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Attenuation of Left Ventricular Hypertrophy by *Psidium guajava* Leaf Extract Via Inhibition of Oxidative Stress and Cardiac Troponin I Levels in Adrenaline-Induced Hypertrophic Rats

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Abstract: Cardiovascular disease (CVD) remains a predominant global cause of mortality, with left ventricular hypertrophy (LVH) representing a significant risk factor for life-threatening cardiovascular incidents. Our study aimed to assess the impact of an ethanolic extract of *Psidium guajava* leaves (PGLE) on adrenaline-induced LVH in rats by evaluating lipid metabolism, cardiac biomarkers, and oxidative stress indicators. Swiss Albino rats were subjected to intraperitoneal injections of adrenaline (100 µL) over 14 days to induce LVH. PGLE was administered via oral gavage at three graded dose levels (100, 200, and 300 mg/kg body weight) for four weeks. The parameters measured included total cholesterol, triglycerides, low-density lipoprotein, high-density lipoprotein, troponin-I, superoxide dismutase, catalase activity, malondialdehyde levels, and liver enzymes (SGPT and SGOT). Adrenaline administration increased total cholesterol, triglycerides, and low-density lipoprotein levels while reducing high-density lipoprotein levels. Treatment with PGLE significantly ameliorated these lipid abnormalities and improved the ratio of left ventricular weight to body weight, suggesting a decrease in LVH. Histological examinations revealed a reduction in cardiac myocyte size in PGLE-treated rats. Furthermore, PGLE administration markedly lowered troponin-I levels, boosted superoxide dismutase and catalase activities, and reduced malondialdehyde concentrations. PGLE also normalized liver enzyme levels, indicating reduced hepatic toxicity. The findings indicate that PGLE exerts potent lipid-lowering and antioxidant properties, mitigating cardiac remodeling in adrenaline-induced hypertrophic rats. Importantly, PGLE significantly decreased troponin-I levels, underscoring its potential as a therapeutic option for the prevention of LVH and associated cardiovascular conditions.

Keywords: left ventricular hypertrophy; cardiovascular disorders; ethanolic extract; adrenaline; lipid metabolism; antioxidant defense

1. Introduction

Cardiovascular diseases (CVDs) remain a leading global health burden, accounting for over 17.9 million deaths annually and nearly half of all non-communicable disease-related mortality [1]. These disorders include coronary artery disease, myocardial infarction, stroke, and heart failure [2]. Among the major risk factors, hyperlipidemia and left ventricular hypertrophy (LVH) play critical roles in disease progression. Hyperlipidemia, characterized by elevated cholesterol and triglycerides, promotes atherosclerosis and increases the risk of cardiovascular complications [3,4].

LVH, defined as the thickening of the left ventricular myocardium, arises primarily from chronic pressure overload such as hypertension, as well as metabolic and neurohormonal disturbances [5–8]. Its development involves complex signaling pathways, where oxidative stress is a key contributing factor. Excessive production of reactive oxygen species (ROS) disrupts redox balance, leading to endothelial dysfunction, inflammation, and

cardiac remodeling [9,10]. Therefore, strategies targeting both lipid abnormalities and oxidative stress are crucial for effective CVD management.

Although conventional lipid-lowering agents such as statins are effective, their long-term use is associated with adverse effects including myopathy and hepatotoxicity [11]. This has encouraged the exploration of plant-based alternatives with improved safety profiles. In this context, *Psidium guajava* (guava) has gained attention due to its rich phytochemical composition, including flavonoids, tannins, and triterpenes, which exhibit antioxidant and cardioprotective properties [12–14]. Previous studies have demonstrated its potential to improve lipid profiles and reduce oxidative stress [15].

The present study investigates the cardioprotective, antioxidant, and antidyslipidemic effects of the ethanolic extract of *P. guajava* leaves in an adrenaline-induced LVH rat model. Adrenaline-induced cardiac remodeling is widely used to mimic hyperlipidemia and oxidative stress conditions [15]. Although adrenaline-induced models are primarily associated with acute cardiac stress, repeated administration has been shown to induce cardiac remodeling, oxidative stress, and hypertrophy-like changes, making it a suitable experimental model for studying early-stage LVH and cardiotoxicity [16]. This study evaluates the effects of the extract on lipid parameters, oxidative stress markers, and cardiac injury biomarkers such as troponin-I [17]. Additionally, its efficacy is compared with atorvastatin, a standard HMG-CoA reductase inhibitor [18], to assess its potential as a natural therapeutic alternative for managing LVH and associated cardiovascular risks.

2. Materials and Methods

2.1. Sourcing of Drugs and Chemicals

The active drug atorvastatin was a generous gift from Square Pharmaceuticals Ltd. (Pabna plant), Bangladesh. Adrinor, Adrenaline 1mg/mL IV, IM, SC Injection (Incepta Pharmaceuticals Ltd. Dhaka, Bangladesh) was purchased and used for producing hyperlipidemia in rats and also for SOD estimation in liver tissue. Total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL) and high-density lipoprotein (HDL), kits were acquired from Human, Germany. Troponin-I (cTnI) kits were purchased from Human, Germany. SGPT (ALT) and SGOT (AST) kits were also purchased from Human, Germany.

