Evaluation of the Antihyperlipidemic and Cardioprotective Effects of Hydroalcoholic Extract of a Polyherbal Combination in High-Fat Diet-Induced Hyperlipidemia and Isoproterenol-Induced Myocardial Infarction in Wistar Rats

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1. Abstract

Cardiovascular diseases (CVDs) continue to be the primary cause of death globally, with hyperlipidemia and myocardial infarction (MI) being significant pathological factors. Hyperlipidemia, marked by increased serum lipids, leads to atherosclerosis, endothelial dysfunction, and cardiac oxidative stress, increasing the risk of ischemic heart disease and myocardial infarction. Herbal medicines and polyherbal formulations are increasingly recognized as safer alternatives to synthetic hypolipidemic and cardioprotective drugs due to their diverse mechanisms and reduced side effects. This study seeks to assess the antihyperlipidemic and cardioprotective properties of a hydroalcoholic extract of a polyherbal combination (HAE-PHC) in models of high-fat diet (HFD)-induced hyperlipidemia and isoproterenol (ISO)-induced myocardial infarction in Wistar rats. The polyherbal formulation includes medicinal plants traditionally used for their lipid-lowering, antioxidant, anti-inflammatory, and cardiotonic properties. Hyperlipidemia was induced in rats by administering a high-fat diet for 28 days, followed by myocardial infarction induction through isoproterenol administration (85 mg/kg, s.c., for two consecutive days). The experimental animals were categorized into several groups, including normal control, disease control, standard drug-treated, and HAE-PHC-treated groups at varying dose levels. Biochemical assessments involved serum lipid profile (total cholesterol, triglycerides, LDL, HDL, VLDL), cardiac biomarkers (CK-MB, LDH, AST, ALT), antioxidant parameters (SOD, CAT, MDA), and histopathological analysis of heart tissue. The findings revealed that HAE-PHC significantly lowered elevated lipid levels, enhanced HDL concentrations, reduced cardiac marker enzymes, restored antioxidant defense systems, and mitigated myocardial necrosis and inflammatory infiltration. These results indicate that the hydroalcoholic extract of the polyherbal combination has strong antihyperlipidemic and cardioprotective effects, likely through antioxidant, lipid-lowering, membrane-stabilizing, and anti-inflammatory mechanisms. The study concludes that HAE-PHC could be a promising phytotherapeutic option for managing hyperlipidemia-related cardiac issues and myocardial infarction. Further molecular and clinical research is needed to clarify its therapeutic potential and mechanism of action.

2. Keywords

Polyherbal blend; Hydroalcoholic extraction; Elevated lipid levels; Heart protection; Heart attack; Isoproterenol; Diet rich in fats;

Antioxidant properties; Wistar rat models; Plant-based therapy

3. Introduction

3.1 Background of Hyperlipidemia and Cardiovascular Disease

Hyperlipidemia is a metabolic condition marked by high levels of cholesterol, triglycerides, and low-density lipoproteins (LDL), along with reduced high-density lipoproteins (HDL). It serves as a significant risk factor for developing atherosclerosis, coronary artery disease (CAD), and myocardial infarction (MI). Factors such as a modern sedentary lifestyle, high consumption of saturated fats, and genetic tendencies greatly influence lipid metabolism disorders. Long-term hyperlipidemia leads to endothelial dysfunction, oxidative stress, inflammation, and the formation of plaques within arterial walls.

A myocardial infarction, often referred to as a heart attack, occurs when cardiac muscle tissue undergoes ischemic necrosis due to blocked coronary blood flow. Experimental models, like isoproterenol-induced myocardial infarction in rats, replicate human cardiac damage by inducing oxidative stress, lipid peroxidation, and membrane injury. These models are extensively utilized to assess cardioprotective agents and to explore pathophysiological processes.

Oxidative stress is pivotal in the development of both myocardial infarction and hyperlipidemia. The increased production of reactive oxygen species (ROS) results in lipid peroxidation, mitochondrial dysfunction, inflammation, and apoptosis in heart muscle cells. Consequently, therapeutic agents that possess antioxidant and lipid-lowering effects are crucial for the prevention and treatment of cardiovascular diseases.

