



RESEARCH ARTICLE

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Investigation of Antioxidant and Antihyperlipidemic Activity of Barleria Terminalis in High Fat Diet Experimental Animals.

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ABSTRACT: The present study was aimed to investigate the antioxidant and antihyperlipidemic activity of Barleria terminalis in High Fat Diet (HFD) induced hyperlipidemic rats. Phytochemical screening revealed the presence of flavonoids, phenols, glycosides, tannins, saponins, and steroids. Treatment with Barleria terminalis extract significantly reduced total cholesterol, triglycerides, LDL, and VLDL while increasing HDL levels. The extract also improved antioxidant parameters and showed protective effects in histopathological studies. The study concluded that Barleria terminalis possesses significant antioxidant and antihyperlipidemic activity and may be useful in the management of hyperlipidemia.

Index Terms: High Fat Diet (HFD), Antioxidant Activity, Antihyperlipidemic Activity, Free Radicals, Hepatoprotective Activity, Nephroprotective Activity

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I. INTRODUCTION

Hyperlipidemia is a metabolic disorder characterized by elevated levels of triglycerides and cholesterol in the blood, increasing the risk of cardiovascular diseases and stroke. Modern antihyperlipidemic drugs produce several side effects; therefore, herbal medicines are considered safer alternatives. Barleria terminalis contains flavonoids, tannins, saponins, sterols, terpenoids, and phenolics which possess important medicinal and antihyperlipidemic activities^[1]

1.1 Classification of hyperlipidemia :

Hyperlipidemia is broadly classified into two main types:

- primary hyperlipidemia
- secondary hyperlipidemia

Primary hyperlipidemia mainly occurs due to genetic defects and may be monogenic or polygenic in nature. It includes disorders such as familial hypercholesterolemia, familial combined hyperlipidemia, and familial hypertriglyceridemia. Secondary hyperlipidemia develops as a result of other medical conditions such as diabetes mellitus, kidney disease, hypothyroidism, alcoholism, and the use of certain drugs like corticosteroids and beta blockers. Unhealthy lifestyle habits, especially consumption of high fat diet rich in saturated fats

and cholesterol, are considered major contributing factors for secondary hyperlipidemia.^[2]

1.2 Lipids

Lipids are fats present in the body that are transported through the blood by lipoproteins. These lipoproteins carry cholesterol and triglycerides to different tissues and organs. The major types of lipids include total cholesterol (TC), triglycerides, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL). Major lipids present in the body are cholesterol, triglycerides, phospholipids, and free fatty acids.^[3,4]

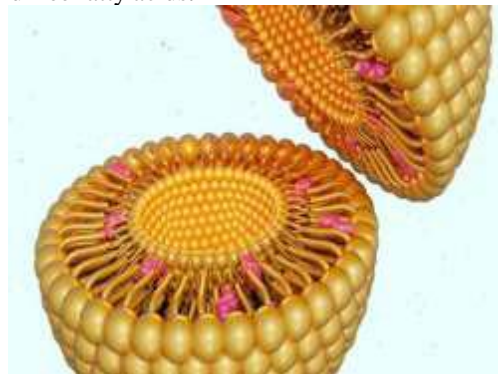


Fig.1: Lipids^[5]

Important lipoproteins are:

- Low Density Lipoprotein (LDL)



- High Density Lipoprotein (HDL)
- Very Low Density Lipoprotein (VLDL) [6]

Pathophysiology of Hyperlipidemia

- Hyperlipidemia is classified into primary and secondary types.
- Primary hyperlipidemia occurs due to genetic defects in lipid metabolism.
- Defect in lipoprotein lipase (LPL) activity causes increased triglycerides and chylomicrons in blood.

There are two types of Pathways

- Exogenous Pathway of Lipids
- Endogenous Pathway of Lipids [9]

Complications of Hyperlipidemia [10]

- Kidney disease
- Hypothyroidism
- Alcohol consumption
- Genetic factors
- Nephrotic syndrome
- Hypertension and stroke
- Cardiovascular diseases

Treatment of Hyperlipidemia [11]

Treatment of hyperlipidemia includes two approaches:

- ✧ Non-Pharmacological Therapy
- ✧ Pharmacological Therapy

Different classes of drugs used for treatment include: [12,13]

- HMG-CoA reductase inhibitors (Statins)
- Bile acid sequestrants
- Fibrates

Table no. 1 Classification of Plasma lipid level
[14, 15]

Total Cholesterol	Values
LDL Cholesterol	< 100mg / dL Optimal 100 - 129mg / dL Near/above Optimal 130 - 159mg / dL Borderline high 160 - 189mg / dL High > 190mg / dL

- Secondary hyperlipidemia develops due to diseases like diabetes, hypothyroidism, liver disease, and nephrotic syndrome.

- Increased synthesis of VLDL and reduced cholesterol metabolism lead to elevated lipid levels. Excess lipid accumulation ultimately increases the risk of atherosclerosis and cardiovascular diseases. [7]

	Very high
Total Cholesterol	<200 mg/dL Desirable Borderline high 200-239 mg/dL High > 240mg / dL
HDL Cholesterol	< 40mg / dL Low 40 - 60mg / Normal dL High > 60mg / dL
Triglycerides	< 150mg / dL Normal Borderline 150 - 199mg / dL high High 200 - 499mg / dL Very high > 500mg / dL

II. PLANT PROFILE

Introduction to Barleria terminalis: Barleria terminalis is a medicinal plant belonging to the family Acanthaceae and is commonly found in tropical and subtropical regions. Traditionally, it has been used for the treatment of inflammation, wounds, infections, and pain. The



plant contains important phytoconstituents responsible for various pharmacological activities including antioxidant, antimicrobial, and antihyperlipidemic effects. Due to its medicinal importance, *Barleria terminalis* is considered a promising plant for further pharmacological research



Fig.3 : The plant of Barleria Terminalis

Scientific classification^[104]

Kingdom: Plantae
Subkingdom: Tracheobionta
Class: Magnoliopsida
Sub-class: Asteridae
Order: Scrophulariales
Family: Acanthaceae
Common name: Blue Barleria
Vernacular name: Mayurpunk , Kurubaksh
Habit: Shrub

Phytochemistry

The plant contains flavonoids, alkaloids, tannins, phenolic compounds, terpenoids, glycosides, and phytosterols responsible for various medicinal activities.

Pharmacological Activities

- Antioxidant activity
- Anti-inflammatory activity
- Antibacterial activity
- Antifungal activity
- Wound healing activity
- Antioxidant enzyme modulation.

III. MATERIALS AND METHODS

3.1 Collection and Authentication of Plant

The leaves of *Barleria terminalis* were collected from the Amravati District of Maharashtra, India. The plant material was identified and authenticated by the Department of Botany. The collected leaves were cleaned, shade-dried, and stored for further experimental studies.

