

Original article

Ameliorative effect of the ethanolic extract of *Nardostachys jatamansi* against high-fat diet-induced dyslipidemia in Wistar albino rats

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Abstract

Background: Dyslipidemia is a key contributor to metabolic disorders such as oxidative stress and is a major risk factor for cardiovascular diseases. Herbal interventions are increasingly preferred due to fewer side effects. *Nardostachys jatamansi* has demonstrated antioxidant and lipid-lowering properties, but its effects on high-fat diet (HFD)-induced dyslipidemia remain insufficiently studied.

Objective: The present study aimed to evaluate the antioxidant and hypolipidemic effects of the ethanol extract of *N. jatamansi* in an HFD-induced obesity model in male Wistar albino rats.

Methods: Thirty-six male Wistar albino rats were randomly divided into 6 groups, each comprising 6 animals. Group I received a normal diet for 9 weeks, whereas Group II was fed an HFD for the same period. Groups III–VI were also fed an HFD throughout the investigation period. From week 5, Group III received rosuvastatin (10 mg/kg), and Groups IV–VI were treated with *N. jatamansi* extract at 100, 200, and 300 mg/kg, respectively, for 4 weeks. All treatments were administered orally. At the end of the study, the animals were fasted overnight, anesthetized, and blood was collected for lipid and hematological analyses. In addition, the organs were excised for histological evaluation.

Results: *N. jatamansi* (100, 200, and 300 mg/kg) reversed HFD-induced increases in body weight, fat pad size, organ weight, total cholesterol, triglycerides, low-density lipoprotein, and very low-density lipoprotein levels while restoring high-density lipoprotein levels in a dose-dependent manner. Moreover, elevated white blood cells, lymphocyte counts, and serum urea, creatinine, and uric acid levels were normalized. Histological improvements in the liver suggest antioxidant and bioactive effects of *N. jatamansi*.

Conclusion: Our results revealed that the herbal drug *N. jatamansi* effectively ameliorates HFD-induced dyslipidemia through its potent antioxidant activity, thereby reducing oxidative stress and restoring lipid homeostasis.

Keywords: Dyslipidemia, ethanol extract of *Nardostachys jatamansi*, high-fat diet, obesity, rosuvastatin.

Obesity is recognized as one of the most widespread metabolic disorders worldwide,⁽¹⁾ with dyslipidemia being a major related complication. While obesity has multifactorial origins, consumption of a high-fat diet (HFD) remains a key driver. Even without visible obesity, excessive dietary fat promotes lipid accumulation in adipose and nonadipose tissues, triggering cellular damage and death through lipotoxicity.⁽²⁾ Cardiovascular diseases are the primary causes of morbidity and mortality linked to dyslipidemia.⁽³⁾ In light of the increasing worldwide

reliance on HFDs, there is a pressing need to explore natural compounds or dietary supplements that can mitigate or prevent their harmful metabolic effects.

Nardostachys jatamansi, commonly known as *jatamansi*, is a well-known natural herb that is widely recognized in orthodox systems such as Ayurveda and valued for its diverse therapeutic effects.⁽⁴⁾ Studies have highlighted its pharmacological activities, including lipid-lowering,⁽⁵⁾ hematopoietic,⁽⁶⁾ antipyretic,⁽⁷⁾ cardioprotective,⁽⁸⁾ and hepatoprotective⁽⁹⁾ properties. These benefits are largely due to its rich phytochemical content, such as lignans, sesquiterpenoids, neolignans, and terpenoids,⁽¹⁰⁾ which possess strong antioxidant potential against oxidative stress and dyslipidemia. The present study aims to evaluate the efficacy of the ethanolic extract of *N. jatamansi* against HFD-induced dyslipidemic rats. In addition, the study aimed

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to assess its effect on lipid profile, oxidative stress, and histopathological changes.

Materials and methods

Animals

Male adult Wistar rats (6 weeks old, weighing 250 ± 20 g) were obtained from an authorized breeder. Before the experiment, the animals were acclimatized for 1 week under standard laboratory conditions, with a temperature of $25 \pm 2^\circ\text{C}$, relative humidity of 50.0–60.0%, and a 12/12 h light/dark cycle. Rats had *ad libitum* access to a standard pellet diet and water. Male Wistar rats were selected for this study because of their well-established susceptibility to diet-induced obesity and associated metabolic alterations.

Ethical approval

All experimental procedures were performed in accordance with the guidelines of the Committee for the Control and Supervision of Experiments on Animals, Government of India. The study protocol was reviewed and approved by the Institutional Animal Ethics Committee (approval number 01/IAEC/2023).

