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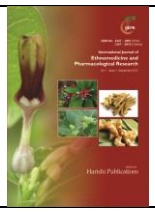

Estimation of cardiac marker enzymes in the isoprenaline-induced myocardial infraction in rats

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ABSTRACT

The present study was conducted to elucidate thecardioprotective effect of *Diospyros discolor* in isoprenaline – induced myocardial infarction in male albino rats.The levels of cardiac marker enzymes (Creatinine kinase,creatinine kinase-MB and homocysteine)in serum of experimental groups of rats were determined. The prior administration of *D.discolor* for 30 days significantly prevented the isoprenaline-induced elevation in the levels of diagnostic myocardial enzymes in experimental rats. *D.discolor* exerted an antioxidant effect against isoprenaline – induced myocardial infarction by blocking the induction of lipid peroxidation. The cardioprotective effect of *D.discolor* might be ascribable to its antioxidant property and membrane stabilizing action.

Keywords: *Arisaema tortuosum*, phytochemical, mineral composition, proximate analysis, Kollihills, Ethnoveterinary.

INTRODUCTION

Cardiac markers are biomarkers measured to evaluate heart function. Levels of cardiac enzymes are principally utilized to help assess the presence or absence of acute myocardial infarction.They are also increasingly being used as predictive markers for short term and long term adverse outcomes particularly among those preventing with acute coronary syndrome.

Cardiovascular diseases including atherosclerosis and cardiac tissue injury after myocardial infarction is due to free radicals generated at the site of damage (Sasikumar and shyamala,2000).Impaired endothelial function is believed to be an early step in the pathogenesis and pathophysiology of atherosclerosis, thrombosis and cardiovascular disease. (Hagar, 2002).Myocardial infarction and the resultant abnormalities in cardiac function represent the leading cause of morbidity and mortality in developed countries (Chada, 1998). However, with changing lifestyle in developing countries, like India, particularly in urban areas. Moreover, with advanced lifestyle in developing countries, like India,,particularly in meteropolitan cities,MI is making an increasingly important contribution to mortality statistics of such countries (Levy and Feinleib, 1984). Despite this understanding appreciation of the cellular processes and mechanistic bases underlying cardiac dysfunction associated with myocardial infarction and most important in applying this knowledge to therapeutic interventions (Kumar and Anandan, 2007).It has been demonstrated that isoprenaline administration produces free radicals that affects membrane integration with disintegration of poly unsaturated fattyacid in the membrane which might be responsible for tissue damage and infarcted heart(Nirmala and Puvanakrishnan,1996;Singal et al,1982).

Isoprenaline is a β-adrenergic agonist that causes severe stress in the myocardium resulting in infarct-like necrosis of the heart muscle (Prabhu*et al.*, 2006). Isoprenaline induced myocardial infarction serves as a well standardized model to study the beneficial effects of many drugs and cardiac dysfunctions (Mohanty*et al*., 2004).

A better understanding of the processes involved in myocardial infarction has stimulated the search for drugs which could limit the myocardial injury.In the present study, an attempt has been made to access the protective effects of D.*discolor* on the antioxidant status in isoprenaline-induced myocardial infarction in rats, a well-established animal model for studying the effects of many drugs on the process of myocardial infarction.

MATERIAL AND METHODS

Chemicals

All the purified chemicals, coenzymes, substrates and standards used for the experiment, were of analytical grade and purchased from Himedia, SD Fine chemicals and Qualigens, India.

Induction

Myocardial stress was induced in experimental rats by intraperitoneal administration of isoprenaline at a single dose of 11 mg(dissolved in physiological saline)/ 100 g b.wt/rat/day) for 2 days.(Anandan*et.al.*,2003).

Statistical Analysis

All numerical data in text, figures and tables are expressed as the mean SD. Statistical analysis was performed by one way analysis of variance (ANOVA) followed by the analysis of level of significance between the groups based on ANOVA using SPSS 10 statistical package. Difference among means was analysed by least significant difference (LSD) at 5% level ($p<0.05$).

RESULT AND DISCUSSION

CK-MB is an isoenzyme of creatine kinase. It is a golden standard marker of myocyte injury/death, leaks out from myocardium due to disintegration of contractile apparatus and increased sarcoplasmic permeability (Mair*et al*., 1994).

Table 1 depicts the activities of myocardial markers namely creatinine kinase (CK) and creatine kinase-MB (CK-MB), in serum of control and experimental rats.

