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Research

Evaluationof Hepatoprotective Activity of *Boerhavia Diffusa* Linn In CCL$_4$-Induced Hepatotoxicityin Male Wistar Rats

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Abstract

Siddha medicine is a traditional healing system that has its roots in southern India and is considered one of the oldest medical practices in the country. However, the Indian Medical Association has labeled degrees in Siddha medicine as "fraudulent" and views its therapies as pseudoscientific, suggesting that they pose a risk to public health due to a lack of scientific training. Boerhavia diffusa (BD) Linn. (Nyctaginaceae) is a prominent medicinal plant recognized in traditional Indian medicine and in various regions worldwide, including South America and Africa. Its different parts, particularly the roots, are utilized for treating gastrointestinal issues, liver protection, and gynecological conditions in these areas and across India. Ayurvedic literature lists over 35 formulations that feature BD as a key ingredient. In Ayurveda, BD is categorized as a "rasayana" herb, known for its anti-aging properties, ability to restore youth, enhance vitality and cognitive function, and prevent diseases. These attributes suggest that it boosts the body's resilience against various challenges, thereby offering hepatoprotective and immunomodulatory benefits. The administration of Boerhavia diffusa extract has been shown to enhance liver conditions in Wistar albino rats. The positive effects of this extract are evident in both the histopathological characteristics and the hepatic serum markers of the rats. Histopathologically, the severity of hepatosteatosis was reduced in the groups receiving Boerhavia diffusa extract, with a clear dose-dependent response observed. Initial qualitative assessment of phytochemicals aimed at detecting the presence of compounds such as phenols, flavonoids, and tannins.

Keywords: Boerhavia diffusa, Hepatoprotective activity, CCL$_4$-induced hepatotoxicity, Siddha medicine, Wistar rats, Phytochemical screening

INTRODUCTION

THE LIVER

The liver, a vertebrate-only organ, is vital for protein synthesis, metabolite detoxification, and the production of biochemicals needed for digestion and development [13]. The liver, which is situated in the right upper quadrant of the human abdomen, beneath the diaphragm, is also responsible for other metabolic processes, such as the synthesis of hormones, the degradation of red blood cells, and the control of glycogen storage [1]. The liver, an auxiliary digestive organ, produces bile, a fluid made up of cholesterol and bile acids that has an alkaline component that helps break down fat. Bile emulsifies lipids, which improves digestion. Before being discharged into the small intestine for full digestion, the bile from the liver is first held in the gallbladder, a little pouch located right underneath it [2]. Hepatocytes, the main component of the liver's highly specialized tissue, control a wide range of high-volume biochemical reactions that produce and degrade small and complex chemicals, many of which are essential for regular physiological processes. Textbooks typically list about 500 different liver functions, while estimates of the overall number of functions vary [3].

There is currently no proven way to make up for long-term loss of liver function, even if liver dialysis procedures may be used for short-term care. The development of artificial livers for long-term replacement in liver failure situations has not yet been successful. The only available treatment for total liver failure as of 2018 is liver transplantation [4].

The liver is a reddish-brown, wedge-shaped organ made up of four lobes that differ in size and form. A human liver's average weight is 1.5 kg (3.3 lb), and its width is roughly 15 cm (6 in) [5]. Individual differences in size are significant; the average weight range for women is 600–1,770 g (1.32–3.90 lb), and for men it is 970–1,860 g (2.14–4.10 lb) [6]. The liver, the body's largest gland and heaviest internal organ, is situated in the right upper quadrant of the abdominal cavity, just below the diaphragm, to the right of the stomach, and above the gallbladder [7]. The hepatic artery and the portal vein are the two main blood arteries that feed blood to the liver. While the hepatic artery transports oxygenated blood from the aorta via the celiac plexus, the portal vein transports blood that is rich in nutrients from the pancreas, spleen, and gastrointestinal tract [8]. These blood channels divide into tiny capillaries called liver sinusoids, which eventually lead to the liver lobules.

The liver is divided into lobules, which are functional units. Millions of hepatic cells, also known as hepatocytes, which are the main cells in charge of metabolism, make up each lobule. A thin, thick, and asymmetrical layer of connective tissue binds these lobules together. Glisson's capsule, a fibrous covering that envelops the whole liver, is the source of this connective tissue layer [9].