3.2 Preparation of Plant Extract

The dried leaves were coarsely powdered and subjected to hydroalcoholic extraction using a Soxhlet apparatus. The extraction process was continued until complete exhaustion of the plant material. The obtained extract was concentrated under reduced pressure and stored in an airtight container for further pharmacological investigations.

3.3 Experimental Animals

Healthy Wistar albino rats weighing between 180–220 g were used for the study. The animals were housed under standard laboratory conditions of temperature, humidity, and a 12 h light/dark cycle, with free access to standard pellet diet and water. All experimental procedures were carried out in accordance with institutional ethical guidelines.

3.4 Induction of Hyperlipidemia

Experimental hyperlipidemia was induced by administering a high-fat diet (HFD) containing cholesterol and coconut oil for a period of 28 days. The HFD-fed animals exhibited elevated serum lipid levels and were used as the disease control model.

3.5 Experimental Design

The animals were randomly divided into five groups, each consisting of six rats.

- Group I: Normal Control
- Group II: Disease Control (HFD)
- Group III: Standard Drug (Atorvastatin, 10 mg/kg)
- Group IV: *Barleria terminalis* Extract (200 mg/kg)
- Group V: *Barleria terminalis* Extract (400 mg/kg)

The treatments were administered orally for 28 consecutive days, and the antihyperlipidemic activity of the extract was evaluated by assessing biochemical, antioxidant, and histopathological parameters.

3.5 Phytochemical Screening: Tests were performed for:

- Alkaloids
- Flavonoids
- Glycosides
- Tannins



- Saponins
- Phenols
- Proteins

3.6 Evaluation Parameters

- Body Weight
- Feed Intake
- Feed Efficiency Ratio
- Serum Lipid Profile
- Liver Function Tests
- Kidney Function Tests
- Oxidative Stress Parameters

- Hepatic Lipid Content

3.7 Histopathological Studies: Microscopic examination was performed for:

- Steatosis
- Inflammation
- Necrosis

3.8 Statistical Analysis

- Data were expressed as Mean \pm SD.
- Statistical analysis was performed using One-way ANOVA followed by Dunnett's test.

IV. RESULTS

4.1 Phytochemical screening

Table 2: Phytochemical screening of ethanolic extract of Barleria Terminalis stem.

Sr.no.	Chemical constituents	Name of the test	Result
1.	Alkaloids	Iodine Test Wagner's Test	+
2.	Saponin	Foam test	+
3.	Carbohydrates	Molish's test Barfoed's test	+
4.	Cardiac Glycosides	Kedde's test Legal test	+
5.	Flavonoids	Shinoda's test	+
6.	Reducing Sugars	Fehling's test	+
7.	Tannins	Ferric chloride test Gelatin test Lead acetate test	+

4.2. Physiological Parameters

- **Effect of Barleria Terminalis extract on the Body weight:**

Table 3: The weekly body weight measurements of control, HFD-induced, standard-treated, and extract-treated groups during the 28-day experimental study. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett's multiple comparison test.

Groups	Initial Day	Day 7	Day 14	Day 21	Day 28
Normal Control	141.00 \pm 0.26	146.00 \pm 0.26	151.00 \pm 0.26	155.00 \pm 0.26	159.00 \pm 0.26
Disease Control	141.00 \pm 0.26	191.00 \pm 0.26	221.00 \pm 0.26	246.00 \pm 0.26	271.00 \pm 0.26
Standard	141.00 \pm 0.26	161.00 \pm 0.26	166.00 \pm 0.26	171.00 \pm 0.26	175.00 \pm 0.26
Test Low Dose	141.00 \pm 0.26 ns	173.00 \pm 0.26 ****	183.00 \pm 0.26 ****	191.00 \pm 0.26 ****	199.00 \pm 0.26 ****
Test High Dose	141.00 \pm 0.26 ns	165.00 \pm 0.26 ****	171.00 \pm 0.26 ****	177.00 \pm 0.26 ****	182.00 \pm 0.26 ****

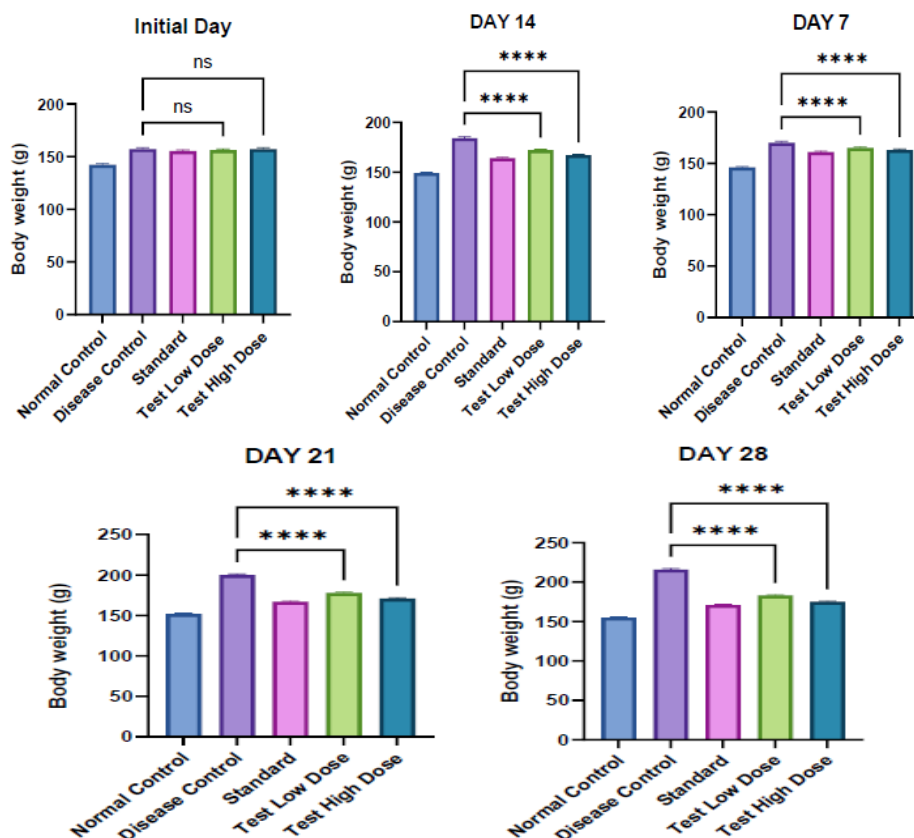


Figure 04: Effect of High Fat Diet (HFD), standard drug, and Barleria terminalis extract (200 mg/kg and 400 mg/kg) on body weight in HFD-induced hyperlipidemic rats during the 28-day experimental period. The HFD group showed a significant increase in body weight, whereas treatment with standard drug and extract significantly reduced body weight gain in a dose-dependent manner. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett's test. ****p < 0.0001 vs. induced/HFD group.

