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MEDICINAL PLANTS USED IN HEPATOPROTECTIVE: A REVIEW

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ABSTRACT: Liver is a vital organ play a major role in the metabolism and excretion of xenobiotics from the body. Liver injury or liver dysfunction is a major health problem that challenges not only health care professionals but also the pharmaceutical industry and drug regulatory agencies. Modern medicine has provided us many drugs that alleviate liver diseases but compared to it herbal medicine; herbal is preferred because they are cost-effective and considered to be a safe approach for treatment with minimal side effects. Herbal medicines have been used in the treatment of liver diseases for a long time. Several herbal preparations are available in the market. Many herbs have been proven to be efficient as hepatoprotective agents while many more are claimed to be hepatoprotective but be deficient in any such scientific substantiation to support such claims. Clinical research in this century has confirmed the efficacy of several plants in the treatment of liver disease. Hence, this review article contributes to the knowledge of reported indigenous plants, which are prevalent for the prevention and treatment of liver disorders.

Keywords: Liver, Metabolism, Hepatoprotective, Herbal medicines

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INTRODUCTION: The liver is a vital organ of vertebrates and some other animals.¹ Terminology related to the liver often starts in *hepar-* or *hepat-* from the Greek word for liver, *hēpar*^{2,3}. In human beings, it is located in the upper right quadrant of the abdomen, below the diaphragm. It is a reddish brown wedge-shaped organ with four lobes of unequal size and shape. A human liver normally weighs 1.44–1.66 kg (3.2–3.7 lb)⁴. It is both the largest internal organ and the largest gland in the human body. The gallbladder, a small pouch that sits just under the liver, stores bile produced by the liver⁵.

The falciform ligament, visible on the front of the liver, divides the liver into a left and a much larger right lobe. From the visceral surface, the two additional lobes are located between the right and left lobes, one in front of the other. A line can be imagined running from the left of the vena cava and all the way forward to divide the liver and gallbladder into two halves⁶. This line is called Cantlie's line.

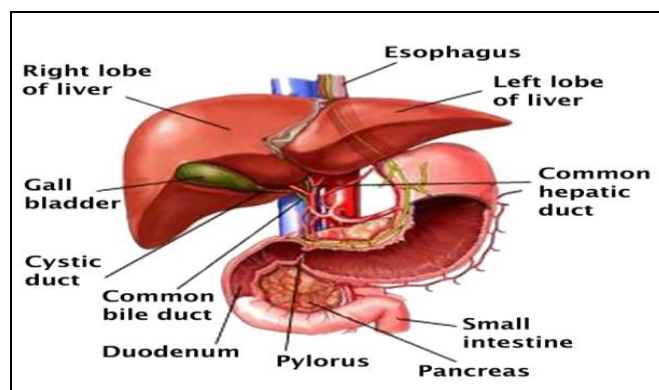


FIG. 1: HUMAN LIVER WITH OTHER ORGANS

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Gross anatomy traditionally divided the liver into two portions— a right and a left lobe, as viewed from the front (diaphragmatic) surface; but the

underside (the visceral surface) shows it to be divided into four lobes and includes the caudate and quadrate lobes⁷.

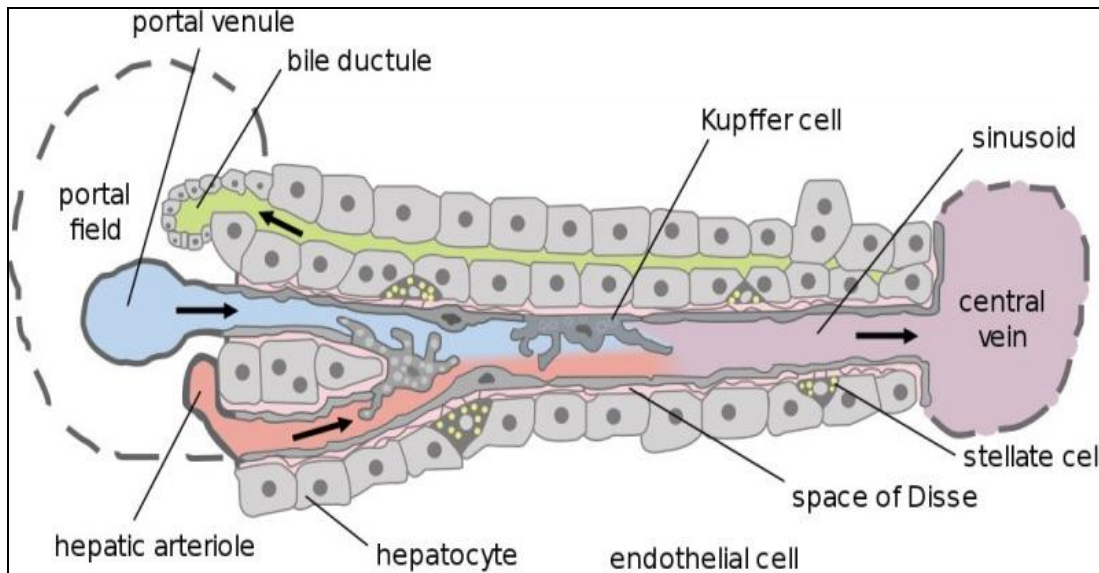


FIG. 2: CELL, DUCTS AND BLOOD VESSELS

The study of microscopic anatomy shows two major types of liver cell: parenchymal cells and non-parenchymal cells. 70–85% of the liver volume is occupied by parenchymal hepatocytes. Non-parenchymal cells constitute 40% of the total number of liver cells but only 6.5% of its volume⁸. The liver sinusoids are lined with two types of cell, sinusoidal endothelial cells, and phagocytic Kupffer cells⁹. Hepatic stellate cells are non-parenchymal cells found in the space of Disse, between a sinusoid and a hepatocyte⁸. Additionally, intrahepatic lymphocytes are often present in the sinusoidal lumen⁸.