Induction of the HFD

The HFD used for the study contained normal laboratory food 67.5%, lard 31.0%, cholesterol 1.0%, di-methionine 0.3%, yeast powder 0.1%, and sodium chloride 0.1%. The HFD consisted of carbohydrates (17.0%), protein (25.0%), and fat (58.0%) as percentages of the total kcal.⁽¹¹⁾

Ethanol extract preparation of *N. jatamansi*

Air-dried *N. jatamansi* powder (100 g) was defatted using petroleum ether in a Soxhlet extractor for 7 days. The dried powder was then extracted with absolute ethanol until fully extracted. The extract was then concentrated under reduced pressure on a rotary evaporator, and the residual ethanol was removed. The final ethanolic extract was stored in a desiccator after noting its color consistency.⁽¹²⁾

Experimental protocol

Thirty-six male Wistar albino rats were randomly divided into 6 groups with 6 animals in each, such that the weight difference within and between groups did not exceed $\pm 20.0\%$ of the average weight of the total rats. Drugs were administered orally. The experimental design is as follows:

- **Group I (Control):** normal diet for 9 weeks
- **Group II (HFD control):** HFD for 9 weeks
- **Group III (Standard drug):** HFD for 9 weeks and treated with rosuvastatin (10 mg/kg body weight (BW)) for the last 4 weeks
- **Group IV (NJ-LD):** HFD for 9 weeks and treated with *N. jatamansi* extract (100 mg/kg BW) for the last 4 weeks
- **Group V (NJ-MD):** HFD for 9 weeks and treated with *N. jatamansi* extract (200 mg/kg BW) orally for the last 4 weeks
- **Group VI (NJ-HD):** HFD for 9 weeks and treated with *N. jatamansi* extract (300 mg/kg BW) for the last 4 weeks

Animals were observed daily for signs of toxicity throughout the experiment.

Parameters measured

The parameters measured on different occasions during the experimental period are as follows:

- **Measurement of anthropometric parameters (BW and abdominal girth)**

BW was measured at the beginning of the study, after 5 weeks, and before sacrifice, using a digital balance. For abdominal circumference measurement, the rat was positioned supine. Determination was made at the midline of the abdomen, i.e., at the level of the kidneys, and measured as an indicator of abdominal fat.⁽¹³⁾

- **Analysis of blood samples and collection of organs for biochemical parameters**

At the end of week 9, animals were fasted overnight and anesthetized with diethyl ether (30–40 s). Blood was collected via the retro-orbital plexus using microcapillary tubes, and serum was separated (centrifuged at 2500 rpm for 10 min) and stored for biochemical analysis.⁽¹⁴⁾ Rats were then sacrificed by cervical dislocation. Thereafter, the liver, kidneys, and adipose tissues were excised, washed with ice-cold saline, dried, weighed, and a portion was fixed in 10.0% formalin for histological analysis.

- **Analysis of biochemical indicators**

Estimation of serum biochemical markers

Serum levels of urea, uric acid, and creatinine were measured.⁽¹¹⁾ Total protein content was determined using the method of Lowry OH, et al.⁽¹⁵⁾

Estimation of the lipid profile

The serum levels of total cholesterol (TC),⁽¹⁶⁾ triglycerides (TG), and high-density lipoprotein (HDL)

were estimated.⁽¹⁷⁾ Friedewald's formula was used to estimate the low-density lipoprotein (LDL) and very LDL (VLDL) levels.⁽¹⁸⁾

Histological analysis

Liver tissue was fixed in 10% formalin, dehydrated with alcohol, embedded in paraffin, sectioned (4–5 μ m) using a rotary microtome, and stained with hematoxylin and eosin for histological evaluation.⁽¹⁹⁾

Statistical analysis

The results are presented as the mean $\pm$ standard deviation (SD). Differences between groups were analyzed using one-way analysis of variance, followed by Tukey's post hoc test for multiple comparisons, in SPSS version 19. $P < 0.05$ was considered statistically significant.⁽²⁰⁾ Graphs were generated using GraphPad Prism software.

Results

Effect of *N. jatamansi* on BW changes in rats

The mean BW of HFD Group II was significantly higher than that of the normal control Group I ($P < 0.0001$). Treatment with HFD + rosuvastatin (10 mg/

kg, Group III) significantly reduced the BW compared to the HFD Group II after 4 weeks of treatment ($P < 0.0001$). The NJ-treated groups (Groups IV–VI) also revealed significant reductions in BW relative to Group II ($P < 0.0001$). Among these, Group VI (300 mg/kg) exhibited the greatest effect, with a highly significant reduction in BW even when compared to the rosuvastatin group (Group III) ($P < 0.0001$) (Figure 1).

Effect of *N. jatamansi* on fat pad body circumference

The mean fat pad circumference was significantly higher in HFD Group II than in the normal control Group I. After 9 weeks of treatment, Group III (HFD + rosuvastatin, 10/ mg/kg) showed a significant reduction in fat pad circumference compared to Group II. Similarly, NJ-treated groups (Groups IV–VI) exhibited a significant decrease in fat pad circumference compared with Group II. Notably, Group VI (300 mg/kg) showed a highly significant reduction compared with Group II and the rosuvastatin-treated Group III (Table 1). These findings indicate that *N. jatamansi* effectively reduces lipid accumulation in body fat pads and abdominal regions.

