Section: Pharmacology

Original Research Article

A STUDY TO EVALUATE THE HEPATOPROTECTIVE EFFECT OF ETHANOLIC EXTRACT OF PERGULARIA DAEMIA LEAVES IN D-GALACTOSAMINE INDUCED HEPATOTOXICITY IN ADULT MALE ALBINO RATS

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ABSTRACT

Background: Herbal remedies play a significant role in traditional Indian medicine for managing liver disorders; however, many such plants require scientific validation. Pergularia daemia, a medicinal plant known for its antioxidant constituents, has been traditionally used for hepatic ailments. The present study evaluated the hepatoprotective potential of the ethanolic extract of Pergularia daemia (EEPD) in rats with D-galactosamine-induced hepatotoxicity. **Materials and Methods:** Thirty adult male albino rats were randomly assigned to five groups (n=6): control, toxic control, silymarin-treated standard, Test-1 (EEPD 100 mg/kg), and Test-2 (EEPD 200 mg/kg). Except for the control group, all animals received D-galactosamine (800 mg/kg, IP) to induce hepatotoxicity. Treatments were administered orally for 21 days. Hepatoprotective efficacy was assessed using serum markers, SGOT, SGPT, and ALP, measured on days 0, 10, and 21. Histopathological evaluation of liver tissue was also performed at the end of the study. **Result:** D-galactosamine significantly elevated the SGOT, SGPT, and ALP levels in the toxic control group, confirming hepatic injury. Silymarin produced a marked reduction in all biochemical parameters ($p < 0.001$). EEPD demonstrated dose-dependent hepatoprotection: Test-1 showed moderate improvement comparable to the standard group, while Test-2 exhibited a more decrease in enzyme levels, approaching near-normal values ($p < 0.001$). Histopathology confirmed these findings, with Test-2 showing preservation of hepatic architecture and reduced necrosis. **Conclusion:** EEPD offers significant hepatoprotective effects against D-galactosamine-induced liver damage, with the 200 mg/kg dose displaying superior protective activity. These findings support its potential as a natural hepatoprotective agent, possibly attributable to its flavonoid-rich antioxidant profile.

INTRODUCTION

The liver, the largest internal organ of the human body, performs several important functions, including the metabolism of carbohydrates, lipids, and proteins, detoxification of xenobiotics, synthesis of plasma proteins and clotting factors, and secretion of bile.[1] Because of its role in metabolism and detoxification, the liver is highly susceptible to toxic injury from various chemicals, drugs, and infectious agents. Liver diseases are recognised as a major global health problem associated with high morbidity and mortality. India alone contributes nearly 18.3% of the two million liver-related deaths that occur worldwide each year.[1,2]

Acute liver failure (ALF) is a severe and quickly progressive form of hepatic injury, with mortality rates ranging from 50% to 75%.[3,4] The common etiological factors include viral hepatitis, drug-induced hepatotoxicity, alcohol abuse, and exposure to environmental toxins.[5] Although liver transplantation is the only treatment with proven survival benefits in irreversible ALF, the lack of donors, high cost, and postoperative complications limit its universal application.[6] Therefore, there is an urgent need to discover effective and safe

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hepatoprotective agents capable of preventing or minimising hepatic injury.

Herbal medicines have been traditionally used in India for the treatment of liver disorders and continue to be the basis of complementary and alternative therapy. About 7,500 plants are estimated to be used in local health traditions in India, which mostly include rural and tribal villages.[7] More than 150 phytoconstituents isolated from 101 plants have been reported to possess hepatoprotective activity, and over 87 medicinal plants are included in 33 proprietary formulations used in India for liver ailments.[8] Analysing the medicinal values of new plant extracts on animal models before using them on humans is the core of animal-based experimental studies. Among the various experimental models used to assess hepatoprotective potential, D-galactosamine (D-GalN)–induced hepatotoxicity remains one of the most reliable and commonly used.9 D-GalN is an amino sugar that causes liver damage similar to human viral hepatitis by interfering with uridine nucleotide metabolism, leading to inhibition of RNA and protein synthesis in hepatocytes.[9]