The visceral surface or inferior surface is uneven and concave. It is covered in peritoneum apart from where it attaches the gallbladder and the porta hepatis¹⁰. Microscopically, each liver lobe is seen to be made up of hepatic lobules. The lobules are roughly hexagonal and consist of plates of hepatocytes radiating from a central vein¹¹. The central vein joins the hepatic vein to carry blood out from the liver. A distinctive component of a lobule is the portal triad, which can be found running along each of the lobule's corners. The portal triad, misleadingly named, consists of five structures: a branch of the hepatic artery, a branch of the hepatic portal vein, and a bile duct, as well as lymphatic vessels and a branch of the vagus nerve¹². Between the hepatocyte plates are liver

sinusoids, which are enlarged capillaries through which blood from the hepatic portal vein and hepatic artery enter via the portal triads, then drains to the central vein¹³. The lobules are held together by fine areolar tissue which extends into the structure of the liver, by accompanying the vessels (veins and arteries) ducts and nerves through the hepatic portal, as a fibrous capsule called Glisson's capsule¹³. The whole surface of the liver is covered in a serious coat derived from the peritoneum and this has an inner fibrous coat (Glisson's capsule) to which it has firmly adhered. The fibrous coat is of areolar tissue and follows the vessels and ducts to support them.

The liver is the only human internal organ capable of natural regeneration of lost tissue; as little as 25% of a liver can regenerate into a whole liver¹⁴. This is, however, not true regeneration but rather compensatory growth in mammals¹⁵. The lobes that are removed do not regrow, and the growth of the liver is a restoration of function, not original form. This contrasts with true regeneration where both original function and form are restored. In some other species, such as fish, the liver undergoes true regeneration by and size of the organ¹⁶. In the liver, large areas of the tissues are formed, but for the formation of new cells, there must be a sufficient amount of material, so the circulation of the blood becomes more active¹⁷.

The liver has a wide range of functions, including detoxification of various metabolites, protein synthesis, and the production of biochemicals necessary for digestion¹⁸. The liver is a gland and plays a major role in metabolism with numerous functions in the human body, including regulation of glycogen storage, decomposition of red blood cells, plasma protein synthesis, hormone production, and detoxification¹⁸. It is an accessory digestive gland and produces bile, an alkaline compound which aids in digestion via the emulsification of lipids

The liver's highly specialized tissue consisting of mostly hepatocytes regulates a wide variety of high-volume biochemical reactions, including the synthesis and breakdown of small and complex molecules, many of which are necessary for normal vital functions¹⁹. Estimates regarding the organ's total number of functions vary, but textbooks generally cite it being around 500²⁰.

The liver supports almost every organ in the body and is vital for survival. Because of its strategic location and multidimensional functions, the liver is also prone to many diseases²¹. The bare area of the liver is a site that is vulnerable to the passing of infection from the abdominal cavity to the thoracic cavity. The classic symptoms of liver damage include the following:

- Pale stools occur when stercobilin, a brown pigment, is absent from the stool. Stercobilin is derived from bilirubin metabolites produced in the liver.
- Dark urine occurs when bilirubin mixes with urine
- Jaundice (yellow skin and whites of the eyes) this is where bilirubin deposits in the skin, causing an intense itch. Itching is the most common complaint by people who have liver failure. Often this itch cannot be relieved by drugs.
- Swelling of the abdomen, ankles, and feet occurs because the liver fails to make albumin.
- Excessive fatigue occurs from a generalized loss of nutrients, minerals, and vitamins.

- Bruising and easy bleeding are other features of liver disease. The liver makes substances which help prevent bleeding. When liver damage occurs, these substances are no longer present, and severe bleeding can occur²².
- Pain in the upper right quadrant can result from the stretching of Glisson's capsule in conditions of hepatitis and pre-eclampsia.



FIG. 3: EVALUATION OF LIVER DISEASE

Few of the liver diseases are as follows:

Alcohol and the Liver: Alcohol affects everyone. When a person has a drink, the alcohol is absorbed directly through the wall of the stomach and intestine into the bloodstream, where it is distributed rapidly throughout the body. The alcohol changes the function of each cell that it enters. The liver processes everything a person ingests, including alcohol. Only a certain quantity of alcohol can be detoxified over some time. In the meantime, excess alcohol affects the brain, heart, muscles, and other tissues of the body. When the liver has too much alcohol to handle, normal liver function may be interrupted, leading to a chemical imbalance.

If the liver is required to detoxify alcohol continuously, liver cells may be destroyed or altered resulting in fat deposits (fatty liver), and more seriously, either inflammation (alcoholic hepatitis), and/or permanent scarring (cirrhosis), moreover, liver cancer can also result from alcohol-induced liver disease and including hepatitis C. Therefore, anyone with hepatitis C, or any other form of liver disease, should not drink alcohol. Symptoms and complications arising from liver damage include fatigue, loss of appetite, lowered resistance to infections, jaundice (yellowing of the skin and eyes), swelling of the abdomen, internal bleeding, confusion, and kidney failure.