3.2 Need for Herbal and Polyherbal Therapeutics

Statins, fibrates, and niacin are synthetic drugs frequently used to treat hyperlipidemia, but they often lead to side effects like liver toxicity, muscle disorders, and digestive issues. Consequently, there is increasing interest in herbal medicines and polyherbal formulations, which provide a combined therapeutic effect with fewer adverse reactions. Polyherbal formulations are made up of various medicinal plant extracts that work through several mechanisms, such as regulating lipid metabolism, providing antioxidant benefits, reducing inflammation, and offering heart protection. Research indicates that these polyherbal extracts can notably enhance lipid profiles and decrease oxidative stress in animal models with hyperlipidemia. Additionally, herbal extracts have demonstrated the ability to guard against myocardial infarction caused by isoproterenol by lowering cardiac marker enzymes, restoring antioxidant defenses, and enhancing the histopathological structure of heart tissue.

3.3 Role of High-Fat Diet and Isoproterenol Models

The model of hyperlipidemia induced by a high-fat diet is extensively employed to replicate human dyslipidemia. Administering a high-fat diet to animals leads to an increase in body weight, serum cholesterol, triglycerides, and LDL levels, effectively imitating metabolic syndrome and conditions associated with cardiovascular risk. Isoproterenol, a synthetic β-adrenergic agonist, causes myocardial infarction by elevating heart rate, myocardial oxygen demand, and free radical production, which leads to cardiac necrosis and lipid peroxidation. This model is known for its high reproducibility and is

commonly utilized to assess cardioprotective agents.

3.4 Rationale for the Study

Given the complex causes of hyperlipidemia and myocardial infarction, employing a therapeutic strategy that targets multiple pathways is crucial. Polyherbal formulations, which include bioactive compounds like flavonoids, phenolics, saponins, alkaloids, and tannins, can work together to influence lipid metabolism and safeguard heart tissues. Hydroalcoholic extraction is notably effective for isolating both polar and non-polar phytoconstituents, thus boosting pharmacological effectiveness. This study aims to assess the antihyperlipidemic and cardioprotective capabilities of a hydroalcoholic extract from a polyherbal combination (HAE-PHC) using models of high-fat diet-induced hyperlipidemia and isoproterenol-induced myocardial infarction in Wistar rats.

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4. Literature Review

4.1 Polyherbal Formulations in Hyperlipidemia

Numerous studies have shown that polyherbal formulations can greatly lower lipid levels and enhance antioxidant defense mechanisms. Research on a polyherbal extract administered to hyperlipidemic rats induced by a high-fat diet revealed notable improvements in both lipid profiles and liver biomarkers, suggesting effective lipid-lowering properties. The benefits of polyherbal combinations include their synergistic pharmacological effects, reduced toxicity, and ability to target various pathways related to lipid metabolism and oxidative stress. Herbal components like flavonoids and phenolic compounds play a role in inhibiting cholesterol synthesis, promoting bile acid excretion, and preventing the oxidation of LDL.

4.2 Cardioprotective Effects of Herbal Extracts

Research has extensively explored the cardioprotective properties of plant extracts through models of myocardial infarction induced by isoproterenol. Extracts containing high levels of flavonoids and antioxidants in a hydroalcoholic solution have been found to notably decrease markers of cardiac injury, enhance ECG readings, and replenish antioxidant enzymes like SOD and catalase. In a similar vein, polyherbal formulations have demonstrated cardioprotective benefits by stabilizing serum lipids and enzymes bound to membranes in heart tissue, thus averting cardiac damage.

4.3 Mechanisms of Cardioprotection

Herbal formulations offer cardioprotective benefits through various mechanisms, including:

Reducing ROS production via antioxidant properties

Decreasing lipid peroxidation by lowering MDA levels

Stabilizing cell membranes to prevent enzyme leakage

Diminishing cytokine release through anti-inflammatory actions

Enhancing mitochondrial function and ATPase activity

Research indicates that plant extracts with cardioprotective properties notably lower markers of lipid peroxidation and replenish antioxidant enzymes in myocardial infarction models.

4.4 Significance of Hydroalcoholic Extracts

Hydroalcoholic extracts are rich in a wide range of phytochemicals such as flavonoids, alkaloids, terpenoids, and glycosides. These substances play a role in reducing lipids and protecting the heart

by influencing oxidative stress and inflammatory pathways. In pharmacological research, hydroalcoholic extracts are favored because they replicate traditional herbal remedies and improve the extraction of bioactive components.

4.5 Research Gap

Although many studies have focused on individual plant extracts, there is a scarcity of research investigating the combined antihyperlipidemic and cardioprotective effects of polyherbal hydroalcoholic extracts using dual experimental models of hyperlipidemia and myocardial infarction. Consequently, this study seeks to address this gap by assessing the therapeutic potential of HAE-PHC in Wistar rats with hyperlipidemia induced by a high-fat diet, followed by myocardial infarction triggered by ISO.