• **Effect of Treatment on Weekly Feed Intake in Hyperlipidemic Rats:**

Table 4: The effect of High Fat Diet (HFD), standard drug, and Barleria terminalis extract (200 mg/kg and 400 mg/kg) on weekly feed intake in hyperlipidemic rats during the 28-day experimental study. Values are expressed as mean \pm SEM (g/day, n = 6) and analyzed using one-way ANOVA followed by Dunnett's multiple comparison test.

Groups	Day 7	Day 14	Day 21	Day 28
Normal Control	8.20 \pm 0.03	8.40 \pm 0.03	8.60 \pm 0.03	8.70 \pm 0.03
Disease Control	12.90 \pm 0.03	14.00 \pm 0.03	14.90 \pm 0.03	15.60 \pm 0.03
Standard	10.10 \pm 0.03	9.80 \pm 0.03	9.10 \pm 0.03	8.90 \pm 0.03
Test Low Dose	11.20 \pm 0.03 ****	10.80 \pm 0.03 ****	10.30 \pm 0.03 ****	9.50 \pm 0.03 ****
Test High Dose	10.50 \pm 0.03 ****	9.90 \pm 0.03 ****	9.70 \pm 0.03 ****	8.80 \pm 0.03 ****

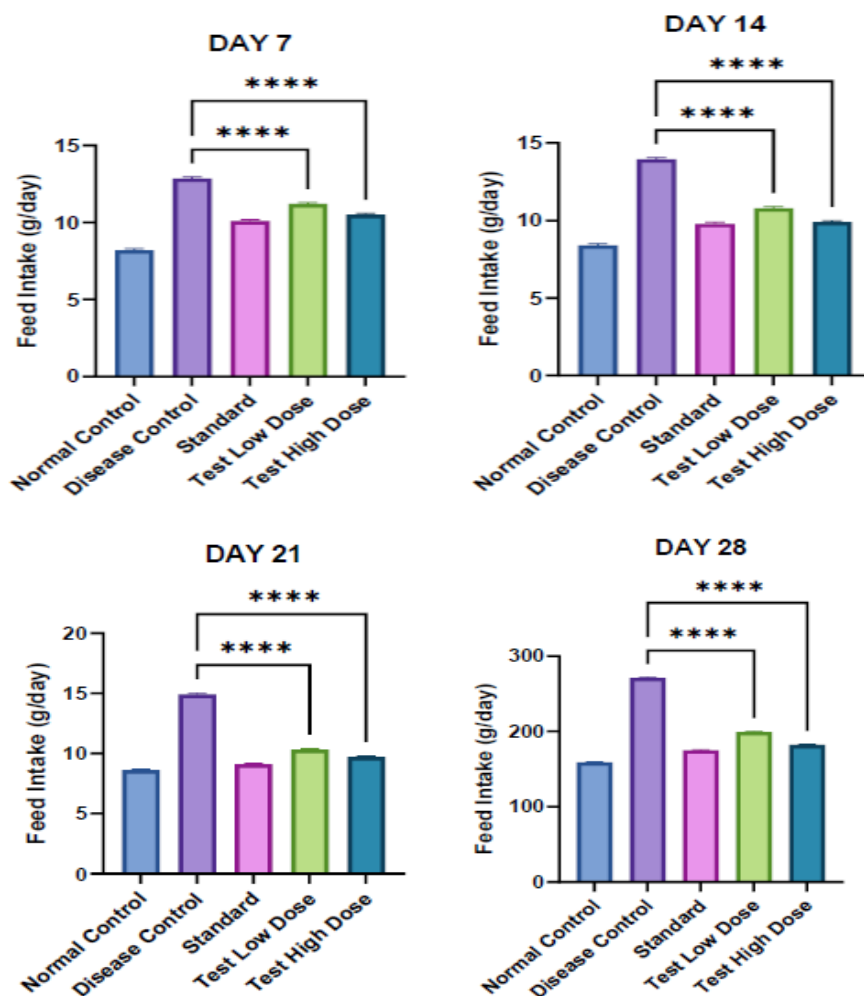


Figure 05: Effect of HFD, standard drug, and Barleria terminalis extract on feed intake in hyperlipidemic rats. The HFD group showed increased feed intake, while treatment groups showed significant reduction in feed intake. Values are expressed as mean \pm SEM (n = 6). ****p < 0.0001 vs. induced/HFD group.

• **Effect of Treatment on Feed Efficiency Ratio (FER) in Hyperlipidemic Rats**

Table 5: Effect of High Fat Diet (HFD), Barleria terminalis extract (200 mg/kg and 400 mg/kg), and standard drug on Feed Efficiency Ratio (FER) in HFD-induced hyperlipidemic rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett's test

Groups	FER
Normal Control	0.53 \pm 0.003
Disease Control	2.25 \pm 0.003
Standard	0.89 \pm 0.003
Test Low Dose	1.38 \pm 0.003 ****
Test High Dose	1.04 \pm 0.003 ****

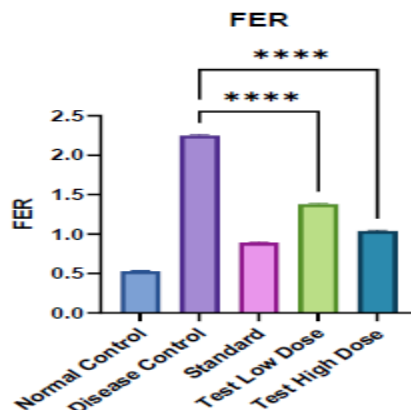
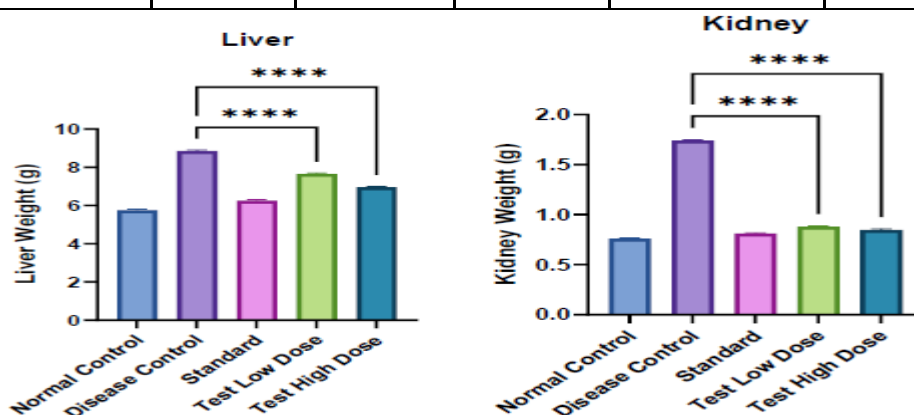


Figure 06: Effect of High Fat Diet (HFD), *Barleria terminalis* extract (200 mg/kg and 400 mg/kg), and standard drug on Feed Efficiency Ratio (FER) in hyperlipidemic rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett’s test. **** significant at $p < 0.0001$ vs. induced/HFD group.

