

Research Article**Effect of Taurine Pre-treatment on Hematological Parameters in Isoproterenol Induced Myocardial Infarction in Albino Rats****M. Sudha^{1*}, D. Rajkumar²**¹Reader, Department of Physiology, Raja Muthaiah Medical College, Annamalai University, Chidambaram, Tamil Nadu, India²Professor & Head, Department of Physiology, Raja Muthaiah Medical College, Annamalai University, Chidambaram, Tamil Nadu, India***Corresponding author**

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Abstract: Cardiovascular diseases form the major health concern in recent years, causing severe illness and death throughout the world. About 16.7 million people around the globe die of myocardial infarction every year (WHO 2004) which forms one third of global deaths. Clinically, taurine has been widely used in variety of conditions including cardiovascular diseases, hypercholesterolemia, macular degeneration, Alzheimer's disease, hepatic disorders, alcoholism and in cystic fibrosis. The present study was designed to investigate the effect of taurine pre treatment on hematological parameters in isoproterenol induced myocardial infarction in albino rats by demonstrating the hematological changes. Wistar male rats were randomly divided into four groups namely Control (G1), taurine (G2), isoproterenol (G3) and Isoproterenol + taurine (G4). Taurine treated group received taurine (100mg/kg body wt) orally for 30 days. Myocardial infarction was induced in rats by isoproterenol administration (100mg/kg) subcutaneously (sc) at an interval of 24 hrs on 31st and 32nd day. Hematological changes were assessed from 24 hrs after the last dose of isoproterenol. There was decrease in RBC count, WBC count, Platelet count, Hb%, Lymphocyte count, Eosinophil count and increase in Neutrophil count in G4 rats as compared to G3 rats. Taurine pretreatment preserved the changes to near normal. Taurine may provide cardioprotection under conditions of ischemia-reperfusion due to its antioxidant properties. It may prevent oxidant-mediated damage of the cardiomyocyte membrane and subsequent intracellular Ca²⁺ overload.**Keywords:** Taurine, Hematological changes, Myocardial infarction, Wistar strain male albino rats

INTRODUCTION

Cardiovascular disease (CVD) is an important cause of mortality and morbidity in India. Mortality statistics and morbidity surveys have indicated substantial regional variations in the CVD prevalence and mortality rates [1]. Disease of coronary artery is almost always because of atheroma and its associated complications [2].

Cross sectional and epidemiological studies performed for the analysis of coronary heart disease has revealed that this condition is increasing both in urban and rural areas [3]. Population based study conducted in Chennai showed a prevalence of CAD of 11% which was 10 times more than what it was in 1970 [4].

Taurine (2-amino ethane sulfonic acid) is a conditionally essential amino acid, not utilized in protein synthesis. It is found free or in simple peptides. It was first discovered as a component of ox bile in 1827 [5].

Low levels of taurine are associated with various pathological lesions including cardiomyopathy, retinal degeneration and growth retardation especially if deficiency occurs during development [5]. Derived from methionine and cysteine metabolism, taurine is known to play an important role in numerous physiological functions [6]. Other metabolic actions of taurine include bile acid conjugation, detoxification, membrane stabilization, osmoregulation and modulation of cellular calcium levels [5].

Isoprenaline (Isoproterenol) is a sympathomimetic drug. It acts almost exclusively on β -adrenergic receptors [7]. It has a powerful stimulating action on heart, increases cardiac output, excitability and rate [8]. Isoprenaline is a potent β -agonist that is widely used as an inhaled bronchodilator for asthma [9]. It also causes peripheral vasodilatation and produces fall in diastolic blood pressure [10] and usually maintains or slightly increases systolic blood

pressure [11]. Isoproterenol is used for inducing myocardial infarction in experimental model [19].

MATERIALS AND METHODS

The sample for this study includes 48 Wistar strain male albino rats weighing 150-200gm. 24 rats were randomly selected for the study and they were divided into 4 Groups G1, G2, G3 and G4 with each group consisting of 6 animals each. Group- 1 (Control group) N=6Rats received a standard diet for a period of 30 days.

Group – 2 (taurine treated group) N=6Rats were orally administered with taurine 100mg/kg body weight / day dissolved in distilled water by intragastric intubation for 30 days

Group- 3 (Isoproterenol treated) N=6Rats were injected with Isoproterenol 100mg/kg body weight /day subcutaneously for 2 consecutive days at an interval of 24 hrs. for induction of Myocardial Infarction. Group 4 (Isoproterenol + taurine) N=6Rats were pretreated with taurine 100mg /kg body weight orally for 30 days and

Myocardial Infarction was induced with Isoproterenol at a dose of 100 mg / kg body weight at an interval of 24 hrs on 31st and 32nd day. At the end of experimental period i.e.24 hrs after the last injection of Isoproterenol the experimental animal were sacrificed. The blood samples were collected and stored in EDTA tubes and divided into two parts. One part was used for total blood cell count and other part was used to obtain plasma.

