muntina calabura	Fruits	Methanol	50, 250, and 500 mg/kg (p.o.)	Acetaminophen	AST, ALT, and ALP	18
Aquilaria Malaccen sis	Leaves	Ethanol	400 mg/kg (p.o.)	PCM	AST, ALT, LDH, ALP, bilirubin, CHL, TP, and ALB	19
Calendul a officinali s	Whole plant	Methanol	500 mg/kg (p.o.)	Acetaminophen/CCl4	ALT, AST, and LDH	20
Taraxacu m officinale	Root	Hydroalcoh olic acid	250 mg/kg (p.o.)	Ethanol	TBARS, GST, GSH, SOD, CAT, GR, and GPx	21
Baliosper mum Montanu m	Root	Methanol	2000 mg/kg (p.o.)	TAA	GOT, GPT, ALP, TB, TC, TB, and albumin	22
Glycosmi s pentaphyl la Corr.	Leaves,bark	Methanol	500 mg/kg (p.o.)	CCl4	ALT/SGPT, AST/SGOT, CHL, bilirubin, and glucose	23
Androgphis lineata Nees	Leaves	Methanol	845 mg/kg (p.o.)	CCl4	SGOT, SGPT, and ALP	24
Wedelia chinensis L.	Leav es	Ethanol	200 mg/kg (p.o.)	CCl4	AST, ALT, ALP, protein, and bilirubin	25
Cassia fistula	Seeds	Methanol	200 and 400 mg/kg (p.o.)	PCM	SGOT, SGPT, ALP, and bilirubin	26
Ziziphus Mucronat a	Leaves	Methanol	200 mg/kg (p.o.)	Dimethoate	SGOT, TBARS, SGPT, GSH,	29

					SOD, tocopherol, HDL, LDL, CHOL, TL, TGA	
Salvia Miltiorrh iza	Dried pulve rized roots	Ethanol	50 mg/kg (p.o.)	CCl4	Induce apoptosis of hepatic stellate cells (HSCs)	30
Dendrop hthoe falcata	Leaves	Ethanol	100 mg/kg (p.o.)	CCl4	AST, TP ALP, and ALT, TB	31
Indigofer a tinctoria	Leaves	Methanol	75, 150, 300 mg/kg (p.o.)	PCM	TBARS, SOD, CAT, and GSH	32
Cestrum Nocturnu m	Leaves	Aqueous and ethanol	250 and 500 mg/kg (p.o.)	PCM	SGOT, SGPT, ALP, AST, ALT, and LDH	33

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