• **Effect of Treatment on Organ Weights in Hyperlipidemic Rats:**

Table 6: Effect of High Fat Diet(HFD), *Barleria terminalis* extract(200mg/kg and 400mg/kg), and standard drug on organ weights [liver, kidney, and heart] in HFD-induced hyperlipidemic rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett’s test.

Organ	Normal Control	Disease Control	Standard	Test Low Dose	Test High Dose
Liver Weight (g)	5.75 \pm 0.02	8.85 \pm 0.02	6.25 \pm 0.02	7.65 \pm 0.02 ****	6.95 \pm 0.02 ****
Kidney Weight (g)	0.76 \pm 0.01	1.74 \pm 0.01	0.81 \pm 0.01	0.88 \pm 0.01 ****	0.85 \pm 0.01 ****
Heart Weight (g)	1.11 \pm 0.01	1.61 \pm 0.01	1.23 \pm 0.01	1.36 \pm 0.01 ****	1.21 \pm 0.01 ****



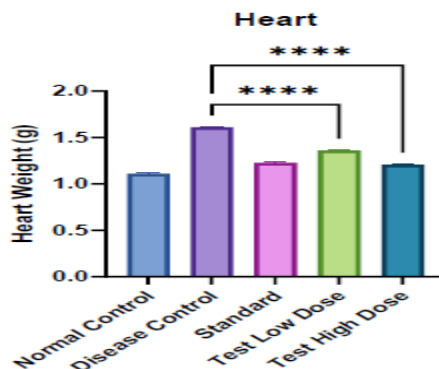
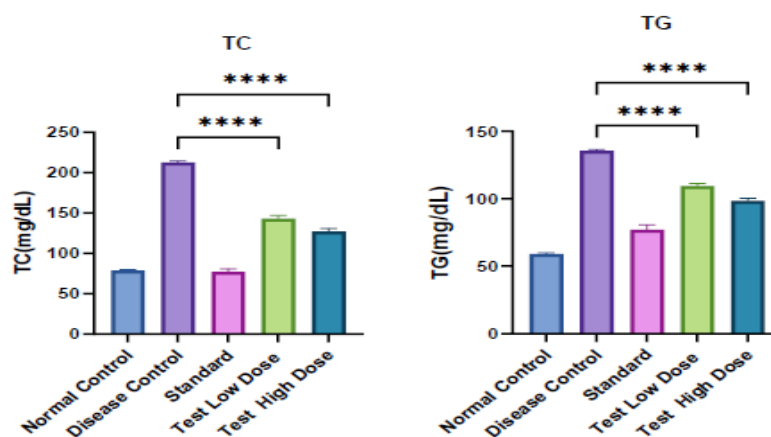


Figure 07: Effect of HFD, standard drug, and Barleria terminalis extract on liver, kidney, and heart weights in hyperlipidemic rats. The HFD group showed increased organ weights, while treatment groups showed significant reduction toward normal values. Values are expressed as mean \pm SEM (n = 6). ****p < 0.0001 vs. induced/HFD group.

•Effect of Barleria terminalis extract on serum lipid profile in HFD-induced hyperlipidemic rats: Table 7
: Effect of Barleria terminalis extract on serum lipid profile parameters in HFD-induced hyperlipidemic rats.

Parameters	Normal Control	Disease control	Standard	Test Low Dose	Test High Dose
TC (mg/dL)	79.33 \pm 0.21	203.67 \pm 0.21	76.50 \pm 0.76	142.50 \pm 0.76 ****	129.50 \pm 0.76 ****
TG (mg/dL)	59.00 \pm 0.37	135.50 \pm 0.43	77.00 \pm 1.53	109.50 \pm 0.76 ****	98.50 \pm 0.76 ****
HDL-c (mg/dL)	30.00 \pm 0.37	15.17 \pm 0.31	39.00 \pm 1.53	26.50 \pm 0.76 ****	30.50 \pm 0.76 ****
LDL-c (mg/dL)	74.00 \pm 0.37	120.50 \pm 0.76	35.00 \pm 1.53	47.00 \pm 1.53 ****	41.00 \pm 1.53 ****
VLDL-c (mg/dL)	12.00 \pm 0.37	28.50 \pm 0.43	19.00 \pm 1.53	22.50 \pm 0.76 ***	19.50 \pm 0.76 ****



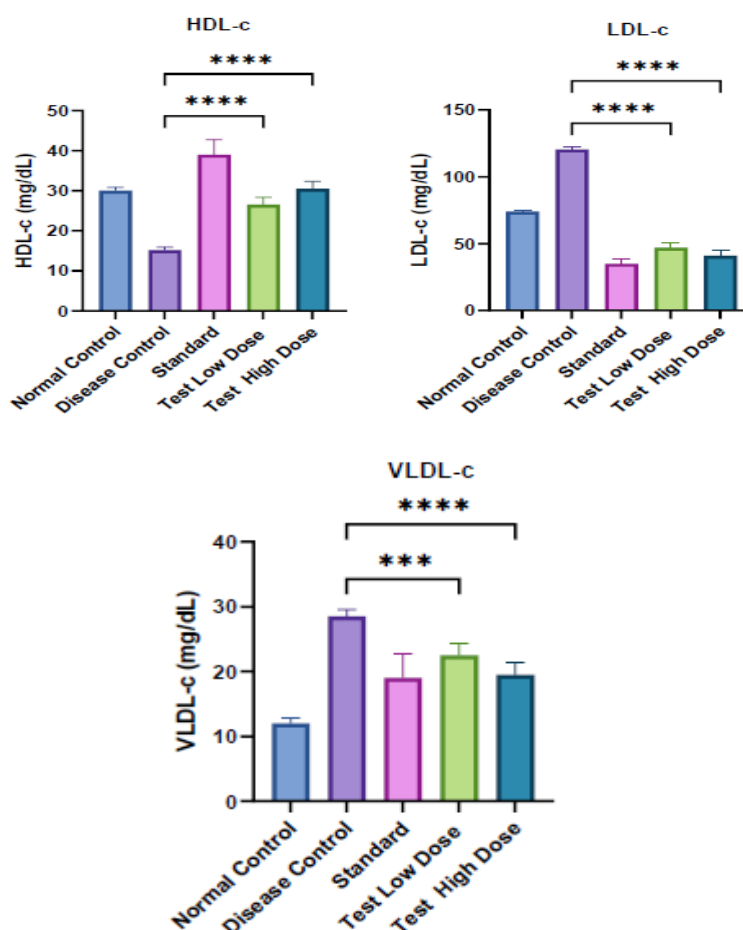


Figure 08: Effect of High FatDiet(HFD),Barleria terminalis extract (200mg/kg and 400 mg/kg), and standard drug on serum lipid profile parameters [Total Cholesterol (TC), Triglycerides (TG), High Density Lipoprotein cholesterol (HDL-c), Low Density Lipoprotein cholesterol (LDL-c), and Very Low Density Lipoprotein cholesterol (VLDL-c)] in rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett’s test. **** significant at $p < 0.0001$ vs. normal control group; ** significant at $p < 0.001$ vs. induced/HFD group.