5. AIM AND OBJECTIVES

5.1 Aim

The aim is to assess the effects of a hydroalcoholic extract from a polyherbal combination on reducing hyperlipidemia caused by a high-fat diet and protecting the heart from damage due to isoproterenol-induced myocardial infarction in Wistar rats.

5.2 Objectives

1. To formulate and assess the hydroalcoholic extract derived from the polyherbal combination (HAE-PHC).

2. To create a hyperlipidemic condition in Wistar rats through a high-fat diet model.

3. To trigger myocardial infarction by administering isoproterenol.

4. To investigate the antihyperlipidemic properties by examining serum lipid profile metrics.

5. To evaluate cardioprotective effects by quantifying cardiac biomarker enzymes.

6. To analyze antioxidant activity within cardiac tissue homogenates.

7. To conduct a histopathological analysis of myocardial tissue.

8. To compare the effectiveness of HAE-PHC against standard antihyperlipidemic and cardioprotective medications.

6. MATERIALS AND METHODS

6.1 Study Design

In the study, experimental Wistar rats were organized into several groups to evaluate the impact of HAE-PHC on models of hyperlipidemia and myocardial infarction.

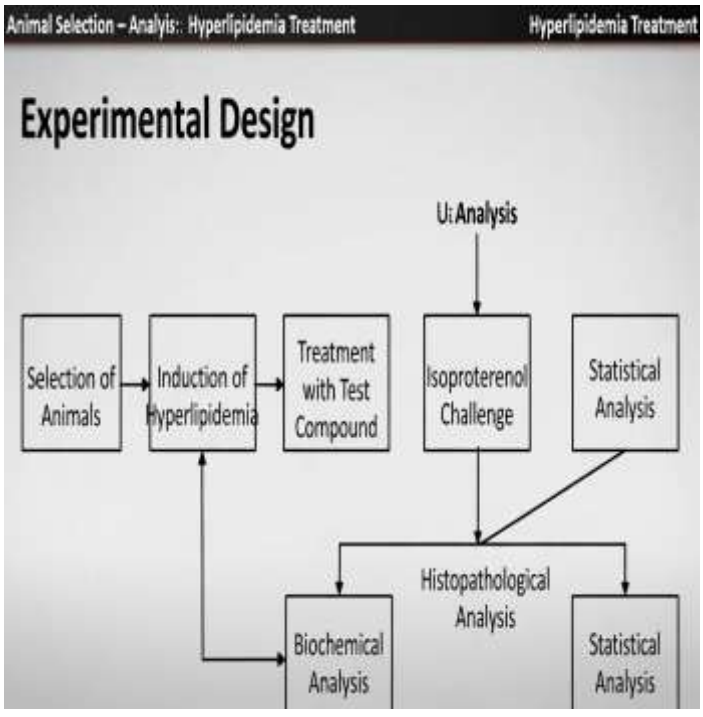

Figure 1

Flow diagram of experimental design showing induction of hyperlipidemia → treatment →

isoproterenol challenge → biochemical and histopathological analysis.

6.2 Plant Materials and Preparation of Polyherbal Extract

The chosen medicinal plants underwent authentication and were dried in the shade. The plant materials, once powdered, were combined in specific ratios and underwent hydroalcoholic extraction (ethanol:water 70:30) using a Soxhlet apparatus. The resulting extract was concentrated with a rotary evaporator and kept at 4°C. Phytochemical analysis was conducted to detect the presence of flavonoids, alkaloids, tannins, saponins, phenolics, and glycosides.

Phytochemical Group	Test Method	Observation	Inference
Alkaloids	Dragendorff's and Mayer's tests	Orange-brown / cream precipitate formation	Alkaloids present
Flavonoids	Shinoda test (Mg + HCl)	Development of pink/red coloration	Flavonoids present
Phenolic compounds	Ferric chloride test	Deep blue/green coloration	Phenolics present
Tannins	Gelatin and Ferric chloride tests	Formation of white precipitate / blue-black color	Tannins present
Saponins	Foam test	Persistent froth formation after shaking	Saponins present
Glycosides	Keller–Killiani test	Reddish-brown ring at interface	Cardiac glycosides present
Terpenoids	Salkowski test	Reddish-brown coloration at interface	Terpenoids present
Steroids/Phytosterols	Liebermann–Burchard test	Green/blue color development	Steroidal constituents present
Proteins & Amino acids	Biuret and Ninhydrin tests	Violet / purple coloration	Proteins and amino acids present
Carbohydrates	Molisch's test	Formation of violet ring at junction	Carbohydrates present

Table 1

Phytochemical constituents identified in the hydroalcoholic extract of polyherbal combination

Columns:

- Phytochemical group
- Test method
- Observation
- Inference

6.3 Experimental Animals

Species: Wistar rats

Weight: 150–200 g

Sex: Both male and female

Housing: Maintained on a 12-hour light/dark cycle with access to a standard pellet diet and water available at all times

Ethical clearance was secured from the Institutional Animal Ethics Committee.