2.2. Collection of Plant Leaves

Fresh, healthy green leaves of *P. guajava* were collected from the Rajshahi University campus (latitude: 24.3682° N, longitude: 88.6376° E) in Bangladesh and verified by Dr. A.H.M. Mahbubur Rahman, a specialist in taxonomy from the Department of Botany at the University of Rajshahi.

2.3. Preparation of Leaf Extract

The collected leaves were washed thoroughly, sun-dried for several days, and ground into a coarse powder using an electric grinder. The powder was stored in airtight containers to prevent moisture and contamination. For extraction, 400 g of powder were placed in each of two amber bottles and soaked in a solvent mixture of 90% ethanol and 10% distilled water. The mixtures were shaken regularly over 14 days to extract bioactive compounds, then filtered through cotton and Whatman paper [19]. The filtrate was concentrated using a rotary evaporator at 40–50 °C to obtain a viscous crude ethanolic extract.

2.4. Qualitative Phytochemical Screening

Qualitative phytochemical analysis of the crude guava leaves extract was conducted to detect the presence of various secondary metabolites described by Shaikh and Patil [20]. The tests performed included those for alkaloids, steroids, cardiac glycosides (Salkowski test), tannins, saponins, phenolic compounds, and reducing sugars.

2.5. Animal Selection and Maintenance

Thirty-five male Swiss albino rats (130–150 g, ~2 months old) were obtained from Jahangirnagar University's Pharmacology Research Laboratory. They were acclimatized for one week under controlled conditions (25 °C, 12-h light/dark cycle) with standard feed and water. All experimental procedures complied with the Institutional Animal Ethics Committee guidelines and ethical standards for animal research.

2.6. Experimental Design and Treatment

The study aimed to evaluate the effects of *Psidium guajava* leaf extract and atorvastatin on adrenaline-induced cardiac hypertrophy in rats (AIHRs). Thirty Swiss albino rats were randomly divided into six groups (n = 5) using a randomization method: Group A served as the normal control and received no treatment, while the remaining groups were subjected to daily intraperitoneal injections of 100 µL adrenaline for 14 days to induce hypertrophy, following the established protocol for hypertrophy induction [21,22]. Group B acted as the hypertrophic control, receiving only adrenaline. Group C was treated with atorvastatin at a dose of 20 mg/kg body weight, prepared in phosphate buffer and administered orally. Groups D, E, and F received guava leaf extract at doses of 100 mg/kg, 200 mg/kg, and 300 mg/kg body weight, respectively, with each dose dissolved in distilled water to appropriate concentrations (10, 20, and 30 mg/mL) and given orally via gavage. Treatments with atorvastatin and guava extract began simultaneously with adrenaline administration and continued for 28 days.

2.7. Measurement of Lipid Profile

After the treatment duration, the rats were sedated with Phenobarbitone Sodium (Barbit, Incepta Pharmaceuticals, Dhaka, Bangladesh). Blood specimens were harvested directly from the thoracic artery and subjected to centrifugation at 4000 rpm for 15 min to obtain serum. The serum specimens were subsequently evaluated for total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) utilizing UV spectrophotometry and commercial diagnostic kits (Human, Germany) such as cholesterol liquicolor test kits, triglycerides liquicolor test kits, HDL cholesterol test kits, and LDL cholesterol test kits.

2.8. Assay of Liver Function

Serum SGPT & SGOT test was performed using SGPT & SGOT test kits purchased from Human, Germany. According to the expert panel of the IFCC (International Federation of Clinical Chemistry), it is a kinetic method for measuring ALT and ASAT activity, respectively, without pyridoxal phosphate activation. The purpose of these tests was to measure liver dysfunction parameters.

2.9. Determination of Antioxidant Activity

To determine the antioxidant activity, superoxide dismutase (SOD), catalase activity, and malondialdehyde (MDA) levels were assessed using homogenates prepared in Tris buffer (pH 7.4). Liver tissue was selected for oxidative stress assessment as a metabolically active organ sensitive to systemic oxidative imbalance. Liver tissue homogenates were prepared using Tris buffer (pH 7.4) made by dissolving 0.6057 g of tris-(hydroxymethyl)-aminomethane in distilled deionized water, adjusting the pH with HCl or NaOH, and making up the volume to 100 mL. Superoxide dismutase (SOD) activity was measured using the Nitro Blue Tetrazolium (NBT) reduction method, where homogenates were reacted with NBT, NADH, and phenazine methosulfate, and absorbance was recorded at 560 nm. Catalase activity was evaluated following the Goth method by adding homogenates to a hydrogen peroxide solution in phosphate buffer and monitoring the decrease in absorbance at 240 nm over time.

2.10. Estimation of Troponin I Levels

Serum cardiac troponin I (cTnI) levels were measured using a commercially available ELISA kit (Human Diagnostics, Wiesbaden, Germany). The test is based on the principle of a solid phase enzyme-linked immunosorbent assay.

2.11. LVW/BW Ratio

An increase in the left ventricular weight to body weight (LVW/BW) ratio is a well-established indicator of cardiac hypertrophy. Therefore, elevated LVW/BW values reflect the development of LVH.