• **Effect of Barleria terminalis extract on liver enzyme parameters in HFD-induced hyperlipidemic rats:**

Table 8: Effect of High Fat Diet (HFD), Barleria terminalis extract (200 mg/kg and 400 mg/kg), and standard drug on liver enzyme parameters [Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), and bilirubin] in HFD-induced hyperlipidemic rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one way ANOVA followed by Dunnett’s test.

Parameters	Normal Control	Disease control	Standard	Test Low Dose	Test High Dose
AST (IU/L)	176.67 \pm 1.36	223.33 \pm 2.32	169.50 \pm 0.76	184.67 \pm 0.88 ****	160.67 \pm 0.88 ****
ALT (IU/L)	90.50 \pm 0.76	118.50 \pm 0.76	90.00 \pm 0.58	106.00 \pm 0.58 ****	98.00 \pm 0.58 ****



ALP (IU/L)	8.98 ± 0.13	13.02 ± 0.14	5.82 ± 0.10	10.10 ± 0.12 ****	7.43 ± 0.09 ****
Bilirubin	12.87 ± 0.12	15.68 ± 0.13	10.38 ± 0.07	12.47 ± 0.11 ****	11.25 ± 0.08 ****

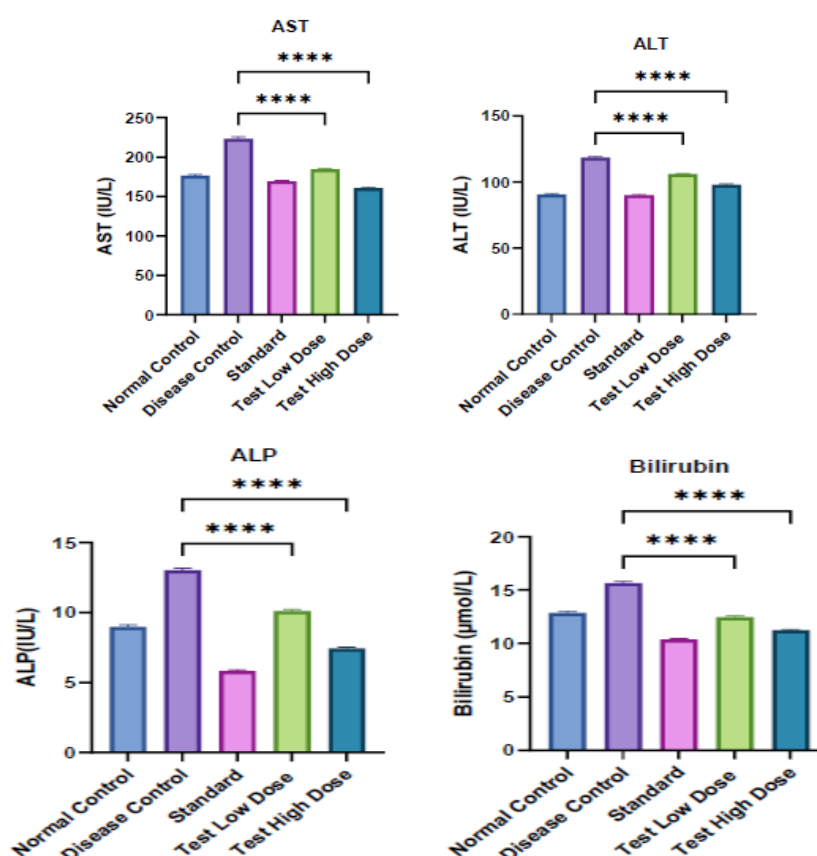


Figure 10: Effect of High Fat Diet (HFD), *Barleria terminalis* extract (200 mg/kg and 400 mg/kg), and standard drug on liver enzyme parameters [Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), and bilirubin] in rats. Values are expressed as mean ± SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett's test. ** significant at p < 0.0001 vs. normal control group; * significant at p < 0.001 vs. induced/HFD group.

• **Effect of Treatment on Renal Biomarkers in HFD-Induced Hyperlipidemic Rats:**

Table 10: Effect of treatment on renal biomarkers in HFD-induced hyperlipidemic rats. Values are expressed as Mean ± SEM (n = 6).

Renal Markers	Normal Control	Disease control	Standard	Test Low Dose	Test High Dose
Urea	25.33 ± 0.49	54.50 ± 0.76	30.33 ± 0.49	42.33 ± 0.49 ****	35.33 ± 0.49 ****
Creatinine	2.30 ± 0.04	3.55 ± 0.04	1.88 ± 0.03	2.88 ± 0.03 ****	2.48 ± 0.03 ****



Total Protein	65.07 ± 0.22	82.55 ± 0.28	71.98 ± 0.19	78.83 ± 0.19 ****	76.97 ± 0.19 ****
Albumin	28.90 ± 0.19	22.00 ± 0.19	33.97 ± 0.18	29.85 ± 0.16 ****	31.85 ± 0.18 ****

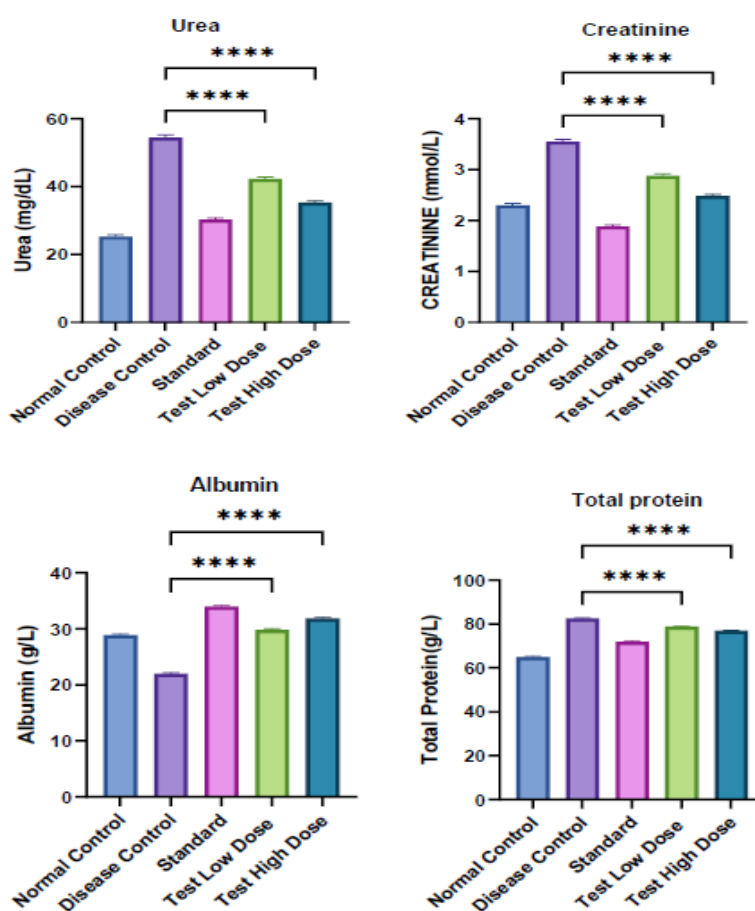


Figure 11: Effect of HFD, standard drug, and treatment extract (200 mg/kg and 400 mg/kg) on renal biomarkers including A) Urea, B) Creatinine, C) Total Protein, and D) Albumin in experimental rats. Values are expressed as mean ± SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett’s test. **** significant at p < 0.0001 vs. control group; ** significant at p < 0.01 vs. induced/HFD group.