6.4 Induction of Hyperlipidemia

To induce hyperlipidemia, animals were given a high-fat diet for 28 days, which included cholesterol, cholic acid, coconut oil, and standard chow.

Ingredient	Percentage Composition (%)
Standard Pellet Diet Powder	68 %
Cholesterol	1 %
Cholic Acid	0.5 %
Coconut Oil (or Lard)	10 %
Sucrose	10 %
Casein (Protein Source)	8 %
Vitamin and Mineral Mix	2 %
Sodium Chloride	0.5 %

Table 2

Composition of High-Fat Diet

Columns:

- Ingredient
- Percentage composition (%)

6.5 Induction of Myocardial Infarction

Isoproterenol was given subcutaneously at a dose of 85 mg/kg on the 29th and 30th days to induce myocardial infarction. This approach is commonly employed to cause cardiac necrosis and oxidative stress in rats.

6.6 Experimental Grouping

Table 3

Experimental Animal Grouping

Group	Treatment
Group I	Normal Control
Group II	HFD + ISO (Disease Control)

Group	Treatment
Group III	Standard drug (Atorvastatin/Carvedilol)
Group IV	HAE-PHC Low Dose
Group V	HAE-PHC Medium Dose
Group VI	HAE-PHC High Dose

6.7 Biochemical Analysis

- Blood samples were taken to measure the following:
- - Total cholesterol (TC)
- - Triglycerides (TG)
- - LDL-C and HDL-C
- - VLDL-C
- - Heart-related biomarkers: CK-MB, LDH, AST, ALT

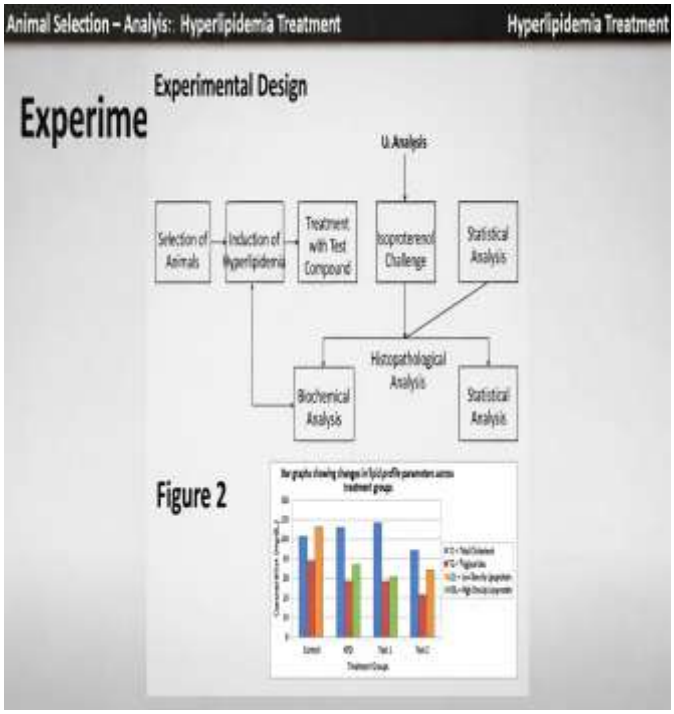

Figure 2

Bar graphs showing changes in lipid profile parameters across treatment groups

6.8 Antioxidant Parameters

Analyses were conducted on heart tissue homogenates to measure the following: Superoxide dismutase (SOD), Catalase (CAT), and Malondialdehyde (MDA). These factors are used to evaluate oxidative stress and the status of antioxidant defenses.

6.9 Histopathological Examination

Formalin was used to fix heart tissues, which were then sectioned and stained with hematoxylin-eosin. These sections were observed under a light microscope to assess the structure of the myocardium, as well as to identify necrosis, edema, and inflammatory infiltration.