2.12. Histopathological Examination

After blood collection, the hearts were excised, fixed in 10% neutral buffered formalin (NBF), and processed for histopathological study [23]. Left ventricular (LV) tissue sections, each 5 µm in thickness, were subjected to staining with hematoxylin and eosin. The mass of the LV and the ratio of LV mass to body weight were subsequently determined to assess hypertrophy. Cardiomyocyte cross-sectional areas were measured at 400× magnification using Olympus BX51TF microscopy (Olympus Corporation, Tokyo, Japan), and Scion Image software (Scion Corporation, Frederick, MD, USA) was used for image analysis. All histological evaluations were conducted by an investigator blinded to the treatment groups to minimize bias.

2.13. Statistical Analysis

Values were presented as mean ± standard error of the mean. Statistical evaluations were conducted using one-way analysis of variance (ANOVA), followed by Dunnett's post hoc test for multiple comparisons or the student's t-test, where appropriate. A *p*-value less than 0.05 was deemed statistically impactful.

3. Results

3.1. Screening of Phytochemical Constituents

The phytochemical analysis of *P. guajava* leaf extract confirmed the presence of several bioactive compounds, including alkaloids, cardiac glycosides, tannins, saponins, phenolic compounds, and reducing sugars, all of which are known for their potential therapeutic effects. However, the test for steroids indicated either their absence or presence in very low concentrations, as the characteristic color change was not observed. These findings highlight the rich phytochemical profile of guava leaves, supporting their traditional medicinal use.

3.2. Effect of Atorvastatin and PGLE on Lipid Profile

The *Psidium guajava* extract demonstrated significant lipid-lowering effects across total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL) in adrenaline-induced hypertrophic rats, closely comparable to atorvastatin. Adrenaline treatment raised TC, TG, and LDL levels significantly while lowering HDL. Treatment with *P. guajava* extract at 100, 200, and 300 mg/kg doses reduced TC levels to 96.06, 70.04, and 54.24 mg/dL respectively, versus atorvastatin's 78.2 mg/dL (Figure 1A). Similarly, TG levels dropped from 120.1 mg/dL in hypertrophic rats to 87.9, 68.72, and 54.92 mg/dL with the extract, nearing atorvastatin's 66.25 mg/dL effect (Figure 1B).

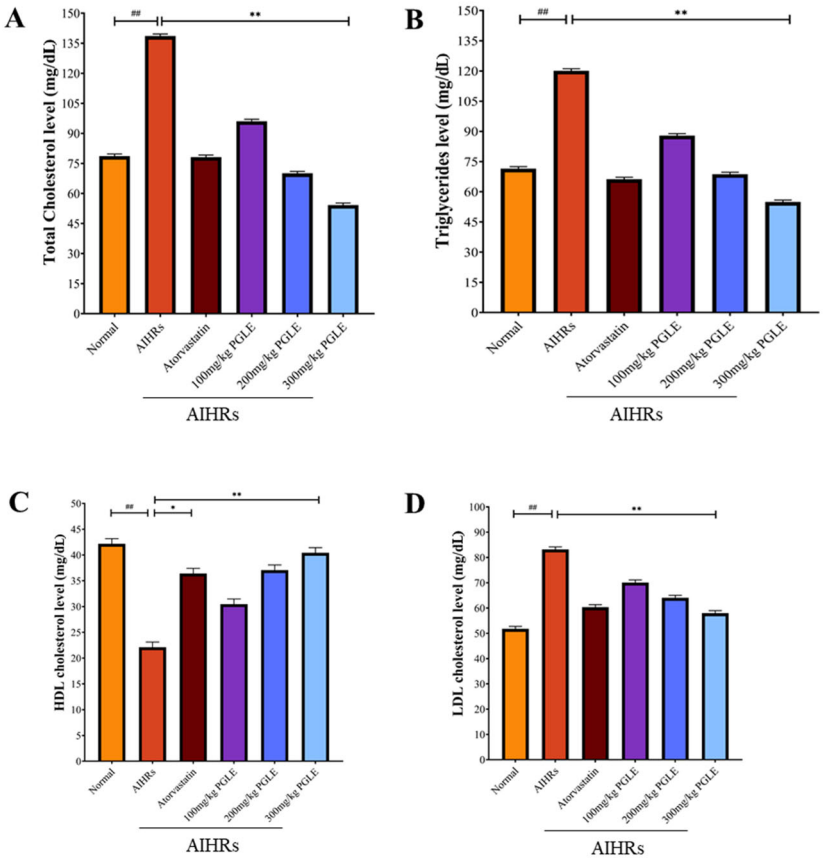

Figure 1. Effects of PGLE on (**A**) TC; (**B**) TG; (**C**) HDL; and (**D**) LDL level in AIHRs for four weeks in comparison with AIHRs. n = 5 in each group. $^{\#\#}$ $p < 0.001$ vs. normal, * $p < 0.01$ vs. AIHRs and ** $p < 0.001$ vs. AIHRs (ANOVA followed by Dunnett's test).

Furthermore, HDL levels, reduced to 22.11 mg/dL by adrenaline, improved with extract treatment to 30.47, 37.06, and 40.43 mg/dL, comparable to atorvastatin's effect at 36.4 mg/dL (Figure 1C). Lastly, LDL levels were

effectively lowered from 83.2 mg/dL in hypertrophic rats to 70.1, 64.03, and 57.98 mg/dL with the extract, aligning with atorvastatin's 60.33 mg/dL (Figure 1D). Collectively, these results show *P. guajava* extract's potential as a natural alternative for managing hyperlipidemia and supporting cardiovascular health.