CIRRHOSIS OF THE LIVER

A chronic form of liver cell deterioration and death is called cirrhosis. Cirrhosis is a disorder in which the liver does not function normally as a result of chronic injury. It is sometimes referred to as liver cirrhosis or hepatic cirrhosis. Scar tissue replaces the normal liver tissue as a result of this damage. The illness usually progresses gradually over several months or years. There are frequently no symptoms in the early stages. As the illness progresses, a person may have fatigue, weakness, itching, lower leg oedema, yellow skin, bruising, abdominal fluid accumulation, or spider-like blood capillaries on their skin [10].

Infections: Antibiotics and other infection treatments may be administered to the patient. Additionally, the doctor is likely to suggest hepatitis, pneumonia, and influenza immunisations.

Elevated risk of liver cancer: To check for liver cancer, the doctor would probably advise routine blood tests and ultrasound examinations.

Encephalopathy of the liver: Because of impaired liver function, doctors may prescribe drugs to assist lower the accumulation of toxins in your blood.

Aim and objective

A variety of alkaloids, flavonoids, glycosides, tannins, saponins, and steroids are present in the powdered ethanolic extract of Boerhavia diffusa. The administration of Boerhavia diffusa extract has been shown to enhance liver conditions in Wistar albino rats. The positive effects of this extract are evident in both the histopathological characteristics and the hepatic serum markers of the rats. Histopathologically, the severity of hepatosteatosis was reduced in the groups receiving Boerhavia diffusa extract, with a clear dose-dependent response observed. This element of the extract may decrease the absorption of CCL4 and hinder its conversion into lipids. Additionally, saponin, another component of the extract, not only modulates lipid metabolism but also helps alleviate hypertriglyceridemia. Furthermore, the plant's polyphenolic and flavonoid compounds, known for their antioxidant properties, provide another rationale for the plant's antihepatosteatosis effects by preventing the secondary progression of the disease. Preliminary qualitative analysis of phytochemicals to identify the presence of substances including tannins, flavonoids, and phenols.

Fig 1: Boerhavia diffusa plant with leaves, flowers, and roots

2. Scientific Classification
Kingdom: Plantae
Subkingdom: Tracheophytes
Superdivision: Angiosperms
Division: Eudicots
Class: Caryophyllales
Order: Nyctaginaceae
Family: Nyctaginaceae
Genus: Boerhavia
Species: B. diffusa

3. Synonyms
English: Hogweed, Spreading hogweed
Sanskrit: Punarnava
Hindi: Raktpunarnava, Lalpunarnava
Tamil: Mukaratti
Telugu: Atikamamidi
Malayalam: Thazhuthama
Kannada: Sanjeevani
Bengali: Punarnava

4. Distribution
Boerhavia diffusa is widely distributed in tropical and subtropical regions, including India, Africa, South America, and Southeast Asia. It is commonly found in sandy or gravelly soils, wastelands, roadsides, and cultivated fields. In India, it is widely available in states like Maharashtra, Tamil Nadu, Kerala, and Karnataka.

5. Traditional Uses
Boerhavia diffusa has a long history of use in traditional medicine systems like Ayurveda, Siddha, and Unani.

- **Ayurveda:** Used as a diuretic, anti-inflammatory agent, and for treating jaundice, edema, and urinary tract infections.
- **Siddha Medicine:** Prescribed for kidney disorders, anemia, and as a general rejuvenator.
- **Unani Medicine:** Applied for liver health, skin diseases, and respiratory conditions.
- **Folk Medicine:** The roots are used to prepare decoctions for treating asthma, fever, and gastrointestinal disorders. The leaves are also consumed as a vegetable in some regions.

MATERIALS AND METHODS

PREPARATION OF PLANT

Plant cultivated and 10 times washed with running water and 10mins soaked with water for remove the soil particles and dirties. And prick the leaves and again washed with water. The leaves are dried in clean area and room temperature without sunlight for 20days. After 20days, the dried leaves are again dried in direct sunlight for 15min and grind fine powder of leaves and the powder was stored in clean container.