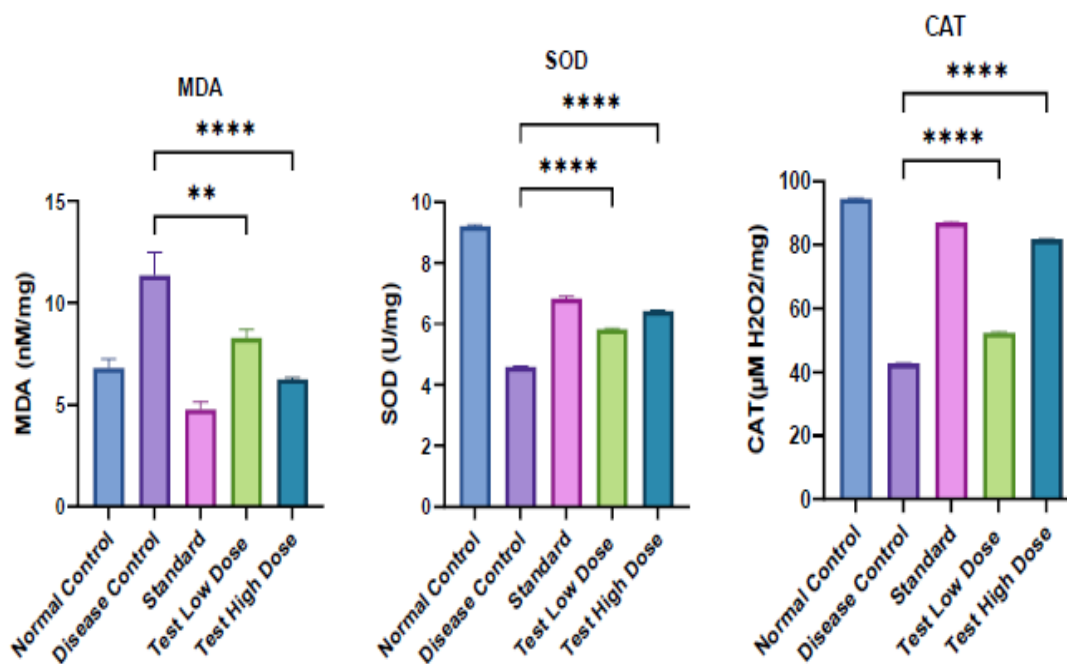
7.3 Effect of treatment on antioxidant parameters:

Table 11: Effect of treatment on antioxidant parameters in HFD-induced hyperlipidemic rats. Values are expressed as Mean ± SEM (n = 6).

Antioxidant Parameters	Normal Control	Disease control	Standard	Test Low Dose	Test High Dose
MDA	6.40 ± 0.04	12.48 ± 0.03	4.42 ± 0.03	6.85 ± 0.02 **	5.18 ± 0.03 ****
SOD	9.20 ± 0.04	4.58 ± 0.03	6.82 ± 0.03	5.82 ± 0.03 ****	6.42 ± 0.03 ****



CAT	94.65 ± 0.17	42.68 ± 0.14	87.12 ± 0.11	52.45 ± 0.18 ****	81.80 ± 0.11 ****
GPX	9.80 ± 0.04	5.42 ± 0.03	9.82 ± 0.03	7.62 ± 0.03 ****	8.52 ± 0.03 ****
GSH	56.28 ± 0.14	21.77 ± 0.11	54.53 ± 0.09	39.60 ± 0.13 ****	51.00 ± 0.58 ****
TAC	9.00 ± 0.06	4.30 ± 0.06	8.20 ± 0.06	6.40 ± 0.06 ****	7.60 ± 0.06 ****