3.3. Hepatoprotective Activity of PGLE

The *P. guajava* extract significantly lowered serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) levels in adrenaline-induced hypertrophic rats, showing effects comparable to atorvastatin. In the case of SGOT, adrenaline administration raised levels to 39.74 ± 0.86 U/L, up from a normal level of 21.89 ± 0.91 U/L. Treatment with *P. guajava* extract at 100, 200, and 300 mg/kg reduced SGOT levels to 36.65 ± 1.37 U/L, 30.5 ± 1.87 U/L, and 29.34 ± 1.89 U/L, respectively, while atorvastatin treatment lowered it further to 29.28 ± 1.35 U/L (Figure 2A). Similarly, SGPT levels rose to 43.77 ± 0.95 U/L in adrenaline-treated rats compared to the normal level of 26.2 ± 0.94 U/L. The extract reduced SGPT levels to 38.68 ± 1.2 U/L, 34.11 ± 1.13 U/L, and 29.54 ± 0.97 U/L at the respective doses, with atorvastatin bringing SGPT to 31.75 ± 0.97 U/L (Figure 2B). Together, these findings suggest that *P. guajava* extract effectively mitigates the increase in both SGOT and SGPT associated with hypertrophic conditions, potentially supporting liver health in conditions of cardiac stress.

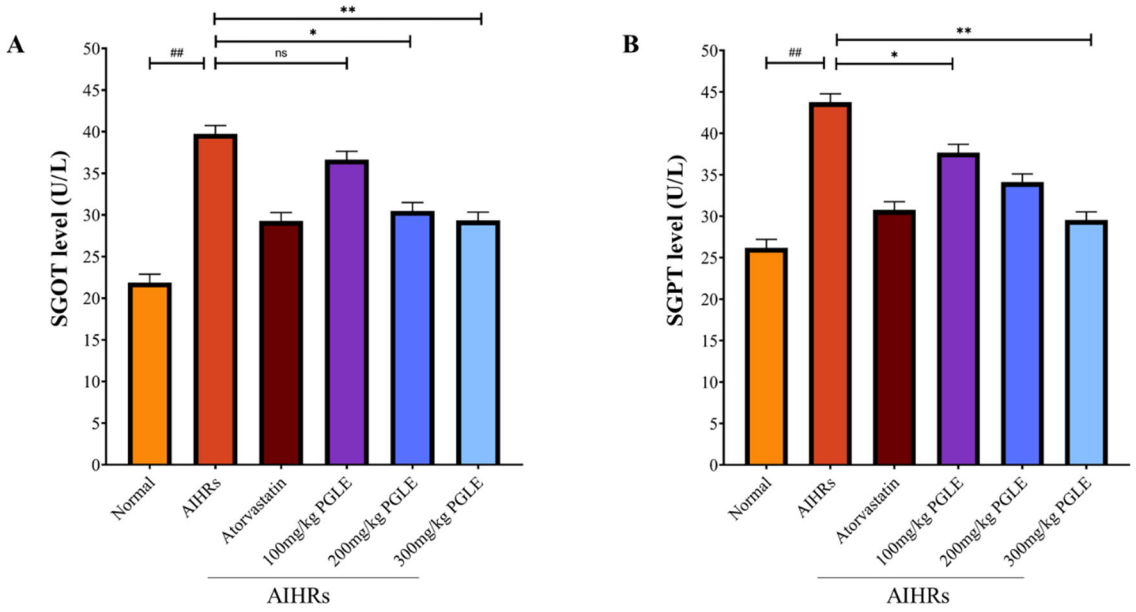

Figure 2. Effects of PGLE on (**A**) SGOT and (**B**) SGPT level in AIHRs for four weeks. n = 5 in each group. ## $p < 0.001$ vs. normal, * $p < 0.01$ vs. AIHRs and ** $p < 0.001$ vs. AIHRs (ANOVA followed by Dunnett's test).

3.4. Antioxidant Activity of PGLE in Rats with Myocardial Hypertrophy

The *P. guajava* extract demonstrated protective antioxidant effects in hypertrophic rats by modulating key enzymes and reducing oxidative stress markers. Adrenaline treatment significantly reduced superoxide dismutase (SOD) activity to 2.49 ± 0.36 U/mg from a normal level of 4.2 ± 0.24 U/mg. The extract at 100, 200, and 300 mg/kg doses restored SOD activity to 3.04 ± 0.15 U/mg, 3.8 ± 0.35 U/mg, and 3.97 ± 0.27 U/mg, respectively, comparable to atorvastatin's effect (3.66 ± 0.17 U/mg) (Figure 3A). Similarly, catalase (CAT) activity, which dropped to 30.96 ± 1.76 U/mg after adrenaline exposure, improved with extract administration to 46.79 ± 0.94 U/mg, 59.18 ± 2.37 U/mg, and 65.82 ± 2.65 U/mg, nearing atorvastatin's level of 63.76 ± 1.57 U/mg (Figure 3B). The extract also effectively reduced malondialdehyde (MDA) levels, a marker of lipid peroxidation, which were elevated to 256.8 ± 10.9 U/mg by adrenaline. Doses of 100, 200, and 300 mg/kg brought MDA levels down to 227.27 ± 8.54 U/mg, 206.96 ± 6.74 U/mg, and 178.58 ± 5.64 U/mg, respectively, in alignment with atorvastatin's reduction to 198.92 ± 5.35 U/mg (Figure 3C). These findings collectively highlight *P. guajava* extract's potent antioxidant activity, mitigating oxidative damage in hypertrophic conditions.