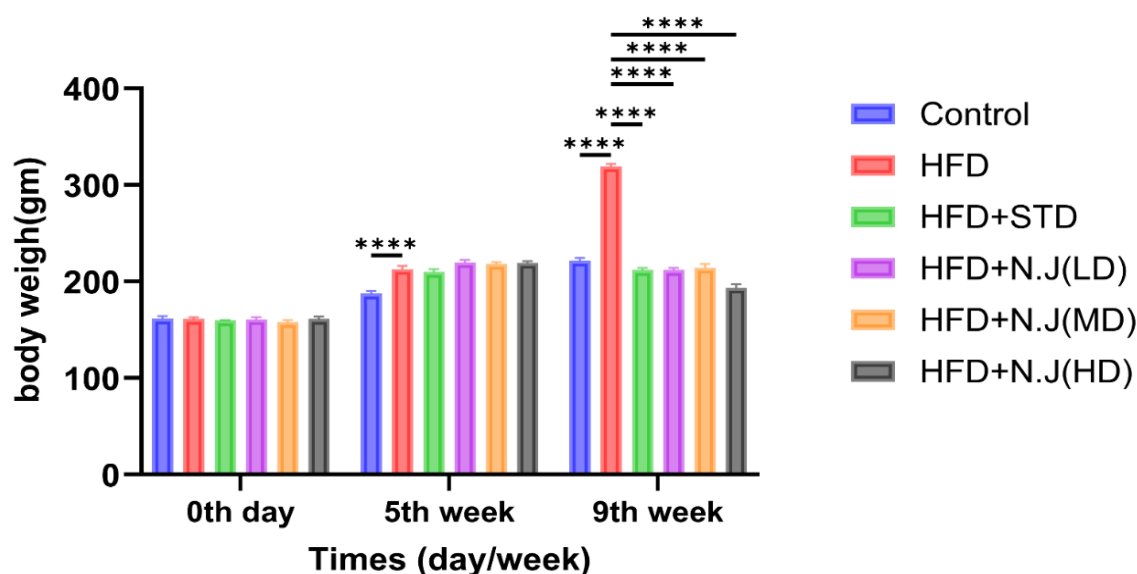


Figure 1. Effect of *N. jatamansi* on body weight changes: Body weight changes were assessed in normal control, HFD, HFD + rosuvastatin (10 mg/kg), and HFD treated with *N. jatamansi* at low (LD; 100 mg/kg), medium (MD; 200 mg/kg), and high (HD; 300 mg/kg) doses. Data are presented as mean $\pm$ standard error of the mean (SEM) (n = 6). Statistical analysis involved two-way ANOVA followed by Dunnett's test comparison test. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$ vs. HFD. HD, high dose; HFD, high-fat diet; LD, low dose; MD, medium dose.

Table 1. Effect of *N. jatamansi* on fat pad circumference and organ weight changes in dyslipidemia-induced rats

Groups	Control	Only HFD	HFD+STD	HFD+NJ (LD)	HFD+NJ (MD)	HFD+NJ (HD)
Fat pad circumference (mm/cm)	16.0±1.9	21.0±2.6**	17.0±2.0 ^c	14.0±1.9 ^a	12.0±2.1 ^a	11.0±2.1 ^a
Liver weight (g)	8.6±1.5	10.7±0.9	7.8±1.4 ^b	8.5±1.2	7.9±1.1 ^b	6.4±0.8 ^a
Kidney weight (g)	2.1±0.4	3.0±0.5**	1.6±0.2 ^a	1.8±0.2 ^a	1.7±0.4 ^a	1.6±0.3 ^a
Adipose tissue weight (g)	3.2±1.1	5.5±0.9**	2.3±1.1 ^a	2.9±1.0 ^b	2.1±0.5 ^a	1.7±1.1 ^a

Mean ± SEM (n = 6). One-way ANOVA followed by Dunnett's comparison test. Significant changes are calculated as **** $P < 0.0001$; *** $P < 0.001$; ** $P < 0.01$ and; * $P < 0.05$ vs. control group. ^a $P < 0.0001$; ^b $P < 0.01$; ^c $P < 0.001$ vs. HFD group. Values with $P < 0.05$ were considered statistically significant. HD, high dose; HFD, high-fat diet; LD, low dose; MD, medium dose; NJ, *N. jatamansi*.

Effect of *N. jatamansi* on organ weight

Liver, kidney, and adipose tissue weights were significantly increased in the HFD group (Group II) compared to the normal control (Group I). Treatment with rosuvastatin (Group III) significantly reduced organ weights compared with Group II ($P < 0.01$). The NJ-treated groups (Groups IV–VI; 100, 200, and 300/ mg/kg, respectively) also exhibited reductions in organ weights relative to Group II. Notably, Group VI (300 mg/kg) exhibited significantly greater reductions than Group IV (100 mg/kg) (Table 1). These findings suggest that *N. jatamansi* exerts hepatoprotective and nephroprotective effects in HFD-fed rats.

