Evaluation of the Anti-Hyperlipidemic Effect of Johrenia Aromatica in Male Albino Rats

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ABSTRACT

Background: Hyperlipidemia is a common cause of cardiovascular diseases (CVD). Although synthetic lipid-lowering agents are effective, interest in herbal therapies with fewer adverse effects has increased.

Aim: This study investigated the antihyperlipidemic effects of *Johrenia aromatica* (Bareza), a traditionally used medicinal plant by Kurdish people for the treatment of various conditions. The current study aims to evaluate the effect of *Johrenia aromatica* (*JA*) on hyperlipidemic rats.

Methods: Thirty albino rats were divided randomly into five groups: normal diet control, high-fat diet (HFD) with atorvastatin, HFD with *JA* extract at 400 mg/kg and 600 mg/kg, and an untreated HFD group. Treatments were continued for four additional weeks. Body weight, lipid profile, liver and renal biomarkers, blood glucose, C-reactive protein (CRP), and blood pressure were evaluated. The data were analyzed using ANOVA followed by Duncan post hoc test, with $P < 0.05$ considered significant.

Results: Treatment with *JA*, particularly at 400 and 600 mg/kg, significantly reduced TC, TG, LDL, and VLDL levels compared with untreated HFD rats. The extract also reduced body weight and demonstrated anti-inflammatory effects. There was a significant reduction in blood glucose, especially in the JA600 group. On the other hand, the renal and liver biomarkers remained unchanged, indicating no adverse effects on either organ.

Conclusion: *Johrenia aromatica* showed significant antihyperlipidemic, anti-inflammatory, and weight-reducing effects in the hyperlipidemic model, with the safest effects on hepatic and renal function, supporting the potential use of *JA* as a natural product in the management of dyslipidemia.

Keywords: Animal study; Antihyperlipidemic; Bareza; *Johrenia aromatica*; Traditional medicine.

INTRODUCTION

Hyperlipidemia or dyslipidemia (used interchangeably) is an abnormal disorder characterized by dysregulation of plasma lipids, including elevated plasma cholesterol and triglycerides (TG) and low-density lipoprotein (LDL), along with reduced high-density lipoprotein (HDL) (Durrington et al., 2026). This condition reflects an imbalance in the metabolism of lipoproteins and is linked with a high risk of CVD. The common features of dyslipidemia are hypertriglyceridemia, hypercholesterolemia, and a disrupted lipoprotein profile, which contribute to atherosclerosis and other lipid- related disorders. The clinically relevant plasma lipids are triglycerides and cholesterol, which serve as constituents of cell membranes (Nelson, 2012). According to the estimation report in 2008, which was published by the World Health Organization (WHO), the incidence of hypercholesterolemia in adults in Europe (53.7%), America (47.7%), Southeast Asia (30.3%), and Africa (23.1%)(Llanes et al., 2025). Dyslipidemia is associated with risk factors, for example, a saturated or trans-fat diet, sedentary lifestyle, smoking, and overweight, which are causative factors in the development of dyslipidemia and exacerbate this disorder, increasing the risk for cardiovascular diseases and other lipid-related disorders. Dyslipidemia can be prevented by controlling the aforementioned risk

factors(Gebreegziabiher et al., 2021). Dyslipidemia may result from changes in lipoprotein metabolism due to genetic, environmental, or other pathologic factors, thereby increasing the risk of cardiovascular disease (Ahmed et al., 2025b, Mosca et al., 2022). The common risk factors, such as insulin resistance (IR), hypertension, obesity, and a sedentary lifestyle, play a crucial role in dyslipidemia during childhood(Sunil and Ashraf, 2020). This is a risk factor for CVD, with elevated blood cholesterol contributing to approximately one-third of ischemic heart disease cases. Globally, each year, about 2.6 million deaths and 29.7 million disabilities occur as a result of dyslipidemia. It represents a significant public health threat and challenge (Xing et al., 2020). According to reports, about 50–80% of hypertensives also have plasma lipid abnormalities; the combination of these two disorders increases the risk of coronary heart disease(Ayoade et al., 2020). Besides dyslipidemia, overweight is another risk factor for hypertension; therefore, an obese person with hyperlipidemia is more prone to hypertension than others(Tang et al., 2022). Obesity decreases the quality of life and increases mortality because it is a causative factor for other abnormalities such as hyperlipidemia, hypertension, diabetes, and CVD(Kim et al., 2021). According to numerous studies, dyslipidemia not only contributes to the mentioned disorders but also exacerbates inflammation, autoimmunity, autoimmune diseases, and atherosclerosis within these conditions (Wang et al., 2020). Although inflammation may cause the progression of Arterial stiffness, in other words, both inflammation and dyslipidemia are linked together. Also, dyslipidemia itself can trigger inflammatory responses, with C-reactive protein (CRP) considered a significant inflammatory marker in clinical practice (Aminuddin et al., 2020).

Moreover, dyslipidemia leads to complications in various organs, including the central nervous system, endocrine system, liver, and kidneys. The liver is closely involved in lipid metabolism. Abnormal lipid levels can alter liver metabolism and lead to hepatic tissue damage(Kathak et al., 2022). To reduce the aforementioned risk factor, it is necessary to control hyperlipidemia, both non-pharmacologically and pharmacologically, using various classes of drugs (Ahmed et al., 2025a). Also, various traditional herbal products have been used to improve the therapeutic effects in curing a specific disease (Ahmed et al, 2025).

Many studies on natural products and complementary medicine have indicated that medicinal plants can reduce hyperlipidemia; on the other hand, some natural products, as adjuvant therapy, play significant roles alongside other antihyperlipidemic agents (Niknafs et al., 2021). The family of Apiece is widely used as a therapeutic agent for various diseases; for example, Johrenia aromatica (Kurdish name: Bareza) is traditionally used to treat digestive and other diseases (Pieroni et al., 2016). This plant belongs to the family of Apiece, and this family of natural products is widely used in the treatment of dyslipidemia; the current study was designed to investigate the effect of JA in treating hyperlipidemia and preventing blood pressure elevation in male albino rats.