Cirrhosis: Cirrhosis is a condition that results from permanent damage or scarring of the liver. This leads to a blockage of blood flow through the liver and prevents normal metabolic and regulatory processes.

Blockage of the normal flow of blood through the liver leads to swelling of the liver and potentially the spleen. Blood from the intestines is then forced to find a new way around the liver through new vessels. Some of these new blood vessels called "varices" which form primarily in the stomach and esophagus become quite large. These variances may rupture due to high blood pressure (portal hypertension) and thin vessel walls, causing bleeding in the upper stomach or esophagus.

Individuals with cirrhosis may bleed and bruise easily due to a decrease in proteins required for blood clotting.

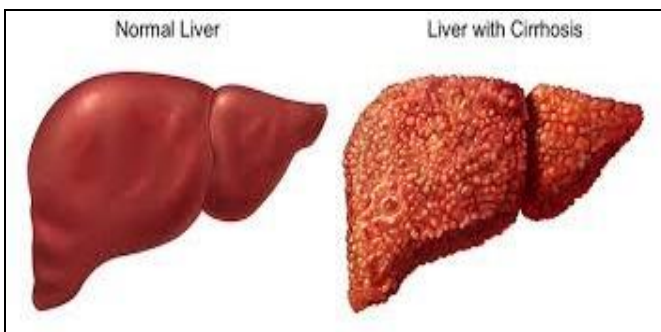


FIG. 4: HEALTHY LIVER AND LIVER WITH CIRRHOSIS

The major causes of cirrhosis are as follows:

- Chronic alcoholism
- Viral infections caused by chronic viral hepatitis (types B, C, and D)
- Metabolic diseases such as alpha-1-antitrypsin deficiency, galactosemia, and glycogen storage disorders
- Inherited diseases such as Wilson disease and hemochromatosis
- Biliary cirrhosis resulting from diseases such as primary biliary cirrhosis (PBC) and primary sclerosing cholangitis (PSC)
- Toxic hepatitis caused by severe reactions to prescribed drugs or prolonged exposure to environmental toxins

- Repeated bouts of heart failure with liver congestion.

People in the early stages of cirrhosis have few symptoms. Some symptoms an individual may notice include:

- Loss of appetite
- Nausea
- Weight loss
- Fatigue
- Weakness
- Exhaustion.

Gallstones: Gallstones are solid lumps of cholesterol crystals or pigment material that form in the gallbladder.

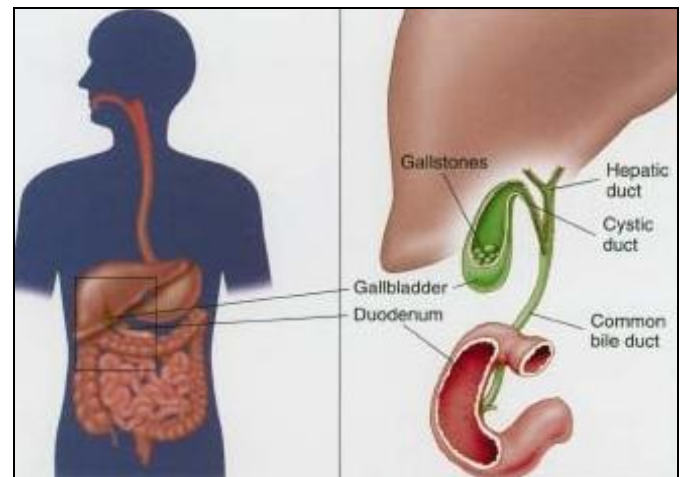


FIG. 5: GALLSTONE

They are formed when some fatty components (such as cholesterol) are not easily dissolved in bile. When there are too much of these components in bile, they precipitate and form solid crystals. This clump together forming gallstones - also known as cholelithiasis. There are different types of gallstones, depending on which component of the bile has solidified. Also, the stones can vary in size ranging from tiny, sand-like particles less than one millimeter in diameter to more than four centimeters in diameter. Patients with symptomatic gallstones experience severe abdominal pain and may suffer further complications such as jaundice (yellowing of the skin and eyes) and inflammation of the gallbladder, bile ducts, liver or pancreas.

However, about 80 percent of people who have gallstones have no symptoms. These people are said to have so-called "silent" gallstones with no associated pain. Gas and indigestion are not specific symptoms of gallbladder or gallstone disease.

Silent gallstones are detected incidentally during the investigation of another problem; Gallstones are usually diagnosed by ultrasound. Other procedures, such as x-rays, may also be used.

Hemochromatosis: It is an inherited disease in which the body absorbs too much iron from the diet. The liver is the first organ to store iron and when its storage capacity is exhausted, the iron continues to accumulate in the heart, the pituitary gland and elsewhere in the body.