Effect of *N. jatamansi* on hematological parameters

Significantly decreased red blood cell (RBC) counts and increased lymphocyte counts were observed in HFD rats (Group II) compared to the control Group I. Rats treated with *N. jatamansi* (Groups IV–VI; 100, 200, and 300/ mg/kg, respectively) showed a significant increase in RBC count compared to the HFD Group II, indicating a potential hematopoietic effect of *N. jatamansi*. Although the white blood cell (WBC) and hemoglobin levels showed trends toward normalization in the NJ-treated groups, these changes were not statistically significant. A significant reduction in lymphocyte percentage was observed in the NJ-treated groups compared with the HFD Group II. At the same time, hemoglobin levels were significantly elevated in the NJ-treated groups (Groups IV–VI) compared to Group II (Figure 2). These findings suggest that *N. jatamansi* may act as a nontoxic hematopoietic and immunomodulatory agent.

Effect of *N. jatamansi* on the lipid profile

At the end of week 5, HFD feeding resulted in a significant increase in LDL, VLDL, TG, and TC levels ($P < 0.0001$), along with a significant decrease in HDL levels ($P < 0.05$) compared to the control Group I. By the end of week 9, the HFD-administered Group II showed significant increases in LDL, VLDL, TG, and TC ($P < 0.001$) and a decrease in HDL levels ($P < 0.05$) compared to the control Group I. Treatment with *N. jatamansi* at low dose (LD, Group IV), medium dose (MD, Group V), and high dose (HD, Group VI) resulted in a significant decrease in TC, TG, LDL, and VLDL levels ($P < 0.001$) compared to the HFD control group (Group II). Furthermore, the NJ-treated groups showed a notable increase in HDL levels, with stronger effects observed at MD and HD ($P < 0.001$), indicating the potent hypolipidemic activity of *N. jatamansi* (Figure 3).

Effect of *N. jatamansi* on serum biochemical markers

In the HFD control Group II, significant increases in protein, creatinine, urea, and uric acid were observed compared with Group I. Rats treated with *N. jatamansi* (Groups IV–VI; 100, 200, and 300 mg/kg, respectively) and rosuvastatin Group III showed reductions in serum protein, creatinine, urea, and uric acid levels compared with the HFD Group II. The extract demonstrated dose-dependent protective effects comparable to those of the standard drug, suggesting its potential as a hypolipidemic and renoprotective agent (Table 2).