MATERIALS AND METHODS

Plant materials

Plant Collection and Extraction Procedure

Johrenia aromatica (JA) was gathered from the Sharbazhir district in the northern parts of the KRG/Iraq. J.A. is traditionally used in cuisine and in the treatment of digestive disorders and nephrology-related diseases. The aerial part of the plant is dried in the shade and then crushed. The crushed material was macerated in a hydroalcoholic solution (75:25) for more than 72 hours, then filtered through Whatman No. 1 filter paper. The process was repeated several times. The filtrate was then evaporated on a rotary evaporator and subsequently lyophilized. The resulting dried powder was obtained and stored until used.

Animal experimental design

Animal modelling

The experimental process was conducted in accordance with the animal care guidelines of the Research and Ethics Committee (Sert et al., 2020). Thirty male albino rats (180- 250 g) were divided into five groups (n=6) randomly. Initially, in stable environmental circumstances, the rats were held for 5 days and housed in the

animal research laboratory. The rats were kept in a cage with a stable temperature of 22-25 °C, light/dark(12/12hours) cycle, and free access to food and water. The experiment started after five days of adaptation. During this period, blood pressure was monitored using the CODA monitor. To confirm the induction of hyperlipidemia, a blood sample was collected via the retro-orbital technique.

At the end of the treatment period, blood pressure was remeasured, and the animals were fasted overnight before sacrifice using ketamine and xylazine anesthesia. Blood samples were collected by cardiac puncture for biochemical analyses.

Induction of Hyperlipidemia and Treatment Protocol

All the animal groups except the normal control group were fed a high-fat diet (HFD) (a combination of saturated fat with the routine diet (60:40)) for a period of six weeks to induce hyperlipidemia.

Experimental Design

In the current study, we divided the animals randomly as follows:

NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg/kg); JA-400: HFD with *Johrenia aromatica (*400 mg/kg); JA-600: HFD with *Johrenia aromatica* (600 mg/kg); HFDG: Received only HFD.

Before and after the study, blood was collected, and body weight was measured at different time points to assess any adverse or toxic effects of this herbal. On the last day of the study, the rats were sacrificed using a mixture of ketamine and xylazine.

Weight of the Rats

The weight of the rats was recorded at three different times throughout the study: at the beginning (Week 0), at Week 6, and at Week 10. Weight change was then calculated using the following equations:

Percent of weight change of rats on day 15: $= \frac{(Finalweight(day15) - intialweight)}{Intialweight} X100$

Percent of weight change of rats on day 30: $= \frac{(Finalweight(day30) - intialweight)}{Intialweight} X100$

Biochemical Analysis

Serum was analyzed for total TC, TG, LDL, HDL, and blood glucose, which were quantified using commercial kits. Serum was analyzed for creatinine, urea, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP) to assess kidney and liver function. All biochemical assays were performed at a controlled temperature of 37 °C to minimize error. Reagents were prepared according to the manufacturer's kit instructions

Histotechnique procedure and Semi-quantitative lesion scoring

Tissues were fixed in 10% formaldehyde for 48 h, dehydrated through graded ethanol (50–100%), and cleared with xylene. Samples were then embedded in paraffin at 60–70°C. Sections (5 µm) were cut using a semi-automated rotary microtome, mounted on slides, and dried. The slides were subsequently deparaffinized with xylene, dried at 50°C for 5 min, and stained with Harris hematoxylin and eosin (H&E).

Semiquantitative histomorphometric analysis of kidney and liver tissues was performed on H&E-stained sections using a light microscope (HighTop, China) equipped with a digital microscope camera (SVPRO, China) for image acquisition. Morphometric measurements were conducted using AmScope software (USA) following prior calibration of the imaging system to ensure measurement accuracy.

For each parameter, measurements were obtained from five tissue sections per experimental group. Representative microscopic fields from each section were examined, and the mean value for each specimen was calculated. Group data were expressed as mean values and used for comparative analysis among the experimental groups.

H&E-stained liver sections were examined under a light microscope at 40× magnification. Three parameters were evaluated: (1) hepatocyte nuclear count in the peripheral region of the hepatic lobule as an indicator of cellular integrity and viability; (2) hepatic steatosis, determined by measuring the area of intracellular fatty vacuoles around the central vein (μm²); and (3) hepatic inflammation, assessed by counting chronic inflammatory cells in periportal and perivascular regions. These measurements were used to quantify hepatocellular preservation, fat accumulation, and inflammatory activity, respectively.

H&E-stained kidney sections were examined under 40× magnification. Three histopathological parameters were evaluated: (1) mesangial cell proliferation, determined by counting hyperplastic mesangial cells surrounding glomerular capillary tufts as an indicator of glomerular injury; (2) tubular dilatation, assessed by measuring the area of dilated renal tubules (μm²) throughout the renal parenchyma; and (3) interstitial inflammation, quantified by counting inflammatory cells within the renal interstitium. These parameters were used to assess glomerular, tubular, and interstitial renal damage, respectively.

Statistical Analysis

All results were analyzed as mean ±standard deviation. SPSS was used to perform ANOVA, followed by Duncan's post hoc test, to compare groups. P-values less than 0.05 were considered significant.

Ethical approval

This experimental study was approved by the ethical committee of the College of Medicine/University of Sulaimani, with reference number (No:20; Date: 05/02/2024)

Results

Body weight changes

The Effect of HFD on Body Weight

Body weight was measured for each rat group three times during the experimental study. Body weight differed significantly ($P < 0.05$) between groups at the end. The animals from the groups ATOR-10, JA-400, and JA-600 exhibited a reduction in body weight ($P<0.05$), as shown in Table 1A

The percentage of weight change:

The percentage change in weight is another measure used in the current study. At week 6, Group NC and JA-400 showed highly significant weight gain ($P < 0.05$); the other groups (ATOR-10, JA-600, and HFDG) also gained weight, but their percentage weight change was less than that of the aforementioned groups ($P < 0.05$).

