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Evaluation Study on Effects of Plant Extract on Liver Toxicity

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Abstract: *Liver is the principal site for clearance and metabolism of xenobiotics which places it in a position to be exposed to potentially toxic, metabolic, microbial, circulatory and neoplastic insults. Dominant primary diseases of liver are alcoholic liver disease, viral hepatitis, and hepatocellular carcinoma. Secondary liver disease may occur due to cardiac decompensation, disseminated cancer, and extra hepatic infections. Herbal preparations or plant derived compounds are used in conjunction with allopathic medicine to treat all types of ailments including liver disease*

Bambusa arundinacea is used in folk medicine as tonic for heart, liver and brain. Bambusa arundinacea contains phytochemicals like Flavonoids, Phenolic compounds, resin, lignin and many others. Flavonoids and Phenolic compounds have shown to possess significant antioxidant and hepatoprotective activity.

So the present study was done to study the protective effect of Bambusa arundinacea on liver toxicity and associated pathology complications.

Methanol Extract of Bambusa arundinacea young shoots was obtained by using soxhelet apparatus and was evaluated for preliminary phytochemical composition which showed the presence of Terpenes, Flavonoids, Saponins, Steroids, Proteins, Carbohydrates, Tannins and Phenolic compound. It was further evaluated for Total Phenolic and Flavonoids content which showed that Methanol Extract had Total Phenolic content 25.5±2.2 mg gallic acid equivalents/g DW, and the Total Flavonoid content was 36 ± 3.7 mg Quercetin equivalents/g DW.

In preliminary study antioxidant activity of Methanol Extract was evaluated by DPPH(2,2-diphenyl-1-picrylhydrazyl), Ferric reducing power assay and Lipid peroxidation. In vitro hepatoprotective activity of Methanol Extracts (25, 50, 100, 200 and 400 µg/ml) against Thioacetamide (100mM) induced cytotoxicity was assessed by monitoring cell viability in HepG2 and Hep3b tumor cell line and also in primary hepatocytes. In vivo hepatoprotective activity of Methanol Extract (50, 100, 200 mg/kg p.o.) was observed against Thioacetamide (100 mg/kg s.c.) induced liver injury in rat by measuring biochemical parameters viz, Aspartate Transaminase, Alanine Transaminase, Alkaline Phosphatase, Total and Direct Bilirubin. The histopathological studies were also performed. Methanol Extract of young shoots showed good antioxidant activity. Thioacetamide caused significant reduction in cell viability in invitro studies. Treatment with Methanol Extract caused significant increase in cell viability. In vivo studies showed that Thioacetamide induced elevation of biochemical parameters in rats were significantly ($p<0.0001$) decreased with Methanol Extract treatment in rats. Histopathology studies also supported the protective effect of Methanol Extract. This concluded that Methanol Extract of young shoots of Bambusa arundinacea has hepatoprotective activity which may be due to its antioxidant activity Methanol Extract (12 gm) was column chromatographed using silica gel (100-200 mesh) as the stationary phase and eluted with solvents of increasing polarity to obtain nhexane, chloroform, Ethylacetate and n-butanol Fractions. These Fractions were evaluated for their antioxidant activity by using DPPH, Lipid peroxidation and Ferric reducing power assay. The Total Phenolic (TPC) and Total Flavonoid contents (TFC) of all the Fractions were also determined. These Fractions (25, 50, 100, 200 µg/ml) were also evaluated for In vitro hepatoprotective activity against

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Thioacetamide (100mM) induced cytotoxicity by monitoring cell viability in HepG2 and Hep3b tumor cell line and also in primary hepatocytes.

The results of the present analysis demonstrated that different Fractions of Methanol Extract of young shoots of Bambusa arundinacea contained appreciable levels of Total Phenolic and Flavonoid contents and also possess good antioxidant activity and in vitro hepatoprotective activity. Of all the Fractions tested, Ethylacetate Fraction was found to be more effective with Total Phenolic content (30.8±1.5 mg gallic acid equivalent /g DW) and Total Flavonoid content (47±2.6 mg Quercetin equivalent/g DW).

Based on these results Ethylacetate Fraction (25, 50, 100 mg/kg p.o.) was further evaluated for in vivo hepatoprotective activity against Thioacetamide (100 mg/kg s.c.) induced liver injury in rat by measuring biochemical parameters viz, Aspartate Transaminase, Alanine Transaminase, Alkaline Phosphatase, gamma glutamyl transferase, Total and Direct Bilirubin. Super oxide dismutase and Catalase were estimated for in vivo antioxidant activity. The histopathological studies were also performed. Ethylacetate Fraction of Methanol Extract of young shoots showed good invivo antioxidant activity. Thioacetamide caused significant reduction in cell viability in in vitro studies. Treatment with Ethylacetate Fraction caused significant increase in cell viability. In vivo studies showed that Thioacetamide induced elevation of biochemical parameters in rats were significantly ($p<0.0001$) decreased with Ethylacetate Fraction treatment in rats. In vivo antioxidant parameters were also significantly improved with treatment with Ethylacetate Fraction. Histopathology studies also supported the protective effect of Ethylacetate Fraction. This concluded that Ethylacetate Fraction of Methanol Extract of Bambusa arundinacea have significant hepatoprotective activity.

Ethylacetate Fraction was Subfractioned by column chromatography to obtain 5 Subfractions by changing different ratio of chloroform, Ethylacetate and n-butanol They were labelled as S.F.1, S.F.2, S.F.3, S.F.4 and S.F.5.

These Subfractions of Ethylacetate were further evaluated for In vitro hepatoprotective activity against Thioacetamide (100mM) induced cytotoxicity by monitoring cell viability in HepG2 and Hep3b cell line and also in primary hepatocytes. Total Phenolic and Flavonoid content were also assessed in the Subfractions. The results of the present analysis demonstrated that

S.F.1 and S.F.2 Subfractions of Ethylacetate Fraction of Methanol Extract of young shoots of Bambusa arundinacea contained appreciable levels of total Phenolic and Flavonoid content and also possess good in vitro hepatoprotective activity. Of all the Subfractions tested, S.F.1 was found to be more effective with Total Phenolic content (14.5±1.45 mg Gallic acid equivalent /g DW) and Total Flavonoid content (88±1.6 mg Quercetin equivalent/g DW)

Based on these results Subfraction 1 (S.F.1(12.5, 25mg/kg p.o.)) was further evaluated for in vivo hepatoprotective activity against Thioacetamide (100 mg/kg s.c.) induced liver injury in rat by measuring biochemical parameters viz, Aspartate Transaminase, Alanine Transaminase, Alkaline Phosphatase, gamma glutamyl transferase Total and Direct Bilirubin. Super oxide dismutase and Catalase were estimated for in vivo antioxidant activity. The histopathological studies were also performed. In vivo studies showed that Thioacetamide induced elevation of biochemical parameters in rats were significantly ($p<0.0001$) decreased with S.F.1 treatment in rats. In vivo antioxidant parameters were also significantly improved. Histopathology studies also supported the protective effect. This concluded that Subfraction 1 of Ethylacetate Fraction of Methanol Extract of Bambusa arundinacea possess significant hepatoprotective activity.

Methanol Extract(200 mg/kg p.o daily), Ethyacetate Fraction (100 mg/kg p.o daily) and S.F.1(25 mg/kg p.o daily) and Silymarin (25mg/kg i.p.) were further evaluated to study their protective action on chronic liver toxicity and associated pathological complications induced by Thioacetamide

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(200mg/kg i.p./3days/week) for 8 weeks. Sensory and motor behavioural tests and avoidance test were used to evaluate hepatic encephalopathy. Serum transaminase (SGOT, SGPT), Alkaline phosphatase, Total Bilirubin , Direct bilirubin, GGT, Lactate dehydrogenase(LDH), Triglycerides (TG), Cholesterol, T.

Protein, Morphological test: Weight of liver and body weight, Collagen, SOD, Catalase, Nitric oxide(NO), Glutathione and Histological studies were done to evaluate hepatic function. Serum Urea, Creatinine, SOD, Catalase, Glutathione and Histological studies were done to evaluate renal function. Methanol Extract of young shoots of Bambusa arundinacea, its Ethylacetate Fraction and S.F.1 showed protective action in chronic liver toxicity and associated pathology complications induced by Thioacetamide by improving hepatic functions, hepatic encephalopathy and renal functions.

So we concluded that young shoots of Bambusa arundinacea has significant antioxidant and invivo, in vitro hepatoprotective activity which may be due to presence of phenolic and flavonoid contents..

Keywords: *Thioacetamide.*

I. INTRODUCTION

Worldwide liver disease is an important cause of mortality and morbidity[1, 2]. Liver plays an important role in clearance and metabolism of xenobiotics due to which it is continuosly exposed to potentially toxic, metabolic, microbial, circulatory and neoplastic insults. Most prominent primary diseases of liver are alcoholic liver disease, viral hepatitis, and hepatocellular carcinoma. Secondary liver disease may occur due to cardiac decompensation, disseminated cancer, and extra hepatic infections.[3,4]

Liver cirrhosis is a critical stage of chronic liver diseases that can produce liver failure, portal hypertension and hepatocarcinoma. Liver cirrhosis is related to high morbidity/mortality rate and it may occur due to viral infection, tissue-immune mediated damage, toxic agents, obstructive jaundice, gene abnormalities, or alcohol and nonalcohol steatohepatitis. Current treatments of liver cirrhosis are limited to the elimination of the underlying injurious stimulus, e.g. viruses in cases of viral hepatitis.[5-6]

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome occurring as a consequence of acute or chronic liver failure. Hepatic encephalopathy affects a large number of patients worldwide with a mortality index ranging from 50 to 90%. Hypothesis suggests a state of hyperammonemia which is responsible for both direct and indirect alterations in cerebral metabolism. Hyperammonemia leads to increased glutamine levels in astrocytes from amidation of glutamate by glutamine synthetase. The accumulation of glutamine in the astrocytes produces osmotic stress and causes the astrocytes to swell. This causes cerebral edema and intracranial hypertension which are described as the main pathophysiological features of HE.[7-18]

Renal function deteriorates in patients with cirrhosis as liver function worsens, which indicates a link between these two organ systems. Apoptotic cell death and increase in adenosine production (potentiating the vascular effects of angiotensin-II) are thought to play an important role in the development of kidney damage during cirrhosis. Generation of free radicals and oxidative stress during cirrhosis may also result in oxidative damage in the kidney. Oxidative stress gains importance in the context of renal failure in cirrhosis, because free radicals such as superoxide can interact with nitric oxide to generate peroxynitrite, which is an oxidant and a nitrosating agent.[19-22] Free radicals, reactive oxygen species and reactive nitrogen species are highly reactive and are linked with the pathophysiology of majority of diseases like aging, carcinogenesis, atherosclerosis, liver cirrhosis, diabetes, and cardiovascular disorders. They damages the macromolecules of the body and produce cellular damage. [23-24] Antioxidants are radical scavengers which protect human body against free radicals by inhibiting the oxidizing chain reactions.[25] An imbalance between Reactive oxygen species and the inherent antioxidant capacity of the body, directed the use of antioxidants from dietary and or medicinal supplements particularly during the disease attack.[26]