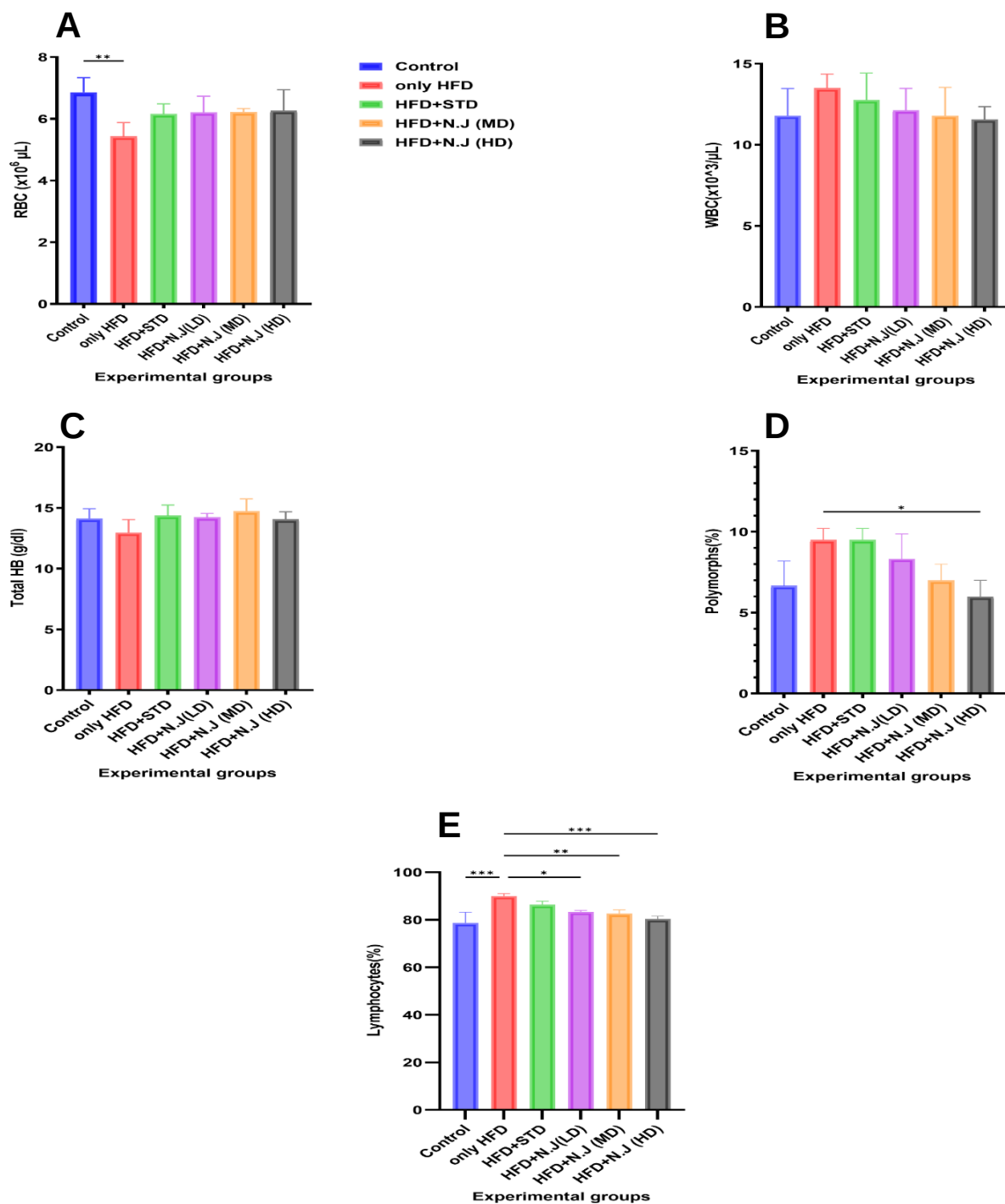


Figure 2. Effect of *N. jatamansi* on haematological parameter values in rats. **(A)** Red blood cell (RBC) count, **(B)** white blood cell (WBC) count, **(C)** hemoglobin concentration, **(D)** polymorph percentage, and **(E)** lymphocyte percentage in control, HFD, HFD + standard drug, and HFD + *N. jatamansi*-treated groups (LD, MD, HD). Mean $\pm$ SEM (n = 6). One-way ANOVA followed by Dunnett's multiple comparison test. Significant changes are calculated as * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; and **** $P < 0.0001$ vs. HFD group. Values with $P < 0.05$ were considered statistically significant. HD, high dose; HFD, high-fat diet; LD, low dose; MD, medium dose; SEM, standard error of the mean.