At week 10, only Group CN and HGDG showed a positive weight change, while all the other treated groups showed negative weight change (weight loss) ($P<0.05$). The negative weight change suggests a potential mitigating effect of the treatments, especially in reducing weight gain induced by a high-fat diet Table 2B

Effect of HFD on blood biochemical analysis and blood pressure

Effect of HFD on blood biochemical analysis

Before initiating treatment, Lipid profiles were measured; the HFD-treated groups showed markedly elevated TC, TG, and VLDL levels compared with the NC group ($P < 0.05$). However, there was no notable change in blood glucose across all groups. Regarding liver function in the HFD-treated groups, AST and ALP levels

showed minimal change (P > 0.05), whereas ALP was significantly elevated (P < 0.01), suggesting potential liver dysfunction in HFD rats. Although Renal function remained normal according to the results of urea and creatinine (P >0.05), Creatinine levels were slightly higher in HFD-treated rats, but not significant (P> 0.05). Concerning inflammatory biomarkers such as CRP, there was a significant increase (P > 0.05) in HFD-treated rats, suggesting increased systemic inflammation. As shown in Table 2A

Effect of HFD on blood pressure

The data from the current study indicate that a high-fat diet for short periods did not significantly (P> 0.05) affect Blood pressure (systolic, diastolic, and mean arterial pressure) and/or heart rate in rats. Despite slightly elevated systolic blood pressure, mABP, and heart rate, which were not statistically significant (P > 0.05), as shown in Table 2B.

Effect of the different regimens of treatment on the biochemicals

Lipid profile

At the end of treatments, the lipid profiles of various groups were analyzed. The findings indicated that the groups NC, ATOR-10, JA-400, and JA-600 demonstrated significantly reduced (P<0.05) levels of TC, TG, LDL, and VLDL compared with the HFDG group. Additionally, HDL levels were significantly elevated (P<0.05) in ATOR-10, JA-400, and JA-600 groups compared with the NC group. Overall, groups JA-400 and JA-600 showed the most noticeable effects on lipid parameters, as shown in Table 3.

Liver enzymes

After initiation of treatment, a significant reduction in AST levels (P< 0.05) was observed in both the JA-400 and JA-600 groups, similar to the NC group, suggesting that these treatments are safe, whereas AST levels were elevated in the ATOR-10 group, similar to the HFDG groups (P> 0.05). Also, ALT levels were significantly reduced (P< 0.05) in the JA400 group, comparable to the NC group; in the JA-600 group, ALT levels decreased, but the difference was not statistically significant (P> 0.05) compared with the ATOR-10 and HFDG groups. Finally, ALP levels were reduced in the JA-400 and JA-600 groups, but this reduction was not statistically significant (P> 0.05), as shown in Table 3.

Renal function test

Regarding renal biomarkers at the end of the study, no significant change (P > 0.05) was observed in urea levels in the treated groups compared with the NC groups, particularly in those that received JA at different doses, whereas creatinine in the JA-400 and JA-600 groups showed significant changes (P< 0.05) compared with NC, ATOR-10, and HFDG groups, as shown in Table 3.

Hypoglycemic effect

Regarding post-treatment blood glucose, a significant (P< 0.05) hypoglycemic effect was observed in the JA-600 group, whereas the other groups maintained glucose levels within the same range (P> 0.05), indicating no notable differences between groups. As illustrated in Table 3.

Inflammatory biomarkers

At the end of treatment, although CRP levels remained below 5 mg/dL across all groups, the groups treated with JA showed responses similar to those of Group ATOR-10 (P > 0.05). This similarity suggests that JA may have anti-inflammatory properties, as shown in Table 3.

Effect of *Johrenia aromatica* on blood pressure

At the end of the study, the results indicated that systolic blood pressure (SBP) remained within normal ranges

across all groups (P> 0.05), with no significant differences observed, except for Group HFDG (P< 0.05), which showed the highest SBP, while the JA-400 group had the lowest levels.

Regarding diastolic blood pressure (DBP), the JA-400 group recorded the lowest value (P< 0.05), followed by JA-600 (P< 0.05), while Group HFDG had the highest DBP (P< 0.05).

Regarding mean arterial blood pressure (mABP), Group HFDG showed the highest mABP (P < 0.05), whereas JA-400 had the lowest. For heart rate (bpm), Group HFDG had the highest rate, while JA-400 and JA-600 had the highest mean heart rates among the experimental groups; however, these differences were not statistically significant, as shown in Table 4

Histopathological results of the effect of *Johrenia aromatica*

Hepatic Tissue

Histopathological examination of liver tissue revealed normal hepatic architecture in the normal group, with no detectable lesions (score 0). In contrast, the HFD group exhibited marked steatosis characterized by extensive intracellular lipid accumulation, with a mean fatty deposit area of 2.58 µm² (score 3). Treatment reduced hepatic lipid deposition, with mean fatty deposit areas of 1.03 µm² and 1.63 µm² in the low- and high-dose groups, corresponding to mild (score 1) and moderate (score 2) steatosis, respectively. The atorvastatin-treated group showed a mean fatty deposit area of 2.00 µm² (score 3), indicating persistent severe fatty changes. Significant differences were observed among all groups ($p < 0.001$).

Assessment of hepatic inflammation demonstrated the absence of inflammatory cell infiltration in the normal group (score 0), whereas the HFD group exhibited severe inflammation with a mean of 57.4 chronic inflammatory cells per 40× field (score 3). Both treatment groups markedly reduced inflammatory infiltration, with mean counts of 13.2 and 18.0 cells per field in the low- and high-dose groups, respectively, corresponding to mild inflammation (score 1). The atorvastatin group showed 22.2 cells per field (score 2), indicating moderate inflammation. Differences among groups were highly significant ($p < 0.001$), demonstrating substantial attenuation of hepatic inflammatory activity following treatment.

Hepatic necrosis was evaluated by counting hepatocyte nuclei in representative microscopic fields, where higher nuclear counts reflected greater cellular viability. The normal group exhibited a mean of 32.6 nuclei per field with no evidence of necrosis (score 0), whereas the HFD group showed a marked reduction to 8.4 nuclei per field, indicative of severe necrosis (score 3). The low-dose and high-dose treatment groups showed mean nuclear counts of 17.3 and 13.0 nuclei per field, corresponding to mild and moderate necrotic changes, respectively. In contrast, the atorvastatin group showed a mean of 9.2 nuclei per field (score 3), indicating persistent severe necrosis. Statistical analysis revealed highly significant differences among groups ($p < 0.001$). Overall, the treatments improved HFD-induced hepatic histopathological alterations, with the low-dose regimen showing the greatest protective effect. All data are summarized in Table 5 and illustrated in the corresponding micrographs (1,2, and 3).