EXTRACTION OF BOERHAVIA DIFFUSA

MATERIALS USED

- Dried leaf of Boerhavia diffusa
- Percolation apparatus
- Ethanol
- Distilled water

The Boerhavia diffusa leaf was extracted and let to dry in the shade. The 3 kg of shade-dried plants were then ground into a coarse powder and sieved through mesh number 60. Using percolation equipment, the whole plant powder was extracted for 72 hours at room temperature using an ethanol solvent. Using 70% ethanol and 30% distilled water; the extraction process was carried out for 50 hours. Using the percolation method, the extraction was carried out at room temperature for 12 hours using 20% ethanol and 80% distilled water. A dry extract (9.8% w/w, in comparison to the powdered substance) was obtained by vacuuming out the solvent. A light greenish brown residue weighing 17.6 grammes was found. Pharmacological screening and qualitative analysis were used to determine the chemical components of the alhocolic extract. Steroid saponins, flavonoids, glycosides, phytosterols, tannins, terpenoids, amide derivatives, amino acids, and proteins were all detected by a phytochemical assay.

PHYTOCHEMICAL EVALUATION

The presence of several phytoconstituents was assessed in an ethanolic extract of the plant material.

ALKALOID TEST

For two minutes, 0.2 grammes of the extracts were heated with 2% H2SO4. After filtering it, a few drops of Dragendorf's reagent were put into each test tube. The presence of alkaloids was revealed by an orange-red precipitate [63].

TANNIN TEST

A little amount of extracts were combined with water in individual test tubes and heated in a water bath. Ferric chloride was added to the filtrates after the mixtures were filtered. Tannins are present when the solution is dark green [64].

A SAPONINS TEST

After shaking 0.2 grammes of the extract with 5 millilitres of distilled water, it was brought to a boil. Saponins are indicated by frothing, which is the appearance of a creamy mass of tiny bubbles [65].

FLAVONOID TEST

After dissolving 0.2 grammes of extracts in diluted NaOH, HCL was added. Flavonoids are indicated by a yellow solution that turns colourless [66].

CARBOHYDRATE TEST

Molisch's Test: Fill a clean test tube with 2 millilitres of the provided sample solution. Slowly add two to three drops of Molisch reagent. Now fill the test tube's sides with concentrated sulphuric acid. At the bottom, an acid layer forms. Take note of where the two levels converge. It is established that carbohydrates are present if a violet ring forms [67].

GLYCOSIDE TEST

Raymond's test:

Add a few millilitres of 50% ethanol and 0.1 millilitres of a 1% m-dinitrobenzene in ethanol solution to the plant powder. Add two to three drops of a 20% sodium hydroxide solution to this mixture. Because of the active methylene group, violet hues are seen.

To check for terpenoids:

Put around 0.5 g of plant extract and 2 ml of chloroform in a different test tube. Then, gently add 5 ml of strong sulphuric acid to create a layer, and look for a reddish brown colour interface, which indicates the presence of terpenoids.

To test for reducing sugars:

Mix two millilitres of crude plant extract with five millilitres of distilled water, then stir and filter. After boiling the filtrate for two minutes with three to four drops of Fehlings solutions A and B, the presence of reducing sugars was detected by looking for an orange-red precipitate.

Steroid Test:

0.5 g of extract was mixed with 2 ml of sulphuric acid and 2 ml of acetic anhydride. The samples were then watched for a colour shift from violet to blue or green, which would indicate the presence of steroids.

Anthocyanin Test:

Sodium Hydroxide Test: In a different test tube, 0.2 grammes of plant extract were weighed, 1 millilitre of 2N sodium hydroxide was added, and the mixture was heated for 5 minutes at 100 ± 2°C. The production of a blue green colour, which denotes the presence of anthocyanin, was then monitored.

The phenol-ferric chloride test involved weighing 0.2 g of plant extract, treating it with 5% ferric chloride, and watching for the development of a deep blue colour, which is a sign that phenol is present.

Ninhydrin Test for Amino Acid:

0.2g of plant extract was weighed, treated with Ninhydrin solution, and checked for the distinctive purple hue that denotes the presence of amino acids.

Protein Test:

Million's Test:

A small amount of plant leaf extract was treated with a few drops of Million's reagent, and the formation of a white precipitate a sign that protein is present—was watched for.

IN VIVO PHARMACOLOGICAL EVALUATION

Rats are particularly suitable for study of physiology of the heart since after partial damage the organ regenarates almost completely in course of week. Albino wister rat (170-225gm) were used for this study. They were housed in a room with control environment (temp 25^{o}C) and 12hrs dark/light cycle with standard laboratory diet and water ad libitum. They were fed on healthy diet and maintained in hygienic environment.