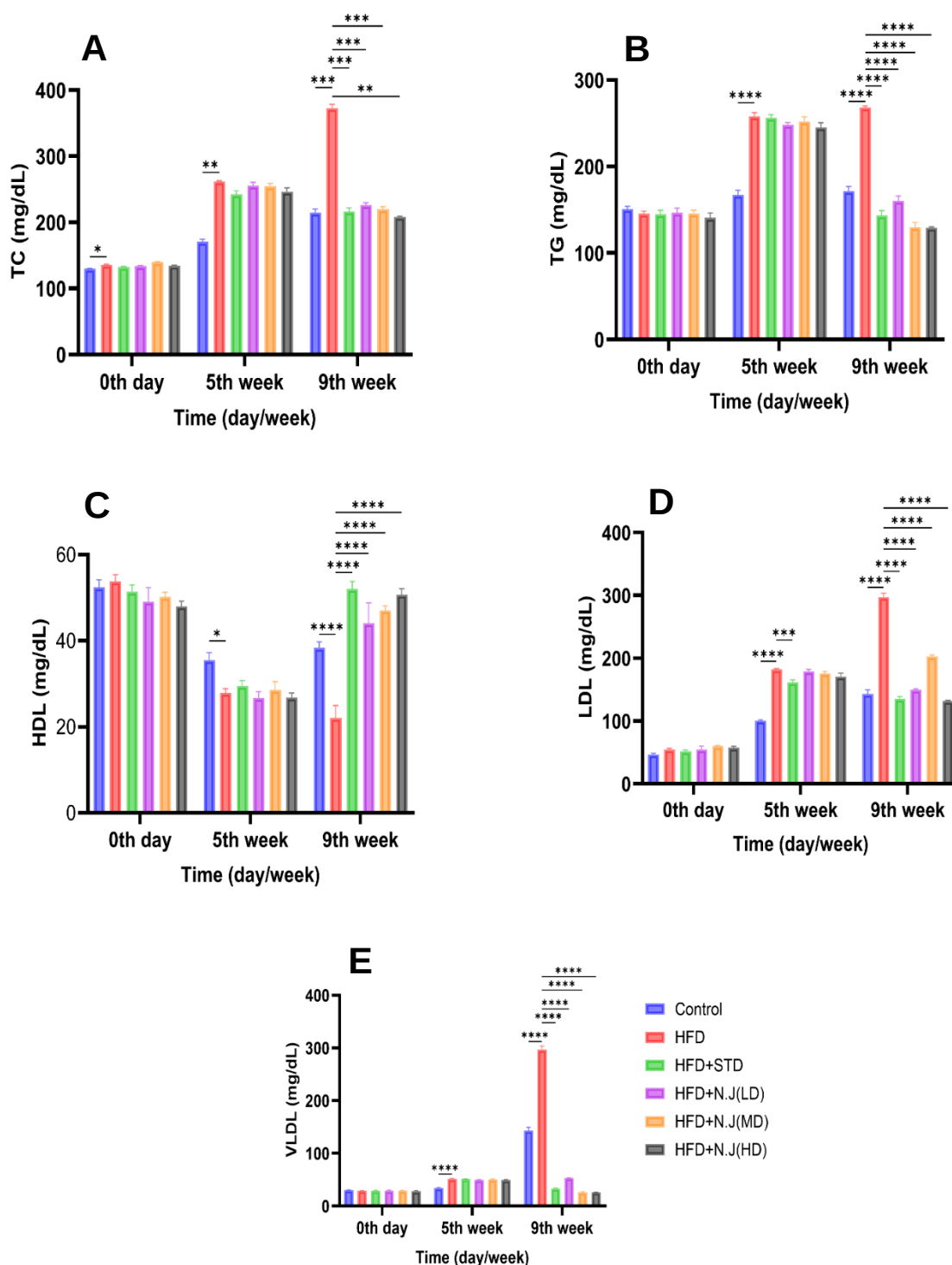


Figure 3. Effect of *N. jatamansi* on serum lipid profile in HFD-fed rats. (A) TC, (B) TG, (C) HDL-C, (D) LDL-C, and (E) VLDL-C measured at 0th day, 5th week, and 9th week. Data are mean $\pm$ SEM (n = 6). Two-way ANOVA followed by Dunnett's multiple comparison test. $P < 0.05$; $**P < 0.01$; $***P < 0.001$; and $****P < 0.0001$ vs. HFD group. Values with $P < 0.05$ were considered statistically significant. HDL, high density lipoproteins; LDL, low density lipoproteins; TC, total cholesterol; TG, triglycerides; SEM, standard error of the mean; VLDL, very low-density lipoproteins.

Table 2. Analysis of the effect of NJ on serum biochemical markers of various groups.

Groups	Control	Only HFD	HFD+STD	HFD+NJ LD	HFD+NJ MD	HFD+NJ HD
Serum protein (mg/dl)	1.8 ± 0.9	8.9 ± 0.7***	1.6 ± 0.9 ^a	5.7 ± 0.6 ^a	1.4 ± 0.5 ^a	1.1 ± 0.2 ^a
Serum urea (mg/dl)	32.7 ± 3.5	105.0 ± 7.0***	44.0 ± 8.0 ^a	65.7 ± 0.6 ^a	40.7 ± 5.5 ^a	34.7 ± 6.7 ^a
Creatinine (mg/dl)	0.6 ± 0.1	1.3 ± 0.5	0.5 ± 0.1 ^c	0.9 ± 0.3	0.6 ± 0.2	0.6 ± 0.3
Uric acid	1.3 ± 0.4	7.9 ± 1.0***	1.7 ± 1.0 ^a	4.1 ± 0.7 ^a	1.6 ± 0.2 ^a	1.5 ± 0.5 ^a

Mean ± SD (n = 6). One-way ANOVA followed by Dunnett's comparison test. Significant changes are calculated as *** $P < 0.001$; ** $P < 0.01$; and * $P < 0.05$ versus control group. ^a $P < 0.0001$; ^b $P < 0.01$; ^c $P < 0.001$ vs. HFD group. Values with $P < 0.05$ were considered statistically significant. HD, high dose; HFD, high-fat diet; LD, low dose; MD, medium dose; NJ, *N. jatamansi*; SD, standard deviation.