The concomitant reduction of intracellular ATP and disruption of calcium homeostasis result in hepatocellular necrosis. Activation of Kupffer cells by D-GalN further releases pro-inflammatory cytokines, intensifying hepatocyte apoptosis and necrosis.[10] One of the broadly studied hepatoprotective agents is Silymarin, which is a naturally occurring bioactive compound extracted from Silybum marianum, commonly known as milk thistle. It acts by neutralising the free radicals generated during the metabolism of harmful substances, such as ethanol, acetaminophen, and carbon tetrachloride. Silymarin enhances hepatic glutathione levels and promotes protein synthesis in liver cells by activating RNA polymerase I, which supports cellular repair and regeneration.[11] Therefore, it is used as a standard reference drug for the comparative evaluation of newer plant-based extracts.

Pergularia daemia (family Asclepiadaceae), known as “Veliparuthi” in Tamil, is a perennial climber commonly distributed throughout tropical India.[12] It has been usually used in traditional medicine for the treatment of several disorders such as fever, asthma, rheumatism, menstrual irregularities, and jaundice.[13] Phytochemical investigations of the leaves have revealed the presence of alkaloids, flavonoids, saponins, tannins, steroids, terpenoids, and glycosides, compounds known for their antioxidant and membrane-stabilising properties.[14] Previous studies have indicated that the ethanolic extract of Pergularia daemia (EEPD) exhibits significant antioxidant and hepatoprotective effects due to its flavonoid constituents.[15]

Considering these pharmacological findings and the traditional use of Pergularia daemia in liver ailments. The present study was conducted to evaluate its hepatoprotective efficacy against D-GalN-induced hepatotoxicity in adult male albino rats. The effect of EEPD leaves was also compared with that of the standard hepatoprotective drug, silymarin.

MATERIALS AND METHODS

Animals

Thirty adult male albino rats weighing 150–250 g were obtained from the Central Animal House, Madurai Medical College, Madurai, for use in the experiment. The animals were housed in polypropylene cages under standard laboratory conditions at normal room temperature, with free access to commercially available pellet feed and distilled water ad libitum. All rats were acclimatised to the laboratory environment for one week before the initiation of the study. Throughout the experimental period, the animals were maintained and monitored in accordance with the CPCSEA guidelines for the care and use of laboratory animals, and every effort was made to ensure proper handling and to minimise stress or discomfort. Ethical approval for the study was obtained from the Institutional Animal Ethics Committee (IAEC) of Madurai Medical College with valid CPCSEA registration (No. 195/GO/REB/S/2000/CPCSEA, valid from 28.10.2020 to 28.10.2025).

Plant material and extract preparation

Fresh leaves of Pergularia daemia were collected in August 2021 from the Melamadai lake region of Madurai and were botanically identified and authenticated by Dr D. Stephen, Assistant Professor, Department of Botany, The American College, Madurai. The collected leaves were shade-dried, cut into small pieces, and coarsely powdered using a mixer grinder, according to the requirements. The powdered plant material was subjected to continuous hot extraction using a Soxhlet apparatus. Petroleum ether extraction was performed by maintaining a temperature between 30 and 40°C until the siphon tube produced a colourless solvent, after which the extract was concentrated, and the percentage yield was calculated. The remaining marc was air-dried and extracted using ethanol at 60–80°C under continuous hot extraction until a colourless solvent appeared in the siphon tube. The ethanolic extract was concentrated, the yield calculated, and the final extract was weighed, labelled, and stored in a refrigerator at temperatures below 10°C until further use.

Procedure

The 30 animals were divided into five groups containing six rats each: Group I served as the control group, Group II as the standard group, Group III as the toxic control, Group IV as Test-1, and Group V as Test-2. Except for the control group, all animals received a single intraperitoneal dose of D-galactosamine (800 mg/kg) on day 0 to induce hepatotoxicity. Following toxin administration, the standard group received oral silymarin at 100 mg/kg, Test-1 received EEPD leaves at 100 mg/kg orally,