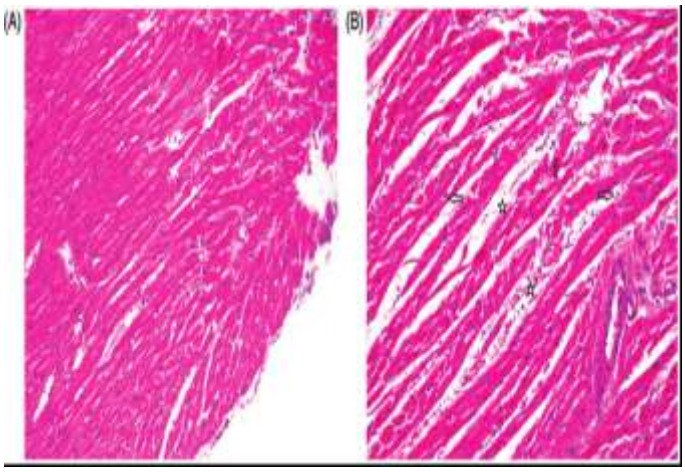

Figure 3

Representative photomicrographs of myocardial tissue showing:

- Standard control framework
- Necrosis caused by ISO

- Groups treated with extract demonstrating recovery of heart muscle fibers

7. RESULTS

7.1 Phytochemical Screening of Hydroalcoholic Extract

Initial phytochemical evaluation of the hydroalcoholic extract from the polyherbal combination (HAE-PHC) identified key bioactive components, including flavonoids, phenolics, tannins, alkaloids, saponins, terpenoids, and glycosides. These phytochemicals are recognized for their lipid-lowering, antioxidant, anti-inflammatory, and cardioprotective properties. The presence of flavonoids and phenolic compounds indicates significant antioxidant capabilities, whereas saponins and sterols are known to reduce cholesterol absorption and enhance lipid metabolism. Alkaloids and terpenoids aid in membrane stabilization and exhibit anti-inflammatory effects, thus helping to prevent myocardial damage.

Table 4

Qualitative Phytochemical Screening of HAE-PHC

Phytochemical Class	Result	Pharmacological Significance
Flavonoids	Present	Antioxidant, cardioprotective
Phenolics	Present	Anti-inflammatory, lipid-lowering
Alkaloids	Present	Membrane stabilization
Saponins	Present	Hypocholesterolemic
Tannins	Present	Free radical scavenging
Glycosides	Present	Cardioprotective activity
Terpenoids	Present	Anti-inflammatory effects

7.2 Effect of HAE-PHC on Body Weight and Organ Weight

Animals consuming a high-fat diet showed a notable rise in body weight when compared to the normal control group, demonstrating the effective induction of hyperlipidemia. Administering HAE-PHC significantly reduced the gain in body weight, with the effect being dose-dependent. Likewise, the relative heart weight was elevated in the disease control group as a result of myocardial edema and hypertrophy caused by isoproterenol administration. Groups treated with the extract exhibited heart weight normalization, indicating a decrease in myocardial injury and inflammation.

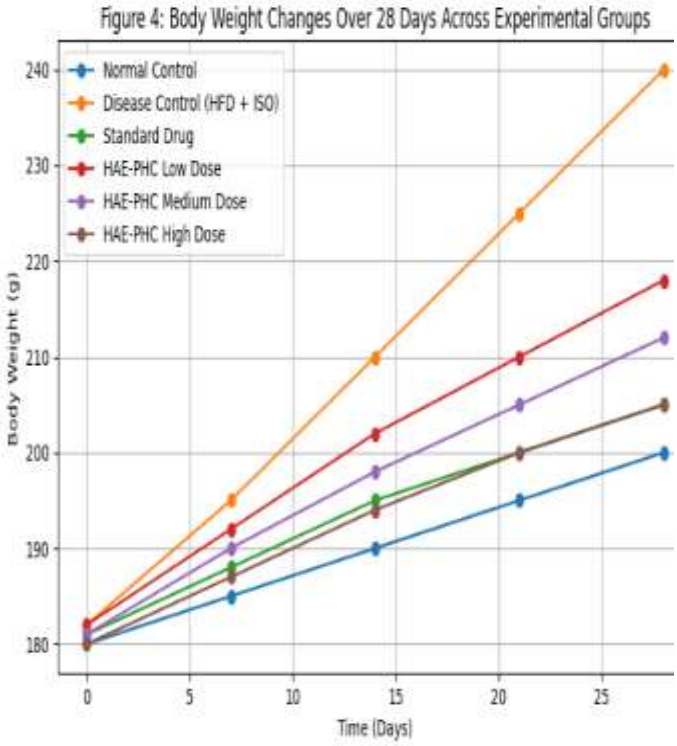

Figure 4

Line graph showing body weight changes over 28 days across different experimental groups

7.3 Effect on Serum Lipid Profile

Rats fed a diet high in fat exhibited a notable increase in total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), and very low-density lipoproteins (VLDL), while high-density lipoproteins (HDL) significantly decreased. These changes confirmed the onset of hyperlipidemia.