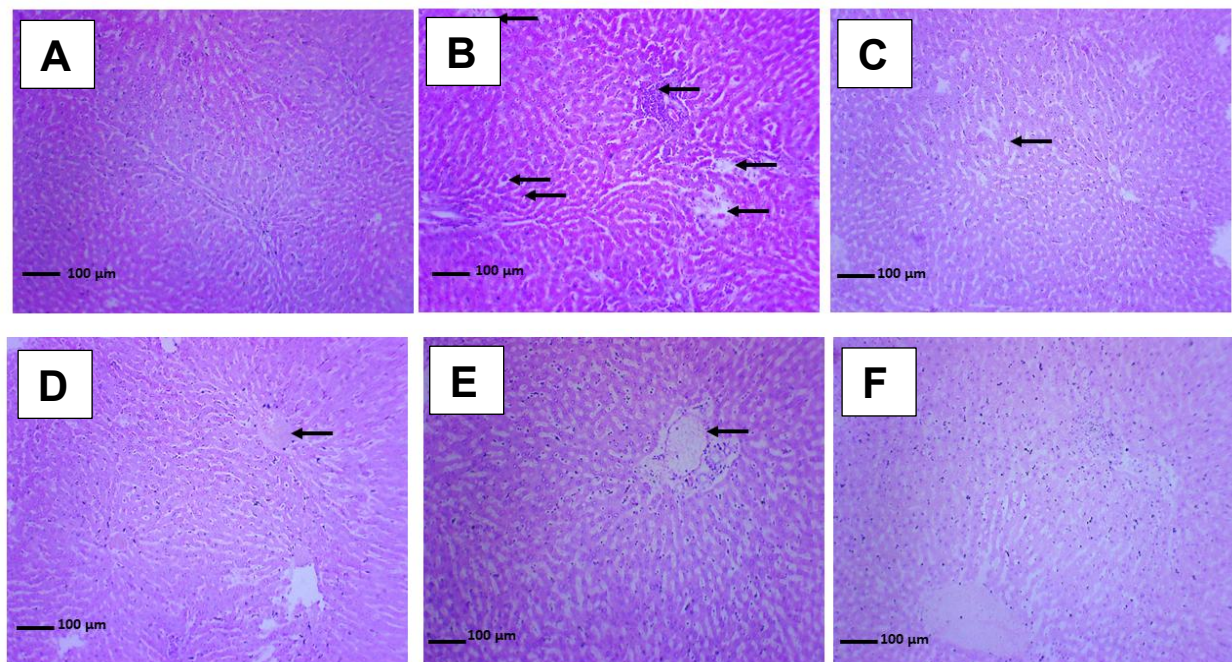


Figure 4. Representative H&E-stained liver sections. (A) Normal control with intact hepatocyte architecture, (B) HFD group showing fatty changes, structural disruption, inflammation, sinusoidal widening, and hepatocyte ballooning, (C) HFD+ STD group showing improved architecture, (D - F) NJ-treated groups demonstrating progressive restoration. Arrows indicate inflammatory cell infiltration, sinusoidal widening, and hepatocellular ballooning in the HFD group. Scale bar = 100 µm. HFD, high-fat diet; NJ, *N. jatamansi*; STD, standard drug.

Effect of *N. jatamansi* on histopathology

Histopathological analysis of the liver tissue was performed in all groups to assess the cytoprotective effect of *N. jatamansi* against HFD-induced damage. Liver sections from normally fed rats (Group I) displayed normal hepatocyte architecture. In contrast, rats subjected to HFD control (Group II) showed signs of fatty liver, disrupted parenchymal structure, and

multiple hepatocellular abnormalities, including central vein dilatation and congestion, periportal inflammation, sinusoidal widening, and hepatocyte ballooning. However, liver tissues from rats treated with the ethanolic extract of *N. jatamansi* (Groups IV–VI) and rosuvastatin (Group III) exhibited notable histological improvement and restoration of hepatocyte architecture (**Figure 4A–F**)

Discussion

Dyslipidemia is characterized by abnormal lipid levels in the bloodstream and is a key contributor to the development of various metabolic disorders.⁽²¹⁾ Although statins and fibrates are widely used as effective hypolipidemic agents, their clinical utility is often restricted by adverse effects such as myalgia, elevated liver enzymes, cholelithiasis, and increased diabetes risk at higher doses, as noted by Hu Y, *et al.*⁽²²⁾

Hence, the present study evaluated the antidyslipidemic effects of *N. jatamansi* by assessing several biologically relevant parameters. In our study, the HFD led to increased BW and abdominal girth, indicating overall visceral fat accumulation. This finding is consistent with previous studies, which suggested that dietary fat delays gastric emptying, stimulates gastrointestinal hormone release, and consequently promotes fat deposition and obesity.⁽²³⁾

Treatment with *N. jatamansi* significantly reduced BW in HFD-fed rats, thereby suggesting its therapeutic efficacy. Similar outcomes reported by Subashini, *et al.* attributed the benefits of *N. jatamansi* to its influence on lipid metabolism and antioxidant potential, likely mediated by its various bioactive phytochemicals.⁽⁵⁾

In addition, in our study, the HFD-fed rats revealed increased liver, kidney, and adipose tissue weights, and these results are consistent with the observations by Rospond B, *et al.*⁽²⁴⁾ Furthermore, a study by Furukawa, S *et al.*⁽²⁵⁾ linked organ enlargement to oxidative stress and dysregulated adipocytokine activity. Overall, our results highlight the hypolipidemic and anti-obesity potential of *N. jatamansi* in mitigating diet-induced metabolic dysfunction.