Renal Tissue

Mesangial cell proliferation was used to assess glomerular injury. The normal group showed normal renal histology with a mean mesangial cell count of 16.20 (score 0), whereas the HFD group exhibited marked mesangial hyperplasia (42.60; score 3; $p < 0.001$). Both low- and high-dose treatments significantly reduced mesangial proliferation to 27.60 and 25.40 cells, respectively (score 2), with no significant difference between them. The atorvastatin group also showed improvement (36.40; score 2) but remained significantly different from the normal and treatment groups ($p < 0.001$).

Tubular dilatation was evaluated by measuring tubular area. The normal group showed intact tubular morphology (1.58 µm²; score 0), while the HFD group exhibited severe tubular injury (4.44 µm²; score 3; $p < 0.001$). Both low- and high-dose treatments restored tubular architecture to near-normal values (1.54 and 1.53 µm²; score 0), with no significant difference between them. The atorvastatin group showed partial

improvement (2.20 μm²; score 1) but remained significantly different from the normal and treatment groups ($p < 0.001$). Interstitial inflammatory cell infiltration was absent in the normal group (score 0) but markedly increased in the HFD group (32.60 cells; score 3; $p < 0.001$). The low-dose group showed mild inflammation (4.00 cells; score 1), whereas the high-dose group demonstrated complete restoration of normal histology with no detectable inflammatory infiltration (score 0). The atorvastatin group exhibited moderate inflammation (12.80 cells; score 2), indicating partial improvement compared with the HFD group. Overall, significant differences were observed among the experimental groups ($p < 0.05–0.001$), demonstrating substantial renoprotective effects of the treatments. All obtained results, along with their statistically significant differences among experimental groups, are summarized in Table (6) and further demonstrated in the representative photomicrographs (Figures 4 and 5).

DISCUSSION

The current study is the first experimental study to evaluate the effect of *Johrenia aromatica* (JA.) extract in the treatment of dyslipidemia. We compared the efficacy of JA with a standard pharmaceutical drug in hyperlipidemic rats. The results demonstrated a significant improvement in the lipid profile following treatment with the JA extract, with no adverse effects on liver or kidney. However, it is important to acknowledge potential confounding factors, such as sample size and medication adherence, which must be carefully considered when interpreting.

After six weeks of HFD feeding to the rats, significant elevations in TC, TG, and VLDL levels were observed, indicating the onset of dyslipidemia. These results agree with the findings of (Kumar et al., 2020), supporting the role of the HFD in inducing hyperlipidemia in rats.

After initiation of treatment regimens for four weeks, there was a change in body weight in all the groups; however, the CN and HFDG groups showed significant weight gain, which agreed with the findings of (Lozano et al., 2016), who showed the effect of HFD on body weight.

The ATOR-10, JA-400, and JA-600 groups showed a negative change in body weight (weight reduction); this weight reduction was more observed in the herbal-treated groups. These results highlight the potential of Johrenia aromatica in body weight management.

Regarding the effect of different doses of JA on hyperlipidemic rats. The results showed that JA significantly reduced TC, TG, LDL, and VLDL levels, while HDL levels changed minimally. The underlying mechanism of the antihyperlipidemic effect of JA is unclear; however, we hypothesize that the different chemical constituents of this plant contribute to these effects through different mechanisms. This finding agrees with the study by (Latifi et al., 2019), which reported similar hypoglycemic and hypolipidemic activity in another species of the same family of this herb, suggesting a potential common mechanism within the family.

Regarding the liver function test, after six weeks of HFD administration, no significant change was observed in AST and ALT; ALT was elevated but not significantly, whereas ALP was significantly elevated. These results agree with the study of (Brahmi et al., 2021).

After initiation of treatment regimens, AST levels decreased in the groups of rats that received different doses of the herbal, paralleling the CN group, indicating no significant liver damage. Alanine aminotransferase (ALT) showed a marked reduction in the JA-400-treated group, consistent with the CN group, suggesting a possible protective effect of JA. At lower dosage. On the other hand, ALT levels increased in all other experimental groups, which indicated potential liver toxicity. Finally, ALP levels decreased significantly only in the group that received the low dose of JA, whereas the ATOR-1 and HFDG groups showed a substantial increase. These findings are consistent with the results reported by (Ashraf et al., 2020, Pasavei et al., 2020). The results of both biochemical and histopathological analyses indicated that JA treatment is safe, with no evidence of adverse effects on the liver's function or structure.

Regarding the renal function test, no significant change was observed in urea and creatinine levels in any rat group after HFD administration. These results agree with the findings of (Amin et al., 2014), who also reported no notable alterations in renal function biomarkers under similar experimental conditions.

After initiation of the treatment regimen, notable changes were observed in both urea and creatinine levels in some groups. However, changes in renal biomarkers across all groups remained within the normal range, as observed in the CN group, suggesting no substantial renal impairment. These findings agree with the findings of (Mojarrad, 2020), which showed results similar to those of other species in the same family and further support the safety of the treatments.

Regarding blood glucose, after administration of HFD, there was no significant change that could be noted; this finding agrees with the finding of (Lozano et al., 2016), which reported an unchanged blood glucose level after HFD feeding under comparable conditions. However, with continued HFD administration to rats, blood glucose increased across all groups, including the control negative group, which was also elevated. This observation is consistent with the findings of (Qi and Wang, 2023), who also reported elevated blood glucose levels in similar experimental conditions.

Conversely, the group of rats that received a high dose of JA exhibited only a slight elevation in blood glucose levels compared to the other groups. This finding suggests that JA may exhibit antihyperglycemic effects at high doses, potentially preventing blood glucose elevation in treated animals.

Regarding inflammatory biomarkers, after five weeks of HFD feeding, CRP levels were significantly elevated in the experimental rats. This finding is consistent with the results reported by (Kalsait et al., 2011), who similarly noted an increase in CRP following administration of an HFD.

After initiation of treatment, CRP levels continued to rise in the CN group, whereas reductions were observed in all hyperlipidemic groups, particularly in the group receiving atorvastatin. These results are consistent with those of (Huang et al., 2014), who likewise reported a reduction in CRP levels following atorvastatin treatment. Notably, a significant decrease in CRP was also observed in the groups receiving JA-400 and JA-600, suggesting that JA shows anti-inflammatory effects. These promising results need further investigation to confirm JA's anti-inflammatory activity.