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and Test-2 received EEPD at 200 mg/kg orally, along with normal feed and water, as described in the treatment table. Oral drug administration was performed using a 1-mL syringe fitted with an 18-gauge hypodermic needle blunted at the tip with a small solder. Each rat was gently grasped at the nape of the neck, and the feeding tube was introduced laterally through the interdental space with gentle rotatory movements until it advanced into the oesophagus, after which the drug was injected slowly to ensure safe delivery. Blood samples were collected by cardiac puncture on days 0, 10, and 21 using red-coloured serum-separating tubes, and all samples were transferred on the same day to the Biochemistry Laboratory, Madurai Medical College, for liver function test analysis. Cardiac puncture was performed under strict aseptic precautions, beginning with intraperitoneal administration of ketamine for anaesthesia. Anaesthetised animals were placed in dorsal recumbency, and a needle attached to a syringe was inserted at a 30–40° angle beneath the sternum, slightly toward the left side, with the bevelled edge parallel to the midline and directed toward the animal's head and left shoulder. The plunger was gently retracted to create a vacuum until blood appeared in the needle hub, after which the required volume of blood was collected while the needle position was maintained. At the end, the animals were euthanised by an overdose of ketamine anaesthesia (400 mg/kg IP), and the liver was immediately excised and fixed in 4% (v/v) paraformaldehyde. The tissues were then processed for histopathological examination by paraffin embedding, sectioning into 4-µm slices, staining with hematoxylin and eosin, and submitting the prepared slides to the Pathology Department of Madurai Medical College for microscopic evaluations. The hepatoprotective effect of EEPD was analysed by comparing the liver function test parameters, SGOT, SGPT, and ALP levels on days 0, 10, and 21 across the groups.

Statistical analysis

Data are expressed as mean ± SD. Differences between the five groups were analysed using one-way ANOVA, followed by a Bonferroni post-hoc test for multiple comparisons. A p-value < 0.05 was considered statistically significant.

RESULTS

At baseline (Day-0), all groups showed similar SGOT, SGPT, and ALP levels. By day 10, the toxic group had a rise in SGOT (283.11 IU/L), SGPT (106.10 IU/L), and ALP (308.01 IU/L). The standard and test groups also increased, but not as much as the toxic group. By Day-21, the toxic group maintained the highest enzyme levels (SGOT 326 IU/L, SGPT 116.26 IU/L, and ALP 328.26 IU/L). In contrast, the standard group had good recovery (SGOT 225.8 IU/L; SGPT 84.43 IU/L), and Test-1 produced moderate recovery. Test-2 had the best hepatoprotection, with SGOT reduced to 188.75 IU/L, SGPT to 58.53 IU/L, and ALP to 188.75 IU/L [Table 1].

Table 1: Comparison among groups on days 0, 10 and 21

Parameters	Groups	Day-0	Day-10	Day-21
SGOT (IU/L)	Control	173.08 ± 2.57	172.28 ± 3.24	174.71 ± 2.3
	Standard	173.48 ± 4.62	249.85 ± 5.42	225.8 ± 4.2
	Toxic	173.3 ± 4.62	283.11 ± 8.9	326 ± 3.7
	Test-1	173.41 ± 3.0	250.63 ± 4.75	232.3 ± 2.57
	Test-2	171.16 ± 3.13	245.68 ± 5.6	188.75 ± 10.26
SGPT (IU/L)	Control	56.78 ± 3.53	55.14 ± 4.45	57.06 ± 2.8
	Standard	53.78 ± 3.22	91.20 ± 3.12	84.43 ± 3.92
	Toxic	55.53 ± 4.22	106.10 ± 3.9	116.26 ± 4.15
	Test-1	56.18 ± 3.02	94.3 ± 2.58	87.53 ± 2.38
	Test-2	53.33 ± 4.2	88.51 ± 4.68	58.53 ± 4.56
ALP (IU/L)	Control	184 ± 4.9	183.11 ± 3.93	185.76 ± 7.03
	Standard	183.2 ± 3.27	288.1 ± 4.7	244.26 ± 7.46
	Toxic	184.68 ± 4.41	308.01 ± 4.6	328.26 ± 8.75
	Test-1	183.53 ± 3.74	290.18 ± 4.25	255.82 ± 8.16
	Test-2	188.93 ± 3.98	281.75 ± 3.61	188.75 ± 10.26

The toxic group had significantly elevated SGOT levels compared to the control group (326.00 ± 3.70 vs. 174.71 ± 2.30 IU/L, p=0.001). Treatment with silymarin significantly reduced SGOT to 225.80 ± 4.20 IU/L (p=0.001 vs. toxic). Test-1 produced a moderate reduction (232.30 ± 2.57 IU/L), while Test-2 showed the highest recovery compared to the standard (p=0.001) [Table 2].