TREATMENT PROTOCOL

The rats were randomly distributed to six groups (n=6). To liver cirrhosis rats, test drug *Boerhavia diffusea* dissolved in sterile water given orally through orally.

[0.75ml of CCL_4(25%) mixed with olive oil]

Group 1: Normal control rats treated with distilled water (6ml/kg).

Group 2: Rats treated with CCL4 (3ml/kg/day) by p.o.

Group 3: CCL_4 induced hepatotoxicity rats treated with silymarin (25mg/kg) by i.p.

Group 4: CCL_4 induced hepatotoxicity rats treated with ethanolic extract of *Boerhavia diffusa* (100mg/kg)

Group 5: CCL_4 induced hepatotoxicity ratstreated with ethanolic extract of *Boerhavia diffusa* (200mg/kg)

Group 6: CCL_4 induced hepatotoxicity rats treated treated with ethanolic extract of *Boerhavia diffusa* (400mg/kg) [69].

METHODS

Biochemical analysis

At the end of the experiment, blood samples were taken from heart and preserved in 37^{o} C for 30min and centrifuged for 15min. The serum was collected and preserved in -20^{0}C until measuring levels of serum marker enzyme Aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), gamma-glutamyltranspeptidase (GGT), bilirubin, protein.

Determination of AST and ALT

Activities of AST and ALT were assayed. In different tubes, 1ml of the buffered substrate was added to 0.1ml of sample and incubated at 37^{o}Cfor 30min. Then 1ml of DNPH reagent was added to arrest the reaction. To the blank tubes, 0.1ml of sample was added only after the addition of DNPH reagent. The tubes were kept aside for 15min, and then 10ml of sodium hydroxide was added and read at 520nm

Determination of ALP

Procedure Serum was obtained by centrifugation of freshly clotted blood. The serum (1 cc.) was removed with a pipette and diluted with 19 cc. of distilled water. 1 cc. of this diluted serum was placed in a 20 cc. test-tube and 5 cc. of the appropriately buffered substrate solution were added. In a separate test-tube, buffered substrate solution alone served as a control for non-enzymatic hydrolysis. The tubes were then incubated at 37.5" for 1 hour in the determination of alkaline phosphatase, and for 2 hours in the determination of acid phosphatase. In the latter determination 4 drops of 1 M sodium carbonate solution were added after the period of incubation, in order to raise the pH to the optimal level for coupling. 1 cc. of the solution of tetrazotizeddiorthoanisidine was added to each tube, and the tubes were agitated vigorously or inverted several times to insure thorough mixing. 3 minutes were allowed to elapse for completion of coupling. To each tube 1 cc. of 40 per cent trichloroacetic acid was added to precipitate protein and favor release of dye from its protein

complex. 10 cc. of ethyl acetate were added from a burette and the tubes were shaken vigorously until an even emulsion was produced. The tubes were then centrifuged for 10 minutes at 2500 rpm, and 5 cc. of the organic layer were transferred with a pipette to a Klett tube. The color did not fade on standing, but evaporation of ethyl acetate in over 1 hour resulted in increased color density. This was avoided by stoppering the tubes when such delay was unavoidable. The color density was measured in a photoelectric calorimeter through a green filter (540 nm).

Determination of GGT

Serum was obtained by centrifugation of freshly clotted blood. The serum (1 cc.) was removed with a pipette and diluted with 19 cc. of distilled water. Pipette in to a clean dry test tube. Working reagent added with test (1ml) and incubates at the assay temperature for 1min and add sample 0.1ml and mix well and read the initial absorbance A_o after 1minute.wavelenth at 405nm.
Calculation: delta A/min x 1158.

Determination of bilirubin

Take two test tubes and label them test and control. Distilled water 1.8ml and serum 0.2ml and diazo reagent 0.5ml methanol 2.5ml with test. And distilled water 1.8ml and serum 0.2ml and diazo blank 0.5ml and methanol 2.5ml with control. Keep the test tubes in dark. Reading taken at 1minute is direct /conjugated bilirubin.
Calculation: S.Bilirubin (mg%) = [ODt-ODc/ODs] x 8.