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A large number of world's population depends on herbal medicine for the primary care. Herbal preparations or plant derived compounds are used in conjunction with allopathic medicine to treat various types of ailments including liver disease [27, 28]. The fruits and seeds of Milk thistle have shown protective activity in liver toxicity in preclinical and clinical studies [29]. Bamboos consist of about 110 genera and 11301140 species. Bambusa arundinacea is a native Indian plant and also cultivated in other countries like Srilanka, Myanmar etc. [30] Various parts of Bambusa arundinacea are used in folk medicine as tonic for heart, liver and brain. It fortifies the heart and calms down heart palpitations. It soothes many types of stomach troubles and its use is also recommended in cases of diarrhoea and chronic liver ailments. It is also used in eye inflammations. It is used as rejuvenator, hemostatic, antispasmodic and suppresses cough and colds, bronchitis, vomiting, convulsions, fever, enuresis, inflammation and gallbladder problems.[31,32] It is reported to cure many types of urinary problem such as urodialysis (suppressed urine), uropenia (scanty urine) and urinary tract infection.[33,34] It is also a part of many ayurvedic and unani formulation like Qursghafis, Sufuftabashir, Drakshadichurna which are used in liver ailments.[35,36] Bambusa arundinacea contains various phytochemicals like resin, lignin, alkaloids, glucoside, silica, uronic acid, galactose, glucose, arabinose, mannose, xylose, Choline, betain. It is also reported to contain urease, nuclease, proteolytic enzymes, diastatic and emulsyfying enzyme.[37,38] Various flavanoids like orientin, homoorientin, vitexin and isovitexin are also present. Also Phytosterols like stigmasterol and β-sitosterol, Stigmast-5, 22-dien-3β-ol, Stigmast-5-en- 3β-ol-β-D glucopyranoside, triterpenes and steroidal glycosides, 17, 20, 20-tri demethyl-20α-isopranyloleanane, eicosanyldicarboxylic acid, α-amyrin acetate, urs-12-en-3β-ol-β-D-glucopyranoside are present.[39-41]

Bambusa arundinacea is reported to possess antifertility, anti-inflammatory, antiulcer, antihyperglycemic, antiarthritic, anthelmintic and antihyperlipidemic activity.[42-49] It has shown good antioxidant and antimicrobial potential in in vitro studies.[50]

In light of the aforementioned properties, we hypothesized that Bambusa arundinacea may overcome the hepatotoxicity mediated through oxidative stress and also have protective effect on associated pathological complications. Hence, we evaluated the protective effect of Bambusa arundinacea in Thioacetamide induced liver injury and associated pathological complications.

Objective:

• To identify Phytochemical composition and anti-oxidant properties of Bambusa arundinacea Methanol extract, its fractions and subfractions.

• To study the protective effects of Methanol extract, its fractions and subfraction in invitro (In HepG2, Hep3b cell lines and primary hepatocytes) and invivo liver toxicity induced by Thioacetamide.

• To assess the protective effect of Bambusa arundinacea on other pathological conditions viz hepatic encephalopathy and hepatorenal syndrome manifested by Chronic liver toxicity induced by Thioacetamide.

II. REVIEW OF THE LITERATURE

2.1. LIVER:

2.1.1. Anatomy of liver:[51]

Liver appears to be simple in function but it proves to be one of the most complicated organ. The right upper quadrant of the abdomen is dominated by the liver and its companion biliary tract and gallbladder. The normal liver weighs 1400 to 1600 gm, representing 2.5% of body weight.

In liver all cells are alike and each can apparently do everything, being a jack of all trades. Liver can take in, build up, break down and cast off .It has a unique power to repair so that if a part of it is cut away, or destroyed it can be replaced with matching parenchymatous tissue, not with the base substitute of fibrous tissue.

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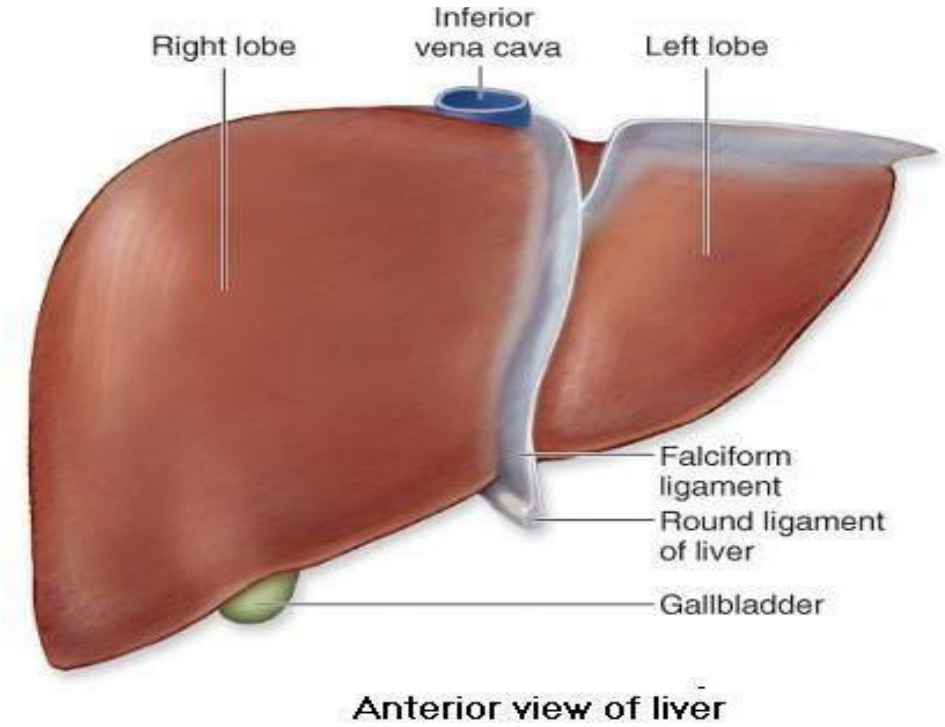

Figure: 1 Anatomy of liver

2.1.2 Microarchitecture of liver [51, 52]

Liver is classically been divided into 1-2mm diameter hexagonal lobules oriented around the terminal tributaries of the hepatic vein. The hepatocytes near the central vein are most remote from the blood supply, are at the periphery of metabolic lobules known as acini. The acini are roughly triangular; the acini have the terminal twigs of hepatic artery and portal vein extending out from the portal tracts at their base and terminal hepatic venoules at their apices. The parenchyma of the hepatic acinus is divided into three zones.

Zone 1 being closest to the vascular supply. Zone 2 being intermediate.

Zone 3 being abutting the hepatic venule.

This zonation is of considerable metabolic consequence since a lobular gradient of activity exists for many hepatic enzymes. The hepatic parenchyma is organized into ciribriform anastomosing sheets of plates of hepatocytes. Hepatocytes immediately abutting the portal tract are referred to the limiting plate, forming a discontinuous rim around the mesenchyme of the portal tract. There is a radial orientation of the hepatocytes cords around the terminal hepatic vein.

Between the cords of hepatocytes are vascular sinusoids. The sinusoids are lined by fenestrated and discontinuous endothelial cells, which demarcate an extra sinusoidal space of disse into which protrudes abundant microvilli of hepatocytes. Scattered Kuffer cells of the monocyte-phagocyte system are attached to the luminal face of endothelial cells .

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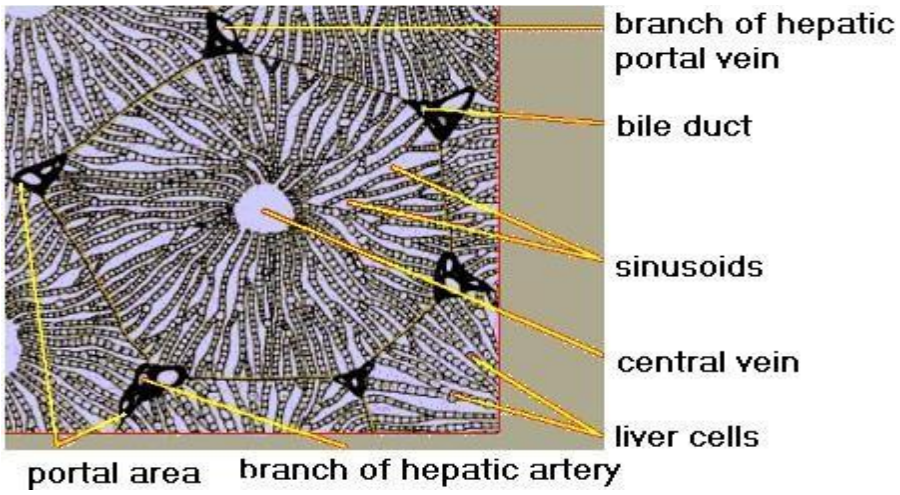

Figure:2 Microscopic structure of liver

2.1.3 Blood supply: [53]

Liver receives blood from two sources:

-Hepatic artery and

-Hepatic portal vein.

Hepatic artery obtains oxygenated blood and hepatic portal vein receives deoxygenated blood. Branches of both the hepatic artery and hepatic portal vein carries blood to liver sinusoids, where the oxygen, most of the nutrients and certain toxins are extracted by the hepatocytes, products manufactured by the hepatocytes and nutrients needed by other cells are secreted back into the blood. The blood then drains into the central vein and eventually passes into a hepatic vein, branches of the hepatic portal vein, hepatic artery and bile duct typically accompany each other in their distribution through the liver.

Collectively these three structures are called a portal triad.

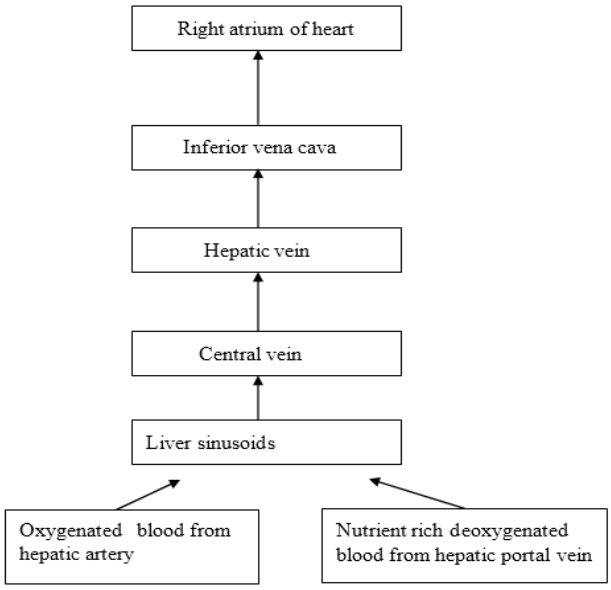

Figure: 3 Blood flow through the liver and return to the heart

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2.1.4. Functions of the liver [53-56]

Liver is the essential organ of the body. Its functions are numerous and briefly summarized as follows:

Blood and its circulation:

- R.B.C. formation-in foetal life.
- R.B.C. destruction in adult life.
- Storehouse of blood and regulates blood volume.
- Relation with blood clotting.

-Manufacture prothrombin and fibrinogen

-Mast cells from heparin and prevent intravascular clotting.

-Manufacture all plasma proteins.

- Stores iron and vitamin B12, helps in the formation of red cells and haemoglobin.

Manufacture bile:

Bile is secreted continuously from liver cells and stored in the gall bladder. Cholesterol is also excreted from the liver. Bile acid, cholic acids, deoxycholic acid and lithocholic acid have been considered to be the derivative of cholanic acid and formed in the liver.