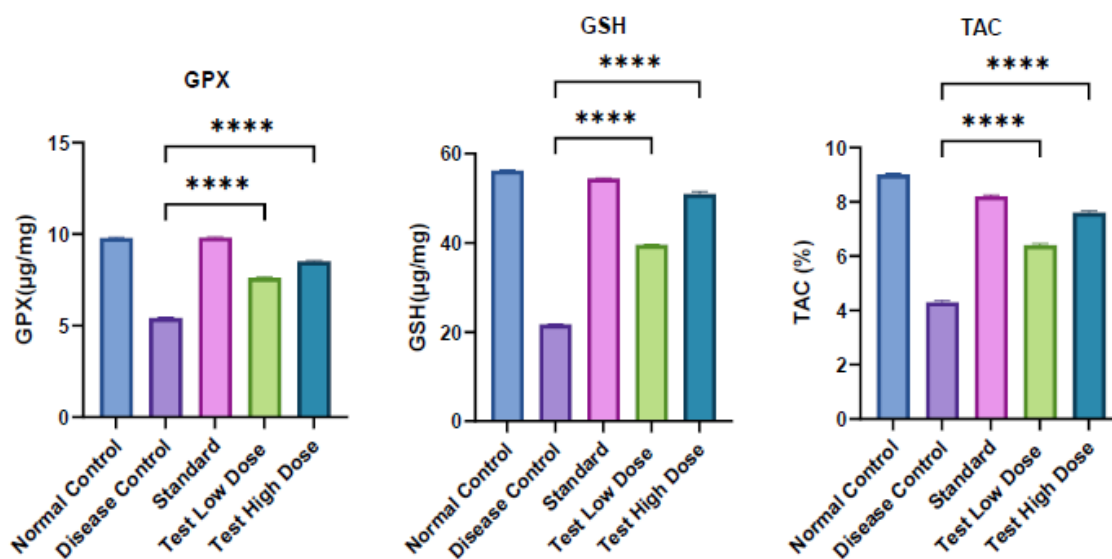
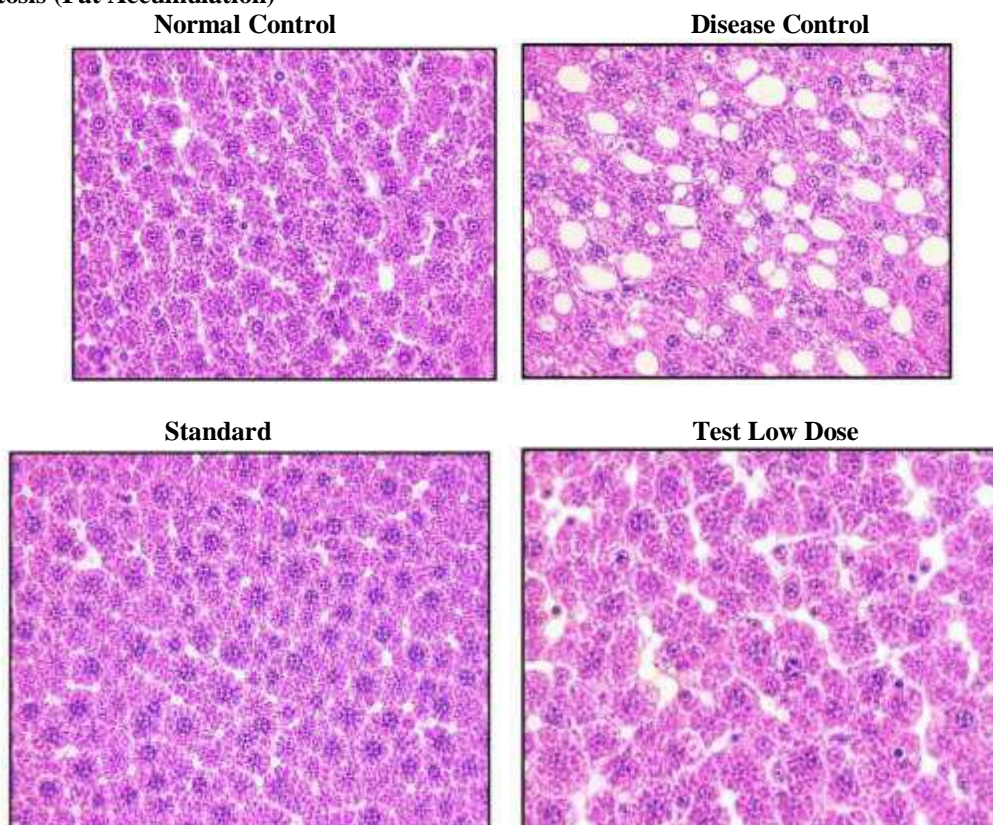


Figure 12: Effect of HFD, standard drug, and treatment extract (200 mg/kg and 400 mg/kg) on ntioxidant parameters including A) MDA, B) SOD, C) CAT, D) GPX, E) GSH, and F) TAC in experimental rats. Values are expressed as mean \pm SEM (n = 6) and analyzed using one-way ANOVA followed by Dunnett’s test. ** significant at $p < 0.0001$ vs. control group; ** significant at $p < 0.01$ vs. induced/HFD group.

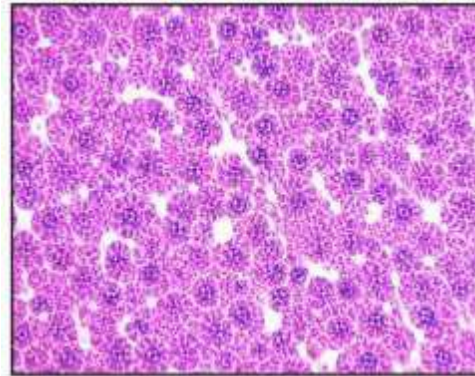
7.4 Histology:

(A) Steatosis (Fat Accumulation)