Determination of protein

Procedure (Standard Assay, 20-150 µg protein; 200-1500 µg/mL) Prepare a series of standards diluted with 0.15 M NaCl to final concentrations of 0 (blank = No protein), 250, 500, 750 and 1500 µg/mL. Also prepare serial dilutions of the unknown sample to be measured. Add 100 µL of each of the above to a separate test tube (or spectrophotometer tube if using a Spectronic 20). Add 5.0 mL of Coomassie Blue to each tube and mix by vortex, or inversion. Adjust the spectrophotometer to a wavelength of 595 nm, using the tube which contains no protein (blank). Wait 5 minutes and read each of the standards and each of the samples at 595 nm wavelength. Plot the absorbance of the standards vs. their concentration. Compute the extinction coefficient and calculate the concentrations of the unknown samples.

Determination of purity of *Boerhavia diffusa*

Preparation of stock solution:

Extraction of *Boerhavia diffusa*100mg was dissolved in 100ml diluted methanol to make 1000µg/ml stocksolution.

Procedure for the Determination of Silymarin standard:

From the above solution aliquots of 0.6 ml, 0.8 ml, 1 ml, 1.2 ml, 1.4 ml, 1.6 ml were taken ina separate 10 ml volumetric flasks then make up the volume with methanol.
And the test solution transferred to uv-spectroscopy (Schimadzu - 1700) at 295nm wavelength.

Histopathological method

The rats are decapitated by using anesthesia and a part of fresh liver was dissect out. The liver tissue was fixed in 10% formalin, embedded in paraffin and finally cut into 5mm section. From each subject, 10section were selected randomly and stained with hematoxylin-cosin and of them, 5 random fields were captured by motic camera for double blinded histological assessment, carried out by two other academic members of anatomical science. The histopathological fatty changes were evaluated using the grading and staging system and degreed from 0 to 4 as follows: 0 = without steatosis, 1= steatosis response, 2=approximately <25% steatosis, 3= approximately 26-55 % steatosis, 4=<78% steatosis, 5=<92% steatosis.
Microscopic examination allows the following grading:
Grade 0: No change
Grade 1: steatosis response
Grade 2:<92% steatosis
Grade 3:<25 % steatosis
Grade 4:<26-55 % steatosis
Grade 5:<78 % steatosis
For each group the main grade is calculated with the standard deviation to reveal significant differences.

Statistical analysis

The result of hepatoprotective activity is expressed as mean "Mean ± SEM from five animals in each group. Results were statistically analyzed using one-way ANOVA followed by Newman Keuls multiple range test for individual comparisons: $p<0.01$ was considered significant. Graph Pad InStat version 3.00 of Graph Pad Software, Inc. (san diego, CA), was used for statistical analysis.

