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RESEARCH PAPER

Effect of Aloe Vera Gel on Lipid Profile in High-Fat Diet Induced Hyperlipidemic Rats

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Abstract

Background: Hyperlipidemia, a major modifiable risk factor for atherosclerosis and cardiovascular diseases, arises from abnormal lipid levels in the blood. Modern dietary habits, high saturated fats and refined sugars, contribute significantly to secondary hyperlipidemia. Evidence suggests that not only total cholesterol but also its distribution among lipoproteins influences disease risk. Aloe vera has been explored for its potential lipid lowering properties.

Objectives: To evaluate the effects of aqueous extract of Aloe vera gel on serum lipid profile in high fatty diet-induced hyperlipidemic rats.

Methods: This experimental animal study was conducted at the Department of Pharmacology and Therapeutics, Sir Salimullah Medical College (SSMC), Dhaka in collaboration with the Institute of Nutrition and Food Science (INFS) Dhaka University, over a 12-month period following ethical approval. Thirty healthy adult male Long Evans rats were randomly divided into five groups (n=6 each). Group A (standard control) received a normal laboratory diet with distilled water and Group B (hyperlipidemic control) received a high-fat diet. Group C received a high-fat diet for the first two weeks followed by aqueous extract of Aloe vera gel (300 mg/kg body weight, orally) along with a high-fat diet for the another four weeks. Group D received a high-fat diet for two weeks followed by atorvastatin (10 mg/kg body weight, orally) with a high-fat diet for four weeks. Group E received a high-fat diet for two weeks followed by a combination of atorvastatin (10 mg/kg body weight) and Aloe vera gel extract (200 mg/kg body weight) along with a high-fat diet for the another four weeks. After 42 days of intervension, all rats were anesthetized with chloroform on the morning of the 43rd day and following overnight fasting, blood samples were collected by cardiac puncture for estimation of the serum lipid profile. Data were analyzed using one-way ANOVA followed by Bonferroni test using SPSS version 26, with $p<0.05$ considered stastitically significant.

Results: Rats in the high-fat diet group developed significant hyperlipidemia compared to normal control ($p<0.001$), with elevated serum TC, TG, and LDL-C levels and reduced HDL-C levels. Intervention with Aloe vera gel extract significantly improved the lipid profile ($p<0.001$), showing marked reductions in TC, TG, and LDL-C and an increase in HDL-C. Atorvastatin produced a similar degree of improvement ($p<0.001$). The combination of Aloe vera gel extract with Atorvastatin showed the greatest lipid lowering effect, although the difference compared to individual intervension was not statistically significant.

Conclusion: Aloe vera gel extract significantly improved the lipid profile in high-fat-diet–induced hyperlipidemic rats. Its effects were comparable to atorvastatin for most parameters, except HDL. Combination therapy showed greater improvement than Aloe vera alone and produced effects comparable to atorvastatin, suggesting that Aloe vera may serve as a potential adjunct therapy for hyperlipidemia.

Keywords: Aloe vera gel, Aqueous extract, Hyperlipidemic rats, High-fat diet, Atorvastatin.

Introduction

Hyperlipidemia is a major public health concern worldwide and is strongly associated with cardiovascular diseases (CVDs).[1] It results from a combination of genetic and secondary factors such as obesity, metabolic syndrome, diabetes mellitus, and sedentary lifestyle.[2] The prevalence of dyslipidemia has been rising globally and in Bangladesh, with high proportion of adults exhibiting elevated triglycerides, total cholesterol, and low-density lipoprotein cholesterol (LDL-C), alongside low high-density

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lipoprotein cholesterol (HDL-C).[3] Dyslipidemia is often asymptomatic but significantly increases the risk of atherosclerosis, coronary artery disease, myocardial infarction, and stroke.[4,5]

Lifestyle modification is the first-line approach to managing dyslipidemia, including a low-fat diet, regular exercise, weight management, smoking cessation, and increased intake of soluble fiber and omega-3 fatty acids.[6] When lifestyle interventions are insufficient, pharmacological therapy is indicated. Statins, such as Atorvastatin, a widely prescribed statin, effectively lowers cholesterol and triglyceride levels by inhibiting HMG-CoA reductase, reducing the risk of cardiovascular events in both primary and secondary hypercholesterolemia.[7] However, long-term use has been associated with adverse effects, including hepatotoxicity, myopathy, and an increased risk of new-onset diabetes.[8,9] These concerns have drawn attention toward safer and more natural lipid-lowering alternatives.

