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Research Article

Pharmacological Evaluation of Cardioprotective Activity of *Phyllanthus niruri* Root Extract in Isoproterenol-Induced Myocardial Infarction in Experimental Rats

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ABSTRACT

Myocardial infarction is associated with oxidative stress, myocardial cellular injury, and impairment of endogenous antioxidant defense. The present study was undertaken to evaluate the cardioprotective activity of hydroalcoholic root extract of *Phyllanthus niruri* against isoproterenol-induced myocardial infarction in Wistar rats. Animals were divided into normal control, isoproterenol control, propranolol-treated, and *P. niruri* extract-treated groups (200 and 400 mg/kg). Myocardial injury was assessed using serum cardiac biomarkers and tissue oxidative stress parameters. Isoproterenol administration markedly increased CK-MB from 116.24 ± 4.08 to 304.52 ± 8.96 U/L, LDH from 228.36 ± 6.14 to 536.48 ± 13.25 U/L, and AST from 82.46 ± 3.05 to 201.35 ± 5.84 U/L. Treatment with *P. niruri* at 400 mg/kg reduced CK-MB, LDH, and AST to 158.42 ± 5.12 , 284.53 ± 9.34 , and 106.28 ± 3.92 U/L, respectively. Isoproterenol also increased MDA from 1.82 ± 0.07 to 6.14 ± 0.18 nmol/mg protein, while decreasing SOD, CAT, and GSH levels. The 400 mg/kg extract restored these parameters to 10.84 ± 0.35 U/mg protein, 50.18 ± 1.62 U/mg protein, and 7.36 ± 0.28 μ g/mg tissue, respectively, and reduced MDA to 2.56 ± 0.10 nmol/mg protein. Histopathological examination showed improved myocardial architecture with reduced necrosis, inflammation, and edema. These findings suggest that *P. niruri* root extract exhibits cardioprotective activity, possibly through reduction of oxidative stress and enhancement of endogenous antioxidant defense.

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INTRODUCTION

1.1 Cardiovascular Diseases

Cardiovascular diseases (CVDs) are among the major causes of morbidity and mortality worldwide. The term cardiovascular diseases includes a group of disorders affecting the heart and blood vessels, with coronary artery disease, myocardial infarction, hypertension,

and heart failure being some of the commonly encountered conditions.^[1] The development of cardiovascular disorders is associated with several risk factors, including hypertension, dyslipidemia, diabetes mellitus, obesity, physical inactivity, unhealthy dietary habits, smoking, and increasing age. These factors may act individually or together to produce structural and functional changes in the cardiovascular system.^[2]

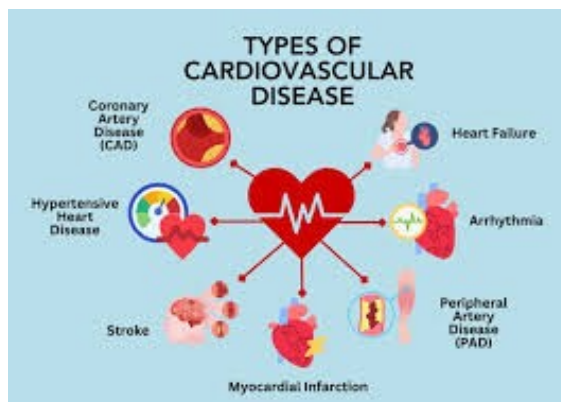


Figure 1: Cardiovascular Diseases (CVDs)

Among the different cardiovascular conditions, coronary artery disease is particularly important because it can reduce the blood supply to the myocardium. Prolonged impairment of coronary blood flow may result in myocardial ischemia and subsequent myocardial infarction.^[3] Damage to cardiac tissue can affect myocardial contractility and may ultimately lead to complications such as arrhythmia, ventricular dysfunction, heart failure, and, in severe cases, death. Therefore, identification of effective approaches for prevention and management of myocardial injury remains an important area of cardiovascular research.^[4]

1.2 Myocardial Infarction

Myocardial infarction (MI) is an acute condition characterized by myocardial injury resulting from an inadequate supply of oxygen to cardiac tissue. The imbalance between myocardial oxygen demand and oxygen supply can cause cellular dysfunction and, when sufficiently prolonged, irreversible damage to cardiomyocytes. The severity of myocardial injury depends on factors such as the duration and extent of ischemia and the ability of the myocardium to maintain adequate oxygenation.^[5]