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Table 2: Association of SGOT among groups on the 21st day

Comparison	Group	Mean ± SD	p-value
Control vs Toxic	Control	174.71 ± 2.30	0.001
	Toxic	326.00 ± 3.70	
Toxic vs Standard	Toxic	326.00 ± 3.70	0.001
	Standard	225.80 ± 4.20	
Toxic vs Test-1	Toxic	326.00 ± 3.70	0.001
	Test-1	232.30 ± 2.57	
Toxic vs Test-2	Toxic	326.00 ± 3.70	0.001
	Test-2	188.75 ± 10.26	
Standard vs Test-1 vs Test-2	Standard	225.80 ± 4.20	0.001
	Test-1	232.30 ± 2.57	
	Test-2	188.75 ± 10.26	

The toxic group showed a rise in SGPT levels (116.26 ± 4.15 IU/L) compared to the control group (57.06 ± 2.80 IU/L, p = 0.001). Silymarin produced a significant reduction to 84.43 ± 3.92 IU/L (p = 0.001 vs toxic). Test-1 also lowered the SGPT level to 87.53 ± 2.38 IU/L, but the change was not significant (p = 0.16). Test-2 showed the highest improvement, reducing SGPT to 58.53 ± 4.56 IU/L, compared to the standard and Test 1 (p = 0.001) [Table 3].

Table 3: Association of SGPT among groups on the 21st day

Comparison	Group	Mean ± SD	p-value
Control vs Toxic	Control	57.06 ± 2.80	0.001
	Toxic	116.26 ± 4.15	
Toxic vs Standard	Toxic	116.26 ± 4.15	0.001
	Standard	84.43 ± 3.92	
Toxic vs Test-1	Toxic	116.26 ± 4.15	0.16
	Test-1	87.53 ± 2.38	
Toxic vs Test-2	Toxic	116.26 ± 4.15	0.236
	Test-2	58.53 ± 4.56	
Toxic vs Test-1 vs Test-2	Toxic	116.26 ± 4.15	0.001
	Test-1	87.53 ± 2.38	
	Test-2	58.53 ± 4.56	
Standard vs Test-1 vs Test-2	Standard	84.43 ± 3.92	0.001
	Test-1	87.53 ± 2.38	
	Test-2	58.53 ± 4.56	

The toxic group showed a rise in ALP levels (328.26 ± 8.75 IU/L) compared to the control group (185.76 ± 7.03 IU/L, p = 0.001). Silymarin significantly reduced ALP to 244.26 ± 7.46 IU/L (p = 0.001 vs toxic). Test-1 also lowered ALP to 255.82 ± 8.16 IU/L, with a significant reduction compared to the toxic group (p = 0.001). Test-2 had the best recovery, reducing ALP to 188.75 ± 10.26 IU/L, better than both the standard and Test-1 groups (p = 0.001) [Table 4].

Table 4: Association of ALP among groups on the 21st day

Comparison	Group	Mean ± SD	p-value
Control vs Toxic	Control	185.76 ± 7.03	0.001
	Toxic	328.26 ± 8.75	
Toxic vs Standard	Toxic	328.26 ± 8.75	0.001
	Standard	244.26 ± 7.46	
Toxic vs Test-1	Toxic	328.26 ± 8.75	0.001
	Test-1	255.82 ± 8.16	
Toxic vs Test-2	Toxic	328.26 ± 8.75	0.001
	Test-2	188.75 ± 10.26	
Toxic vs Test-1 vs Test-2	Toxic	328.26 ± 8.75	0.001
	Test-1	255.82 ± 8.16	
	Test-2	188.75 ± 10.26	
Standard vs Test-1 vs Test-2	Standard	244.26 ± 7.46	0.001
	Test-1	255.82 ± 8.16	
	Test-2	188.75 ± 10.26	