Aloe vera, a succulent plant, has been widely used for its medicinal properties, including laxative, anti-inflammatory, and wound-healing effects. Recently, it has gained attention for its potential metabolic benefits. Its inner gel contains bioactive compounds, including polysaccharides, flavonoids, vitamins, and phytosterols, which may contribute to hypolipidemic and antioxidant properties.[10,11] Experimental studies have demonstrated that Aloe vera gel supplementation can significantly reduce serum total cholesterol, triglycerides, and LDL-C while increasing HDL-C in animal models.[13]

Considering these findings, the present study was designed to evaluate the lipid-lowering effect of Aloe vera gel in high-fat diet-induced hyperlipidemic rats, with Atorvastatin as a reference standard. The study aims to explore the potential of Aloe vera as a natural adjunct or alternative therapy for hyperlipidemia.

Materials and Methods

This was an experimental study (animal) carried out in the Department of Pharmacology and Therapeutics of Sir Salimullah Medical College (SSMC), Dhaka, in collaboration with the Institute of Nutrition and Food Science (INFS), University of Dhaka, from February 2023 to January 2024. A total of 30 healthy adult male Long Evan rats (weight approximately 180 to 200 grams) and aged 10 to 12 weeks were enrolled in this study. The rats were obtained from the Animal House, Department of Pharmacy, Jahangirnagar University. Diseased or female rats were excluded. Animals were housed at INFS in a different metallic cage under standard laboratory conditions (temperature: 22 ± 2°C, 12-hour light/dark cycle, and 50–60% humidity) and acclimatized for one week. Ethical approval was obtained from the Institutional Ethical Committee of SSMC. The rats were randomly allocated into 5 groups (n=6).

Group-A (Normal Control): Standard pellet diet and distilled water.

Group B (High-Fat Diet Control): Fed high-fat diet (HFD) and distilled water.

Group C (Aqueous extract of Aloe vera gel treated group): HFD for 2 weeks, then Aloe vera gel extract (300 mg/kg/day) with HFD and distilled water for subsequent 4 weeks.

Group D (Atorvastatin treated group): HFD for 2 weeks, then Atorvastatin (10 mg/kg/day) with HFD and distilled water for subsequent 4 weeks.

Group E (Combination): HFD for 2 weeks then Atorvastatin (10mg/kg/day) and Aloe vera gel (200mg/kg/day) together with HFD and distilled water for subsequent 4 weeks.

The HFD (40% coconut oil and 60% vanaspati ghee) was freshly prepared daily. Alo vera gel was extracted from fresh leaves, homogenized, and stored at 8°C before oral administration. After 42 days of intervention, rats were fasted for 18 hours with free access to water. Blood (3–5 ml) was collected by cardiac puncture under chloroform anesthesia. Serum was separated by centrifugation and analyzed serum TC, TG, LDL, HDL in the Biochemistry Department of SSMC. Data was expressed as mean ± SD. Statistical analysis was performed using One-way ANOVA followed by Bonferroni test by using SPSS version 26. A p-value of <0.05 was considered statistically significant.

Results

A total of 30 healthy adult male Long Evan rats were enrolled in this study in five groups. Among them 6 rats enrolled as Normal control group (Group-A), 6 rats in Hyperlipidemic control group (Group-B), 6 rats in Aqueous extract treated group (Group- C), 6 rats in Atorvastatin intervened group (Group-D) and 6 rats in Combination group (Group-E).

Table I: Comparison of serum lipid profile (mg/dl) in rats among different study groups after completion of 42 days experiment (N=30).

Lipid profile	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)	Group E (n=6)	p-value
TC (mg/dl)	91.1±1.17 (90–93)	186.33±1.37 (185–188)	94.17±1.72 (92–96)	92.50±1.22 (91–94)	91.5±1.38 (90–92)	0.00013
TG (mg/dl)	94.33±1.37 (92–96)	136.33±1.51 (135–138)	96.17±3.43 (90–99)	95.67±0.82 (95–97)	94.5±0.55 (94–95)	0.00011
HDL-C (mg/dl)	36.2±1.17 (35–38)	19.83±0.75 (19–21)	34.50±0.55 (34–35)	36.17±0.75 (35–37)	36.8±0.75 (36–38)	0.0001
LDL-C (mg/dl)	36.0±2.37 (33.0–39.2)	139.17±1.87 (137.4–142)	40.43±1.49 (38.2–42.4)	37.73±2.65 (35.6–42)	34.9±1.46 (33.2–37.0)	0.0001

Note: Values are expressed as mean ± SD and range in parenthesis. One-way ANOVA was performed to compare among different groups.