Regarding blood pressure, after six weeks of HFD feeding, blood pressure remained stable across all rat groups, with no significant change observed. This finding disagrees with the results reported by (Islamiana et al., 2021), who noted significant alterations in blood pressure following similar dietary interventions.

After four weeks of different treatment regimens in hyperlipidemic rats, both systolic and diastolic blood pressure increased significantly in the ATOR-10 and HFDG groups; these results align with the findings of (Nemoto et al., 2020), who suggested that hypertension in HFD-exposed rat models may be associated with elevated corticosterone levels, contributing to the observed blood pressure change. Meanwhile, systolic and diastolic blood pressure in the groups receiving different doses of JA remained within the normal range and unchanged, similar to the CN group. This suggests that JA may inhibit the elevation of blood pressure. However, to confirm these findings, further studies are necessary.

CONCLUSION

The results of this study indicated that *Johrenia aromatica* exhibits significant antihyperlipidemic effects. Specifically, the plant extract was found to reduce levels of TC, TG, and LDL, while also preventing hyperglycemia and mitigating weight gain. Importantly, *JA* demonstrated no adverse effects on renal or liver function, suggesting its safety in these organs. Additionally, the extract maintains blood pressure within the normal range, further supporting its potential as a therapeutic agent for managing metabolic disorders. However, to confirm these findings and elucidate the underlying mechanisms, further studies are granted.

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None.

Authors' contributions

All authors contributed to the research, writing process, and data analysis.

Conflict of interest

The authors declare that there is no conflict of interest.

Data availability

All data supporting the findings of this study are available within the manuscript.

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Table 1A: Body weight of the rats at different Times.

Weeks	NC	ATOR-10	JA-400	JA-600	HFDG	P-Value
wk-0	237±64.7	284±33.7	200±13.6	227±24.2	248.5±48.2	0.004
wk-6	315.2±30	319±18.7	280±10.3	297±12.8	336±21.7	0.002
wk-10	325.6±14.7	286±24	244±10.3	262±18.5	345±16.7	0

Note: wk: week, NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD. The data were expressed as mean ±SD. Statistical comparison among groups: Mean values with different letters have significant differences ($P < 0.05$).

Table 1B: The percent body weight change of the rats in weeks 6 and 10.

Weeks	NC	ATOR-10	JA-400	JA-600	HFDG
wk-6	38.4±27.3[a]	13.1±9.3[b]	40.2±7.8[a]	32±11.2 [b]	18.1±6.5 [a.b]
wk-10	4.1±11[a]	-10.4±3.2 [b]	-12.8±3.7 [b]	-11.6±4.5 [b]	2.7±4.5 [b]

Note: wk: week, NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD. The data were analyzed using ANOVA followed by Duncan correction, and expressed as mean ±SD. Statistical comparison among groups: Mean values with different letters have significant differences ($P < 0.05$).

Table 2A: The difference in blood parameters in week 0 and week 6

Biomarkers	NC group	HFD induction Groups	P-value
Lipid profile mg/dL			
TC	91.8±6.7	389.1±126.2	0.0001
TG	44.0±3.7	373±100.4	0.0001
HDL	16±1.4	23±2.3	0.0001
LDL	20.5±1.3	25.9±9.1	0.128
VLDL	8.9±0.9	73.1±17.3	0.0001
Liver function biomarker IU/L			
AST (sGOT)	130.3±7.9	130.4±18.8	0.995
ALT (sGPT)	63.3±7.4	98.7±14.4	0.056

ALP	183.3±9.4	915.4±221.4	0.0001
Renal function biomarkers mg/dl			
Urea	28±2.8	28±0.82	0.25
Creatinine	0.45±0.8	0.52±0.05	0.027
B. Glucose mg/dL	112.3±3.2	121.8±13.4	0.991
CRP mg/dL	0.91±0.1	2.73±0.6	0.001

Note: NC: Normal Control Group, HFD: High fatty diet; TC: Total cholesterol, TG: Triglyceride; LDL: Lower density lipoprotein; VLDL: Very low-density lipoprotein; HDL: High density lipoprotein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; B. Glucose: blood glucose; CRP: C-reactive protein

Table 2B: The difference in blood pressure in week 0 and week 6

blood pressure mmHg	**NC group**	**HFD induction Groups**	**P-value**
Systolic	98.4±5.2	104.2±9.5	0.41
Diastolic	78.4±2.4	76±5.7	0.67
m ABP	81.8±2.8	85±5.7	0.53
Heart rate bpm	366.2±7.5	378.6±44.7	0.43

Note: NC: Normal Control Group, HFD: high-fat diet, mABP: mean arterial blood pressure

Table 3: Effect of different regimens on blood parameters of hyperlipidemic rats

Biomarkers	**NC**	**ATOR-10**	**JA-400**	**JA-600**	**HFDG**
Lipid profile mg/dl					
TC	81±4.5 [a]	87.2±5.1 [a]	97.2±2.3 [a]	90.4±2.7[a]	348.2±71.2 [b]
TG	49.2±10.4 [a]	61.8±11.8 [a]	149.8±128.7 [a]	138.6±97.5 [a]	332±80.4 [b]
HDL	18.2±2.6 [a]	21.2±4.2 [b]	23.2±5c	21.3±1.1 [b, c]	20.2±4.3 [b]
LDL	19.9±1.7 [a]	13.4±3.1 [b]	16.8±1.1c	16±1.6 [b, c]	25±2.7 [d]
VLDL	9.8±2.1 [a]	11.6±3.3 [a]	29.9±25.7 [a]	27.8±19.5 [a]	65.6±15.9 [b]
Liver function biomarker IU/L					
AST (sGOT)	166.6±20.5 [a]	184.8±22 [b]	131.8±40.6 [a]	136.8±20.7 [a]	192.2±59.4 [b]
ALT (sGPT)	89.4±7.9 [a]	208.4±63.4 [b]	63.8±10.8 [a]	159.2±133.4 [b]	188.4±6.8 [b]
ALP	217.6±27.6 [a]	871±155.7 [c]	367.6±84.4 [a, b]	514.2±227 [b]	575.6±86.2 [c]
Renal function biomarker mg/dl					
Urea	28.8±2.2 [a]	25.6±3.2 [a]	24.4±2.3 [a]	25.6±2.9 [a]	23.2±4.5 [b]
Creatinine	0.53±0.8 [a]	0.55±0.03 [a]	0.45±0.33 [b]	0.46±0.01 [b]	0.53±0.3 [a]