RESULTS

EVALUATION OF BIO CHEMICAL PARAMETER

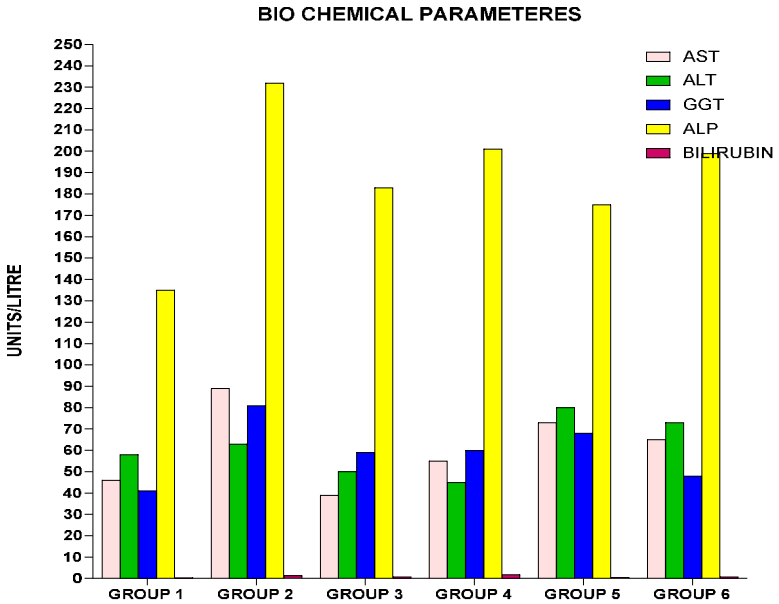
Serum Marker Enzyme

S.NO	AST (UNITS/L)	ALT (UNITS/L)	GGT (UNITS/L)	ALP (UNITS/L)	BILIRUBIN (UNITS/L)
GROUP 1	46.57±1.51	58.46±1.2	41.4±2.32	135.1±1.5	0.35±0.02
GROUP 2	89.37±2.7	63.02±2.3	81.42±1.33	232.6±4.8	1.36±0.2
GROUP 3	39.36±2.3	50.61±0.1	59.15±1.2	183.1±2.3	0.81±0.02
GROUP 4	55.35±2.6	45.41±1.3	60.32±01	201.5±4.1	1.89±0.06
GROUP 5	73.23±1.9	80.29±2.1	68.23±1.02	175±5.7	0.45±0.02
GROUP 6	65.43±2.5	72.23±2.3	48.12±2.5	199.25±5.3	0.76±0.04

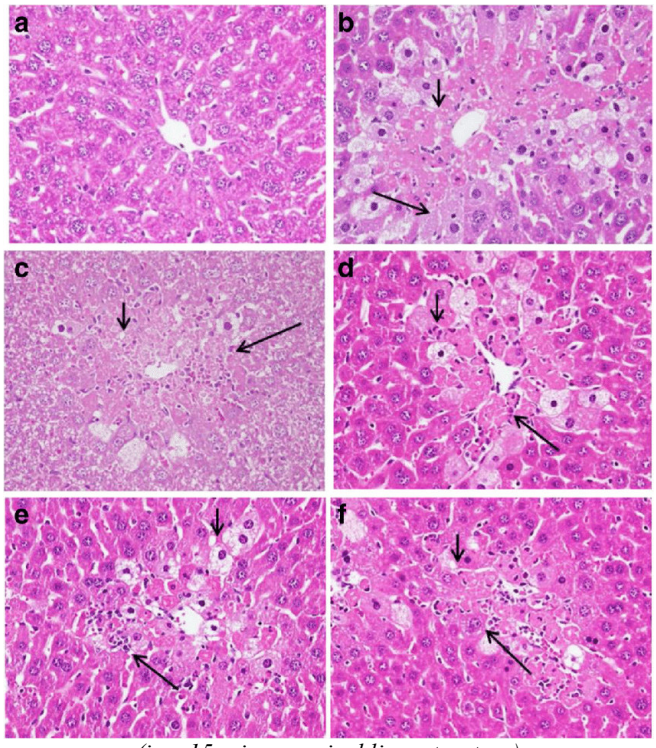
Group 1: Normal control rats treated with distilled water
Group 2: Rats treated with CCL4 (3ml/kg/day) by p.o.
Group 3: CCL_4 induced hepatotoxicity rats treated with of silymarin (25mg/kg) by i.p
Group 4: CCL_4 induced hepatotoxicity rats treated with ethanolic extract of *Boerhavia diffusa* (100mg/kg)
Group 5: CCL_4 induced hepatotoxicity rats treated with ethanolic extract of*Boerhavia diffusa* (200mg/kg)
Group 6: CCL_4 induced hepatotoxicity rats treated with ethanolic extract of *Boerhavia diffusa*(400mg/kg)

Comparison study

Group 1. is a normal control, treated with distilled water .
Group 2. is a negative control, treated with CCL_4.
Group 3.is a standard drug compared with Group 1. Group 3 enzyme unit value almost equal to group 1.
Group 4 compared with group 1 , the value shows group 4 units increased than group 1.
Group 5 compared with group 1 , the value shows group 5 units increased than group 1.
And decreased than group 4.
Group 6 compared with group 1 , the value shows group 6 units increased than group 1.
andgroup 5.
And the group 3 standard drug compared with Group 4,5,6. But the group 5 units similar to group 3.



HISTOPATHOLOGICAL RESULT



(img.15-microscopical liver structure)

Hepatic histological analyses of *Boerhavia diffusa*and silymarin on CCl 4 -induced acute liver damage in mice. Liver tissues were subjected to hematoxylin and eosin staining (400×). (a) Control group; (b) animals treated with 25% CCl 4; displayed cell necrosis (long arrow) and vacuole formation (short arrow) (c) animals pretreated with silymarin (200 mg/kg) and then treated with CCl 4 ; (d–f) animals pretreated with *Boerhavia diffusa*(100,200,400gm/kg) and then treated with CCl 4.

Description

The group 1 is a normal control; histopathology report shows the liver tissue cells are normal and no cell destruction or cell injury.