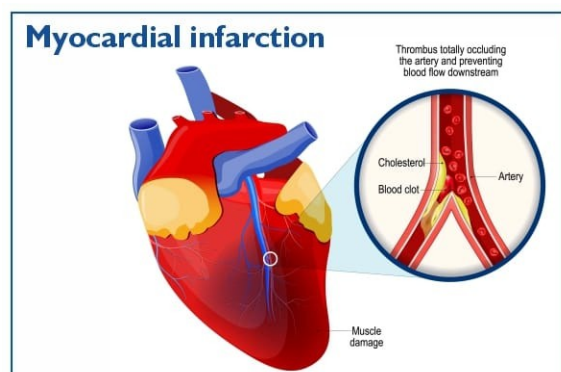


Figure 2: Myocardial Infarction (MI)

Oxidative stress plays an important role in the development of myocardial injury. Excessive generation of

reactive oxygen species (ROS) can cause oxidation of cellular lipids, proteins, and other important

biomolecules. Increased oxidative damage can disturb the normal structure and function of cardiomyocytes and may promote inflammatory responses and cellular death. Thus, oxidative stress is considered one of the important mechanisms involved in myocardial damage.^[6]

Biochemical markers are commonly used to assess myocardial injury experimentally. Enzymes such as creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) may show altered activity following cardiac tissue injury.^[7] An increase in these markers reflects leakage of intracellular components into the circulation as a consequence of myocardial cell damage. Their estimation therefore provides useful biochemical evidence for evaluating the extent of cardiac injury and the protective effect of test treatments^[8].

1.3 Experimental Model of Myocardial Infarction

Experimental animal models are widely used to investigate the mechanisms involved in myocardial injury and to evaluate potential cardioprotective agents. Such models provide an opportunity to study biochemical, physiological, and histopathological changes associated with cardiac damage under controlled experimental conditions.^[9]

Isoproterenol (ISO), a synthetic β -adrenergic agonist, is commonly used to produce experimental myocardial injury in laboratory animals. Administration of a suitable dose of isoproterenol produces characteristic biochemical and structural changes resembling several features of myocardial infarction. The model is therefore useful for screening compounds and plant extracts with potential cardioprotective activity.^[10]

The cardiotoxic effect of isoproterenol is associated with excessive β -adrenergic stimulation of the myocardium. Increased cardiac workload and oxygen demand, together with enhanced generation of reactive oxygen species, contribute to oxidative damage of myocardial tissue. Isoproterenol-induced oxidative stress can promote lipid peroxidation, damage cellular membranes, alter antioxidant defense mechanisms, and result in leakage of cardiac enzymes. These changes

can be evaluated through biochemical parameters and histopathological examination of cardiac tissue.^[11]

1.4 Oxidative Stress in Myocardial Injury

Oxidative stress occurs when the production of reactive oxygen species exceeds the capacity of the endogenous antioxidant defense system to neutralize them. The myocardium is particularly susceptible to oxidative damage because of its high metabolic activity and continuous requirement for oxygen. Excessive ROS generation can adversely affect membrane lipids, proteins, enzymes, and cellular structures.^[12]

One of the important consequences of increased ROS generation is lipid peroxidation. Oxidation of membrane lipids can alter membrane integrity and permeability, resulting in cellular dysfunction and leakage of intracellular components. Malondialdehyde (MDA) is commonly used as an indicator of lipid peroxidation and provides an indirect measure of oxidative damage to cellular membranes.^[13]

The body possesses several enzymatic and non-enzymatic antioxidant mechanisms to maintain redox balance. Superoxide dismutase (SOD) converts superoxide radicals into hydrogen peroxide, while catalase (CAT) helps in the decomposition of hydrogen peroxide into water and oxygen.^[14] Reduced glutathione (GSH) is an important endogenous antioxidant that participates in the neutralization of reactive species and maintenance of cellular redox status. During myocardial injury, excessive oxidative stress may reduce the activity or availability of these antioxidant defenses. Therefore, evaluation of SOD, CAT, GSH, and MDA can provide useful information regarding oxidative changes associated with myocardial damage and the possible antioxidant effect of a test extract.^[15]