B. Glucose mg/dL	268±79.8 [a]	255±17.3 [a]	265.2±36.2 [a]	166±28.4 [b]	259.6±26.6 [a]
CRP mg/dL	1.42±0.6 [a]	1.04±0.3 [b]	1.1±0.7 [b]	1.22±0.41 [a, b]	1.9±1 [a]

Note: wk: week, NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD; TC: Total cholesterol, TG; Triglyceride; LDL: lower density lipoprotein; VLDL: very lower density lipoprotein; HDL: high density lipoprotein; AST: aspartate aminotransferase; ALT; alanine amino transferase; ALP: alkaline phosphatase; B. Glucose: blood glucose; CRP: C-reactive protein; The data were analysed using ANOVA following Duncan's correction, expressed as a mean ±SD; mean values with different letters have a significant difference ($P < 0.05$)

Table 4: Effect of different regimens on blood pressure of hyperlipidemic rats.

Biomarkers	NC	ATOR-10	JA-400	JA-600	HFDG
SBP	118.8±4[a]	129±8.4 [b]	112±7.7 [c]	120.5±4.6 [a, b]	140.2±14 [d]
DBP	89±5.3 [a]	100.2±12.3 [a]	74±14 [c]	88.9±5.1 [a]	105.8±10.6 [c]
m ABP	98.2±5 [a]	110±10.2 [b, c]	87±11.3 [a]	100.2±4.3 [b]	117.6±11 [c]
Heart rate bpm	353.6±23.5 [a]	366.8±13 [a, b]	390.6±29.2 [c]	370±11.4 [a, b]	419±19.5 [c]

Note: SBP: systolic blood pressure (DBP), DBP: diastolic blood pressure, mABP: mean arterial blood pressure NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD. The data were analyzed using ANOVA followed by Duncan correction, and expressed as mean ±SD. Statistical comparison among groups: Mean values with different letters have significant differences ($P < 0.05$).

Table 5: Evaluation of Liver Sections through Histological Analysis

	NC	ATOR-10	JA-400	JA-600	HFDG
Fatty Deposits (µm²)	0.00 µm² ± 0.00[a]	2.00 µm² ± 0.04[e]	1.03 µm² ± 0.01[c]	1.63 µm² ± 0.04[d]	2.58 µm² ± 0.05[b]
Inflammation	0.00 ± 0.00[a]	22.2 ± 1.30[e]	13.2 ± 1.30[c]	18.0 ± 1.00[d]	57.4 ± 1.51[b]
Remaining hepatocyte nuclei per high-power field.	32.6 ± 1.14[a]	9.20 ± 1.30[d]	17.8 ± 0.83[c]	13.0 ± 0.70[d]	8.4 ± 0.89[b]

Note: NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD. The data were analyzed using ANOVA followed by Duncan correction, and expressed as mean ± SD. Statistical comparison among groups:). Means with different superscript letters (a–e) differ significantly at $p < 0.05$.

Table 6: Evaluation of Kidney Sections through Histological Analysis

Kidney	NC	ATOR-10	JA-400	JA-600	HFDG
Proliferation	16.20 ±	36.40 ±	27.60 ±	25.40 ±	42.60 ±

	1.92^{a}	1.14^{d}	1.14^{c}	1.14^{c}	1.14^{b}
Tubular Dilatation (µm²)	1.58 µm² ± 0.02^{d}	2.20 µm² ± 0.02^{b}	1.54 µm² ± 1.01^{c}	1.53 µm² ± 1.01^{c}	4.44 µm² ± 0.03^{a}
Inflammation	0.00 ± 0.00^{d}	12.80 ± 0.83^{b}	4.00 ± 0.70^{c}	0.00 ± 0.00^{d}	32.6 ± 1.14^{a}

Note: NC: Normal control group, received routine diet only; ATOR-10: received HFD with Atorvastatin (10 mg); JA-400: HFD with Johrenia aromatica 400 mg; JA-600: HFD with Johrenia aromatica 600 mg; HFDG: Received only HFD. The data were analyzed using ANOVA followed by Duncan correction, and expressed as mean ± SD. Statistical comparison among groups:). Means with different superscript letters (a–e) differ significantly at $p < 0.05$.

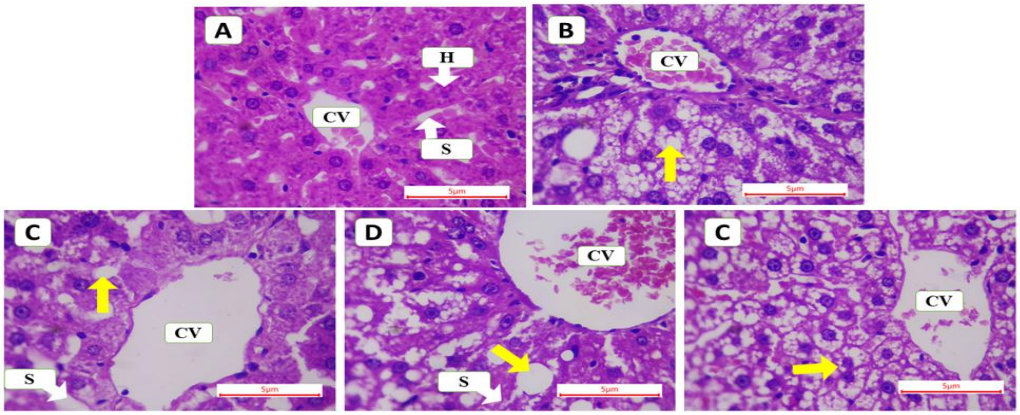


Figure (1). Representative photomicrographs of liver tissue sections from the normal, model, and treatment groups stained with hematoxylin and eosin (H&E) and examined at ×40 magnification (scale bar = 5 µm). **Panel A (NC)** shows normal hepatic architecture with a central vein (CV) surrounded by radiating hepatic sinusoids (S) lined by cords of hepatocytes (H), with no significant histopathological alterations. **Panel B (HFDG group)** demonstrates severe hepatic steatosis (score 3), characterized by extensive cytoplasmic lipid vacuolation within hepatocytes (yellow arrows), resulting in peripheral displacement of nuclei and a characteristic signet-ring appearance. Marked hepatocellular swelling and distortion of the hepatic cords are also evident, accompanied by compression and partial obliteration of the sinusoidal spaces. **Panel C (JA-400 group)** reveals noticeable histological improvement, with preservation of the central vein and surrounding sinusoids and only mild fatty change (score 1) in hepatocytes (yellow arrows). **Panel D (JA-600 group)** shows moderate fatty degeneration (score 2) within hepatocytes (yellow arrows), with slight distortion of the hepatic sinusoids surrounding the central vein. **Panel E (ATOR-10 group)** exhibits persistent severe steatosis (score 3), characterized by marked intracellular lipid accumulation, peripheral nuclear displacement producing signet-ring hepatocytes, and pronounced loss of normal sinusoidal architecture.