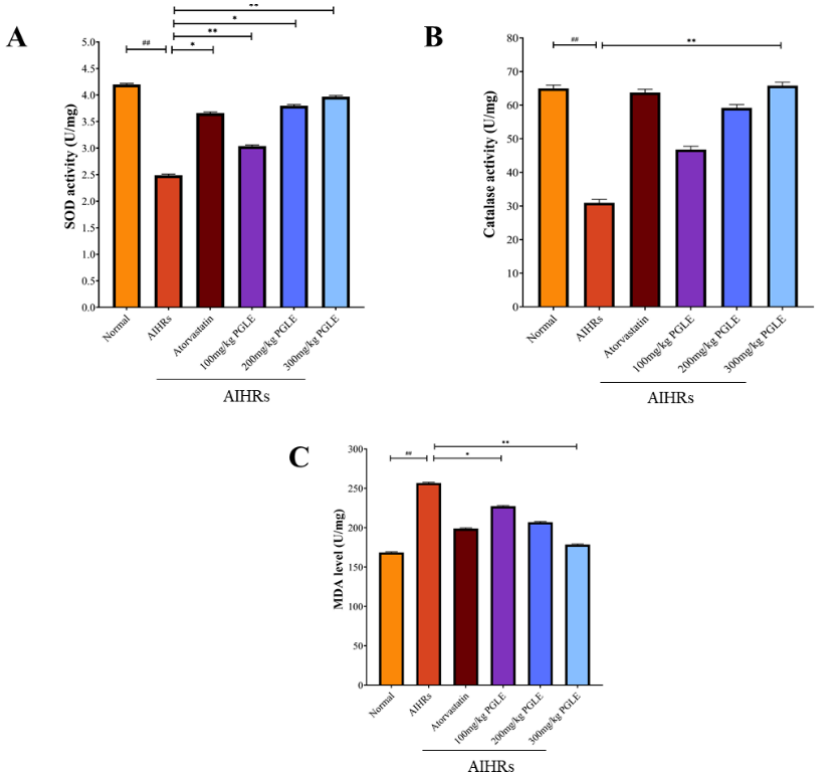

Figure 3. Effects of PGLE on (**A**) SOD; (**B**) CAT; and (**C**) MDA level in AIHRs for four weeks. n = 5 in each group. $^{\#\#}p < 0.001$ vs. normal, * $p < 0.05$ vs. AIHRs and ** $p < 0.001$ vs. AIHRs (ANOVA followed by Dunnett's test).

3.5. Effect of PGLE on Troponin-I (cTnI) Levels

Following adrenaline treatment, cTnI levels increased to 0.66 ± 0.038 ng/mL compared to normal rats, which had a cTnI level of 0.2 ± 0.008 ng/mL. Guava leaves extract treatment at 100, 200, and 300 mg/kg doses lowered cTnI levels to 0.48 ± 0.024 ng/mL, 0.28 ± 0.022 ng/mL, and 0.21 ± 0.022 ng/mL, respectively. The drug showed a comparable reduction to 0.66 ± 0.038 ng/mL showed in Figure 4.

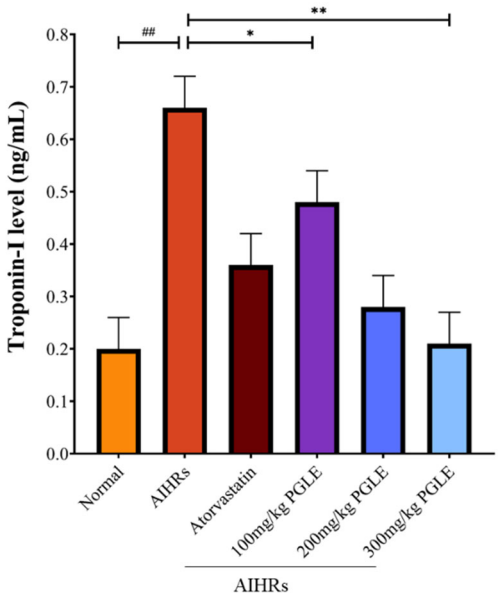

Figure 4. Effects of PGLE on cTnI level in AIHRs for four weeks in comparison with AIHRs. n = 5 in each group. $^{\#\#}p < 0.001$ vs. normal, * $p < 0.01$ vs. AIHRs and ** $p < 0.001$ vs. AIHRs (ANOVA followed by Dunnett's test).

3.6. Activity of PGLE on LVH in Hypertrophy-Induced Rats

The proportion of left ventricular mass to total body mass increased to 0.046 ± 0.0021 in hypertrophic rats, while the normal control had a ratio of 0.022 ± 0.006. *P. guajava* extract at doses of 100, 200, and 300 mg/kg reduced this ratio to 0.037 ± 0.0013, 0.029 ± 0.0011, and 0.024 ± 0.0009, respectively. Atorvastatin also significantly decreased the ratio to 0.025 ± 0.006. Figure 5A displays the responses graphically.