DISCUSSION

This study evaluated the hepatoprotective effects of EEPD in a D-galactosamine-induced hepatotoxicity rat model using silymarin as the standard reference control. D-galactosamine is known to induce liver damage by reducing uridine nucleotides and disrupting RNA synthesis, causing hepatocyte necrosis and leakage of cytosolic enzymes.[16] In the present study, administration of D-galactosamine at 800 mg/kg produced hepatocellular injury, as observed by significant elevations in SGOT, SGPT, and ALP by day 21. A previous study had observed a similar pattern of enzyme elevation among rats with GalN-induced hepatotoxicity.[17]

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We have set treatment with silymarin as standard, and a study has reported a significant reduction in hepatic enzymes, confirming its hepatoprotective effect through membrane stabilisation, free-radical scavenging, and enhanced ribosomal protein synthesis.[14] As for the experimental treatment, the ethanolic extract of EEPD produced dose-dependent hepatoprotection for the rats. The 100 mg/kg EEPD group (Test-1) showed partial recovery, with SGOT and SGPT levels lower than those of the toxic group on day 21 but still higher than those of the standard control group. However, the 200 mg/kg EEPD group (Test-2) produced the highest hepatoprotective effect, by reducing the liver enzyme levels close to the control group. Studies suggest that Pergularia daemia extracts possess antioxidant and hepatoprotective activity in rodent models of toxic hepatic injury.[15,18] The hepatoprotective effect observed with EEPD is similar to the mechanism of other medicinal plants rich in flavonoids, alkaloids, and triterpenoids that can counteract oxidative stress and mitochondrial damage in hepatotoxicity.[19,20] Similarly, studies have reported that Pergularia daemia have antioxidant activity, suppression of lipid peroxidation, and enhancement of endogenous enzymatic antioxidants such as superoxide dismutase and catalase.[15,21]

In our study, both EEPD-treated groups showed significant reductions in ALP, with Test-2 producing a better outcome than Test-1. When compared with the standard group, Test-1 had a moderate decrease, while Test-2 had a more noticeable reduction ($p < 0.001$). Thus, both 100 mg/kg and 200 mg/kg doses produced a hepatoprotective effect; however, the 200 mg/kg dose provided better results, as reported by Bhaskar et al,[18] Studies have reported that both ethanolic and aqueous extracts of Pergularia daemia possess significant hepatoprotective effects, yet higher doses have the best outcome.[18,21] Administration of solvent extracts of Pergularia daemia prevents inflammation and various diseases, including arthritis and cancer. The ethanolic extracts not only reduced the SGOT, SGPT, and ALP but also the total bilirubin, total protein, total albumin, and total cholesterol levels.[21] Plants such as Phyllanthus niruri, Andrographis paniculata, and Azadirachta indica have a similar antioxidant pathway for producing hepatoprotective effects.[22-24] Phytochemical analyses of these species have revealed the presence of constituents including carbohydrates, cardiac glycosides, amino acids, flavonoids, alkaloids, phenols, saponins, steroids, and tannins. Further, a significant positive correlation was reported between antioxidant activity and polyphenolic content.[22] The dose-dependent character of EEPD in our study can also be due to this phenomenon.

The limitations of the present study are that oxidative stress markers such as malondialdehyde, glutathione, catalase, and superoxide dismutase were not measured. We did not evaluate the dose-response beyond 200 mg/kg, which may have helped to identify the maximum effective dose.

Certain herbs used in the Indian system of medicine are claimed to possess protective activity against liver diseases. In the modern scientific era, such claims require systematic evaluation. Hence, the present study assessed the hepatoprotective effects of Pergularia daemia. The extract proved a clear protective effect, with the 100 mg/kg dose (Test-1) showing improvements comparable to the standard silymarin-treated group. The 200 mg/kg dose (Test-2) produced results better than the standard, with enzyme levels reaching near normal values.

Histopathological findings supported these biochemical results, showing better preservation of hepatic architecture in the 200 mg/kg EEPD group compared with the 100 mg/kg group. The hepatoprotective activity of Pergularia daemia is probably due to the presence of flavonoids, which are known to reduce oxidative stress, stabilise cellular membranes, and protect hepatocytes from toxin-induced injury.

CONCLUSION

EEPD offers significant hepatoprotective effects against D-galactosamine-induced liver damage, with the 200 mg/kg dose displaying superior protective activity. These findings support its potential as a natural hepatoprotective agent, possibly attributable to its flavonoid-rich antioxidant profile.

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