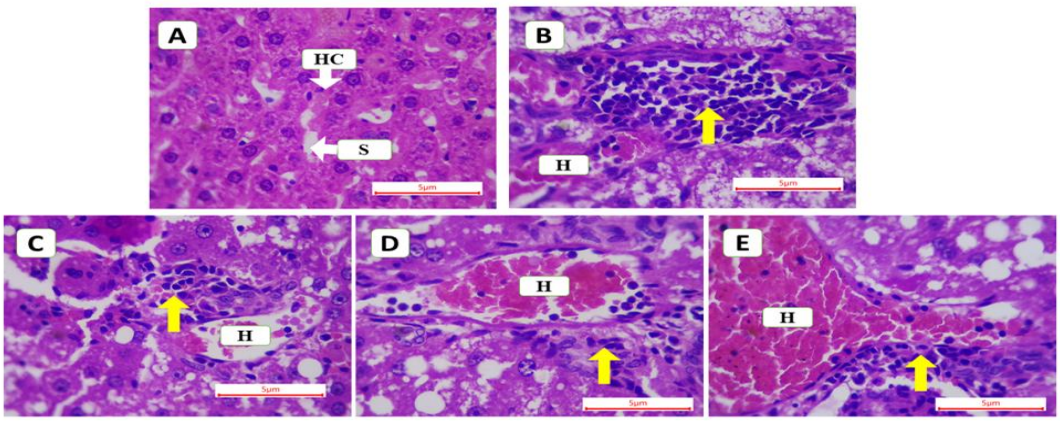


Figure (2). Representative photomicrographs of liver tissue sections stained with hematoxylin and eosin (H&E) and examined at ×40 magnification (scale bar = 5 µm). Panel A (NC) shows normal hepatic

parenchyma composed of hepatocytes (HC) arranged around patent hepatic sinusoids (S), with no significant histopathological alterations. Panel B (HFDG) demonstrates marked hepatic injury characterized by hyperemic blood vessels (H) associated with severe chronic inflammatory cell infiltration (score 3) throughout the examined sections (yellow arrows). Panels C and D (JA-400 and JA-600, respectively) reveal hyperemic blood vessels (H) surrounded by only mild chronic inflammatory cell infiltration (score 1), indicating a significant reduction in inflammatory changes compared with the model group. Panel E (ATOR-10) shows hyperemic blood vessels (H) accompanied by moderate chronic inflammatory cell infiltration (score 2), reflecting partial improvement relative to the model group but greater inflammatory activity than that observed in the low- and high-dose treatment groups.

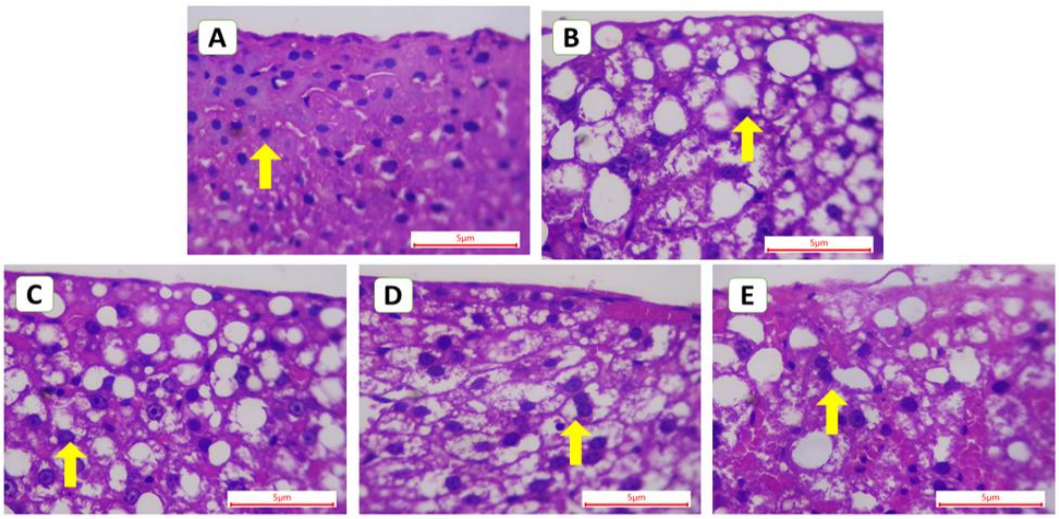


Figure (3). Representative photomicrographs of liver tissue sections from the normal, model, and treatment groups stained with hematoxylin and eosin (H&E) and examined at ×40 magnification (scale bar = 5 µm). Panel A (NC**)** demonstrates normal hepatic architecture with well-preserved hepatocytes containing centrally located nuclei and no significant histopathological abnormalities. Panel B (HFDG) exhibits a marked reduction in the number of hepatocytic nuclei, with many nuclei showing pyknosis (yellow arrows), indicative of severe hepatocellular necrosis (score 3). Panel C (JA-400) reveals partial preservation of hepatocytic nuclei, corresponding to a mild degree of necrosis (score 1). Panel D (JA-600) shows a greater degree of nuclear preservation, with moderate necrotic changes observed (score 2), indicating substantial improvement compared with the model group. Panel E (ATOR-10) demonstrates a pronounced reduction in the number of hepatocytic nuclei (score 3), with numerous pyknotic nuclei (yellow arrows), consistent with severe hepatocellular necrosis and marked tissue injury.