1.5 Herbal Cardioprotection

Medicinal plants have traditionally been used for the management of several cardiovascular disorders and continue to attract interest as potential sources of pharmacologically active compounds. Plant-derived preparations contain diverse groups of phytoconstituents, including phenolic compounds, flavonoids, tannins, alkaloids, terpenoids, and other secondary metabolites. Several of these compounds have been



reported to possess antioxidant, anti-inflammatory, and membrane-protective properties.^[16]

Oxidative stress and inflammation are closely associated with the progression of myocardial injury. Consequently, plant extracts capable of reducing oxidative damage and supporting endogenous antioxidant defenses may have potential cardioprotective effects. Phenolic and flavonoid constituents are of particular interest because of their ability to interact with reactive oxygen species and contribute to the overall antioxidant activity of plant extracts.

The cardioprotective potential of medicinal plants can be evaluated using experimental models of myocardial injury. Changes in cardiac biomarkers, antioxidant enzymes, lipid peroxidation products, and myocardial histology provide useful evidence for determining whether a plant extract can attenuate cardiac damage.^[17]

1.6 *Phyllanthus niruri*

Phyllanthus niruri is a medicinal plant belonging to the family Phyllanthaceae and has been traditionally used in various systems of herbal medicine. Different parts of the plant have been investigated for a range of pharmacological activities, including antioxidant, hepatoprotective, anti-inflammatory, and other biological effects. The plant contains several classes of secondary metabolites that may contribute to its pharmacological properties.^[18]

Table 1: Botanical Profile of *Phyllanthus niruri*

Parameter	Description
Botanical Name	<i>Phyllanthus niruri</i> Linn.
Family	Phyllanthaceae
Common Name	Bhui Amla
Habit	Annual herb
Distribution	Tropical regions
Part Used	Root/whole plant
Major Constituents	Flavonoids, lignans, phenolics
Major Uses	Liver, kidney and inflammatory disorders

The antioxidant potential reported for *P. niruri* provides a rationale for investigating its possible protective effect against conditions in which oxidative stress plays an important role. Phytochemical constituents

such as phenolic compounds, flavonoids, lignans, tannins, and other bioactive constituents have been associated with various biological activities of the plant. However, the pharmacological activity of a plant extract may vary according to the plant part, extraction solvent, geographical source, and method of preparation.^[19]

In the present study, the root of *P. niruri* was selected for preparation of a hydroalcoholic extract. The use of a hydroalcoholic solvent system allows extraction of a broad range of polar and moderately polar phytoconstituents. Considering the reported antioxidant properties of *P. niruri* and the important role of oxidative stress in myocardial injury, investigation of its root extract in an experimental model of myocardial infarction is scientifically relevant.^[20]

Phyllanthus Niruri

Colour: Green to yellowish green in colour
Taste: Bitter
Odour: None
Stem: 1-1.5cm length
Leaves: Short stalked oblong in shape



Figure 3: *Phyllanthus niruri*

1.7 Research Gap

Although *Phyllanthus niruri* has been investigated for several pharmacological activities, comparatively limited experimental information is available regarding the cardioprotective potential of its root extract in myocardial infarction. In particular, evidence concerning the effect of a hydroalcoholic root extract against isoproterenol-induced myocardial injury remains limited. Further experimental investigation is therefore required to determine whether the root extract can attenuate biochemical and oxidative changes associated with myocardial damage.

The present study was undertaken to evaluate the cardioprotective potential of the hydroalcoholic root extract of *Phyllanthus niruri* against isoproterenol-induced myocardial infarction in Wistar rats. The study focuses on biochemical markers of myocardial injury,

antioxidant defense parameters, lipid peroxidation, and histopathological changes to obtain a comprehensive assessment of the protective effect of the extract.

MATERIALS AND METHODS

2.1 Plant Material

Roots of *Phyllanthus niruri* were collected from [indore], authenticated by [botanist], washed, shade-dried, powdered, and stored in an airtight container until extraction.

2.2 Instruments and Apparatus

Soxhlet apparatus, rotary evaporator, electronic balance, centrifuge, UV-Visible spectrophotometer,

homogenizer, pH meter, microscope, microtome, water bath, refrigerator/freezer, micropipettes, and standard laboratory glassware were used.

RESULTS AND DISCUSSION

3.1 Effect of *Phyllanthus niruri* Root Extract on Cardiac Biomarkers

The cardioprotective effect of the hydroalcoholic root extract of *Phyllanthus niruri* was evaluated by estimating serum CK-MB, LDH, and AST levels. The administration of isoproterenol produced a marked elevation in all three cardiac biomarkers compared with the normal control group, indicating the development of myocardial injury.