Bile pigments biliverdin and bilirubin are the excretory products of the haemoglobin of broken down R.B.C. and are formed in the reticular endothelium system in various parts of the body.

Carbohydrate metabolism:

Liver maintains normal blood glucose levels, when blood glucose level is low; the liver breaks the glycogen to glucose and release glucose into the blood stream. Liver also converts amino acids and lactic acids to glucose and convert other sugars, such as fructose and galactose into glucose. When glucose level is high liver convert's glucose to glycogen for storage.

Lipid metabolism:

Liver stores some triglycerides, breaks down fatty acids into acetyl coenzyme A, through a process called beta oxidation, and converts excess acetyl coenzyme A into ketone bodies (ketogenesis). It synthesizes lipoproteins, which transports fatty acids, triglycerides, and cholesterol and use cholesterol to make bile salts.

Protein metabolism:

Liver synthesizes all plasma proteins also, liver can perform transamination, the transfer of an amino group from an amino acid to another substance to convert one amino acid to another. Liver also deaminates amino acids so that they can be used for the production of ATP or converted to carbohydrates or fats. It converts the ammonia to much less toxic urea for excretion in urine.

Hormone metabolism:

Liver reduces the circulating adrenal cortical and sex hormones by degradation and conjugation. Inactivation of insulin, glucagon, antidiuretic hormone (ADH) and anterior pituitary trophic hormones.

Vitamin manufacture and storage:

- Manufactures prothrombin
- Forms vitamin A from carotene and stores vitamin A and D.
- Liver converts the folate to tetrahydrofolate which is the storage form of folic acid.
- Liver is the principle storage organ for vitamin B12.

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Removal of drugs:
Liver can detoxify substance such as alcohol or excrete drugs such as penicillin, erythromycin, and sulphonamides into bile.
Excretion of bilirubin:
The bilirubin forms from the heme of the worn out R.B.C. is absorbed by the liver from the blood and excreted into bile.
Storage:
Liver stores, glycogen fat, protein, various vitamins (A and B12), minerals
Phagocytosis:
The kuffer cells of the liver phagocytize worn-out red and white blood cells and some bacteria.

3.1.5. Liver Pathology:[51,52,57,58]
I. Hepatic injury:
Liver suffers from general responses
 Degeneration and intracellular accumulation:
Toxic or immunologic damage may cause hepatocytes to take on a swollen edematous, appearance with irregularly clumped cytoplasm and large clear spaces. Substances may accumulate in viable hepatocytes, including, iron and copper.
 Necrosis and Apoptosis.
Any significant insult to the liver may cause hepatocyte necrosis. This form of cell death is apoptosis. Necrosis frequently exhibits a zonal distribution. The most obvious is necrosis of hepatocytes immediately around the terminal hepatic vein, an injury that is characteristic in ischemic injury and a number of drug and toxic reactions.
The necrosis may be divided into:
a. Diffuse necrosis in which all the cells in group of lobules are affected as in acute yellow atrophy.
b. Zonal necrosis, in which only the cells of a certain area in each lobule are affected.
c. Focal necrosis, in which small areas of no uniform distribution are affected.
 Inflammation
Injury to the liver associated with an influx of acute or chronic inflammatory cells is termed hepatitis. Hepatocyte necrosis may precede the onset of inflammation, and vice versa. Attack on viable cells by sensitized T cells is a common cause of liver damage. Inflammation may be limited to the site of leukocyte entry (portal tracts) or spill over into the parenchyma.
 Regeneration
The liver has enormous reserve, and regeneration occurs in all but the most fulminant diseases.
 Fibrosis
Fibrous tissue is formed in response to inflammation or direct toxic insult to the liver. Fibrosis is irreversible.
II. Jaundice
Hepatic bile promotes dietary fat absorption in the lumen of the gut and also the elimination of waste products. When bile formation is disrupted it is clinically evidenced as yellow discoloration of the skin and sclerae is jaundice.
Jaundice may be either
 Obstructive jaundice:
The purest examples of obstructive jaundice are cases of obstruction of the common bile duct by cancer of the head of the pancreas, stone in the duct, or structure of the duct.
 Hemolytic jaundice:
When there is excessive hemolysis the bilirubin carried to the liver cannot be excreted entirely so that some remains in the blood. So this bilirubin cannot pass the kidney filter, so that the jaundice is of the acholuric type, although there is a great increase of urobilinogen in the urine.

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 Hepatic jaundice:
This form also called toxic jaundice, is the jaundice of infectious hepatitis, or of liver necrosis

III. Cholestasis
It is characterized by the systemic retention of not only bile but also other solutes eliminated in the bile. It generally results from hepatocellular dysfunction or intrahepatic or extrahepatic biliary obstruction.

IV. Hepatic failure
It may result from sudden and massive hepatic destruction. Often it is the endpoint of progressive damage to the liver, either by insidious destruction of hepatocytes or by repetitive discrete waves in parenchymal damage. Morphologic alterations that causes liver failure fall into 3 categories:
 Massive hepatic necrosis:
It is most often due to fulminant viral hepatitis. Drugs and chemicals like paracetamol, halothane, antituberculasis may also induce massive necrosis.
 Chronic liver disease:
This is the most common route to hepatic failure and is the endpoint of relentless chronic hepatitis.
 Hepatic dysfunction without overt necrosis:
Hepatocytes are less commonly viable but unable to perform normal metabolic function.

V. Cirrhosis:
It largely results due to alcohol abuse, other major contributors are chronic hepatitis, biliary disease and iron overload.

VI. Portal hypertension:
Increased resistance to portal blood flow may be categorize intraprehepatic, intrahepatic and posthepatic causes.
The major prehepatic cause is obstructive thrombosis and narrowing of portal vein. Dominant intrahepatic cause is cirrhosis.
The major posthepatic cause include severe right sided heart failure, constrictive pericarditis, and hepatic vein outflow obstruction.

VII. Infectious disorders:
The foremost hepatic infections are viral in origin.

 Viral Hepatitis:
Acute viral hepatitis is a systemic infection affecting the liver predominantly. Almost all cases of acute viral hepatitis are caused by one of five viral agents: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), the HBV-associated delta agent or hepatitis D virus (HDV), and hepatitis E virus (HEV). Other transfusiontransmitted agents, e.g., "hepatitis G" virus and “TT" virus, have been identified but do not cause hepatitis. All these human hepatitis viruses are RNA viruses, except for hepatitis B, which is a DNA virus.
 Bacterial, parasitic and helminthic infections:
Extrahepatic bacterial infections, particularly sepsis can induce mild hepatic inflammation and varying degree of hepatocellular cholestasis. A number of bacteria which can infect the liver include Staphylococcus aureus, Salmonella typhi. Liver fluke like fasciola hepatica, Clonorchis sinensis, and Opisthorchis viverrini can also affect the liver.
VIII. Auto immune hepatitis
Autoimmune hepatitis (formerly called autoimmune chronic active hepatitis) is a chronic disorder characterized by continuing hepatocellular necrosis and inflammation, usually with fibrosis, which tends to progress to cirrhosis and liver failure.

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IX. Hemochromatosis

It is characterized by the excessive accumulation of body iron. It results either from a genetic defect causing excessive iron absorption as a consequence of parenteral administration of iron usually in the form of transfusions.

X. Wilson Disease

This is an uncommon inherited disorder of copper metabolism. Wilson's disease presents clinically in adolescence or young adulthood by which time there is excess copper accumulation in the liver and other tissues. Deficiency of the plasma copper protein ceruloplasmin is a characteristic feature. The accumulation appears to result from impaired copper excretion due to a mutation in a gene that encodes a P-type ATPase copper transport.

XI .α1-antitrypsin deficiency

Patients with homozygous deficiency of serum α1-antitrypsin (α1AT) are prone to develop emphysema in adult life. The disease is suggested by the absence of alpha1 globulin on serum electrophoresis (α1AT makes up 90% of this fraction normally) and confirmed by direct measurement of α1AT.

XII. Urler's syndrome

This is an uncommon hereditary disease that is characterized by the widespread tissue deposition of mucopolysaccharide in many tissues. The liver is frequently enlarged and firm.

2.1.6. Drug induced liver Damage [59, 60]

The liver plays an important role in the metabolism of a great variety of ingested substances which may include drugs taken for therapeutic purposes and environmental toxins. It is therefore uniquely susceptible to these agents. Drugs and toxins can affect the liver in one of two ways. The effect may be predictable and dose related i.e. intrinsic, or it maybe unpredictable and idiosyncratic.

Intrinsic hepatotoxins

It include drugs that predictably produce liver damage when taken in sufficient quantities. The type of damage is usually characteristic of a particular drug, and often zonal necrosis follows a short latent period.

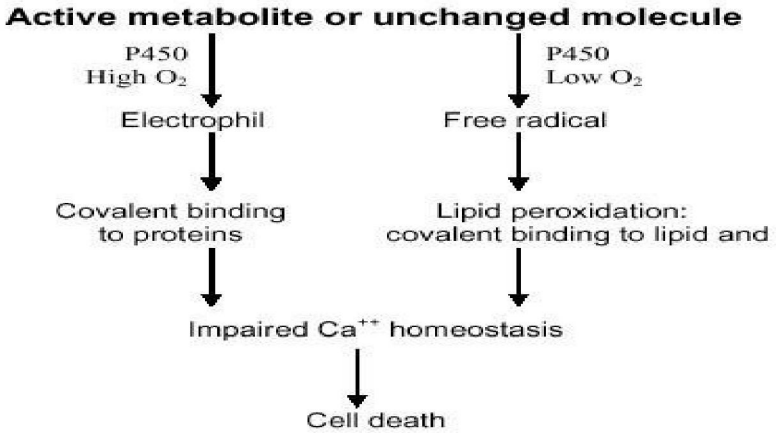

Figure: 4 Mechanism of intrinsic hepatotoxins

Idiosyncratic hepatotoxins

An idiosyncratic reaction is due to either hypersensitivity or genetically determine differences in drug metabolism or lymphocyte reactivity. The overall incidence tends to be low, the hepatic lesions are not dose related and they occur after a relatively long and variable latent period.

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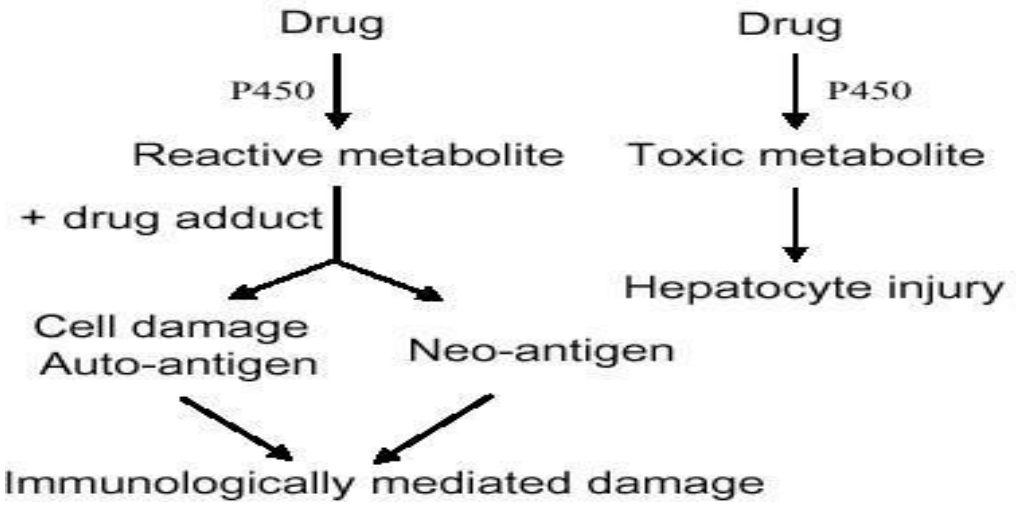

Figure: 4 Mechanism of intrinsic hepatotoxins

Idiosyncratic hepatotoxins

An idiosyncratic reaction is due to either hypersensitivity or genetically determine differences in drug metabolism or lymphocyte reactivity. The overall incidence tends to be low, the hepatic lesions are not dose related and they occur after a relatively long and variable latent period.