Differences in serum lipid levels were observed among the groups ($p<0.001$). The high-fat diet group (B) showed markedly elevated TC (186.33±1.37), TG (136.33±1.51), and LDL-C (139.17±1.87), along with reduced HDL-C (19.83±0.75), compared with the normal control. Treatment with Aloe vera (C), atorvastatin (D), and their combination (E) effectively improved the lipid profile, reducing TC (94.17±1.72; 92.50±1.22; 91.5±1.38), TG (96.17±3.43; 95.67±0.82; 94.5±0.55), and LDL-C (40.43±1.49; 37.73±2.65; 34.9±1.46), while increasing HDL-C (34.50±0.55; 36.17±0.75; 36.8±0.75), bringing values close to normal (Table I).

Table II: Comparison of serum total cholesterol levels (mg/dl) in rats among different groups after completion of 42 days experiment (N=30).

Variable	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)	Group E (n=6)	p-value
S. TC (mg/dl)	91.1±1.17	186.33±1.37	94.17±1.72	92.50±1.22	91.5±1.38	
Bonferroni test						
Group A vs Group B						0.0001
Group A vs Group C						0.01
Group A vs Group D						0.864^{ns}
Group A vs Group E						1.000^{ns}
Group B vs Group C						0.0011
Group B vs Group D						0.0023
Group B vs Group E						0.0013
Group C vs Group D						0.348^{ns}
Group C vs Group E						0.027
Group D vs Group E						0.936^{ns}

Data are expressed as mean ± SD. One-way ANOVA followed by the Bonferroni test was performed to compare between groups.

Table III: Comparison of serum triglyceride levels (mg/dl) in rats among different groups after completion of 42 days experiment (N=30).

Variable	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)	Group E (n=6)	p-value
S. TG (mg/dl)	94.33±1.37	136.33±1.51	96.17±3.43	95.67±0.82	94.5±0.55	
Bonferroni test						
Group A vs Group B						0.0018
Group A vs Group C						0.962^{ns}
Group A vs Group D						1.000^{ns}
Group A vs Group E						1.000^{ns}
Group B vs Group C						0.00012
Group B vs Group D						0.00021
Group B vs Group E						0.00015
Group C vs Group D						1.000^{ns}
Group C vs Group E						1.000^{ns}
Group D vs Group E						1.000^{ns}

Note: Data were expressed as mean ± SD. One-way ANOVA followed by the Bonferroni test was performed to compare between groups.

Table IV: Comparison of serum HDL-C levels (mg/dl) in rats among different groups after completion of 42 days experiment (N=30).

Variable	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)	Group E (n=6)	p-value
S.HDL-C (mg/dl)	36.2±1.17	19.83±0.75	34.50±0.55	36.17±0.75	36.8±0.75	
Bonferroni test						
Group A vs Group B						0.00012
Group A vs Group C						0.017
Group A vs Group D						1.000^{ns}
Group A vs Group E						1.000^{ns}
Group B vs Group C						0.0011
Group B vs Group D						0.0012
Group B vs Group E						0.0021
Group C vs Group D						0.017
Group C vs Group E						0.0001
Group D vs Group E						1.000^{ns}

Note: Data were expressed as mean ± SD. One-way ANOVA followed by the Bonferroni test was performed to compare between groups.

Table V: Comparison of serum LDL-C levels (mg/dl) in rats among different groups after completion of 42 days experiment (N=30).

Variable	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)	Group E (n=6)	p-value
S.LDL-C (mg/dl)	36.0±2.37	139.17±1.87	40.43±1.49	37.73±2.65	34.9±1.46	
Bonferroni test						
Group A vs Group B						0.0014
Group A vs Group C						0.021
Group A vs Group D						1.000^{ns}
Group A vs Group E						1.000^{ns}
Group B vs Group C						0.0031
Group B vs Group D						0.0021
Group B vs Group E						0.0011
Group C vs Group D						0.296^{ns}
Group C vs Group E						0.022
Group D vs Group E						0.536^{ns}

Data were expressed as mean ± SD. One-way ANOVA followed by the Bonferroni test was performed to compare between groups.

Serum total cholesterol differed significantly among the groups (Table II). The high-fat diet group (B) showed a marked increase (186.33±1.37) compared with the normal control (A). Intervension with Aloe vera (C), atorvastatin (D), and their combination (E) significantly reduced cholesterol levels versus group B ($p<0.01$ to $p<0.001$). Cholesterol values in groups D and E were comparable to group A ($p>0.05$). No significant differences were observed between groups C and D or between D and E, while group E showed a modest but significant reduction compared with group C ($p<0.05$).

Serum triglycerides were markedly higher in the high-fat diet group B (136.33±1.51) compared with the control group A (94.33±1.37). Intervention with Aloe vera (C: 96.17±3.43), atorvastatin (D: 95.67±0.82), and their combination (E: 94.5±0.55) significantly lowered TG levels versus group B ($p<0.001$). TG values in groups C, D, and E were similar ($p>0.05$) to each other and to the control (Table III).