Table 2: Effect of *Phyllanthus niruri* Root Extract on Cardiac Biomarkers

Group	CK-MB (U/L)	LDH (U/L)	AST (U/L)
Normal Control	116.24 ± 4.08	228.36 ± 6.14	82.46 ± 3.05
Disease Control (ISO)	304.52 ± 8.96	536.48 ± 13.25	201.35 ± 5.84
Propranolol + ISO	146.38 ± 5.24	268.42 ± 8.72	98.15 ± 4.22
PN 200 mg/kg + ISO	204.18 ± 6.34	356.27 ± 10.84	132.46 ± 4.98
PN 400 mg/kg + ISO	158.42 ± 5.12	284.53 ± 9.34	106.28 ± 3.92

RESULTS

The CK-MB level increased from **116.24 ± 4.08 U/L** in the normal control group to **304.52 ± 8.96 U/L** following isoproterenol administration. Treatment with *P. niruri* at 200 mg/kg reduced CK-MB to **204.18 ± 6.34 U/L**, whereas the 400 mg/kg dose reduced it to **158.42 ± 5.12 U/L**.

Similarly, LDH increased from **228.36 ± 6.14 U/L** in the normal control group to **536.48 ± 13.25 U/L** in the disease control group. Treatment with 200 and 400

mg/kg of the extract reduced LDH levels to **356.27 ± 10.84** and **284.53 ± 9.34 U/L**, respectively.

AST also increased considerably after isoproterenol administration, from **82.46 ± 3.05 U/L** to **201.35 ± 5.84 U/L**. Treatment with the extract reduced AST to **132.46 ± 4.98 U/L** at 200 mg/kg and **106.28 ± 3.92 U/L** at 400 mg/kg.\

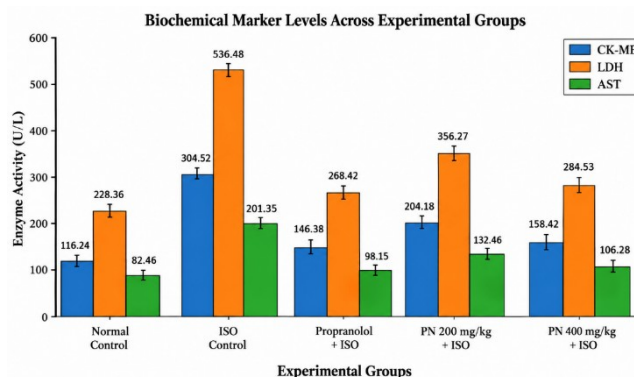


Figure 4: Effect of *Phyllanthus Niruri* Root Extract on Cardiac Biomarkers



DISCUSSION

The marked elevation of CK-MB, LDH, and AST in the disease control group indicates myocardial cellular injury and leakage of intracellular enzymes into the circulation. Pretreatment with *P. niruri* reduced these enzyme levels in a dose-dependent manner. Compared with the disease control group, the 400 mg/kg dose produced approximately **47.98% reduction in CK-MB**, **46.96% reduction in LDH**, and **47.22%**

reduction in AST. The higher dose therefore produced a greater reduction in cardiac enzyme leakage and showed values approaching those observed with propranolol.

3.2 Effect on Oxidative Stress and Antioxidant Parameters

Oxidative stress was evaluated by estimating MDA as a marker of lipid peroxidation and SOD, CAT, and GSH as indicators of endogenous antioxidant defense.

Table 3: Effect of *Phyllanthus niruri* Root Extract on Oxidative Stress Markers

Group	MDA (nmol/mg protein)	SOD (U/mg protein)	CAT (U/mg protein)	GSH (μ g/mg tissue)
Normal Control	1.82 $\pm$ 0.07	12.64 $\pm$ 0.44	59.36 $\pm$ 1.92	8.58 $\pm$ 0.34
Disease Control (ISO)	6.14 $\pm$ 0.18	4.12 $\pm$ 0.18	22.48 $\pm$ 0.88	2.96 $\pm$ 0.12
Propranolol + ISO	2.18 $\pm$ 0.09	11.42 $\pm$ 0.36	53.26 $\pm$ 1.74	7.86 $\pm$ 0.26
PN 200 mg/kg + ISO	3.86 $\pm$ 0.13	8.24 $\pm$ 0.28	40.36 $\pm$ 1.32	5.94 $\pm$ 0.24
PN 400 mg/kg + ISO	2.56 $\pm$ 0.10	10.84 $\pm$ 0.35	50.18 $\pm$ 1.62	7.36 $\pm$ 0.28