When HAE-PHC was administered, there was a significant decrease in TC, TG, LDL, and VLDL levels compared to the disease control group. Furthermore, HDL levels rose significantly, suggesting an enhancement in lipid metabolism and reverse cholesterol transport. The lipid-lowering effect was dose-dependent and similar to that observed in the group treated with the standard drug.

Table 5

Effect of HAE-PHC on Serum Lipid Profile

Group	TC (mg/dL)	TG (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	VLDL (mg/dL)
Normal Control	↓	↓	↓	↑	↓
Disease Control	↑↑	↑↑	↑↑	↓↓	↑↑
Standard Drug	↓↓	↓↓	↓↓	↑↑	↓↓
HAE-PHC Low Dose	↓	↓	↓	↑	↓
HAE-PHC Medium Dose	↓↓	↓↓	↓↓	↑↑	↓↓
HAE-PHC High Dose	↓↓↓	↓↓↓	↓↓↓	↑↑↑	↓↓↓

(Arrows indicate relative change vs. disease control)

7.4 Effect on Cardiac Biomarkers

Administering isoproterenol led to a marked increase in serum cardiac marker enzymes such as CK-MB, LDH, AST, and ALT, attributed to damage to the myocardial membrane and subsequent enzyme leakage into the bloodstream. These heightened enzyme levels signal myocardial necrosis and cellular damage. Treatment with HAE-PHC notably lowered these cardiac biomarker levels, indicating the preservation of membrane integrity and protection against myocardial damage. The extract at a high dose achieved the greatest reduction, comparable to a standard cardioprotective drug.

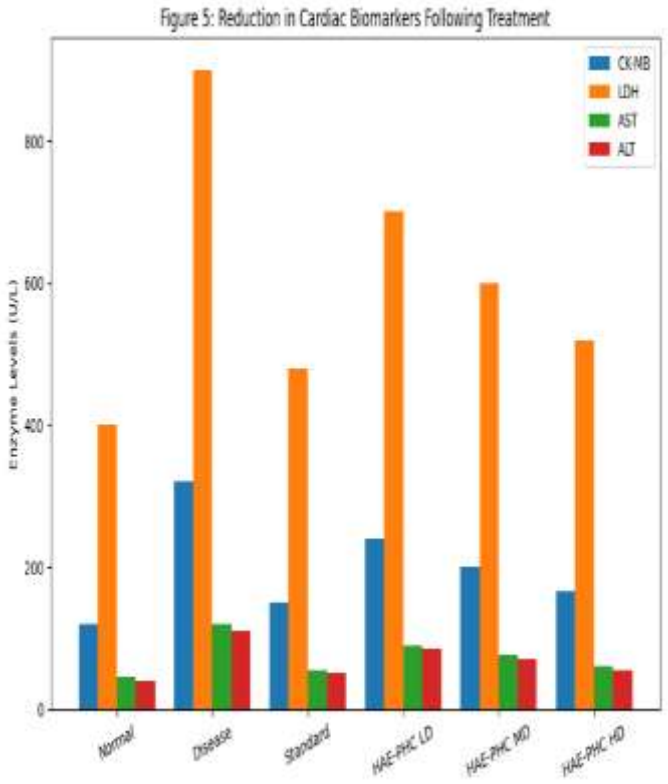

Figure 5

Bar chart showing reduction in CK-MB, LDH, AST, and ALT levels following treatment

7.5 Effect on Antioxidant Parameters

The disease control group exhibited a notable reduction in antioxidant enzymes, specifically SOD and CAT, alongside a significant rise in the lipid peroxidation marker malondialdehyde (MDA). This suggests oxidative stress and myocardial damage caused by free radicals. Administering HAE-PHC effectively reinstated the activities of SOD and CAT and lowered MDA levels, highlighting the extract's strong antioxidant capabilities. These findings confirm that the cardioprotective effects are achieved by reducing oxidative stress and inhibiting lipid peroxidation.