Estimation of blood parameters

Haemoglobin (Hb) concentration, Red blood cell (RBC) count, White blood cell (WBC) count, Neutrophil, Lymphocyte and Eosinophil percentages were estimated using an automatic hematological analyzer (Sysmex XS-1000IXS - 800 I automated hematology analyzer - Sysmex Corporation Kobe, Japan). The cells were counted based on the principle of electronic impedance [6].

Statistical analysis

The data was expressed as mean \pm SD. Statistical comparisons were performed by student's t-test. The results were considered as significant if the P values were 0.05 or less.

Table 1: Complete blood count in control and experimental group of rats

Parameters	Control Group I	Taurine Group II	Isoproterenol Group III	Taurine + isoproterenol Group IV
RBCx10 ⁶ μc^{-1}	5.7 \pm 0.06	6.35 \pm 0.8	7.1 \pm 0.8	6.15 \pm 1.5*
WBCx10 ³ μc^{-1}	5.11 \pm 0.7	5.5 \pm 0.8	6.1 \pm 0.7	5.3 \pm 0.6*
Platelet x10 ⁵ μc^{-1}	879.5 \pm 7.7	725.4 \pm 6.9	980 \pm 6.4	728 \pm 5.4*
Hb g %	13.4 \pm 0.6	13.6 \pm 0.5	14.6 \pm 0.6	12.9 \pm 0.6*
Lymphocytes %	74.1 \pm 4.7	72 \pm 8.3	79.7 \pm 6.9	70 \pm 7.3*
Neutrophil %	29.1 \pm 6.7	36.3 \pm 6.2	19.3 \pm 2.7	30.3 \pm 6.0*
Eosinophil %	2.0 \pm 0.2	1.0 \pm 0.1	0.03 \pm 0.03	0.01 \pm 0.00*

** - Significant at p- value < 0.001 level, * - Significant at P- value < 0.05 level

Table 1 shows the hematological parameters in normal and experimental groups of rats. Significant increase in RBC count, WBC count, platelet count, taurine administration significantly preserved the hematological parameters to near normal

DISCUSSION

Taurine may provide cardio protection due to its antioxidant properties. They may prevent oxidant-mediated damage of the cardiomyocyte membrane and subsequent intracellular Ca²⁺ overload [12]. During acute coronary events (unstable angina and myocardial infarction, often before the onset of ischemic damage), neutrophils secrete proteolytic enzymes in latent forms that are activated by hypochlorous acid (HOCl) generated by myeloperoxidase [12, 13]. The major function of taurine in leukocytes is to trap chlorinated oxidants (HOCl). It converts them into less toxic chloramines and taurine chloride production is found to

result in decreased NO production [14, 15] thereby causing increase in neutrophil count and decrease in lymphocyte count.

Neutrophils have been observed to infiltrate with the onset of ischemic injury and increase their numbers progressively throughout the process associated with evolution of myocardial infarction [13].

Decreased platelet aggregability has found to be associated with an increase in platelet taurine and glutathione concentrations, as well as a decrease in thromboxane release on platelet aggregation [12, 16].

Thus, taurine has been demonstrated to stabilize platelets against aggregation. It has been observed that during taurine depletion, platelets become overly sensitive to aggregation. Taurine depresses this tendency to aggregate [12]. Platelets from taurine-

depleted cats had been reported to be twice as sensitive to aggregation as platelets from cats that received taurine. In humans with normal taurine level had shown an increase in resistance to aggregation by 30 to 70% with taurine supplement of 400 mg/day or 1600 mg/day, respectively [16].

Erythropoietin production and erythroid differentiation are regulated by reactive oxygen species, especially by H₂O₂, which are involved in redox-sensitive signaling pathways indicating that the ROS generation can suppress erythropoietin synthesis. On the other hand, antioxidants can stimulate the synthesis [17]. Only few antioxidants can modulate hematological parameters [15].

Taurine is a physiological antagonist of HOCl, protects the heart by diminishing the inflammatory response in cases of acute coronary artery disease [13].

Platelet activation, adhesion and aggregation at sites of vascular endothelial disruption caused by atherosclerosis are key events in arterial thrombus formation [18]. The status of taurine has been reported to exert a significant effect on aggregation of platelets [12, 19].

CONCLUSION

Taurine pretreatment preserved the hematological changes to near normal. Taurine having antioxidant properties may provide cardioprotection under conditions ischemia-reperfusion. It may prevent oxidant-mediated damage of the cardiomyocyte membrane and subsequent intracellular Ca²⁺ overload [12].

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