RESULTS

Isoproterenol administration markedly increased MDA from **1.82 $\pm$ 0.07** to **6.14 $\pm$ 0.18 nmol/mg protein**, indicating increased lipid peroxidation. Treatment with *P. niruri* reduced MDA to **3.86 $\pm$ 0.13** and **2.56 $\pm$ 0.10 nmol/mg protein** at 200 and 400 mg/kg, respectively.

SOD activity decreased from **12.64 $\pm$ 0.44 U/mg protein** in normal animals to **4.12 $\pm$ 0.18 U/mg protein** after isoproterenol administration. Treatment with the extract increased SOD activity to **8.24 $\pm$ 0.28 U/mg protein** at 200 mg/kg and **10.84 $\pm$ 0.35 U/mg protein** at 400 mg/kg.

CAT activity decreased from **59.36 $\pm$ 1.92** to **22.48 $\pm$ 0.88 U/mg protein** in the disease control group. The extract increased CAT activity to **40.36 $\pm$ 1.32** and **50.18 $\pm$ 1.62 U/mg protein** at 200 and 400 mg/kg, respectively.

Similarly, GSH decreased from **8.58 $\pm$ 0.34** to **2.96 $\pm$ 0.12 μ g/mg tissue** following isoproterenol administration. Treatment with the extract increased GSH levels to **5.94 $\pm$ 0.24** and **7.36 $\pm$ 0.28 μ g/mg tissue** at 200 and 400 mg/kg, respectively.

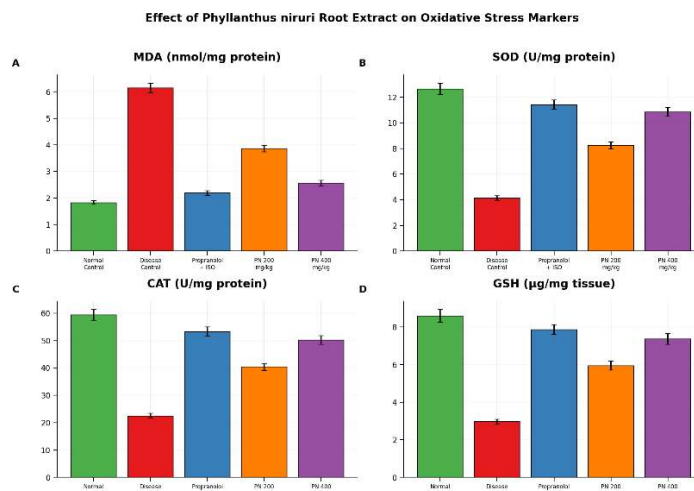


Figure 5: Effect on Oxidative Stress and Antioxidant Parameters



DISCUSSION

The increased MDA level in the isoproterenol-treated group indicates enhanced lipid peroxidation and oxidative damage to myocardial membranes. Treatment with *P. niruri* markedly reduced MDA, with the 400 mg/kg dose producing approximately **58.31% reduction** compared with the disease control group.

The reduction in SOD, CAT, and GSH following isoproterenol administration indicates depletion of endogenous antioxidant defense. Treatment with *P. niruri* restored these parameters in a dose-dependent manner. At 400 mg/kg, SOD, CAT, and GSH reached

10.84 ± 0.35 U/mg protein, 50.18 ± 1.62 U/mg protein, and 7.36 ± 0.28 μ g/mg tissue, respectively. These findings indicate that the extract was able to attenuate oxidative stress and improve the antioxidant status of myocardial tissue. The effect may be related to antioxidant phytoconstituents such as flavonoids, tannins, and phenolic compounds reported in the extract.

3.3 Histopathological Evaluation of Cardiac Tissue

Histopathological examination was performed to determine structural alterations in the myocardium following isoproterenol administration and treatment with *P. niruri* root extract.