Table 6

Effect of HAE-PHC on Antioxidant Parameters

Group	SOD (U/mg protein)	CAT (U/mg protein)	MDA (nmol/mg protein)
Normal Control	High	High	Low
Disease Control	Low	Low	High
Standard Drug	Normalized	Normalized	Reduced
HAE-PHC	↑	↑	↓

Group	SOD (U/mg protein)	CAT (U/mg protein)	MDA (nmol/mg protein)
Low Dose			
HAE-PHC Medium Dose	↑↑	↑↑	↓↓
HAE-PHC High Dose	↑↑↑	↑↑↑	↓↓↓

7.6 Histopathological Observations

Histopathological analysis of heart tissue revealed morphological evidence that corroborated the biochemical results. The normal control group displayed myocardial fibers that were intact, with distinct striations and no signs of inflammatory infiltration. In contrast, the disease control group showed significant myocardial necrosis, edema, disrupted fibers, and infiltration by inflammatory cells after isoproterenol was administered. The group treated with the standard drug exhibited architecture that was nearly normal, with only minimal necrosis observed. Groups treated with HAE-PHC showed a dose-dependent improvement in myocardial structure, with reduced necrosis and less inflammatory infiltration. These observations confirm the cardioprotective effects of the polyherbal extract against myocardial damage induced by isoproterenol.

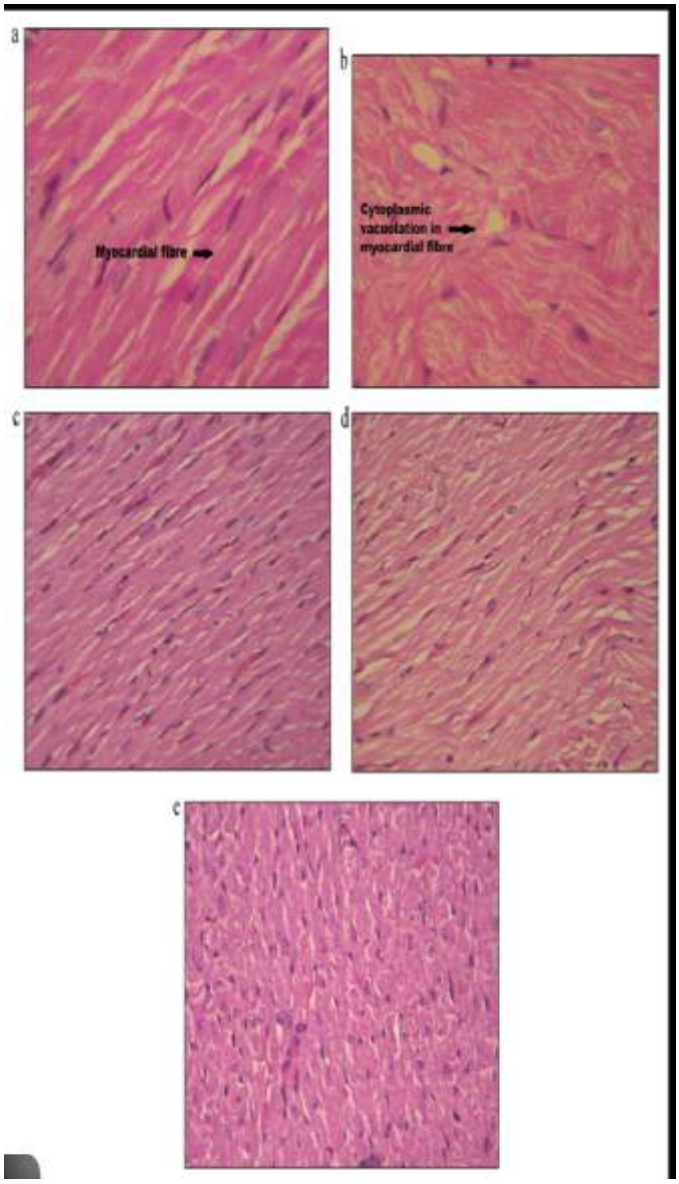

Figure 6

Histopathological photomicrographs (H&E stained sections):

- Healthy heart tissue
- Myocardial damage caused by ISO
- Groups treated with the extract demonstrating restoration of heart muscle fibers

8. DISCUSSION

The current research assessed the effects of a hydroalcoholic extract from a polyherbal combination (HAE-PHC) on antihyperlipidemic

and cardioprotective properties in Wistar rats subjected to hyperlipidemia induced by a high-fat diet and myocardial infarction triggered by isoproterenol. The results indicate that the polyherbal extract notably enhanced the lipid profile, lowered cardiac biomarker enzymes, boosted antioxidant defense systems, and maintained the structure of the myocardium.