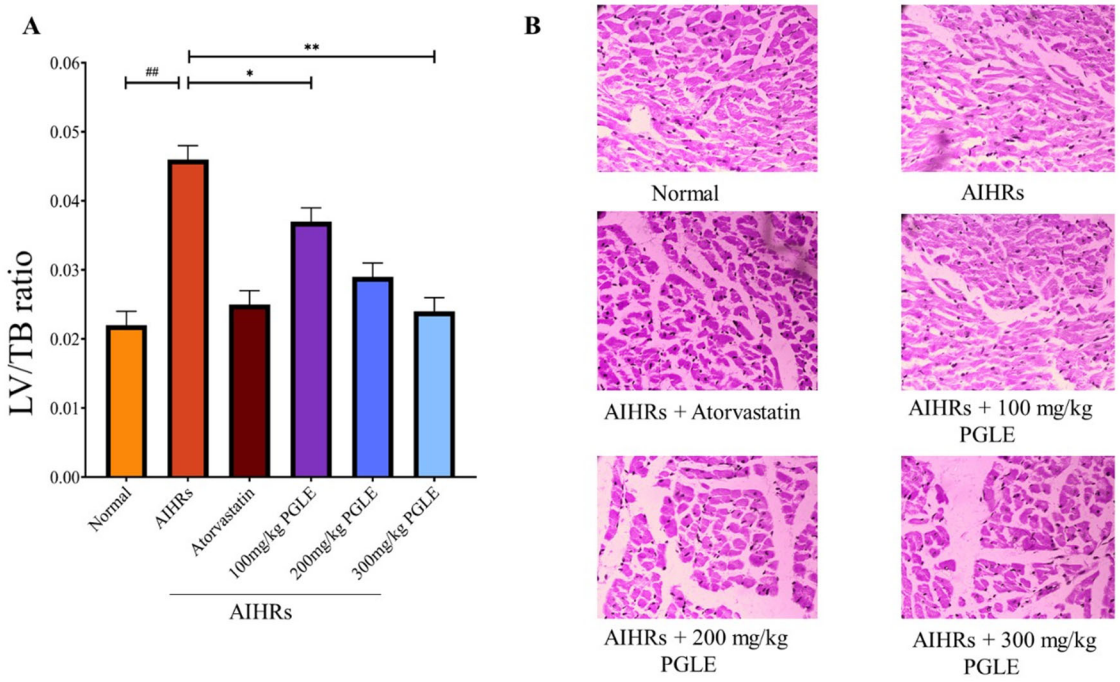

Figure 5. (**A**) Effects of PGLE on LVH in AIHRs for four weeks. n = 5 in each group. $^{\#\#}p < 0.001$ vs. normal, * $p < 0.05$ vs. AIHRs and ** $p < 0.001$ vs. AIHRs (ANOVA followed by Dunnett's test). (**B**) Microscopic view of cardiac myocytes.

3.7. Histopathology of Left Ventricle of Rat Heart

The microscopic analysis confirms that *Psidium guajava* leaf extract significantly protects against adrenaline-induced cardiac hypertrophy in a dose-dependent manner. The 300 mg/kg dose effectively restored normal myocardial architecture, showing superior cardioprotective effects compared to atorvastatin (Figure 5B).

4. Discussion

The present study evaluated the antidyslipidemic, antioxidant, and cardioprotective effects of *P. guajava* leaf extract in an adrenaline-induced hypertrophic rat model. Cardiovascular diseases (CVDs), with hyperlipidemia as a major risk factor, remain a significant global health burden, and LVH is strongly associated with adverse cardiac outcomes [24].

Adrenaline administration successfully induced dyslipidemia and cardiac hypertrophy, as evidenced by elevated TC, TG, and LDL levels along with reduced HDL levels [25]. Treatment with *P. guajava* extract significantly improved these lipid parameters in a dose-dependent manner, with the highest dose (300 mg/kg) showing effects comparable to atorvastatin. This lipid-lowering effect may be attributed to enhanced cholesterol clearance and modulation of lipid metabolism [13].

Liver enzyme analysis revealed that adrenaline increased SGPT and SGOT levels, indicating hepatic stress. However, *P. guajava* extract significantly reduced these levels, suggesting hepatoprotective activity, likely mediated by its antioxidant constituents [26].

Oxidative stress plays a central role in the progression of LVH. In the present study, adrenaline administration reduced antioxidant enzyme activities (SOD and CAT) and increased MDA levels, indicating enhanced oxidative damage. Treatment with *P. guajava* extract significantly restored SOD and CAT activities while reducing MDA levels, demonstrating strong antioxidant potential. These effects were comparable to, or in some cases greater than, those of atorvastatin, possibly due to the presence of bioactive flavonoids such as quercetin. However, it should be noted that oxidative stress markers were assessed in liver tissue, which reflects systemic redox status but may

not directly represent cardiac oxidative stress. Therefore, future studies should include cardiac tissue analysis to provide more precise mechanistic insights.

Cardiac injury was further confirmed by elevated troponin-I levels following adrenaline administration. The extract significantly reduced cTnI levels, particularly at 300 mg/kg, indicating mitigation of myocardial damage [27]. Additionally, the reduction in LVW/BW ratio and improvement in histological architecture demonstrate its antihypertrophic effect. However, it should be noted that the adrenaline-induced model reflects acute-to-subacute cardiac stress rather than chronic pressure overload, which is a limitation when extrapolating to long-term LVH conditions.