Table 4: Histopathological Findings

Group	Myocardial Structure	Necrosis	Inflammation	Edema
Normal Control	Normal architecture	Absent	Absent	Absent
Disease Control (ISO)	Severe myocardial damage	Severe	Severe infiltration	Severe
Propranolol + ISO	Near-normal structure	Mild	Mild	Mild
PN 200 mg/kg + ISO	Moderate protection	Moderate	Moderate	Mild
PN 400 mg/kg + ISO	Well-preserved structure	Mild/minimal	Mild	Minimal

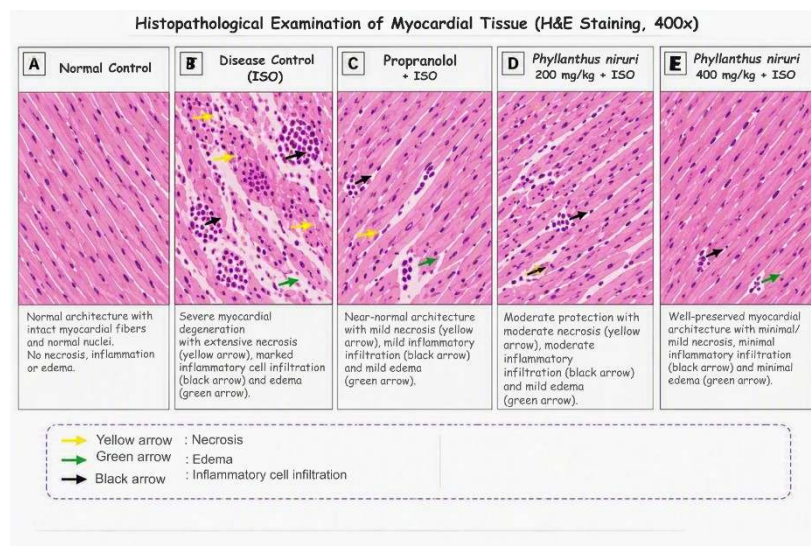


Figure 6: Histopathological Findings

RESULTS AND DISCUSSION

The normal control group showed normal myocardial architecture with intact cardiac muscle fibers and no apparent necrosis, inflammation, or edema. In contrast, the isoproterenol-treated disease control group showed severe myocardial damage accompanied by marked necrosis, inflammatory cell infiltration, edema, and degeneration of cardiac tissue.

Treatment with *P. niruri* root extract produced noticeable improvement in myocardial architecture. The 200 mg/kg dose showed moderate protection with a reduction in tissue damage, whereas the 400 mg/kg dose produced better preservation of myocardial structure with only mild or minimal necrosis, inflammation, and edema. The findings were broadly consistent with the biochemical results, particularly the reduction in

cardiac biomarkers and MDA together with restoration of SOD, CAT, and GSH.

CONCLUSION

The present study demonstrated the cardioprotective potential of the hydroalcoholic root extract of *Phyllanthus niruri* against isoproterenol-induced myocardial injury in Wistar rats. Isoproterenol markedly increased CK-MB from 116.24 ± 4.08 to 304.52 ± 8.96 U/L and LDH from 228.36 ± 6.14 to 536.48 ± 13.25 U/L. Treatment with *P. niruri* at 400 mg/kg reduced CK-MB to 158.42 ± 5.12 U/L and LDH to 284.53 ± 9.34 U/L. The extract also reduced MDA from 6.14 ± 0.18 to 2.56 ± 0.10 nmol/mg protein and improved SOD, CAT, and GSH levels. Histopathological examination further showed improved myocardial architecture at the higher dose. Overall, the findings indicate that *P. niruri* root extract possesses promising cardioprotective activity, possibly through attenuation of oxidative stress and lipid peroxidation.

4. FUTURE SCOPE

Further studies should focus on identification and quantification of the active phytoconstituents responsible for the cardioprotective activity. Dose-response studies, molecular investigations involving oxidative stress and inflammatory pathways, and isolation of individual bioactive compounds may provide better understanding of the mechanism. Long-term toxicity, pharmacokinetic studies, and further validated animal studies are also required before considering potential clinical application.