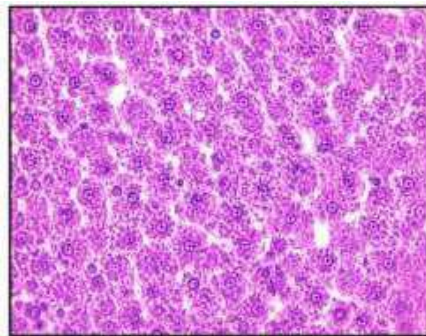


Test High Dose



(B).Necrosis

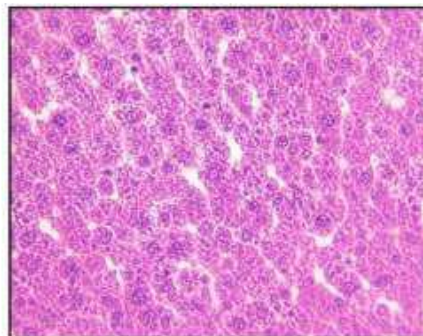
Normal Control



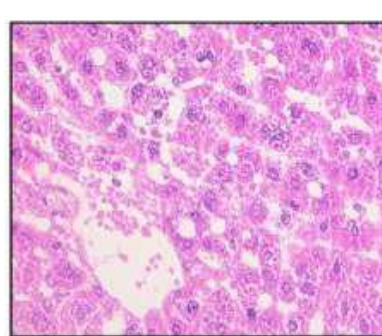
Disease Control



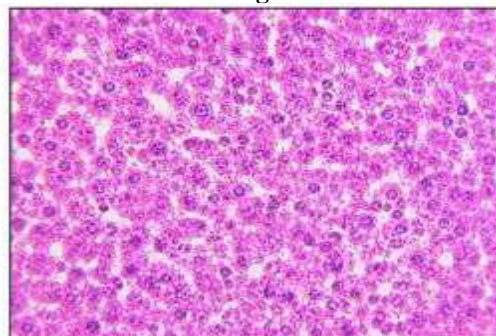
Standard



Test Low Dose

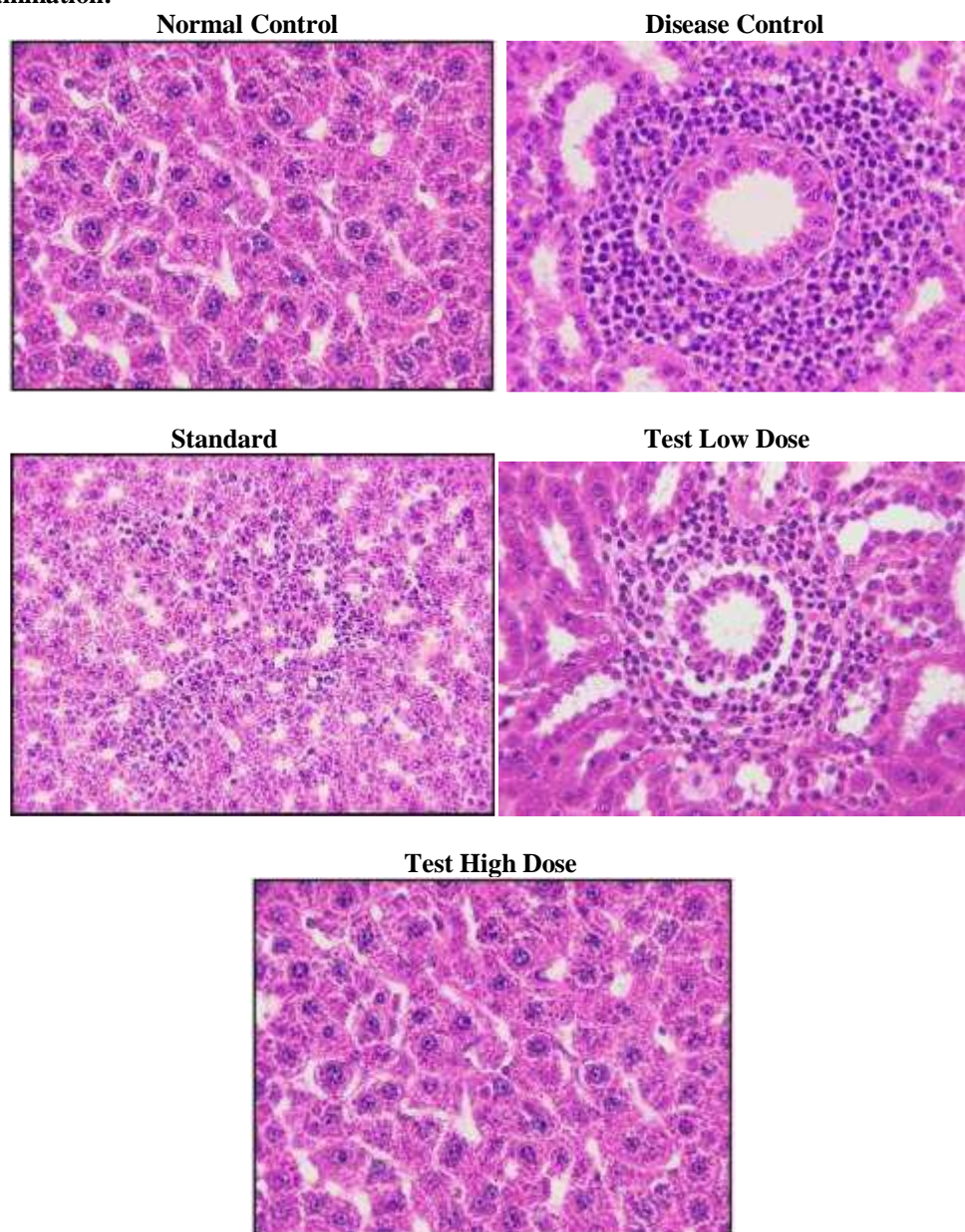


Test High Dose





(C). Inflammation:



V. DISCUSSION

High-fat diet induced hyperlipidemia caused significant increase in body weight, serum lipid levels, hepatic lipid accumulation, oxidative stress, and liver dysfunction in experimental animals. Treatment with *Barleria terminalis* extract significantly reduced total cholesterol, triglycerides, LDL, VLDL, and body weight while increasing HDL levels. The extract also improved antioxidant status, liver and kidney function parameters, and reduced hepatic lipid

accumulation. Histopathological studies showed restoration of normal liver architecture with reduced fatty degeneration and inflammation. The study concluded that *Barleria terminalis* possesses significant antihyperlipidemic, antioxidant, hepatoprotective, and nephroprotective activities.



VI. CONCLUSION

The present study concluded that *Barleria terminalis* possesses significant antihyperlipidemic and antioxidant activity in high-fat diet induced hyperlipidemic animals. The extract reduced TC, TG, LDL, and VLDL levels while increasing HDL levels. It also improved liver, kidney, antioxidant, and histopathological parameters. The 400 mg/kg dose showed better protective activity comparable to atorvastatin.

Future Perspective

Further studies are required to isolate active phytoconstituents and evaluate the molecular mechanism, safety, and clinical efficacy of *Barleria terminalis* for development as a herbal antihyperlipidemic agent.

REFERENCES

- [1]. Lakhne R, Gupta R, Gupta RS. Active phytoconstituents for controlling hypercholesterolemia: a review. *Int J Clin Endocrinol Metab.* 2015;1(1):1-9.
- [2]. Shattat GF. A review article on hyperlipidemia: types, treatments and new drug targets. *Biomed Pharmacol J.* 2015;7(2):399-409. doi:10.13005/bpj/504.
- [3]. Dargel R. Lipoproteins and the etiopathogenesis of atherosclerosis. *Zentralbl Allg Pathol* 1989; 135: 501-504.
- [4]. Kritchevsky D. Cholesterol vehicle in experimental atherosclerosis. A brief review with special reference to peanut oil. *Arch Pathol Lab Med* 1988; 112:1041-4.
- [5]. Lehninger Principles of Biochemistry. Nelson DL, Cox MM. Lehninger Principles of Biochemistry. 8th ed. New York: W.H. Freeman; 2021.
- [6]. Ferrier DR. Lippincott Illustrated Reviews: Biochemistry. 6th ed. Philadelphia: Wolters Kluwer; 2017.
- [7]. Roopa R, Banu Z, Rahman A. Comprehensive review of hyperlipidemia: pathophysiology, diagnosis, and management [Internet]. 2024 [cited 2026 May 6].
- [8]. Hill MF, Bordoni B. Hyperlipidemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. Available from: NCBI Bookshelf.
- [9]. Castilla-Guerra L, Fernández-Moreno Mdel C, Alvarez-Suero J. Secondary stroke prevention in the elderly: new evidence in hypertension and hyperlipidemia. *Eur J Intern Med* 2009;20:58690. 10. Keane WF, Peter J, Kasiske BL. Is the aggressive management of hyperlipidemia in nephritic syndrome mandatory *Kidney Int.* 1992, (Suppl 38), S 134-41
- [10]. Ray KK, Seshasai SR, Erqou S. Statins and all cause mortality in high-risk primary prevention: a meta analysis of 11 randomized controlled trials involving 65,229 participants. *Arch Intern Med,* 2010, 170(12), 1024-1031.
- [11]. Abourbih S, Filion KB, Joseph L, Schiffrin EL, Rinfret S, Poirier P, Pilote L, Genest J, Eisenberg MJ. Effect of fibrates on lipid profiles and cardiovascular outcomes: a systematic review. *Am J Med,* 2009, 122(10), 962-962.
- [12]. Wong NN. Colesevelam: a new bile acid sequestrants. *Heart disease,* 2001, 3(1), 63-70. K. Nirosha et al., A Review on hyperlipidemia VOLUME 4 | NUMBER 5 SEP 2014 192
- [13]. Shahid akbar *Cuscuta Chinensis* Lam. for C. *epithimum* L] [Convolvulaceae] Published in springerlink 15. Kazemipoor M. Radzi CW. Cordell GA, Yaze L. Safety, efficacy and metabolism of traditional medicinal plants in the management of obesity: a review. *Int J Chem Eng Appl* 2012;3(4):288-292.