The group 2 is a CCL_4 treated, histopathology report shows the liver tissue cells are abnormal and cell destruction or cell injury.

The group 3 is standard drug treated, histopathology report shows the liver tissue cells are normal and cell destruction or cell injury reduced when compared to group 2.

The group 4 is 100mg/kg *Boerhavia diffusa*extract treated, histopathology report shows the liver tissue cells are normal and cell destruction or cell injury reduced when compared to group 2.

The group 5 is 200mg/kg *Boerhavia diffusa*extract treated, histopathology report shows the liver tissue cells are normal and cell destruction or cell injury reduced when compared to group 2.

The group 6 is 400mg/kg *Boerhavia diffusa* extract treated, histopathology report shows the liver tissue cells are normal and cell destruction or cell injury reduced when compared to group 2.

The group 4, 5, 6 is a ethanolic extract of*Boerhavia diffusa*, the three groups are compared to group 3 standard silymarin. The report shows group 6 400mg/kg of *Boerhavia diffusa* extract similar to group 3standard.

The liver tissue cells of group 5 similar to group 3. And decrease the liver cell damage.

Table 1: *Boerhavia diffusa* **leaf extract Phytochemical test**

S.No	Phytochemical Test	Ethanolic extract
1.	Alkaloids	–
2.	Flavonoids	+
3.	Tannins	+
4.	Phenols	+

5	Carbohydrates	_
6.	Proteins	_
7.	Steroids	_
8.	Saponins	_

[- absence ; + presence]

DISCUSSIONS

In this study, we assessed the protective effects of Boerhavia diffusa ethanolic extract against liver toxicity induced by CCL4 in rats. The findings revealed that CCL4 significantly increased liver weight. Additionally, the study indicated that CCL4 led to liver cirrhosis, which should be viewed as a result of microsomal enzyme induction, causing increased lipid storage, peroxisome proliferation, and heightened liver activity.[11] Furthermore, the administration of Boerhavia diffusa extract resulted in a decrease in both body and liver weight in the rats, along with a reduction in the severity of steatohepatitis.

Liver function was evaluated through blood assays, which provided insights into its functionality and its relationship with the biliary tract. The groups treated with Boerhavia diffusa showed that the extract affected hepatocellular or secretory liver functions in a dose-dependent manner.[12]

The five key biomarkers associated with liver damage include AST, ALT, ASP, GGT, and bilirubin, which collectively indicate liver injury. The AST/ALT ratio can be utilized for disease diagnosis; a ratio exceeding 1 may suggest myocardial infarction, while a ratio greater than 2 points to alcoholic hepatitis or cirrhosis. A ratio between 1.2 and 1.4 indicates abnormal liver function.

In this study, the extract demonstrated a protective effect against hepatic damage, as evidenced by blood chemistry analysis and histopathological evaluation. Significant changes were observed in the liver damage indicators (ALT, ALP, AST, GGT, bilirubin) and the steatosis ratio.[13]

The extract of Boerhavia diffusa is rich in flavonoids, which are recognized for their insulin-mimicking effects and their ability to inhibit the lipogenase enzyme. These flavonoids enhance cell viability and reduce the leakage of AST and ALT from hepatocytes. Additionally, research indicates that flavonoids can lower insulin resistance and the insulin needs in adipose and muscle tissues. In addition to flavonoids, the plant also contains alkaloids, glycosides, and steroid saponins, all of which exhibit hypolipidemic properties.

Alkaloids play a role in decreasing the absorption and metabolism of carbohydrates. Consequently, this extract component may lower the absorption of CCL4 and hinder its conversion into lipids. Another component, saponin, not only modulates lipid metabolism but also helps alleviate hypertriglyceridemia. Additionally, the plant's polyphenolic and flavonoid compounds, known for their antioxidant properties, contribute to the antihepatosteatosis effects by preventing the progression of the disease through a secondary mechanism.[14,15]

CONCLUSION

It has been demonstrated that giving Wistar albino rats Boerhavia diffusa extract improves their liver health. The rats' histological features and hepatic serum indicators both demonstrate the beneficial benefits of this extract. Histopathologically, the groups who received Boerhavia diffusa extract showed less severe hepatosteatosis; the results were dose-dependent. Initial qualitative assessment of phytochemicals aimed at detecting the presence of compounds such as phenols, flavonoids, and tannins.

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