Normally, only enough iron to meet the body's daily requirements is absorbed; the remainder is usually excreted through the bowels. In hemochromatosis, however, iron continues to be absorbed and stored in different organs and tissues long after body needs are met. If untreated, damage to the liver, heart, and pancreas may eventually lead to death. Hemochromatosis is hereditary. The liver begins to retain iron at birth, but it may take 20 to 30 years before symptoms manifest themselves. In the early stages of hemochromatosis, there may be no signs, but when symptoms begin to appear, they include:

- Fatigue
- Swelling in the joints (arthritis), particularly in the knuckles of the middle and index fingers

In the later stages of the disease, symptoms include:

- Abdominal pain or tenderness
- Yellowing of the skin and eyes (jaundice)
- Distention of the abdomen
- Bleeding from dilated veins in the esophagus development of diabetes
- The skin may have a bronze or grey color
- Excessive hunger and thirst

- Frequent urination.

Liver Cancer: Many factors may play a role in the development of cancer. Because the liver filters blood from all parts of the body, cancer cells from elsewhere can lodge in the liver and start to grow. Cancers that begin in the gut often spread to the liver. The ability of the liver to regenerate may also be linked to the development of liver cancers. There are many types of liver tumors, only some of which are cancers. The most important classification is whether the tumours are benign (relatively harmless) or malignant (capable of spreading from the liver and thus more serious).

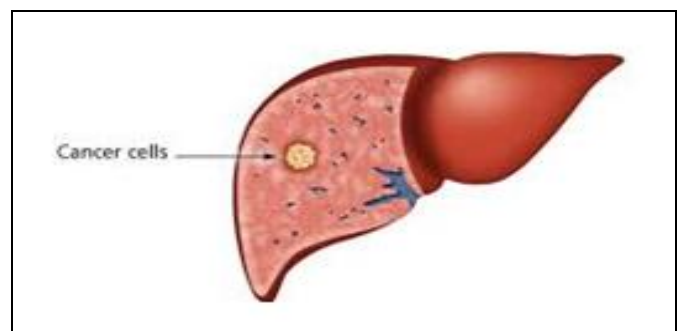


FIG. 6: LIVER CANCER

Benign Tumours, Hemangioma is the most common type of benign liver tumor. It is an abnormal growth of blood vessels of the liver that begins in the fetus. More than 10% of the normal population has hemangiomas in the liver. Most people with hemangiomas have no symptoms and require no treatment. Some hemangiomas may rarely enlarge and bleed, in which case they may require surgical removal. Hepatic adenomas are benign tumors of liver cells. Most do not cause symptoms and do not require treatment. However, if they are large, they may cause pain or blood loss and may need to be removed. Hepatic adenomas occur more frequently in women and seem to be triggered in some cases by the birth control pill or by pregnancy. Focal nodular hyperplasia (FNH) is a tumor-like growth of several cell types. Although FNH tumors are benign, it can be hard to distinguish them from liver cancers.

Malignant Tumours, the most common form of primary liver cancer (cancer that starts in the liver) in adults is called hepatocellular carcinoma (HCC). It is a cancer of liver cells. This type of cancer can have different growth patterns. Some begin as a single tumor that grows larger. It may spread to

other parts of the liver in the later stages of the disease.

Liver cancer may also develop in more than one site in the liver and may grow into multiple tumors. This pattern is most often seen in people with liver cirrhosis.

Another liver cancer is called cholangiocarcinoma. It originates in the small bile ducts which are tubes that carry bile to the gall bladder.

Most often, however, when cancer occurs in the liver, it did not start there but spread to the liver from cancer that began somewhere else in the body. These types of cancers are named after the place where they began (primary site) and are considered secondary liver cancers or cancer metastases. For example, cancer that started in the lung and spread to the liver is called metastatic lung cancer with spread to the liver. Secondary liver cancers are 30 times more prevalent than primary liver cancers.

In the early stages, liver cancer does not cause symptoms. Some common symptoms of advanced liver cancer include:

- Weight loss
- Loss of appetite
- Abdominal pain
- Jaundice
- Fluid in the abdomen.

Alagille Syndrome: It is an inherited disorder that mimics other forms of prolonged liver disease seen in infants and young children. Specifically, Alagille syndrome is also associated with cardiac disease, eye, and skeletal findings, and a characteristic facial appearance. Alagille syndrome is caused by changes, or mutations, in one of two genes, usually JAGGED1 or occasionally NOTCH2. Each affected adult or child may have all or only a few of the features of the syndrome.

Typically, symptoms of the illness are jaundice and poor growth within the first three months of life. Later, there is persistent jaundice, severe itching, fatty deposits in the skin (xanthomas), and poor

growth during early childhood. Frequently the disease stabilizes between ages five and eight with an improvement in symptoms. There is also a specific facial appearance shared by children with Alagille syndrome that makes them easily recognizable. The features include a prominent, broad forehead, deep-set eyes, a straight nose, and a small pointed chin. The majority of children with Alagille syndrome have an abnormality of the eyes in which an extra, circular line on the surface of the eye can be detected by a specialized eye examination. However, it does not lead to any vision problems.

Autoimmune Hepatitis: It is a disease characterized by chronic inflammation of the liver. Autoimmune refers to the body's immune system attacking another part of the body. The exact mechanism whereby the body's immune system attacks the liver is not yet known. It appears that certain types of white blood cells (the type of blood cell that usually fights infection), in addition to attacking foreign substances (e.g., germs and viruses) misread liver cells as foreign substances and start attacking these cells. The type of damage that follows is known as chronic hepatitis. Several other conditions can cause identical patterns of liver damage.