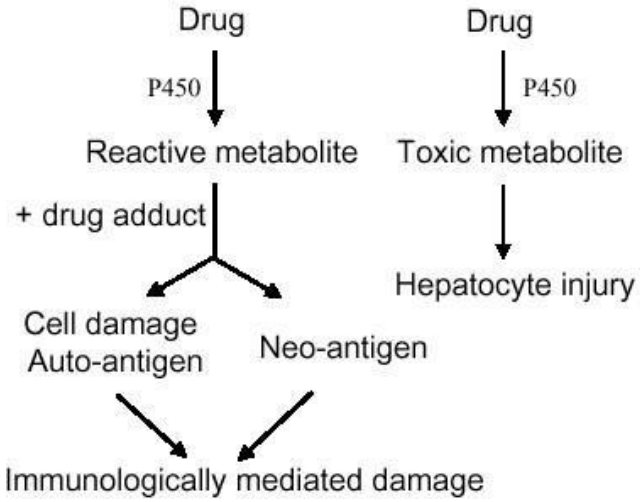

Figure: 5 Mechanism of idosyncratic injury

Factors modulating hepatotoxicity

The risk of drug hepatotoxicity is influenced by various acquired and genetic factors summarized in Tables below.

Table: 1 Principal alterations of hepatic morphology produced by some commonly used drugs and chemicals [58]

Principal Morphologic Change	Class of Agent	Example
Cholestasis	Anabolic steroid	Methyl testosterone
	Anti-inflammatory	Sulindac
	Antithyroid	Methimazole
	Antibiotic	Erythromycin estolate, nitrofurantoin, rifampin,

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		amoxicillin-clavulinic acid, oxacillin
	Oral contraceptive	Norethynodrel with mestranol
	Oral hypoglycemic	Chlorpropamide
	Tranquilizer	Chlorpromazine
	Oncotherapeutic	Anabolic steroids, busulfan, tamoxifen
	Immunosuppressive	Cyclosporine
	Anticonvulsant	Carbamazine
	Calcium channel blocker	Nifedipine, verapamil
Fatty liver	Antibiotic	Tetracycline
	Anticonvulsant	Sodium valproate
	Antiarrhythmic	Amiodarone
	Antiviral	Dideoxynucleosides (e.g., zidovudine) protease inhibitors (e.g., indinavir, ritonavir)
	Oncotherapeutic	Asparaginase, methotrexate
	Anesthetic	Halothane
	Anticonvulsant	Phenytoin, carbamazine
	Antihypertensive	Methyldopa, captopril, enalapril
	Antibiotic	Isoniazid, rifampin, nitrofurantoin
	Diuretic	Chlorothiazide
	Laxative	Oxyphenisatin
	Antidepressant	Iproniazid, amitriptyline, imipramine
	Anti-inflammatory	Ibuprofen, indomethacin, diclofenac, sulindac
	Antifungal	Ketoconazole, fluconazole, itraconazole
	Antiviral	Zidovudine, dideoxy inosine
	Calcium channel blocker	Nifedipine, verapamil, diltiazem
	Antiandrogen	Flutamide
	Immunosuppressive Lipid-lowering	Azathioprine Nicotinic acid, lovastatin
	Hydrocarbon	Carbon tetrachloride
	Metal	Yellow phosphorus
	Mushroom	Amanita phalloides
	Analgesic	Acetaminophen
	Solvent	Dimethylformamide

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	Anti-inflammatory	Phenylbutazone
	Antibiotic	Sulfanomides
	Xanthine oxidase inhibitor	Allopurinol
	Antiarrhythmic	Quinidine
	Anticonvulsant	Carbamazine

Acetaminophen hepatotoxicity[58,61]

Acetaminophen is a widely used analgesic and antipyretic drug. It causes severe centrilobular hepatic necrosis when ingested in large amounts in suicide attempts or accidentally by children. A single dose of 10 to 15g, occasionally less, may produce clinical evidence of liver injury. Fatal fulminant disease is usually associated with ingestion of 25g or more.

APAP is metabolized to a minor electrophilic metabolite, N-acetyl-p-benzoquinoneimine (NAPQI), which during APAP overdose depletes glutathione and initiates covalent binding to cellular proteins. These events lead to the disruption of calcium homeostasis, mitochondrial dysfunction, and oxidative stress and may eventually culminate in cellular damage and death.

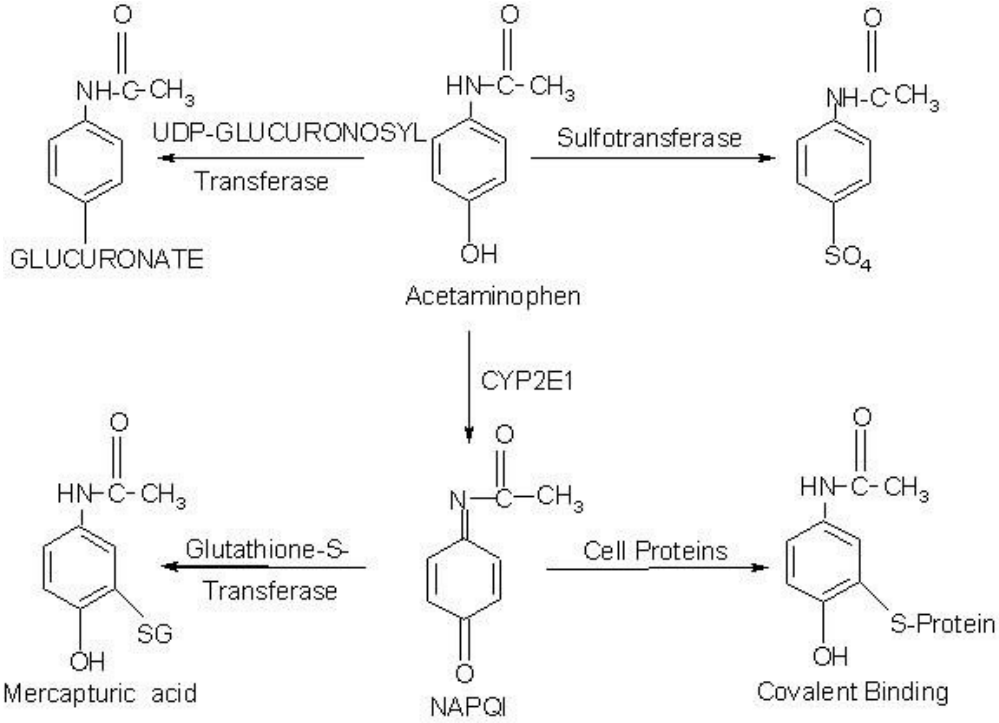

Figure: 6 Metabolic pathway of Acetaminophen

Rifampicin[62-64]

Several anti-tuberculosis agents have been implicated as being hepatotoxic viz.. rifampicin, thioacetamide, isoniazide etc. Rifampicin is a broad spectrum antibiotic agent most commonly used as antituberculin. It is largely metabolized to desacotyl rifampicin which undergo enteroheptic circulation, which actively binds to RNA polymerases and thereby inhibits the synthesis of nucleic acid and protein synthesis Rifampicin may cause transient hyperbilirubinaemia, due to interference with bilirubin excretion.

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Isoniazid[65-69]
Isoniazid is metabolized to monoacetyl hydrazine, which is further metabolized to a toxic product by cytochrome P450 leading to hepatotoxicity. Studies have shown that cytochrome P4502E1 (CYP2E1) is involved in anti tubercular drug hepatotoxicity. Rat studies showed that Isoniazid and Hydrazine induce CYP2E1 activity. Isoniazid causes inhibition of on CYP1A2, 2A6, 2C19 and 3A4 activity. CYP1A2 is found to be involved in hydrazine detoxification. Isoniazid can induce its own toxicity, possibly by the induction or inhibition of these enzyme.

Thioacetamide
Chronic thioacetamide exposure produces liver injury. Mechanism of thioacetamide toxicity is due to the formation of thioacetamide-5-oxide which is responsible for the change in cell permeability, increased intracellular concentration of Ca++, increase in nuclear volume and enlargement of nucleoli and also inhibits mitochondrial activity which leads to cell death.

2.1.7. Liver Function test:[67,68,70]
Several biochemical tests are useful in the evaluation and management of patients with hepatic dysfunction. These tests can be used to (1) detect the presence of liver disease; (2) distinguish among different types of liver disorders; (3) gauge the extent of known liver damage; and (4) follow the response to treatment.
The liver function tests have been divided into different categories as:

I. Tests based on detoxification and excretory functions Serum Bilirubin:
Bilirubin, is a breakdown product of the porphyrin ring of heme containing proteins it is found in the blood in two fractions conjugated and unconjugated. The van den Bergh assay, used in most clinical chemistry laboratories to determine the total serum bilirubin level.
Elevation of unconjugated bilirubin is seen primarily in hemolytic disorders and in a number of genetic conditions. Conjugated hyperbilirubinemia almost always implies liver or biliary tract disease. In most liver diseases, both conjugated and unconjugated fractions of the bilirubin tend to be elevated. The serum of normal adults contains less than 1mg/dl of total bilirubin, out of which less than 0.25 mg/dl is conjugated bilirubin.

Urine Bilirubin :
Unconjugated bilirubin always binds to albumin in the serum and is not filtered by the kidney. Therefore, any bilirubin found in the urine is conjugated bilirubin; the presence of bilirubinuria implies the presence of liver disease. A urine dipstick test is used to estimate urine bilirubin.

Bilirubin in faeces:
Excretion of bilirubin in stools is assessed by inspection of stools. Clay colored stool due to absence of faecal excretion of the pigment indicates obstructive jaundice.

Blood Ammonia:
The liver plays an important role in the detoxification of ammonia by converting it to urea, which is excreted by the kidneys. Patients with advanced liver disease typically have significant muscle wasting, which contributes to hyperammonemia in these patients. Some physicians use the blood ammonia for detecting encephalopathy or for monitoring hepatic synthetic function.

Serum Enzymes:

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Determination of certain serum enzymes is useful in various types of liver injury. Serum enzyme tests can be grouped into three categories: (1) enzymes whose elevation in serum reflects damage to hepatocytes; (2) enzymes whose elevation in serum reflects cholestasis; and (3) enzyme tests that do not fit precisely into either pattern.

 Enzymes that Reflect Damage to Hepatocytes

The aminotransferases (transaminases) are sensitive indicators of liver cell injury. They include the aspartate aminotransferase (AST) and the alanine aminotransferase (ALT). Any type of liver cell injury can cause modest elevations in the serum aminotransferases. Levels of up to 300 IU/L are nonspecific and may be found in any type of liver disorder. Striking elevations i.e., aminotransferases (1000 IU/L) occur almost exclusively in disorders associated with extensive hepatocellular injury such as (1) viral hepatitis, (2) ischemic liver injury (prolonged hypotension or acute heart failure), or (3) toxin or drug-induced liver injury.