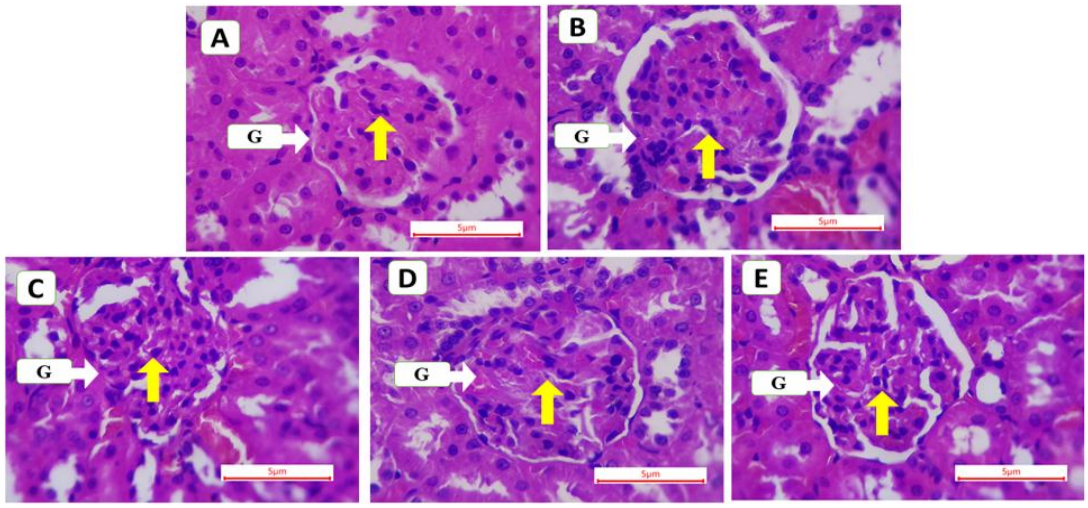


Figure (4). Hematoxylin and eosin (H&E)-stained renal cortex sections of experimental groups at ×40 magnification (scale bar = 5 µm). The photomicrographs demonstrate glomeruli (G) within the renal cortex

across all experimental groups.Panel (A) shows the renal cortex of the normal control group with intact glomerular architecture and normal mesangial cellularity within the capillary tuft (yellow arrow), with no detectable pathological changes, corresponding to score 0 (normal grade). Panel (B) illustrates the HFDG group, demonstrating marked mesangial cell hypercellularity within the glomerular tuft (yellow arrow), indicative of severe mesangial hyperplasia corresponding to score 3. Panels (C) and (D) represent the low-dose and high-dose treatment groups, respectively, both showing a reduction in mesangial cellularity compared with the HFD group (yellow arrows), corresponding to score 1 (mild mesangial hyperplasia). Panel (E) illustrates the atorvastatin-treated group, which shows persistent moderate mesangial hyperplasia with increased cellularity within the glomerular tuft (yellow arrow), corresponding to score 2.

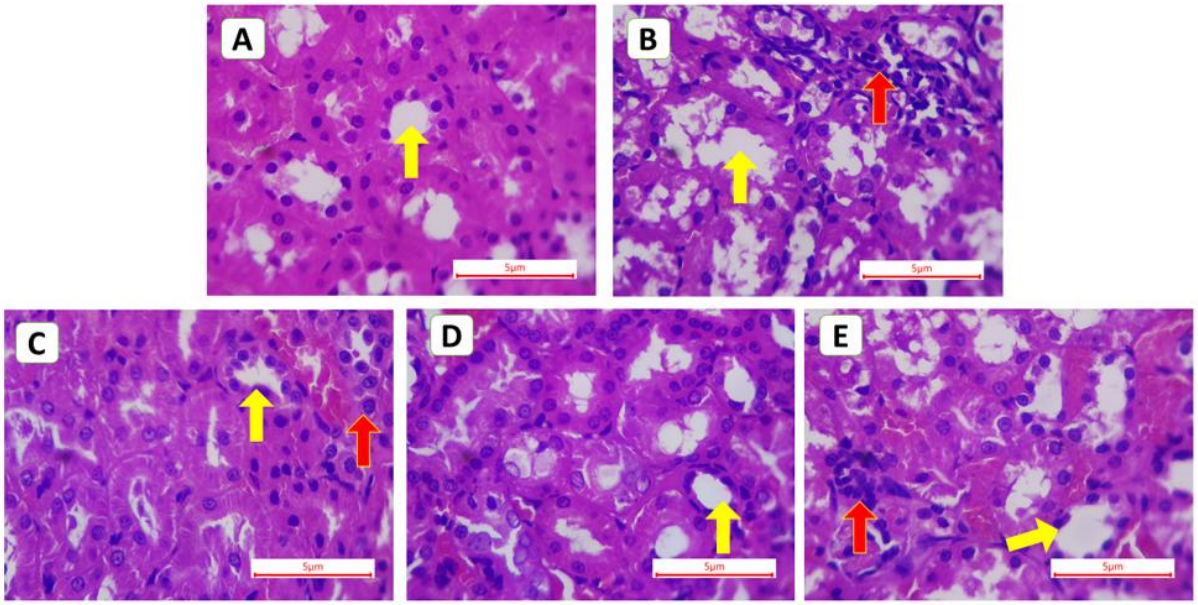


Figure (5). Hematoxylin and eosin (H&E)-stained sections of renal cortex from different experimental groups at ×40 magnification (scale bar = 5 µm). The photomicrographs demonstrate renal cortical architecture, including renal tubules, across all experimental groups. In panel (A), the normal control group shows preserved tubular structure with normal tubular spaces and no histopathological alterations, corresponding to score 0 (normal grade) for both tubular dilation and inflammatory cell infiltration. In panel (B), the HFD group exhibits severe tubular dilation and marked chronic inflammatory cell infiltration within the interstitium, corresponding to score 3 (severe grade) for both parameters. In panels (C) and (D), the low-dose and high-dose treatment groups demonstrate restoration of near-normal tubular architecture, with preserved tubular spaces and score 0 (normal grade) for tubular dilation; however, mild inflammatory cell infiltration is observed in the low-dose group, whereas the high-dose group shows normal inflammatory status. In panel (E), the atorvastatin-treated group shows mild tubular dilation (score 1) and moderate chronic inflammatory cell infiltration (score 2). In all panels, yellow arrows indicate tubular spaces, while red arrows indicate inflammatory cell infiltration within the renal interstitium.