These include viruses such as hepatitis B and hepatitis C, certain types of drugs, and overload of certain metals such as copper and iron in the liver. In many patients, there will be no symptoms at all. The patient will feel perfectly healthy. The condition may be detected on a routine blood test by an elevation in levels of certain enzymes that the liver makes. Other patients may experience fatigue, decreased appetite, drowsiness, or even aches or pains in the muscles or joints. Some patients may notice jaundice or yellowish discoloration of the skin and whites of the eyes as the first symptom.

Biliary Atresia: In this, the bile duct that leads from the liver to the intestine becomes damaged, preventing bile from leaving the liver. In the early stages, the bile duct outside the liver is mainly affected, but in later stages, bile ducts inside the liver are also damaged. This can lead to building up of bile in the liver, which can be harmful to the liver. Unless bile flow can be established, liver function is gradually lost and affected children

rarely survive beyond two years of age. It is a relatively rare disease that begins in early infancy.

Bile is a yellow-green fluid made in the liver and stored in the gallbladder. It helps with digestion and absorption of dietary fats and fat-soluble vitamins. It is also necessary for removing waste products from metabolism and toxins from the body.

The cause of biliary atresia is not known. It is not contagious, and it cannot be passed from one child to another. Biliary atresia is usually diagnosed during the first two months of life. Common signs are:

- Newborn jaundice that does not improve by two weeks of age, or jaundice that appears after two weeks of age
- Dark urine and pale, clay-colored stools
- Enlarged liver or swollen abdomen.

Galactosemia: This disease is caused by elevated levels of galactose (a sugar in milk) in the blood resulting from a deficiency of the liver enzyme required for its metabolism (breakdown). It is a rare hereditary disease that can lead to cirrhosis in infants, and early, devastating illness if not diagnosed quickly. The disease usually appears in the first few days of life following the ingestion of breast milk or formula. Vomiting, liver enlargement, and jaundice are often the earliest signs of the disease, but bacterial infections (often severe), irritability, failure to gain weight, and diarrhea may also occur. If unrecognized in the newborn period, the disease may produce liver, brain, eye, and kidney damage.

Reye's Syndrome: It is a rare complication of common childhood respiratory infections, including chickenpox. Reye's syndrome is most common in school-aged children and teenagers, but cases also occur in infants. The illness is rare in adults.

Reye's syndrome should be suspected when vomiting begins three to seven days after the onset of the flu or chickenpox. Usually, the vomiting becomes increasingly severe over eight to 12 hours. When vomiting persists for more than 12 hours, or vomiting is associated with signs of brain disorder,

such as staring spells, stupor, delirium, or strange behavior, call your doctor. In the later stage of the disease, the child shows personality changes such as aggressive behavior and disorientation. Other symptoms may include confusion, slurred speech, agitated delirium with screaming and struggling, and an inability to recognize parents. This stage is a medical emergency.

Although Reye's syndrome can occur anytime, it is most frequent in January, February, and March, in association with influenza and similar respiratory infections. About one-third of the cases of Reye's syndrome occurs as a complication of chickenpox, usually three or four days after the rash appears.

Vomiting on the first day of illness, especially when accompanied by diarrhea, is not typically due to Reye's syndrome. In such cases, children are usually alert and active. They may be suffering from acute infectious gastroenteritis.

Above are few of the popular and known diseases which affect the liver. Other than these some hepatotoxins majorly damage liver. Hepatotoxicity, in most cases is due to free radical. Free radicals generated by the metabolism of toxicants initiate the toxicity cascade²³. Hepatotoxins like Carbon tetrachloride, Paracetamol, D-Galactosamine, and Thioacetamide.

Carbon Tetrachloride: The hepatotoxicity of CCl₄ is due to the formation of the highly reactive trichloromethyl free radical in the body which attacks the polyunsaturated fatty acids of the membrane of the endoplasmic reticulum. Carbon tetrachloride poisoning leads rapidly to the cessation of movement of large quantities of triglycerides from the liver to the plasma leading to fatty liver²⁴. If the damage is severe, it leads to an abnormal increase in liver enzymes followed by hepatocellular necrosis.

Paracetamol: Paracetamol is metabolically activated by cytochrome P450 to a reactive metabolite that covalently binds to protein²⁵. The reactive metabolite responsible for hepatotoxicity is N-acetyl-p-benzoquinoneimine, which reacts with N-acetyl cysteine²⁶. Although considered safe at therapeutic doses, in overdose, it produces centrilobular hepatic necrosis that can be fatal²⁷.

Various mechanisms leading to paracetamol toxicity includes

- a. Increased formation of superoxide anions which cause lipid peroxidation (oxidative stress) via hydrogen peroxide formation²⁸.
- b. Decreased glutathione concentrations in centrilobular cells²⁹.

D-galactosamine: Galactosamine administration induces an inflammatory response in the liver that biochemically and histologically resembles viral hepatitis³⁰. A single administration causes hepatocellular necrosis and fatty liver³¹. It causes the appearance of specific lesions in liver cells, characterized by inhibition of nuclear RNA and protein synthesis³².

Thioacetamide: Thioacetamide, originally used as a fungicide is a potent hepatotoxic and is bioactivated by CYP450 and flavin-containing monooxygenase (FMO) systems to sulfinic (sulfoxide) and sulfenic (sulfone) metabolites, which causes centrilobular necrosis^{33, 34}. This metabolite causes liver fibrosis. Thioacetamide interferes with the movement of RNA from the nucleus to cytoplasm, which may cause membrane injury³⁵.