Overall, *P. guajava* leaf extract demonstrated significant improvements in lipid metabolism, attenuation of oxidative stress, hepatoprotective effects, and reduction of cardiac hypertrophy in this experimental model. These findings highlight its potential as a natural therapeutic candidate for managing hyperlipidemia-associated LVH. However, the relatively small sample size (n = 5 per group) may limit the statistical power of the study. Nevertheless, this sample size is consistent with ethical considerations for animal experimentation and comparable to similar preclinical studies. Further investigations are required to isolate the active constituents and elucidate the underlying molecular mechanisms.

5. Conclusions

The study demonstrates that the ethanolic extract of *P. guajava* leaves exerts significant cardioprotective, lipid-lowering, hepatoprotective, and antioxidant effects in adrenaline-induced hypertrophic rats. The extract improved lipid profiles, reduced liver enzyme levels, enhanced antioxidant enzyme activities, lowered MDA and troponin-I levels, and showed comparable or superior efficacy to atorvastatin in several parameters. These findings suggest that *P. guajava* extract holds promise as a natural therapeutic agent for managing left ventricular hypertrophy (LVH) and related cardiovascular risks. Further research is needed to isolate the active compounds and elucidate their mechanisms of action.

Author Contributions: R.K.: methodology, formal analysis, and interpretation; J.S.: data curation, writing—original draft and validation; F.S.: writing—review & editing; M.S.H.: writing—review & editing; A.B.M.A.: software; A.H.M.K.A.: writing—review & editing and validation; M.R.: conceptualization, supervision and investigation. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Institute of Biological Sciences (IBSc), Rajshahi University, Bangladesh (license no: 72 (23)/320/IAMEBBC/IBSc).

Informed Consent Statement: Not applicable.

Data Availability Statement: All data generated or analyzed during this study are included in this published article.

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Conflicts of Interest: The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies: During the preparation of this work, the authors used ChatGPT to enhance readability and language. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the final manuscript.

Abbreviations

Full term	Abbreviation
Cardiovascular disease	CVD
Left ventricular hypertrophy	LVH
Psidium guajava leaves extract	PGLE
Alanine Aminotransferase	SGPT
Aspartate Aminotransferase	SGOT
World Health Organization	WHO
Reactive Oxygen Species	ROS
Low-density lipoprotein	LDL
High-density lipoprotein	HDL
Superoxide dismutase	SOD
Catalase enzyme	CAT
Malondialdehyde	MDA
Nitro blue tetrazolium	NBT
Hydrogen peroxide	H_2O_2
3-hydroxy-3-methylglutaryl coenzyme A	HMG-CoA
Intravenous	IV

Intramuscular	IM
Subcutaneous	SC
Total cholesterol	TC
Triglyceride	TG
Thiobarbituric acid reactive substances	TBARS
Thiobarbituric acid	TBA
Mili-liter	mL
Cardiac troponin-I	cTnI
Very low-density lipoprotein	VLDL
Enzyme-linked immunosorbent assay	ELISA
Malondialdehyde tetrabutylammonium	MDA
Ethanol	EtOH
Nitro Blue Tetrazolium	NBT
Thiobarbituric acid	TBA
Trichloroacetic acid	TCA
One-way analysis of variance	ANOVA