Table: 2 The relative incidence of liver injury and elevated transaminase levels for various drug classes[70]

Class	Hepatic effects	Mechanism of injury
Nonsteroidal anti-inflammatory drugs	Incidence of acute liver injury is about 3.7 per 100,000 NSAID users. Transaminase levels abnormal in 5–15% of patients taking a particular drug.	Mostly idiosyncratic responses.
Anticancer drugs	Hepatotoxic manifestations include venoclusive disease, hepatocellular necrosis, and fatty change. Incidence of 1.3% in 980 cases of hepatic injury in one study.	Diverse injuries, both intrinsic and idiosyncratic.
Antibacterials	Estimated frequency of hepatotoxic reactions is between 1 and 10 per 100,000 drug prescriptions. Antibiotics accounted for one-third of all suspected drug-induced hepatic injury in one study. Mild transient transminase elevations reported in up to 30–50% of recipients for some antibiotics.	Injuries include necrosis, cholestasis, cholangitis, and other. Mostly idiosyncratic except intrinsic for tetracyclines and some macrolides.
Cardiovascular	In one study, cardiovascular agents were implicated in 27.3% of 980 cases of liver injury. Diuretics accounted for 13.9% in another study. Mild ALT/AST elevations are common at the onset of treatment.	Autoimmune or hypersensitivity reactions with antiarrhythmic agents; heptocellular pattern with antihypertensive agents.

 Enzymes that Reflect Cholestasis

The activities of three enzymes α-alkaline phosphatase, 5α-nucleotidase, and gamma glutamyl transpeptidase (GGT) are usually elevated in cholestasis.

Alkaline phosphatase elevations greater than four times normal occur primarily in patients with cholestatic liver disorders, liver cancer, and also in bone conditions like rapid bone turnover, gamma glutamyl transpeptidase (GGT) is rarely increased in any conditions other than liver disease.

 Other serum enzyme like

Lactate Dehydrogenase (LDH) is elevated in patients with metastatic liver. Choline esterase is diminished in hepatocellular disease.

II. Tests for metabolic functions:

Amino Acid and plasma protein metabolism

Based on the metabolic functions of the liver, serum estimation of proteins, immunoglobulins are employed to assess the liver cell damage.

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 Serum Albumin

Serum albumin is synthesized exclusively by hepatocytes has a long half-life of 15 to 20 days. Because of this slow turnover, the serum albumin is not a good indicator of acute or mild hepatic dysfunction; only minimal changes in the serum albumin are seen in acute liver conditions such as viral hepatitis, drug-related hepatoxicity and obstructive jaundice.

 Serum Globulins

Serum globulins include gamma globulins (immunoglobulins) and alpha and beta globulins, Gamma globulins are increased in chronic liver disease, such as chronic hepatitis and cirrhosis.

 Coagulation Factors

With the exception of factor VIII, the blood clotting factors are made exclusively in hepatocytes. Serum prothrombin time(measures factors II, V, VII, and X.) may be elevated in hepatitis and cirrhosis as well as in disorders that lead to vitamin K deficiency such as obstructive jaundice or fat malabsorption.

 Immunoglobulins

The levels of serum immunoglobulins produced by lymphocytes and plasma cells show nonspecific abnormalities in liver diseases and represent inflammatory or immune response rather than liver cell dysfunction.Liver diseases are associated with various immunologic abnormalities .

Non specific immunologic reactions:

It includes

o Smooth muscle antibody to actin component of muscle is formed in certain hepatic disorders with hepatic necrosis. o Mitochondrial antibody develops in patients with primary biliary cirrhosis.

o Antinuclear antibody is present in some patients of chronic hepatitis.

Antibodies to specific etiological agents:

These vary according to the etiologic agents causing liver injury. Hepatitis B surface antigen (HBsAg) can be demonstrated in cases of serum hepatitis. Hepatitis B core antigen (HBcAg) can be detected in all patients with hepatitis B Hepatitis Be antigen (HBeAg) can be found in chronic varieties of hepatitis. Amoeba antibodies to entamoeba histolytica develop in patients with amoebic liver disease.

Lipid and Lipoprotein Metabolism

There is a rise in total serum cholesterol in cholestasis, serum triglycerides are also elevated in cholestasis. There values are lowered in acute and chronic diffuse liver diseases and in malnutrition.

Carbohydrate Metabolism

Blood glucose level is lowered in fulminant acute hepatic necrosis in chronic liver diseases, there is impaired glucose tolerance and relative resistance.

III. Other diagnostic tests

Radiologic testing and procedures are often necessary to make the proper diagnosis. The two most commonly-used ancillary tests are

Percutaneous Liver Biopsy

Percutaneous biopsy of the liver is a safe procedure that can be easily performed at the bedside with local anesthesia. It is valuable in diagnosis of any no of liver disorder.

Ultrasonography

Ultrasonography is the first diagnostic test, to use in patients whose liver tests suggest cholestasis, to look for the presence of a dilated intrahepatic or extrahepatic biliary tree, or to identify gallstones.

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2.1.8. Free radicals/ reactiveoxygen species[71]

The free radical can be defined as a chemical species, an atom or molecule that has one or more unpaired electrons in valance shell and is capable of existing independently. As free radical, contain an odd number of electron, which make it unstable, short lived and highly reactive; therefore it can react quickly with other compound, trying to capture the needed electron to gain stability. Generally, free radical attacks the nearest stable molecule, "stealing" its electron. When the attacked molecule looses its electron, it becomes a free radical itself, beginning a chain reaction cascade resulting in disruption of a living cell.

Free radicals can be formed by following mechanism

A) Covalent bond cleavage of normal molecule or atom: Atoms are bounded together when they share or transfer electron to form molecule. A covalent bond is formed when a pair of electron is shared.

B) Electron transfer: Electron transfer is a far more common an important source of generation of free radicals in biological system.

i) Oxidation reaction : By loss of a single electron from a normal molecule ii) Reduction reaction : By addition of a single electron to a normal molecule.

Reactive oxygen species

The most important free radicals in biological system are radical derivatives of oxygen. Oxygen is required to transfer various substances for the release of the energy and detoxify xenobiotics. During this process oxygen acts as terminal electron acceptor and is eventually converted to more stable compound, water. This reduction of one molecule of O via the cytochrome oxidase system of respiratory chain requires 4 electrons. Such type of reduction is known as transvalent reduction of oxygen to water.

Oxidative stress: Normally there is an equilibrium between free radical formation and endogenous antioxidant defense mechanisms. An imbalance leads to oxidative stress, which can be mainly because of two reasons either due to excessive generation of reactive oxygen species (ROS) or this state of oxidative stress can result injury to both cellular as well as extra cellular components of cell and can cause cell death as shown in figure.

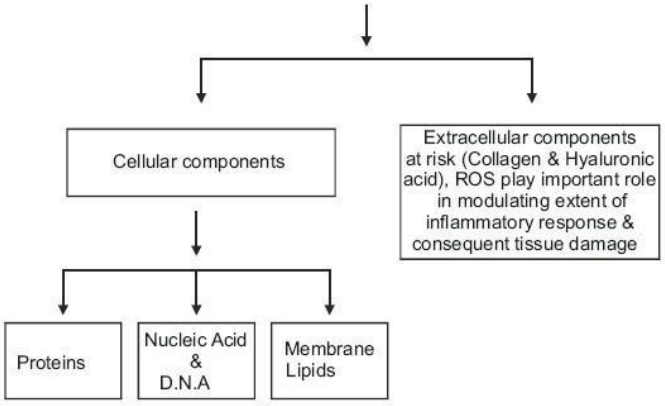

Figure: 7 Cellular and extracellular components at risk from reactive oxygen species mediated damage

There are compelling evidences for the involvement of reactive species in number of pathophysiological states as shown in figure.

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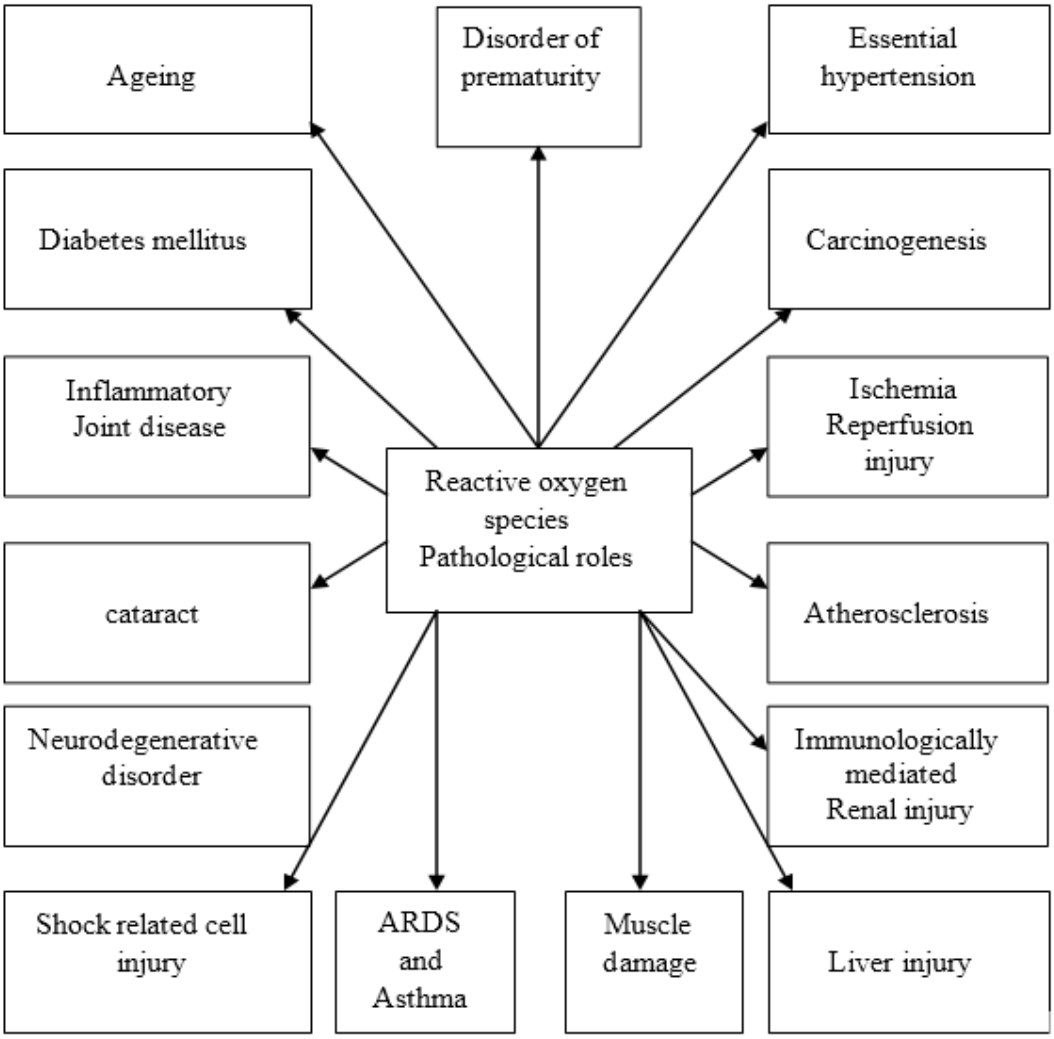

Figure: 8 Involvement of reactive species in number of pathophysiological states

2.1.9. Therapeutic agents in liver disorders

No single curative therapeutic agent is found completely effective in hepatic disorder. Agents from many fields are used to cure liver disorders .they can be described as follows:

Allopathic medicines[72]

The conventional drugs used in the treatment of liver diseases are corticosteroids, interferon, colchicines, penicillamine, antiviral and immunosuppressant agents but sometimes they are inadequate and inconsistent at best. Often treatment with them is worse than the disease which may lead to serious side effects.