Serum HDL-C was markedly reduced in the high-fat diet group B (19.83±0.75) compared with the control group A (36.2±1.17). Treatment with Aloe vera (C: 34.50±0.55), atorvastatin (D: 36.17±0.75), and their combination (E: 36.8±0.75) significantly increased HDL-C levels versus group B ($p<0.01$–0.001). HDL-C values in groups D and E were comparable to the control ($p>0.05$). Although group C showed slightly lower HDL-C than groups D and E, differences were statistically significant but small (Table IV).

LDL-C was markedly elevated in the high-fat diet group B (139.17±1.87) compared with the control group A (36.0±2.37). Intervention with Aloe vera (C: 40.43±1.49), atorvastatin (D: 37.73±2.65), and combination therapy (E: 34.9±1.46) significantly reduced LDL-C versus group B ($p<0.01$–0.001). LDL-C levels in groups D and E were comparable to the control ($p>0.05$). Group C showed slightly higher LDL-C than group E, though both were lower than group B (Table IV).

Discussion

This experimental study was conducted to evaluate the antihyperlipidemic effect of aqueous Aloe vera gel extract in high-fat diet (HFD)-induced hyperlipidemic rats. A total of 30 adult male Long Evans rats weight 180–200 g and aged 10–12 weeks were enrolled and divided into five groups. The study was carried out in the Department of Pharmacology and Therapeutics, Sir Salimullah Medical College, Dhaka, in collaboration with the Institute of Nutrition and Food Science, Dhaka University, from February 2023 to January 2024.

In this study, rats fed a HFD for six weeks (group B) developed significant hyperlipidemia compared to normal controls (group A), as evidenced by elevated serum total cholesterol (186.33±1.37 mg/dl vs 91.1±1.17 mg/dl), triglycerides (136.33±1.51 mg/dl vs 94.33±1.37 mg/dl), LDL-C (139.17±1.87 mg/dl vs 36.0±2.37 mg/dl), and reduced HDL-C (19.83±0.75 mg/dl vs 36.2±1.17 mg/dl). These results were statistically significant ($P<0.001$) and consistent with previous reports showing that HFD induces dyslipidemia in rodents.[14,15]

Intervention with aqueous Aloe vera extract at 300 mg/kg (group C) and 200 mg/kg combined with atorvastatin 10 mg/kg (group E) significantly improved lipid profiles in hyperlipidemic rats. Group C showed reductions in total cholesterol (94.17±1.72 mg/dl), triglycerides (96.17±3.43 mg/dl), LDL-C (40.43±1.49 mg/dl), and an increase in HDL-C (34.50±0.55 mg/dl). Group E showed similar improvements: total cholesterol 91.5±1.38 mg/dl, triglycerides 94.5±0.55 mg/dl, LDL-C 34.9±1.46 mg/dl, and HDL-C 36.8±0.75 mg/dl. The lipid-lowering effect of Aloe vera extract was comparable to that of atorvastatin alone (group D), which had total cholesterol 92.5±1.22 mg/dl, triglycerides 95.67±0.82 mg/dl, LDL-C 37.73±2.65 mg/dl, and HDL-C 36.17±0.75 mg/dl.

A significant negative correlation exists between high-fat diet–induced hyperlipidemia and serum HDL-C, while positive correlations were observed with TC, TG, and LDL-C. These findings align with previous studies.[16-20] The hypolipidemic effect of Aloe vera may be attributed to inhibition of HMG-CoA reductase, increased clearance of LDL-C, reduced bile acid absorption via glucomannan polysaccharides, and decreased hepatic lipogenesis through the AMPK pathway.[21,22]

Comparison between the Aloe vera-only group (C) and combination therapy group (E) showed that combination therapy produced slightly greater improvement, particularly in HDL-C levels, although the differences were not statistically significant for most lipid parameters. These observations suggest that Aloe vera gel can serve as an effective adjunct to conventional lipid-lowering therapy.

The study demonstrates that aqueous Aloe vera gel extract possesses significant antihyperlipidemic

activity in HFD-induced hyperlipidemic rats and shows potential as a natural, low-cost alternative or supplement to standard therapy for dyslipidemia.

Conclusion

Aloe vera gel extract significantly improved the lipid profile in high-fat-diet–induced hyperlipidemic rats. Its effect was comparable to atorvastatin for most parameters, except HDL. Combination therapy showed greater improvement than Aloe vera alone and produced effects comparable to atorvastatin, suggesting that Aloe vera can serve as a potential adjunct therapy for hyperlipidemia.

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Conflict of Interest: There are no conflicts of interest.

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Ethical Clearance: Ethical clearance taken from the Institutional Review Board (IRB) of SSMC.

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