Therefore, maintenance of a healthy liver is essential for the overall well being of an individual³⁶. Modern medicines have a little to offer for the alleviation of hepatic diseases, and it is chiefly the plant-based preparations which are employed for their treatment of liver disorders³⁷.

Herbal medicines are the most lucrative form of traditional medicine on which about 80% of the population depends (WHO traditional medicine facts sheet no 134. Dec 2008).

Herbal drugs have gained importance and popularity in recent years because of their safety, efficacy, and cost-effectiveness. The association of medical plants with other plants in their habitat also influences their medicinal values in some cases.

One of the important and well-documented uses of plant products is their use as hepatoprotective agents. Hence, there is an ever-increasing need for safe hepatoprotective agent³⁸. It has been reported that about 160 phytoconstituents from 101 medicinal plants have hepatoprotective activity³⁹.

In this review, majorly hepatoprotective activity contained plant names are detailed

TABLE 1: HEPATOPROTECTIVE ACTIVITY CONTAINED PLANTS

Plants	Family	Part used
<i>Casuarina equisetifolia</i> ⁴⁰	Casuarinaceae	Leaf and Bark
<i>Cajanus cajan</i> ⁴⁰	Papilionaceae	Whole plant
<i>Glycosmis pentaphylla</i> ⁴⁰	Rutaceae	Leaf, Bark
<i>Bixa orellana</i> ⁴⁰	Bixaceae	Seed
<i>Physalis minima</i> ⁴⁰	Solanaceae	Whole plant
<i>Argemone Mexicana</i> ⁴⁰	Papaveraceae	Leaf and flower
<i>Caesalpinia bonduc</i> ⁴⁰	Caesalpiniaceae	Leaf and bark
<i>Carthamus tinctorius</i> ⁴¹	Compositae	Flower
<i>Ardisia solanacea</i> ⁴²	Myrsinaceae	Leaves
<i>Delonix regia</i> ⁴³	Caesalpiniaceae	Aerial parts
<i>Aphanamixis polystachya</i> ⁴⁴	Meliaceae	Leaves
<i>Solanum pubescens</i> ⁴⁵	Solanaceae	Whole plant
<i>Coriandrum sativum</i> ⁴⁶	Apiaceae	Whole plant
<i>Plumbago zeylanica</i> ⁴⁷	Plumbaginaceae	Aerial parts
<i>Cardiospermum helicacabum</i> ⁴⁸	Sapindaceae	Stem
<i>Luffa acutangula</i> ⁴⁹	Cucurbitaceae	Leaves
<i>Epilobium divaricatum</i> ⁵⁰	Compositae	Whole plant
<i>Tagetes erecta</i> ⁵¹	Asteraceae	Flower branches
<i>Ziziphus rotundifolia</i> ⁵²	Rhamnaceae	Leaves
<i>Millettia aboensis</i> ⁵³	Fabaceae	Roots
<i>Ficus carica</i> ⁵⁴	Moraceae	Leaves