Herbs as potential therapeutic agents

Plants constitute the group having effective therapeutic agents with a low incidence of side effects.

Silymarin[73-78]

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Silybum marianum is the scientific name for Milk thistle or St. Mary's thistle and belongs to the Asteraceae family. It is characterized by thorny branches and a milky sap, with its oval leaves reaching up to 30 cm. The flowers are bright pink and can measure up to 8 cm in diameter.

Milk thistle has been used in plague and for congestive conditions of the liver and spleen. In modern times the use of milk thistle has been focused on liver conditions. The hepatoprotective activity of Silymarin rests with the strong antioxidant and free radicals scavenging activity. Providing sylimarin with ability to protect the hepatocyte membranes against oxidative damage. Silymarin inhibits lipid peroxidation of hepatocytes, microsomal and erythrocyte membranes in rats. Silymarin was found to protect hepatocytes against ethanol induced liver peroxidation by increasing hepatic glutathione levels and superoxide dismutase expression and activity was increased in liver cells taken from the patients with chronic liver disease.

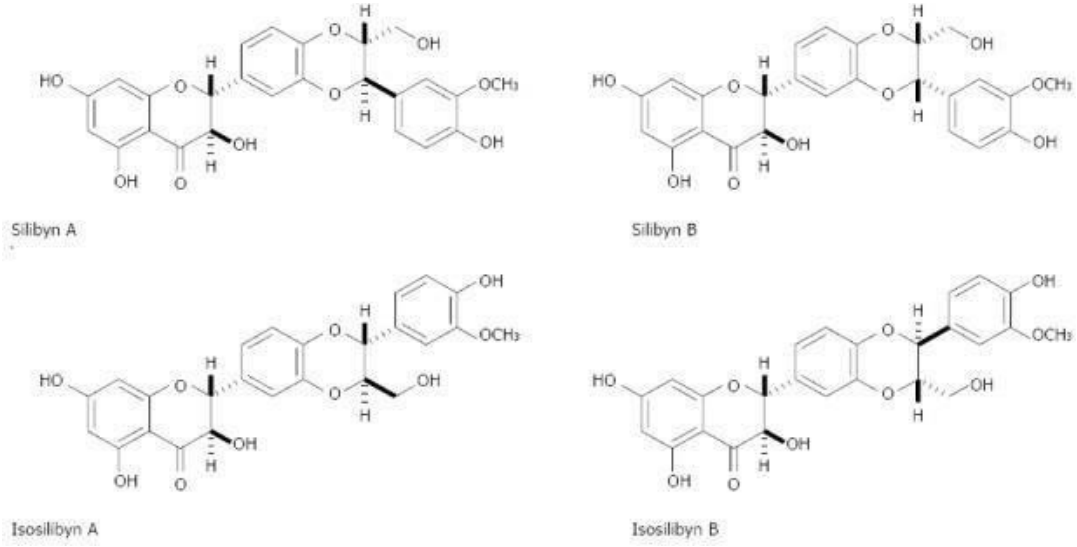

Figure: 9 The chemical structure of silbyna A, silbyna B, isosilbyna A, and isosilbyna B, the extract of silymarin.

Multiplant preparation

44 proprietary formulations with 93 medicinal plants belonging to 44 families have been used in hepatic disorders[79] Herbal formulation like rhinax,[80] Amalkadhi ghrita,[81] liver 52,[82] liv100,[83] Kamilari[84] were found to be effective in CCl4 induced heptotoxicity. Livex [85] found effective in erythromycin estolate induced hepatotoxcicity. HD-03 [86] is found effective in paracetamol, thioacetamide, isoniazide induced hepatotoxicity.

Single plants

Plants commonly used in liver disorders are Andrographis paniculata, Boerhaavia diffusa, Eclipta alba, Picrohiza kurroa and Tinospora cardifolia[79]

- Andrographis paniculata[82]

Out of 40 Indian multiplant formulations, Andrographis paniculata occurred in 26 formulation, it has shown effectiveness against many models like BHC,CCl4 induced liver damage.

- Eclipta alba[79]

Other

It is considered one of the best plants in traditional medicine for jaundice and cirrhosis. It is effective against carbontetrachloride and galactosamine induced cytotoxicity.

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Plants for which interesting results have been obtained are Tinosporia cardifolia,[77,88] Phyllanthus niruri,[89-91] Ailanthus excels,[92] Lawsonia alba,[93] Entanda pursaetha,[94] Acanthus ilicifolius,[95] Foeniculm vulgare,[96] Emblica officinalis, [97] Artemisia maritime,[98] Cichorium intybus,[99] Aronia melanocarpa,[100] Bupleurum kaoi Liu,[101] Pergularia daemia Forsk,[102] Pterocarpus marsupium,[103] Careya arborea,[104] Phoenix dactylifera L,[105] Aspalathus linearis,[106] against carbon tetrachloride induced hepatotoxicity. Gingko biloba,[107] Azardhicta indica,[108] Ambrosia maritime,[109] Nigella sativa,[110] Caseria esculenta,[111] against paracetamol induced hepatotoxicity. Boerhavia diffusa,[112] Jigrin,[113] Nardostachys jatamansi,[114] against thioacetamide induced hepatotoxicity. Vegetables like Beta ulgaris,[115] Daucus carola,[116] Raphnus sativus,[117] against carbontetrachloride.

Isolated agents as hepatoprotective

Some agents isolated from plants e.g., emodin,[118] bergenin,[119] 3,4,5- trihydroxy benzoic acid[120] are found to be effective in carbon tetrachloride induced hepatotoxicity monomethyl fumarate,[121] Alantolactone,[122] umaric acid,[123] in paracetamol and rifampicin induced liver toxicity. β-carotene[124] in paractamol induced liver injury shows protective effects.

Emergence of trace elements as potential agents for liver ailments

Trace elements like Gadolinium Chloride,[125,126] alters thioacetamide induced hepatotoxicity. Selenium[127,128] protects against dietary liver necrosis, boron and copper are also reported to protect against liver injury.

Alternate therapies in liver diseases.

 Thymosin therapy[129-131]

It involves, using, hormones normally secreted by the thymus gland. These hormones appear to stimulate the body's production of interferon. People with low levels of these hormones are susceptible to liver infection.

 Megadose vitamin therapy[132-135]

It is based on the theory that the higher the dose of vitamins , the faster the cure, a consistent low dose of vitamins, in conjugation with a healthy diet, is much more effective as a preventative measure than megadoses, once damage is done as this puts much strain on the liver.

 Alpha-lipoic Acid therapy[136, 137]

It uses the antioxidant enzyme helper of Alpha lipoic acid, and is beneficial in protecting the liver against mushroom or acetaminophen overdose if administered intravenously.

2.2 THIOACETAMIDE-INDUCED HEPATOTOXICITY

2.2.1 Thioacetamide

Thioacetamide (TAA) is an organosulfur compound with formula C2H5NS. It is a carcinogen (class 2B), designated as a human carcinogen. It is a white crystalline solid, soluble in water & alcohol. It serves as a source of sulfide ions in the synthesis of organic and inorganic compounds (rubber chemicals, metallurgy, pesticides, crosslinking agents and pharmaceuticals).[138-140]

It is reported that TAA can damage different organs, including the [141-145]

- Liver
- Lungs
- Intestine
- Kidneys
- Spleen
- Thymus
- Pancreas

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Metabolism of TAA

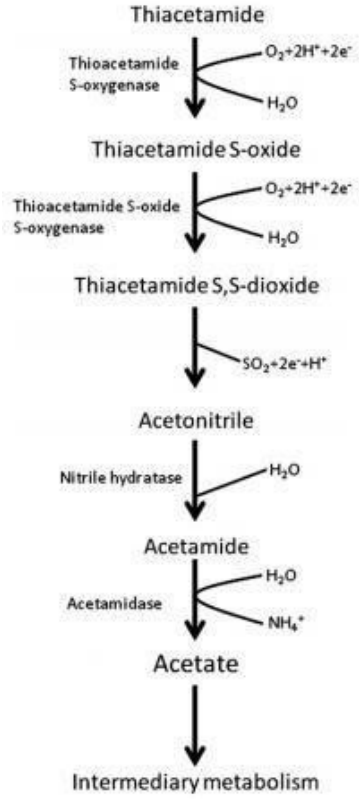

Figure:10 Metabolism of TAA

Bioactivation of TAA

TAA undergoes 2 step bioactivation interceded by microsomal CYP2E1 to sulfine, and Then to sulfene, a reactive metabolite. Cyt-P450 system is responsible for TAA metabolism in rat liver. TAA is bio-transformed or metabolized by flavin-containing monooxygenase (FMOs) systems and cytochrome P450 (Cytp450). Cyt-p450 enzymes are present in the liver microsomes, which converts TAA to thioacetamide S-oxide (TAASO), a reactive intermediate with toxic nature; which induces oxidative stress in the hepatic cells that finally leads to centrilobular necrosis and liver injury.

The mode of action of TAA during liver injury

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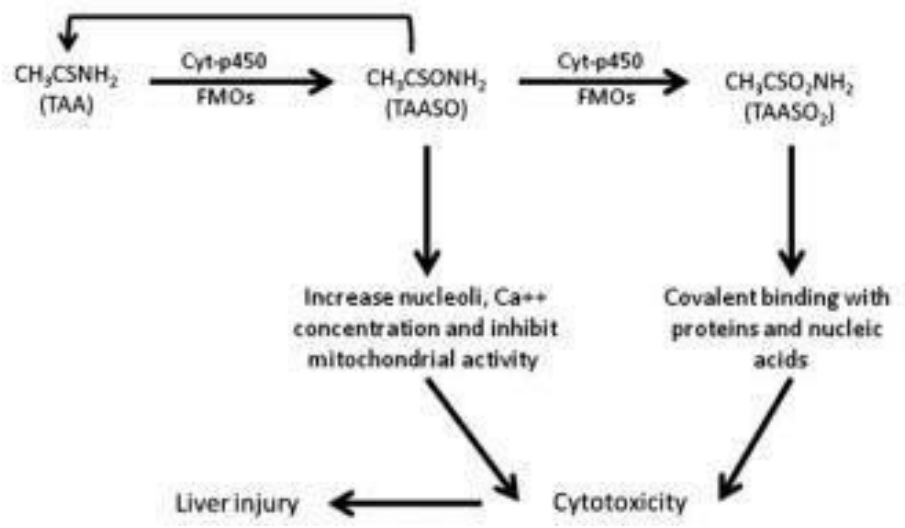

Figure:11 The mode of action of TAA during liver injury

According to the pattern and doses of administration, thioacetamide (TAA) induces acute liver failure or hepatic cirrhosis [149,150].