Baker identified elevated serum uric acid as a strong predictor of obesity.⁽²⁶⁾ Tsushima Y, *et al.* showed that HFDs increase uric acid levels due to fat accumulation and related metabolic changes, such as insulin resistance and chronic inflammation, which disrupt uric acid metabolism and impair renal excretion. These diets may also increase uric acid production via xanthine oxidase activity while reducing the kidney's ability to eliminate uric acid and creatinine.⁽²⁷⁾ Consistent with these findings, our study observed elevated levels of uric acid, urea, and creatinine in the HFD group (Group II). In contrast, the NJ-treated groups (Groups V–VII) showed a significant reduction in these markers.

Unruh, *et al.* reported that HFDs elevate phosphatidylserine and cholesterol levels in RBC membranes, triggering chemokine release and subsequent macrophage activation, thereby initiating inflammation and increasing WBC and lymphocyte counts.⁽²⁸⁾ In contrast, the NJ-treated groups in our study showed increased RBC and hemoglobin levels, indicating enhanced erythropoiesis, along with reduced WBC and lymphocyte counts, which suggests potential anti-inflammatory or immunomodulatory effects.⁽⁶⁾

Dyslipidemia is closely associated with obesity and leads to coronary artery disease. The main characteristics of dyslipidemia are increases in TC, TG, LDL, and VLDL and decreases in HDL levels. According to Wang, *et al.*, an HFD disrupts lipid homeostasis by increasing intestinal cholesterol absorption, stimulating hepatic cholesterol synthesis, and impairing LDL clearance by downregulating LDL receptors, thereby elevating LDL, VLDL, and TC levels.⁽²⁹⁾ Consistent with these findings, our study observed significant increases in TC, TG, LDL, and VLDL in the HFD group (Group II). Notably, treatment with *N. jatamansi* (Groups IV–VI) effectively reversed these alterations, likely due to its antioxidant properties that mitigate oxidative stress.⁽³⁰⁾

Liver tissues from the HFD group exhibited marked histopathological alterations, including disrupted parenchymal architecture, central vein dilatation and congestion, periportal inflammation, sinusoidal widening, and hepatocyte necrosis. In contrast, treatment with *N. jatamansi* (Groups IV–VI) effectively restored hepatic architecture, closely resembling that of the normal control group (Group I). *N. jatamansi* exhibited better results at 300 mg/kg than at other doses.

N. jatamansi is recognized for its ability to lower blood lipid levels, particularly TC and TG, which are key contributors to cardiovascular disease. Its lipid-lowering effect is mediated by the regulation of liver enzymes involved in lipid synthesis and breakdown. In preclinical studies, the oral administration of *N. jatamansi* extract at 500 mg/kg for 7 days before doxorubicin exposure resulted in notable improvements in lipid profiles and restoration of lipid-metabolizing enzyme activity.^(5, 8) Our histological examination of the liver tissues further supported these findings, indicating the extract's protective and regulatory role in lipid metabolism.

N. jatamansi has demonstrated considerable potential in managing hyperlipidemia via its antioxidant

and lipid-lowering properties. The ethanolic extract of *N. jatamansi* reduced lipid accumulation in liver tissue and improved serum lipid profiles, particularly by increasing HDL levels in rats fed an HFD. These effects were dose-dependent and likely attributable to the active phytochemicals present, including lignans, alkaloids, phenols, and tannins. In addition, *N. jatamansi* may stimulate key enzymes such as lecithin-cholesterol acyltransferase and lipoprotein lipase, which play crucial roles in cholesterol transport and metabolism. Thus, *N. jatamansi* effectively counteracts diet-induced lipid abnormalities.

This study has certain limitations. It was conducted in a single animal model (male Wistar albino rats), which may limit direct extrapolation to human conditions. However, the HFD-induced dyslipidemia model used in this study is well established and widely accepted. The study duration was limited to 9 weeks, which may not fully capture the long-term effects. Nevertheless, the study duration was sufficient to demonstrate significant biochemical and histopathological improvements. In addition, the study primarily focused on biochemical and histopathological parameters and did not explore detailed molecular mechanisms. Future studies incorporating molecular and genetic analyses would provide deeper insights into the mechanism of action. Despite these limitations, this study provides important preliminary evidence supporting the antihyperlipidemic potential of *N. jatamansi*, thereby establishing a basis for further experimental and clinical investigations.

Conclusion

The findings of the current study revealed that *N. jatamansi* is effective in treating hyperlipidemia in HFD-induced rats, which demonstrates its potential antioxidant activity. Furthermore, its hepatoprotective effects are due to its important antioxidants, anti-inflammatory, and lipid-lowering properties.

Author contributions

UP contributed substantially to the study concept and design, data acquisition, literature review, analysis and interpretation of results, and critical editing of the manuscript for important intellectual content. GS contributed substantially to data acquisition. NS contributed to drafting the manuscript. All authors approved the final version submitted for publication and take responsibility for statements made in the published article.

Acknowledgments

Not applicable

Conflict of interest statement

The authors declare no conflict of interest.

Data sharing statement

The datasets used and/or analyzed in the current study are available from the corresponding author on reasonable request for noncommercial purposes.

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