<i>Morus alba</i> ⁵⁵	Moraceae	Leaves
<i>Alchornea cordifolia</i> ⁵⁶	Euphorbiaceae	Leaves
<i>Leucophyllum frutescens</i> ⁵⁷	Scrophulariaceae	Aerial parts
<i>Carissa carandas</i> ⁵⁸	Apocynaceae	Roots
<i>Sesamum indicum</i> ⁵⁹	Pedaliaceae	Seeds
<i>Flacourtia indica</i> ⁶⁰	Flacourtiaceae	Leaves
<i>Hippophae rhamnoides</i> ⁶¹	Elaeagnaceae	Leaves
<i>Apium graveolens</i> ⁶²	Apiaceae	Seeds
<i>Croton oblongifolius</i> ⁶²	Euphorbiaceae	Whole plant
<i>Hypericum japonicum</i> ⁶³	Hypericaceae	Whole plant
<i>Cinnamomum zeylanicum</i> ⁶⁴	Lauraceae	Bark
<i>Launea intybacea</i> ⁶⁵	Asteraceae	Aerial parts
<i>Mimosa pudica</i> ⁶⁶	Mimosaceae	Leaves
<i>Polygala javana</i> ⁶⁷	Polygalaceae	Whole plant
<i>Marsilea minuta</i> ⁶⁸	Marsileaceae	Whole plant
<i>Ficus bengalensis</i> ⁶⁹	Moraceae	Leaves
<i>Chenopodium album</i> ⁷⁰	Chenopodiaceae	Aerial parts
<i>Psidium guajava</i> ⁷¹	Myrtaceae	Leaves
<i>Rhododendron arboretum</i> ⁷²	Ericaceae	Leaves
<i>Diteracanthus patulus</i> ⁷³	Acanthaceae	Leaves
<i>Cuscuta reflexa</i> ⁷⁴	Cuscutaceae	Aerial parts
<i>Crassocephalum crepidioides</i> ⁷⁵	Asteraceae	Whole plant
<i>Glycyrrhiza glabra</i> ⁷⁶	Fabaceae	Roots
<i>Gundelia tourenfortii</i> ⁷⁷	Asteraceae	Footstalks
<i>Coptidis rhizome</i> ⁷⁸	Ranunculaceae	Whole plant
<i>Carica papaya</i> ⁷⁹	Caricaceae	Seeds
<i>Cichorium intybus</i> ⁸⁰	Asteraceae	Leaves
<i>Scoparia dulcis</i> ⁸¹	Scrophulariaceae	Whole plant
<i>Indigofera tinctoria</i> ⁸²	Leguminosae (Papilionatae)	Whole plant
<i>Solanum trilobactum</i> ⁸³	Solanaceae	Whole plant
<i>Pterocarpus marsupium</i> ⁸⁴	Papilionaceae	Stem bark
<i>Pterocarpus santalinus</i> ⁸⁵	Fabaceae	Stem bark
<i>Curculigo orchoides</i> ⁸⁶	Amaryllidaceae	Rhizomes
<i>Phoenix dactylifera</i> ⁸⁷	Palmae	Fruit
<i>Asteracantha longifolia</i> ⁸⁸	Acanthaceae	Whole plant
<i>Strychnos potatorum</i> ⁸⁹	Longaniaceae	Seeds
<i>Vitex trifolia</i> ⁹⁰	Verbenaceae	Leaves
<i>Capparis spinosa</i> ⁹¹	Capparidaceae	Root bark
<i>Lawsonia alba</i> ⁹²	Lythraceae	Bark
<i>Carissa opaca</i> ⁹³	Apocynaceae	Leaves
<i>Azima tetracantha</i> ⁹⁴	Salvadoraceae	Leaves
<i>Dragea volubilis</i> ⁹⁵	Asclepiadaceae	Leaves
<i>Coccinia indica</i> ⁹⁶	Curcubitaceae	Fruits
<i>Sida rhombifolia</i> ⁹⁷	Malvaceae	Whole plant
<i>Tabebuia rosea</i> ⁹⁸	Bignoniaceae	Leaves
<i>Ichnocarpus frutescens</i> ⁹⁹	Apocynaceae	Whole plant
<i>Vanilla planifolia</i> ¹⁰⁰	Orchidaceae	Beans
<i>Nilgiranthus ciliates</i> ¹⁰¹	Acanthaceae	Bark
<i>Phyllanthus amarus</i> ¹⁰²	Phyllanthaceae	Leaves
<i>Aegle marmelos</i> ¹⁰²	Rutaceae	Leaves
<i>Aloe vera</i> ¹⁰²	Xanthorrhoeaceae	Leaves
<i>Eclipta alba</i> ¹⁰²	Asteraceae	Leaves

<i>Solanum Indicum</i> ¹⁰²	Solanaceae	Leaves
<i>Maytenus emarginata</i> ¹⁰²	Celastraceae	Leaves
<i>Aerva lanata</i> ¹⁰³	Amaranthaceae	Whole plant
<i>Aerva sanguinolenta</i> ¹⁰⁴	Amaranthaceae	Leaves
<i>Gymnosporia emerginata</i> ¹⁰⁵	Celastraceae	Whole plant
<i>Marsdenia volubillis</i> ¹⁰⁵	Asclepiadaceae	Whole plant
<i>Carissa carandas</i> ¹⁰⁶	Apocyanaceae	Roots
<i>Asparagus racemosa</i> ¹⁰⁷	Liliaceae	Roots
<i>Kigelia Africana</i> ¹⁰⁸	Bignoniaceae	Leaves
<i>Anogeissus acuminate</i> ¹⁰⁸	Combretaceae	Leaves
<i>Abelmoschus moschatus</i> ¹⁰⁹	Malvaceae	Seeds
<i>Cyperus articulatus</i> ¹¹⁰	Cyperaceae	Rhizomes
<i>Thymus capitatus</i> ¹¹¹	Lamiaceae	Essential oils
<i>Salvia officinalis</i> ¹¹¹	Lamiaceae	Essential oils
<i>Solidago microglossa</i> ¹¹²	Compositae	Leaves
<i>Macrotyloma uniflorum</i> ¹¹³	Fabaceae	Seeds
<i>Tridax procumbens</i> ¹¹⁴	Compositae	Whole plant
<i>Orthosiphon stamineus</i> ¹¹⁵	Lamiaceae	Leaves
<i>Desmodium oojainens</i> ¹¹⁶	Fabaceae	Bark
<i>Trianthema portulacastrum</i> ¹¹⁷	Aizoaceae	Whole plant
<i>Acacia chatechu</i> ¹¹⁸	Mimosaceae	Heartwood
<i>Feronia limonia</i> ¹¹⁹	Rutaceae	Fruits
<i>Tecomella undulate</i> ¹²⁰	Bignoneaceae	Leaves
<i>Baliospermum montanum</i> ¹²¹	Euphorbiaceae	Roots
<i>Cyathea gigantean</i> ¹²²	Cyatheaceae	Leaves
<i>Operculina turpethum</i> ¹²³	Convolvulaceae	Roots
<i>Tamarindus indica</i> ¹²⁴	Caesalpiniaceae	Fruits, seeds, leaves
<i>Berberis tinctoria</i> ¹²⁵	Berberidaceae	Leaves
<i>Azadirachta indica</i> ¹²⁶	Meliaceae	Leaves
<i>Ceiba pentandra</i> ¹²⁷	Bombacaceae	Stem bark
<i>Plumbago zeylanica</i> ¹²⁸	Plumbaginaceae	Roots
<i>Phyllanthus emblica</i> ¹²⁹	Euphorbiaceae	Fruits
<i>Pittosporum neelgherrense</i> ¹³⁰	Pittosporaceae	Stem bark
<i>Sphaeranthus amaranthoides</i> ¹³¹	Compositae	Whole plant
<i>Oenlandia herbacea</i> ¹³²	Rubiaceae	Whole plant
<i>Calotropis gigantean</i> ¹³³	Asclepiadaceae	Root bark
<i>Coldenia procumbens</i> ¹³⁴	Boraginaceae	Whole plant
<i>Portulaca oleraceae</i> ¹³⁵	Portulacaceae	Whole plant
<i>Betula utilis</i> ¹³⁶	Betulaceae	Bark
<i>Pterocarpus santalinus</i> ¹³⁷	Fabaceae	Heartwood
<i>Santolina chamaecyparissus</i> ¹³⁸	Asteraceae	Whole plant
<i>Polygala arvensis</i> ¹³⁹	Polygalaceae	Leaves
<i>Enicostemma axillare</i> ¹⁴⁰	Gentianaceae	Whole plant
<i>Solanum tuberosum (purple potato)</i> ¹⁴¹	Solanaceae	Tubers
<i>Garcinia kola</i> ¹⁴²	Guttifera	Seeds
<i>Fumaria indica pugsley</i> ¹⁴³	Fumariaceae	Whole plant
<i>Leucas lavandulaefolia</i> ¹⁴⁴	Labiatae	Leaves
<i>Pisonia aculeate</i> ¹⁴⁵	Nyctaginaceae	Whole plant
<i>Phyllanthus niruri</i> ¹⁴⁶	Phyllanthaceae	Whole plant
<i>Vitex negundo</i> ¹⁴⁷	Lamiaceae	Leaves
<i>Momordica tuberosa</i> ¹⁴⁸	Cucurbitaceae	Tubers
<i>Tinispora crispa</i> ¹⁴⁹	Menispermaceae	Stem