2.2.2 Single dose intravenous thioacetamide administration as a model of acute liver damage in rats

Acute liver failure(ALF) is an important challenge for hepatologists. ALF can rapidly lead to multiorgan failure and death. Its Common causes include acute viral hepatitis, and drug- or toxin-induced liver injury. Despite advances in intensive care and the development of new treatment modalities, ALF remains a condition associated with high mortality rates. Liver transplantation remains the only effective treatment. Thioacetamide (TAA) has been used extensively in the development of ALF in animal models.

Thioacetamide (TAA) has been used since several years to induce a model of acute liver injury in rats. TAA is a potent centrilobular hepatotoxicant, which undergoes a two-step bioactivation mediated by microsomal CYP2E1 to thioacetamide sulphoxide (TASO), and further to a reactive metabolite thioacetamide-S, S-dioxide (TASO2). It can release inducible nitric oxide synthase (iNOS) and nuclear factor-κB (NF-κB), leading to centrilobular necrosis.

Thioacetmide is used in dose ranging from (50, 70, 100, 150, 200, 250, 280,300, 350,400,500 mg/kg)[156-168] intraperitoneal or subcutaneous route to induce acute liver failure.

2.2.3 Liver cirrhosis induced by Thioacetamide

Liver cirrhosis is a critical stage of chronic liver diseases that can produce liver failure, portal hypertension and also hepatocarcinoma. It is related to high morbidity and mortality, which may be due to viral infection, toxic agents, tissue-immune mediated damage, obstructive jaundice, gene abnormalities, or alcohol and nonalcohol steatohepatitis. The current treatments of liver cirrhosis are limited to the removal of the underlying injurious stimulus,

e.g. viruses in cases involving viral hepatitis. Fibrosis results from activation of stellate cells by cytokines and oxidative stress. It replaces damaged cells with an extracellular matrix. The induction of oxidative stress, mitochondrial dysfunction and depletion of antioxidant status is a relevant feature in the progression of liver cirrhosis and fibrosis. [169-176] Thioacetamide is biotransformed by CYP2E1 enzymes found in the microsomes of liver cells and transform it to a highly reactive toxic intermediate known as thioacetamide sulphur dioxide by oxidation, inducing hepatotoxicity in experimental animals and different grades of liver damage including nodular cirrhosis, production of pseudolobules, proliferation of hepatic cells, and necrosis of parenchyma cells.[5-6]

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Hepatic encephalopathy

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome occurring as consequence of acute or chronic liver failure. It affects a considerable number of patients worldwide with a mortality index from 50 to 90%. Its pathogenesis is still not well understood. The main hypothesis suggests a state of hyperammonemia which is responsible for both direct and indirect alterations in cerebral metabolism. Hyperammonemia increases the levels of glutamine in astrocytes from amidation of glutamate by glutamine synthetase. This accumulation of glutamine in the astrocytes produces osmotic stress and causes the astrocytes to swell. This leads to cerebral edema and intracranial hypertension which are the main pathophysiological features of HE. Other mechanisms are also proposed to explain the pathogenesis of HE, including dysfunction of the immune and neurotransmitter systems. Several drugs have been used to induce liver failure in experimental models, such as thioacetamide (TAA), D-galactosamine, acetominophen, carbon tetrachloride and concanavalin A. TAA is frequently used in experimental studies due to its efficacy in inducing hepatic failure in rats. Following hepatic failure, TAA is able to induce HE.[7-18]

Hepatorenal syndrome:

Renal function deteriorates in patients with cirrhosis as liver function worsens, which indicates a link between these two organ systems. Apoptotic cell death and increases in adenosine production (potentiating the vascular effects of angiotensin-II) are thought to play an important role in the development of kidney damage during cirrhosis. Generation of free radicals and oxidative stress during cirrhosis may also result in oxidative damage in the kidney. Oxidative stress gains importance in the context of renal failure in cirrhosis, because free radicals such as superoxide can interact with nitric oxide to generate peroxynitrite, which is an oxidant and a nitrosating agent. Reports have shown that nitric oxide synthase is increased in renal glomeruli of rats with cirrhosis.[19-22] Thioacetmide is used in various dose of (50, 100, 200 mg/kg)[177-183] is used to induce liver cirrhosis.

2.3 PLANT: Bambusa arundinacea

2.3.1. Introduction

Bamboo consist of fresh leaves & dried fruits of Bambusa arundinacea. Bamboos are members of the Graminae (Poaceae) family other member from this family include corn, sugar cane and other grasses. Bamboos differ from the other members of the grass family by the presence of branches at each node. A bamboo culm consists of an internode (which is hollow for most bamboo) and a node, which is solid and provides structural integrity for the plant. At the node are one or more buds (depending on the species) which produce side branches.

Figure: 12 Young Shoots Of Bambusa arundinacea

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2.3.2. Vernacular name [186]
English: Bamboo, Bamboo manna, Giant Thorny Bamboo
Hindi: Bans-lochana, Banskapur, Vanoo, Banz
Sanskrit: Vanshalochana, Venulavanam
Gujarati: Toncor, Wans, Vanskapur, Vas-nu-mitha
Marthi: Bansa, Baambii, Bansamitha
Bengali: Bans-Kapur, Baans, Baansh, Baroowa Bans Telugu: Veduruppu, Mulkas Veduru, Mullu Veduru Tamil: Munga-luppa, Mullumangila, Mulmunkil, Mungil
Maliyalam: Moleuppa
Kannad: Bidaruppu, Tavakshira
Arab: Tabashir
Unani: Tabashir, Tawashir
Burma: Vd-chha, Vathega-kiyo, Vasan, Vathe gasu

2.3.3. Distribution[187-192]
The common bamboo is found to be distributed throughout the moist parts of India, upto an altitude of 1250m most commonly near river banks, in Central and South India ascending upto an altitude of 1100m on the Nilgiri. They are also cultivated in many places in North-West India and Bengal. It is also found other countries like Sri Lanka, Malaya, Peru and Myanmar (Burma).

2.3.4. Botanical description
Thorny tree, with many stems, tufted on a stout root-stock, they grows upto 30 meter high; culms 15-18cm across; nodes prominent, the lower emiting horizontal is almost naked shoots armed at the nodes with 2-3 stout recurved spines; internodes upto 45 cm. long. Leaves are 17.5 – 20.5 X 2-2.5 cm, linear or linear – lanceolate, tip stiff, glabrous or puberulous beneath, margins scabrous, base cilliate, mid-rib narrow, leaf-sheath ending on a thick callus and shortly bristly auricle. Inflorescence, an enormous panicles often occupy the whole stem. Caryopsis (grain) oblong, 5-8 mm long, grooved on one side. Flowers and Fruits : Once in life time, mostly during September – May.

Figure: 13 Bambusa arundinacea

2.3.5. Macroscopic Characteristic
They are characterized by woody, pointed stems called culms arising from the underground woody jointed rhizomes. Culms are round and smooth. Diameter is few mm to > 30cm. The number of fiber bundles and the manner of their scattering are responsible for the hardness of the culm. Also the thickness of the outer shell & the deposit of silica in outer cortical layer make it very hard. Fresh cums are green colour. Generally culms don't bear any branches to a considerable height. Rhizomes is of pachymorph type, they are woody in nature, slightlyarched, upturned sharply at the

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tip just like a walking stick handle, they become become thick & broad at the end bearing the culms & narrow at the proximate end which is called neck where it attached to the older rhizoms.

2.3.6. Phytochemicals[39-41]

Bambusa arundinacea contains phytochemicals like Choline, betain, urease, nuclease, proteolytic enzymes, diastatic and emulsyfying enzyme, alkaloids and glucoside, resins, lignin, waxes, silica, uronic acid, reducing sugars such as galactose, glucose, arabinose, mannose and xylose. Phytosterols like stigmasterol and β-sitosterol, Stigmast-5, 22-dien-3β-ol, Stigmast-5-en- 3β-ol-β-D glucopyranoside, triterpenes and steroidal glycosides, 17, 20, 20-tri demethyl-20α-isopranyloleanane, eicosanyldicarboxylic acid, α-amyrin acetate, urs-12-en-3β-ol-β-D-glucopyranoside. Flavonoids like orientin, homoorientin, vitexin and isovitexin are also present.

2.3.7 Pharmacological studies

Antidiabetic activity [45]

• The Ethanolic extract of the root of Bambusa arundinacea was evaluated for its antidiabetic activity in normoglycemic rats by alloxan and glucose loaded hyperglycemic rats by single dose and multidose administration. Results showed that the plant extract significantly ($p<0.01$) reduces blood glucose level both in normoglycemic and hyperglycemic rats induced by alloxan and oral glucose loaded methods.

• Aqueous extract of the leaves of Bambusa arundinacea were evaluated for the hypoglycemic activity. The extract was administered via oral route at the dose of 500 mg/kg to normal and streptozotocin-induced diabetic rats. The hypoglycemic effect of the extract was significant in euglycemic rats at 30 minutes and in hyperglycemic rats at 3 hours, the results were comparable to that of standard antidiabetic agent, glibenclamide 0.9 mg/kg.

Antifertility effect

An ethanolic extract of Bambusa arundinacea tender shoots (BASE) caused a significant decrease in fertility of male rats. After the administration of 300 mg/kg per day of extract for 7 days, the fertility index reduced to 15% for control rats and to 23% after a 7day recovery period, respectively. The number of cohabited females being successfully inseminated was reduced prominently after 4 days of treatment. Complete recovery of mating behavior was evident 8 days after the withdrawal of ethanolic extract. The number of spermatozoa in the caput and cauda epididymis was reduced concomitant with a reduction in the motility of spermatozoa collected from the cauda epididymis. The weights of testes, epididymides, vas deferens and prostate were also significantly decreased. The serum profile of protein and oxaloacetic/pyruvic transaminase activity showed that the extract was non-toxic .

Antibacterial activity

Water-phase extract of Bambusa shavings (WEBS), was evaluated for antimicrobial activity against the range of food borne and food spoilage pathogens. The Water-phase extract exhibited significant antimicrobial activity against Staphylococcus aureus, Escherichia coli ,Bacillus subtilis, Penicilliun citrinum, Aspergillus niger and Saccharomyces cerevisiae with a concentrationdependent relationship. The minimum inhibitory concentrations (MICs) of the extract against the tested bacterial strains were found in the range of 4.9 - 32 mg/ml.

Antiinflammatory and Antiulcer

Methanol extract of the leaves of Bambusa arundinacea was evaluated for The antiinflammatory effect against carrageenin-induced and immunologically induced paw oedema and also for antiulcer activity in albino rats. Methanol extract was found to be significant in reducing inflammatory activity when compared to the standard drugs. The combination of methanol extract and phenylbutazone has also been studied and found to be the most potent with least toxic (no ulcerogenic) activity.

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Protective effects[200]

Bamboo-derived pyrolyzates were evaluated for Two biological activities 1) the protective effects against N-methyl-daspartate (NMDA)-induced cell death in primary cultured cortical neuron. 2) The anti-plasmin effects determined by using fibrin and fibrinogen degradation products (FDPs) assay. Neuronal cells treated with pyrolyzates of Phyllostachys pubescens, Phyllostachys nigra and Phyllostachys bambusoides restored cell viability when compared with untreated cells in an NMDA-induced neuronal cell death assay. Treatment of Cortical neurons with Phyllostachys pubescens and Phyllostachys nigra showed a decrease of apoptosis following exposure to NMDA. Phyllostachys nigra pyrolyzates also exhibited anti-plasmin action in a fibrinogen degradation products assay.