REFERENCES

1. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990-2019: update from the GBD 2019 study. J Am Coll Cardiol. 2020;76 (25):2982-3021.
2. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40 (3):237-269.
3. Dhalla NS, Temsah RM, Netticadan T. Role of oxidative stress in cardiovascular diseases. J Hypertens. 2000;18 (6):655-673.
4. Rona G. Catecholamine cardiotoxicity. J Mol Cell Cardiol. 1985;17 (4):291-306.
5. Rathore N, John S, Kale M, Bhatnagar D. Lipid peroxidation and antioxidant enzymes in isoproterenol induced oxidative stress in rat tissues. Pharmacol Res. 1998;38 (4):297-303.
6. Rathore N, Kale M, John S, Bhatnagar D. Lipid peroxidation and antioxidant enzymes in isoproterenol induced oxidative stress in rat erythrocytes. Indian J Physiol Pharmacol. 2000;44 (2):161-166.
7. Sharma M, Kishore K, Gupta SK, Joshi S, Arya DS. Cardioprotective potential of Ocimum sanctum in isoproterenol induced myocardial infarction in rats. Mol Cell Biochem. 2001;225 (1):75-83.
8. Rajadurai M, Stanely Mainzen Prince P. Preventive effect of naringin on lipids, lipoproteins and lipid metabolic enzymes in isoproterenol-induced myocardial infarction in Wistar rats. J Biochem Mol Toxicol. 2006;20 (4):191-197.
9. Rajadurai M, Stanely Mainzen Prince P. Preventive effect of naringin on lipid peroxides and antioxidants in isoproterenol-induced cardiotoxicity in Wistar rats: biochemical and histopathological evidences. Toxicology. 2006;228 (2-3):259-267.
10. Rajadurai M, Stanely Mainzen Prince P. Preventive effect of naringin on cardiac markers, electrocardiographic patterns and lysosomal hydrolases in normal and isoproterenol-induced myocardial infarction in Wistar rats. Toxicology. 2007;230 (2-3):178-188.
11. Bagalkotkar G, Sagineedu SR, Saad MS, Stanslas J. Phytochemicals from Phyllanthus niruri Linn. and their pharmacological properties: a review. J Pharm Pharmacol. 2006;58 (12):1559-1570.
12. Lee NYS, Khoo WKS, Adnan MA, Mahalingam TP, Fernandez AR, Jeevaratnam K. The pharmacological potential of Phyllanthus niruri. J Pharm Pharmacol. 2016;68 (8):953-969.



13. Kaur N, Kaur B, Sirhindi G. Phytochemistry and pharmacology of *Phyllanthus niruri* L.: a review. *Phytother Res.* 2017;31 (7):980-1004.
14. Patel JR, Tripathi P, Sharma V, Chauhan NS, Dixit VK. *Phyllanthus amarus*: ethnomedicinal uses, phytochemistry and pharmacology: a review. *J Ethnopharmacol.* 2011;138 (2):286-313.
15. Thippeswamy AHM, Shirodkar A, Koti BC, Sadiq AJ, Praveen D, Viswanatha Swamy AHM, et al. Protective role of *Phyllanthus niruri* extract in doxorubicin-induced myocardial toxicity in rats. *Indian J Pharmacol.* 2011;43 (1):31-35.
16. Chikku AM, Rajamohan T. Coconut haustorium maintains cardiac integrity and alleviates oxidative stress in rats subjected to isoproterenol-induced myocardial infarction. *Indian J Pharm Sci.* 2012;74 (5):397-402.
17. Zhang B, Wang H, Yang Z, Cao M, Wang K, Wang G, et al. Protective effect of alpha-pinene against isoproterenol-induced myocardial infarction through NF- κ B signaling pathway. *Hum Exp Toxicol.* 2020;39 (12):1596-1606.
18. Abukhalil MH, Hussein OE, Aladaileh SH, Althunibat OY, Al-Amarat W, Saghir SA, et al. Visnagin prevents isoproterenol-induced myocardial injury by attenuating oxidative stress and inflammation and upregulating Nrf2 signaling in rats. *J Biochem Mol Toxicol.* 2021;35 (11):e22906.
19. Mi X, Zhang Z, Cheng J, Xu Z, Zhu K, Ren Y. Cardioprotective effects of Schisantherin A against isoproterenol-induced acute myocardial infarction through amelioration of oxidative stress and inflammation via modulation of PI3K-AKT/Nrf2/ARE and TLR4/MAPK/NF- κ B pathways in rats. *BMC Complement Med Ther.* 2023;23 (1):277.
20. Ohkawa H, Ohishi N, Yagi K. Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Anal Biochem.* 1979;95 (2):351-358.

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