<i>Zizyphus jujube</i> ¹⁵⁰	Rhamnaceae	Fruits
<i>Phoenix dactylifera</i> ¹⁵¹	Aracaceae	Fruits
<i>Gardenia gummifera</i> ¹⁵²	Rubiaceae	Roots
<i>Albizia lebbek</i> ¹⁵³	Fabaceae	Leaves
<i>Wedelia calendulaceae</i> ¹⁵⁴	Compositae	Leaves
<i>Ceiba pentandra</i> ¹⁵⁵	Bombacaceae	Root
<i>Ipomoea aquatic</i> ¹⁵⁶	Convolvulaceae	Leaves
<i>Boerhaavia diffusa</i> ¹⁵⁷	Nyctaginaceae	Stem, leaves
<i>Anisochilus carnosus</i> ¹⁵⁷	Nyctaginaceae	Leaves
<i>Phyllanthus acidus</i> ¹⁵⁸	Euphorbiaceae	Leaves
<i>Artemisia aucheri</i> ¹⁵⁹	Compositae	Flowered
<i>Nigella sativa</i> ¹⁶⁰	Ranunculaceae	Seed oil
<i>Curcuma longa</i> ¹⁶¹	Zingiberaceae	Rhizome
<i>Capparis deciduas</i> ¹⁶²	Capparaceae	Root bark
<i>Bosenbergia rotunda</i> ¹⁶³	Zingiberaceae	Rhizomes
<i>Feronia elephantum</i> ¹⁶⁴	Rutaceae	Leaves
<i>Optunia ficus indica</i> ¹⁶⁵	Cactaceae	Fruits
<i>Citrus paradise</i> ¹⁶⁵	Rutaceae	Fruits
<i>Matricaria chamomilla</i> ¹⁶⁵	Asteraceae	Essential oil
<i>Silybum marianum</i> ¹⁶⁵	Asteraceae	Crude extract
<i>Trigonella foenum graecum</i> ¹⁶⁶	Leguminosae	Seeds
<i>Jatropha curcas</i> ¹⁶⁶	Euphorbiaceae	Leaves
<i>Coccinia grandis</i> ¹⁶⁶	Curcubitaceae	Fruits
<i>Morinda citrifolia</i> ¹⁶⁶	Rubiaceae	Juice
<i>Ficus benjamina</i> Linn ¹⁶⁷	Moraceae	Leaves
<i>Bauhinia purpurea</i> Linn ¹⁶⁸	Cesalpiniaceae	Leaves
<i>Solanum nigrum</i> ¹⁶⁹	Solanaceae	Whole plant
<i>Ficus religiosa</i> Linn ¹⁷⁰	Moraceae	Stem bark
<i>Melia azhadirecta</i> Linn ¹⁷¹	Piperaceae	Leaves
<i>Withania frutescens</i> ¹⁷²	Solanaceae	Leaves
<i>Valeriana wallichii</i> ¹⁷³	Valerianaceae	Roots

CONCLUSION: Liver is a vital organ play a major role in metabolism and excretion of body. Liver injury or liver dysfunction is a major health problem that challenges not only health care professionals but also the pharmaceutical industry and drug regulatory agencies. Few of the popular liver diseases are listed above. Herbal medicines have been used in the treatment of liver diseases for a long time. Several herbal preparations are available in the market. Many herbs have been proven to be efficient as hepatoprotective agents, and maximum of them are listed above.

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