III. MATERIALS AND METHODS

3.1 Drugs and Chemicals

2,2-diphenyl-1-picrylhydrazyl (DPPH), Fetal Bovine Serum FBS, MTT (3-(4,5Dimethylthiazol-2-yl)-2,5-Diphenyltetrazolium Bromide) were purchased from Hi-media. TCA (trichloroacetic acid), TBA (thiobarbituric acid),Thioacetmide, Ascorbic acid was purchased from Sigma. SGOT, SGPT, ALP, LDH, Triglyceride, T.Protein and Bilirubin, Cholestrol kits were brought from span diagnostics. GGT kit from Tulip and all other chemicals were of analytical grade.

HepG2 and Hep3b cell lines were procured from National Centre For Cell Science, Pune.

3.2 Plant Collection and preparation of Extract

Young shoots of Bambusa arundinacea were collected in the month of August from Assam. They were dried under shed at room temperature (25-30oC). After drying the shoots (10 Kg) were powdered with dry grinder and sieved through sieve 40 mesh. The powder was packed in soxhlet apparatus and defatted with petroleum ether and then Extracted with Methanol to obtain Methanolic Extract. The percentage yield was found to be 15.5% (w/w).

3.3 Primary Phytochemical screening of Methanolic Extract[201-207]

3.3.1 Test for Terpenes

2 ml of Chloroform and 3 ml of concentrated H2SO4 were added to 5 ml of the Methanol Extract. Reddish brown ring formation confirms the presence of terpenes.

3.3.2 Test for Flavonoids

To a small amount of the Methanol Extract a few drops of concentrated hydrochloric acid were added. Immediate development of a red colour indicate the presence of flavonoids.

3.3.3 Test for Saponins

Frothing test: 0.5 g of the Methanol Extract was dissolved in distilled water in a test tube. Frothing which persisted on warming indicate the presence of saponins.

3.3.4 Test for Steroids

Liebermann–Burchard reaction: To 5 ml of the Methanol Extract 2 ml of acetic anhydride and 2 ml Concentrated H2SO4 were added. Color change from violet to blue confirms the presence of steroids.

3.3.5 Test for Tannins

10 ml of bromine water was added to the 0.5 g of Methanol Extract. Decoloration of bromine water showed the presence of tannins.

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3.3.6 Test for cardiac glycosides

To 2 ml of Methanol Extract 2 ml of glacial acetic acid containing 1 drop of ferric chloride solution and 1 ml of conc. H2SO4 were added. Appearance of a brown ring indicates the presence of cardiac glycosides.

3.3.7 Test for proteins

Biuret test: To 3 ml of the Methanol Extract 4% of NaOH and few drops of 1% CuSO4 solution were added. Formation of violet or pink color shows the presence of proteins.

3.3.8 Test for carbohydrates: Monosaccharide.

Barfoed's test: Equal volumes of Barfoed's reagent and the Methanol Extract solution were mixed and heated for 1–2 min in a boiling water bath and then cooled. Red color indicate presence of monosaccharide.

3.3.9 Test for reducing sugars

Fehling test: 1 ml of Fehling's A and Fehling's B solutions was boiled with equal volume of Methanol Extract for 5–10 min. First a yellow followed by a brick red precipitates showed presence of reducing sugars.

Molisch test: To 2–3 ml of the Methanol Extract, two drops of alpha napthol solution in alcohol were added and then it was shaken. Concentrated H2SO4 from the sides of test tube was added. Formation of Violet ring indicate presence of reducing sugars.

3.3.10 Test for alkaloids

Methanol Extract was warmed with 1% HCl for 2 minutes. This mixture was filtered and few of Dragendorff's reagents were added. A reddish-brown colour and turbidity indicated the presence of alkaloids.

IV. DISCUSSION

Liver is one of the most versatile and biologically active organs. Its key functions include: a) Filtration and storage of blood; b) Metabolism of carbohydrates, lipids, proteins and hormones, synthesis of albumin and coagulation factors; c) Detoxification and biotransformation of foreign chemicals; and d) Excretion of protein-bound/lipid-soluble waste products.

Hepatic fibrosis is characterized by the deposition of excess extracellular matrix (ECM). It is a wound-healing response to chronic or repeated liver injury which may be due to biological factors or chemical drugs such as carbon tetrachloride and Thioacetamide (TAA). Persistent fibrosis can lead to liver cirrhosis and hepatocellular carcinomas. Hence early diagnosis and prevention of hepatic fibrosis can effectively hinder liver cirrhosis and its complications. Despite tremendous advancements in modern medicine, very few drugs are available for the treatment of liver disorders. Therefore, developing new drugs with hepatoprotective effects has become more important and urgent.

Hepatic encephalopathy (HE) is a clinical condition with several types regarding chronicity and clinical diversity. HE can develop as a complication of both acute and chronic liver failure. The wide spectrum of the clinical presentations add to the complexity of HE. Encephalopathy is one of the hallmark symptom in patients suffering from acute liver insufficiency and may progress from altered mental status to coma within few days. It has very high rate of mortality. Supportive care until spontaneous recovery is the only treatment but does not occur in many patients. To prevent death, liver transplantation is the only effective option available, necessitating the preventive therapy.

Hepatorenal syndrome (HRS) is a multiorgan condition affecting both the kidneys and the liver. It is a cause of acute kidney injury that can be seen in patients with acute or chronic liver disease. Oxidative stress and the generation of reactive oxygen and nitrogen species in the brain has been associated with HRS. Studies suggest that impaired mitochondrial function and down regulation of the expression of key antioxidation enzymes contributes to an increased oxidative damage to membrane lipids, protein, and DNA.

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Recent studies have highlighted the importance of oxidative damage in the pathogenesis of Hepatic encephalopathy and Hepatorenal syndrome, with various experimental treatments aimed at decreasing reactive oxygen/nitrogen species or restoring the activity of anti-oxidative enzymes such as catalase , superoxide dismutase, thioredoxin, or glutathione peroxidase.

Reactive oxygen and nitrogen species (ROS and RNS) are produced by metabolism of normal cells. However, in liver diseases, redox is increased and thereby damaging the hepatic tissue; ROS/RNS can activate hepatic stellate cells, characterized by the increased production of extracellular matrix and accelerated proliferation. Cross-talk between parenchymal and nonparenchymal cells is one of the most important events in liver injury and fibrogenesis; ROS play an important role in fibrogenesis by increasing platelet-derived growth factor. Most of the hepatocellular carcinomas occur in cirrhotic livers, and the common mechanism for hepatocarcinogenesis is thought to be chronic inflammation associated with severe oxidative stress, while other risk factors include dietary aflatoxin B1 consumption, cigarette smoking, and heavy drinking. Ischemia–reperfusion injury affects directly on hepatocyte viability, more particularly during transplantation and hepatic surgery; ischemia can activate Kupffer cells which are the main source of ROS during the reperfusion period. The toxic mechanism of action of paracetamol is through metabolic activation of the drug, depletion of glutathione, and covalent binding of the reactive metabolite N-acetyl-pbenzoquinone imine to cellular proteins as the main cause of hepatic cell death; intracellular steps critical for cell death include mitochondrial dysfunction, formation of ROS and peroxynitrite. Infection with hepatitis C is associated with increased levels of ROS/RNS and reduced antioxidant levels. So antioxidants have been proposed as an adjunct therapy for various liver diseases.

The use of natural remedies for the treatment of liver diseases has a long history and medicinal plants and their derivatives are still used all over the world in different form for this purpose. Liver protective plants are reported to contain a various phytochemicals like phenols, coumarins, monoterpenes, glycosides, alkaloids and xanthenes. Flavonoids and phenolic compounds widely distributed in plants, have shown multiple biological effects, including antioxidant and free radical scavenging abilities. Due to very few reliable liver-protective drugs in modern medicine, a large number of medicinal preparations are recommended for the treatment of liver diseases and quite often claimed to offer significant relief. Attempts are being made globally to get scientific evidences for these traditionally reported herbal drugs.

Bambusa arundinaceae is used in folk medicine as tonic for heart, liver and brain. It is also one of the important constituent of many unani and ayurvedic formulation like Qursghafis, Sufuftabashir, Drakshadichurna used in liver ailments.[35,36] Bambusa arundinaceae contains various phytochemicals like resin, lignin, alkaloids, glucoside, silica, uronic acid, galactose, glucose, arabinose, mannose, xylose, Choline, betain. It also reported to contain urease, nuclease, proteolytic enzymes, diastatic and emulsyfying enzyme.[37,38] Presence of flavanoids like orientin, homoorientin, vitexin and isovitexin are reported along with phytosterols like stigmasterol and β-sitosterol,

Stigmast-5, 22-dien-3β-ol, Stigmast-5-en- 3β-ol-β-D glucopyranoside, triterpenes and steroidal glycosides, 17, 20, 20-tri demethyl-20α-isopranyloleanane, eicosanyldicarboxylic acid, α-amyrin acetate, urs-12-en-3β-ol-β-D-glucopyranoside.[39-41]

Thioacetamide (TAA), a well-known hepatotoxin, which can disrupt liver metabolism and cause hepatocellular necrosis. TAA has negative effects on DNA, RNA, and protein synthesis of enzymes in liver cells, prolongs the cell mitosis process and can obstructs the transfer of RNA from the nucleus to the cytoplasm. The TAA-induced hepatic fibrosis injury model in rats is most similar to the pathology in humans, and also to the pathology of liver cirrhosis caused by virus. TAA-induced hepatic cirrhosis has been observed to be associated with reduction of antioxidants and enhancement of lipid peroxidation. Studies have shown that reducing oxidative stress play a positive role in preventing or reversing hepatic fibrosis. Hence hydroxyl radical scavengers like selenium, curcumin, and taurine have been applied in the treatment of TAA-induced liver cirrhosis. Hence in present study Thioacetamide was selected to induce liver cirrhosis in rats to study the protective effect of Bambusa arundinacea in liver toxicity and other associated pathology complications viz hepatic encephalopathy and hepatorenal syndrome.

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V. CONCLUSION

Methanolic extract, Ethylacetate fraction and S.F.1 showed good antioxidant, invivo and invitro protective effect against thioacetamide induced liver toxicity. They also improved liver toxicity associated pathology complications viz.. hepatic encephalopathy and hepatorenal syndrome. Preliminary screening showed that Phytochemicals like terpenes, flavanoids, saponins, steroids, proteins, carbohydrates, tannins and phenolic compounds were present in methanolic extract. Quantitative analysis of Methanolic extract, Ethylacetate fraction and S.F.1 extract showed presence of significant amount of flavanoids. Studies have shown that flavanoids possess good antioxidant and hepatoprotective activity. So the protective effect of Bambusa arundinacea aagaint thioacetmide induced liver toxicity and pathology complications may be due to presence of phytochemicals like flavanoids. Collectively, the current study reveals hepatoprotective role of Bambusa arundinacea. Flavonoid is key anti-oxidant component of Bambusa arundinacea. It decreases hepatic cells death rate and chemical induced damage in rat liver by protecting against oxidative stress. Other than this, flavonoids also attenuates hepatitis induced secondary complications such as hepatic encephalopathy and hepatorenal syndrome. The young shoots of Bambusa arundinacea are a source of novel nutraceuticals.

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