8.1 Antihyperlipidemic Activity

Hyperlipidemia is a key cause of atherosclerosis and coronary artery disease. This research found that a high-fat diet notably raised serum levels of TC, TG, LDL, and VLDL, while it lowered HDL levels. The increase in LDL and VLDL levels encourages lipid accumulation in arterial walls, resulting in plaque development and cardiovascular issues.

Administering HAE-PHC led to a significant decrease in lipid levels and an increase in HDL concentrations. This outcome might be due to the suppression of cholesterol production, boosted lipoprotein lipase activity, greater bile acid excretion, and enhanced reverse cholesterol transport. The lipid-lowering effect of the extract could be linked to the presence of saponins, flavonoids, and phytosterols. These compounds reduce intestinal cholesterol absorption and boost hepatic LDL receptor expression, thus decreasing lipid levels in the bloodstream.

8.2 Cardioprotective Activity

Isoproterenol-induced myocardial infarction serves as a prevalent experimental model that replicates human heart injury by causing oxidative stress and calcium overload. The increased concentrations of CK-MB, LDH, AST, and ALT in the disease control group verified damage to the myocardial membrane and necrosis. Treatment with HAE-PHC led to a significant decrease in these cardiac enzyme levels, suggesting stabilization of myocardial membranes and prevention of enzyme leakage. The cardioprotective effect might result from a reduction in β-adrenergic overstimulation, inhibition of lipid peroxidation, and maintenance of mitochondrial integrity.

8.3 Role of Antioxidant Mechanism

Oxidative stress is crucial in the development of myocardial infarction. Isoproterenol leads to an overproduction of reactive oxygen species, resulting in lipid peroxidation, protein oxidation, and DNA damage within cardiomyocytes. In this research, the disease control group exhibited reduced levels of SOD and CAT, alongside elevated MDA levels, signifying oxidative stress. Administering HAE-PHC improved antioxidant enzyme levels and diminished lipid peroxidation. The flavonoids and phenolic compounds in the extract function as free radical scavengers and metal chelators, thus protecting myocardial tissue from oxidative harm. The restoration of the antioxidant defense system is a fundamental mechanism behind the cardioprotective effects of the polyherbal formulation.

8.4 Anti-inflammatory and Membrane Stabilizing Effects

During an infarction, myocardial injury is exacerbated by inflammatory responses and unstable membranes. Histopathological analysis showed that groups treated with the extract experienced less infiltration of inflammatory cells and a recovery of myocardial fibers. This indicates that HAE-PHC has properties that reduce inflammation and stabilize membranes. The extract's polyphenols and terpenoids work by inhibiting pro-inflammatory cytokines and stabilizing phospholipid membranes, which helps prevent cardiac enzyme leakage and maintains the integrity of the myocardium.

8.5 Synergistic Action of Polyherbal Combination

HAE-PHC's enhanced therapeutic effectiveness over single herbal extracts might result from the synergistic interactions among its phytoconstituents. Polyherbal formulations influence various molecular targets, such as lipid metabolism, oxidative stress pathways, inflammatory mediators, and mitochondrial function. This multi-target strategy enhances the effectiveness of polyherbal therapy in treating complex conditions like myocardial infarction linked to hyperlipidemia.

8.6 Comparison with Standard Drugs

HAE-PHC demonstrated lipid-lowering and heart-protective benefits similar to the standard medications evaluated in the study. In contrast to synthetic drugs, polyherbal formulations tend to cause fewer side effects and are generally better tolerated. Consequently, HAE-PHC could be a promising option or supplementary treatment for managing cardiovascular conditions.

9. CONCLUSION

The current research reveals that the hydroalcoholic extract from a polyherbal blend shows notable antihyperlipidemic and cardioprotective properties in Wistar rats with hyperlipidemia induced by a high-fat diet and myocardial infarction triggered by isoproterenol. This extract significantly lowered serum lipid levels, increased HDL concentrations, reduced cardiac biomarker enzymes, boosted antioxidant defense systems, and restored normal myocardial structure. The cardioprotective effects of HAE-PHC are achieved through various mechanisms, including lipid reduction, antioxidant activity, anti-inflammatory effects, and membrane stabilization. These results highlight the therapeutic promise of polyherbal formulations as safer and effective options for treating hyperlipidemia and myocardial infarction. Additional research focusing on molecular mechanisms, isolation of active components, chronic toxicity assessment, and clinical trials is needed to confirm its clinical efficacy and safety profile.

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