Table-1. Activities of cardiac marker enzymes in the serum of control and experimental rats

Groups	CK	CK-MB
Group I (Control)	274.81 ± 1.5	2.58 ± 0.12
Group II (ISO)	530.76± 1.04a*	5.33 ± 8.16a*
GroupIII (*D.discolor* extract + ISO)	298.6 ± 1.16b*	3.78 ± 0.11b*
Group IV (Atorvastatin + ISO)	276.27 ± 0.3c*	3.25 ± 0.13c*

Values are expressed by mean ± SD (n=6)
Units: CK - IU/L CK - MB - ng/ml
Statistical comparison:
a-Group II is compared with group I;
b-Group III is compared with group II
c-Group IV is compared with group II

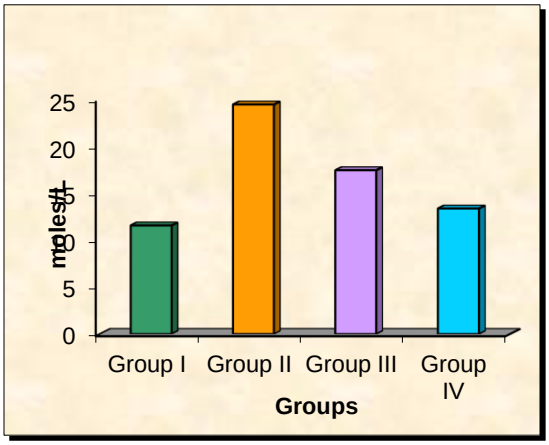
Statistical significance: * - significant ($p<0.05$)

A significant ($p<0.05$) increase was observed in the activities of both creatine kinase and creatine kinase-MB (CK-MB) in the serum of isoprenaline - induced rats (group II) as compared to control rats (group I). The oral pretreatment with ethanolic extract (group III) showed a significant ($p<0.05$) decrease in their activities as compared to the isoprenaline administered group of rats (group II). The administration of atorvastatin to group IV rats brought back their levels to near normalcy, when compared with the group II rats.

The significant increase in CK levels suggests the occurrence of considerable, necrotic damage to the myocardial membrane by isoproterenol leading to their leakage.As the size of infarct shows an increase, the release of CK and CKB also increase proportionately, thereby probably helping to get an idea of the magnitude of infarction (Shah and Haridas, 2007).

The release of CK reflects the non-specific alterations in the plasma membrane integrity and permeability, as a response to β-adrenergic stimulation. The decrease in their levels might be due to the prevention of isoprenaline induced release of these enzymes from the myocardium into the systemic circulation and maintained the rats of near normal status, indicating the cardio protective action of *D. discolor* (Kumar and Anandan, 2007).

Figure 1 shows the activities of homocysteine in serum of control and experimental rats.



Homocysteine (Hcy) is a branch point metabolite, the biological fate of which is linked to vitamin B_{12}reduces folates and vitamin B_6. It is a non-protein forming amino acid, whose metabolism is at the intersection of two metabolic pathways: remethylation and transulfuration. Homocysteine levels in serum was significantly ($p<0.05$) increased in isoprenaline treated rats (group II) when compared to control rats (group I). Ethanolic *D.discolor* extract (group III) pretreated rats showed a significant decrease ($p<0.05$) in the serum homocysteine level as compared toisoprenaline administered group (group II). The atorvastatin treated rats (group IV) also showed a similar trend by decreasing the level of homocysteine, as compared to the group II rats. A significant ($p<0.05$) difference was noticed in their activities in the *D.discolor* treated rats (group III) and atorvastatin treated rats (group IV).

This increase in the serum homocysteine levels, might be attributed to the inhibition of the production of monocyte macrophage derived interleukins, which triggers from the adhesion of rolling monocytes to vascular endothelium, a necessary prelude to the initiation of atherosclerosis (Berwanger*et al*., 1995).

CONCLUSION

The levels cardiac marker enzymes namely creatinine kinase, creatinine kinase – MB and homocysteine) were increased in the toxicity induced rats and their levels were reversed on treatment with the ethanolic leaf extract of *D.discolor* which indicated the counteracting ability of the free radicals by the antioxidants present in the extract. A similar results were obtained with atorvastatin treated group. The results of the present study indicate the cardio protective effect of *D.discolor* on isoprenaline – induced myocardial infarction may probably related to a counteraction of free radicals by its antioxidant nature, to a strengthening of myocardial membrane by its membrane stabilizing action.

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