References

1. Soares, L.; Leal, T.; Faria, A.L.; et al. Cardiovascular Disease: A Review. *Biomed. J. Sci. Tech. Res.* **2023**, *51*, 42696–42703.
2. Khan, S.U.; Saeed, S.; Sheikh, A.N.; et al. Crafting a Blueprint for MicroRNA in Cardiovascular Diseases (CVDs). *Curr. Probl. Cardiol.* **2023**, *48*, 102010.
3. Abbasi, S.; Khan, A.; Choudhry, M.W. New Insights into the Treatment of Hyperlipidemia: Pharmacological Updates and Emerging Treatments. *Cureus* **2024**, *16*, e62789.
4. Blesso, C.N.; Fernandez, M.L. Dietary Cholesterol, Serum Lipids, and Heart Disease: Are Eggs Working for or against You? *Nutrients* **2018**, *10*, 426.
5. Shenasa, M.; Shenasa, H. Hypertension, Left Ventricular Hypertrophy, and Sudden Cardiac Death. *Int. J. Cardiol.* **2017**, *237*, 60–63.
6. Kim, T.-H.; Yang, P.-S.; Yu, H.T.; et al. Effect of Hypertension Duration and Blood Pressure Level on Ischaemic Stroke Risk in Atrial Fibrillation: Nationwide Data Covering the Entire Korean Population. *Eur. Heart J.* **2019**, *40*, 809–819.
7. Oh, G.C.; Cho, H.-J. Blood Pressure and Heart Failure. *Clin. Hypertens.* **2020**, *26*, 1.
8. Shad, M.; Shah, F.S.; Zulqarnain, M.; et al. Analyzing and Identifying the Molecular Targets and Regulators Controlling Cardiac Hypertrophy Progression. *NUST J. Nat. Sci.* **2020**, *5*. https://doi.org/10.53992/njns.v5i2.47.
9. Yan, Q.; Liu, S.; Sun, Y.; et al. Targeting Oxidative Stress as a Preventive and Therapeutic Approach for Cardiovascular Disease. *J. Transl. Med.* **2023**, *21*, 519.
10. Donia, T.; Khamis, A. Management of Oxidative Stress and Inflammation in Cardiovascular Diseases: Mechanisms and Challenges. *Environ. Sci. Pollut. Res.* **2021**, *28*, 34121–34153.
11. Abdul-Rahman, T.; Bukhari, S.M.A.; Herrera, E.C.; et al. Lipid Lowering Therapy: An Era beyond Statins. *Curr. Probl. Cardiol.* **2022**, *47*, 101342.
12. Gutierrez-Montiel, D.; Guerrero-Barrera, A.L.; Chávez-Vela, N.A.; et al. *Psidium guajava* L.: From Byproduct and Use in Traditional Mexican Medicine to Antimicrobial Agent. *Front. Nutr.* **2023**, *10*, 1108306.
13. Kumar, M.; Tomar, M.; Amarowicz, R.; et al. Guava (*Psidium guajava* L.) Leaves: Nutritional Composition, Phytochemical Profile, and Health-Promoting Bioactivities. *Foods* **2021**, *10*, 752.
14. Kareem, A.T.; Kadhim, E.J. *Psidium guajava*: A Review on Its Pharmacological and Phytochemical Constituents. *Biomed. Pharmacol. J.* **2024**, *17*, 1079–1090.
15. Bumrungpert, A.; Lilitchan, S.; Tuntipopipat, S.; et al. Ferulic Acid Supplementation Improves Lipid Profiles, Oxidative Stress, and Inflammatory Status in Hyperlipidemic Subjects: A Randomized, Double-Blind, Placebo-Controlled Clinical Trial. *Nutrients* **2018**, *10*, 713.
16. El-Marasy, S.A.; El Awdan, S.A.; Hassan, A.; et al. Cardioprotective Effect of Thymol against Adrenaline-Induced Myocardial Injury in Rats. *Heliyon* **2020**, *6*, e04371.
17. Aladağ, N.; Asoğlu, R.; Ozdemir, M.; et al. Oxidants and Antioxidants in Myocardial Infarction (MI): Investigation of Ischemia Modified Albumin, Malondialdehyde, Superoxide Dismutase and Catalase in Individuals Diagnosed with ST Elevated Myocardial Infarction (STEMI) and Non-STEMI (NSTEMI). *J. Med. Biochem.* **2021**, *40*, 286–293.
18. Saeedi Saravi, S.S.; Saeedi Saravi, S.S.; Arefidoust, A.; et al. The Beneficial Effects of HMG-CoA Reductase Inhibitors in the Processes of Neurodegeneration. *Metab. Brain Dis.* **2017**, *32*, 949–965.
19. Sarker, J.; Reza, A.S.M.A.; Ashraful, A.B.M.; et al. *In Vitro* Antioxidant and p53-Targeted Anticancer Activity of *Aerva sanguinolenta* (L.) Blume across Different Human Cancer Cell Lines and Ehrlich Ascites Carcinoma Model. *J. Chem. Health Risks* **2026**, *16*, 1292–1316.
20. Shaikh, J.R.; Patil, M. Qualitative Tests for Preliminary Phytochemical Screening: An Overview. *Int. J. Chem. Stud.* **2020**, *8*, 603–608.

21. Ali, Y.; Islam, M.; Alam, A.; et al. Inhibitory Effects of *Nigella sativa* Seed Extract on Adrenaline-Induced Dyslipidemia and Left Ventricular Hypertrophy in Rats. *J. Sci. Res.* **2013**, *5*, 325–334.
22. Majewski, H.; Tung, L.-H.; Rand, M. Adrenaline-Induced Hypertension in Rats. *J. Cardiovasc. Pharmacol.* **1981**, *3*, 179–186.
23. Constantin, I.; Tăbăran, A.F. Dissection Techniques and Histological Sampling of the Heart in Large Animal Models for Cardiovascular Diseases. *J. Vis. Exp.* **2022**, *184*, e63809.
24. Saeed, A.; Virani, S.S.; Mulukutla, S.; et al. Dyslipidemia and Cardiovascular Disease Prevention in South Asians: A Review and Discussion of Causes, Challenges and Management Strategies. *Curr. Diabetes Rev.* **2021**, *17*, 74–86.
25. Khodir, S.A.; Sweed, E.; Gadallah, M.; et al. Astaxanthin Attenuates Cardiovascular Dysfunction Associated with Deoxycorticosterone Acetate-Salt-Induced Hypertension in Rats. *Clin. Exp. Hypertens.* **2022**, *44*, 382–395.
26. Vijayakumar, K.; Arumugam, V.A.; Ramasamy, M.; et al. Hepatoprotective Effects of *Psidium guajava* on Mitochondrial Enzymes and Inflammatory Markers in Carbon Tetrachloride-Induced Hepatotoxicity. *Drug Dev. Ind. Pharm.* **2020**, *46*, 2041–2050.
27. Wen, C.; Liu, C.; Li, Y.; et al. Ameliorative Potentials of the Ethanolic Extract from *Lycium chinense* Leaf Extract against Diabetic Cardiomyopathy. Insight into Oxido-Inflammatory and Apoptosis Modulation. *Biomed. Pharmacother.* **2022**, *154*, 113583.