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Histopathological Observations on Protective Effects of Vitamin E on Endosulfan Induced Cardiotoxicity in Rats

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Abstract: The protective effects of vitamin E was investigated on the cardiotoxicity induced by endosulfan administration. Male rats in different groups were given endosulfan (2 mg per kg body weight per day in corn oil through gavage), vitamin E (200 mg per kg body weight twice a week in corn oil through gavage) and endosulfan and vitamin E at the same dose and route, to the control group corn oil is given at the dose rate of 2 mL per rat per day through gavage, for a period of 28 days. The animals were sacrificed and heart tissues were collected and subjected to histopathology. The result indicated, sever congestion, haemorrhages with interstitial oedema. In some places there was diapedesis of leukocytes. Myocardium showed different degrees of degeneration, some of the myofibrils were found to be granular with pyknotic nuclei. Thickening of wall of arteries were seen. In the Vitamin E and endosulfan treated group the above mentioned lesions were significantly decreased in their severity. This study brought to light the protective effects of vitamin E on the toxic pathological lesions caused by endosulfan administration.

Key words: Vitamin E, endosulfan, cardiotoxicity, histopathology

INTRODUCTION

Protective role of vitamin E as an antioxidant is well documented, its multivarious activities like, scavenger of free radicals, inhibition of lipooxygenases and reduction of peroxidase in association with lipoxygenases were reported (Kumar *et al*., 2004). Cardioprotective activity of vitamin E against doxorubicin alone and in combination with cyclophosphamide in mice were studied Bjelogrlic *et al*., 2005) . Protective action of vitamin E treatment against lipid peroxidation and cardiac dysfunction associated with ischaemia-reperfusion was reported (Venditti *et al*., 1999) . It is possible that vitamin E being present at cell membrane site may prevent organophosphate-induced oxidative damage (John *et al*., 2001), on the other hand it is reported that vitamin E is able to minimize oxidative damage caused by exercise in rats (Kelle *et al*., 1999) . The protective effects of vitamin E against damage caused by formaldehyde in the testes of rats (Zhou *et al*., 2006) and damage of hepatic tissue caused by CCl_4 in cattle (Yonezawa *et al*., 2005) were reported. In this study endusulfan a broad spectrum insecticide and acaricide is used to induce cardiotoxicity and the protective effects of vitamin E was evaluated by histopathology.

MATERIALS AND METHODS

Male wistar rats with average body weight of 150-180 g were obtained from laboratory animal centre of college of veterinary medicine of Urmia University. They were housed in plastic cages under standard conditions. They were provided with commercial pellete and water was given ad libitum. The room temperature of 20-22°C , 50% relative humidity and 12 h light/dark cycle were maintained.

The rats were divided into 4 groups each including 6 animals which were as follows:

- Control goup: Corn oil is given at the dose rate of 2 mL per rat per day through gavage.
- Endosulfan treated group: Endosulfan at the dose rate of 2 mg per kg body weight per day in corn oil through gavage.
- Vitamin E treated group: Vitamin E is given at the dose rate of 200 mg per kg body weight twice a week in corn oil through gavage.
- Vitamin E plus endosulphan treated group: In this group E was given along with endosulphan at the same dose levels through the same routes.

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All the groups were given the treatments for a period of 28 days. At the end of the experiment the rats were euthanized using diethyl ether. Necropsy were done on the animals and the heart tissues were fixed in 10% buffer formal saline for histopathology. Tissues were fixed and processed, embedded in paraffin and 4-5 μ sections were cut using microtome. The sections were stained by hematoxylin and eosin and were examined under light microscope and observations were recorded.

RESULTS

The histopathological observations in different experimental groups were presented in Table 1, in control and vitamin E treated groups there was no detectable histopathological changes (Fig.1). In the endosulfan treated group there was sever congestion, haemorrhages with interstitial oedema (Fig. 2) in some places there was diapedesis of leukocytes (Fig. 3). Myocardium showed different degrees of degeneration, some of the myofibrils were found to be granular (Fig. 4) with pyknotic nuclei. Another histopathological lesion was the thickening of wall of arteries (Fig. 5). In the Vitamin E and endosulfan treated group the above mentioned lesions were significantly decreased in their severity (Fig. 6).

Table 1: Extent of histopathological heart lesions in different experimental groups

Type of lesions	Group I	Group II	Group III	Group IV
Congestion	-	+++	-	++
Haemorrhages	-	+++	-	++
Oedema	-	+++	-	++
Thickening of arterial walls	-	+++	-	++
Leukocytic infiltration	-	++	-	+
Degeneration	-	+++	-	++

Group I: Control group, Group II: Endosulfan treated, Group III: Vitamin E treated, Group IV: Vitamin E plus endosulfan treated, No abnormalities detected (-), Mild lesion (+), Moderate lesion (++), Sever lesion (+++)

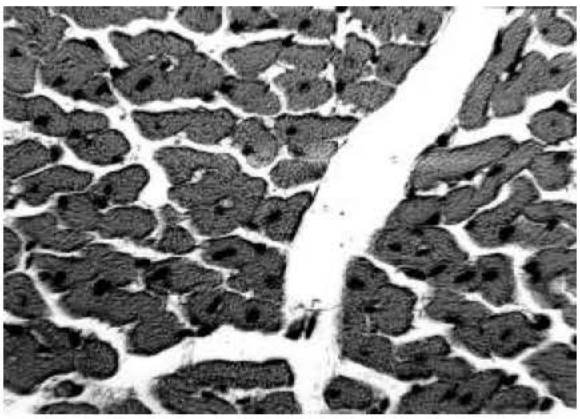

Fig. 1: Myocardium of rat, Vitamin E treated group, no histopathological lesion (H & E X 400)

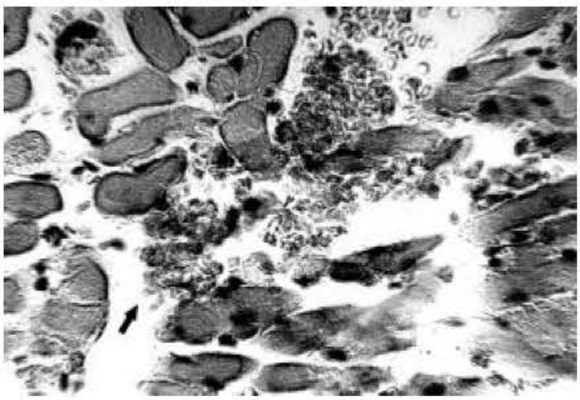

Fig. 2: Myocardium of rat, endosulfan treated group, haemorrgages (arrow) and interstitial oedema (H & E X 400)

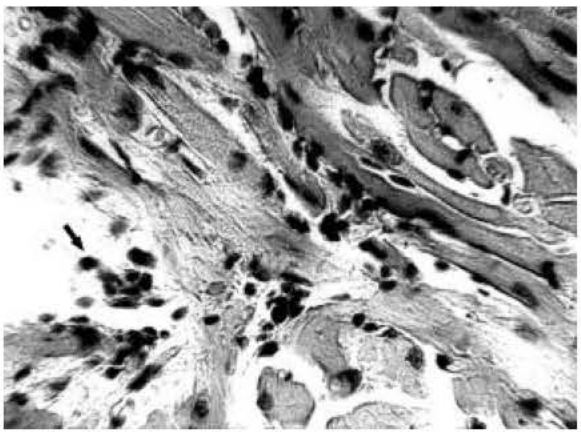

Fig. 3: Myocardium of rat, endosulfan treated group, leukocytic infiltration (arrow), oedema and degenerative changes (H & E X 400)

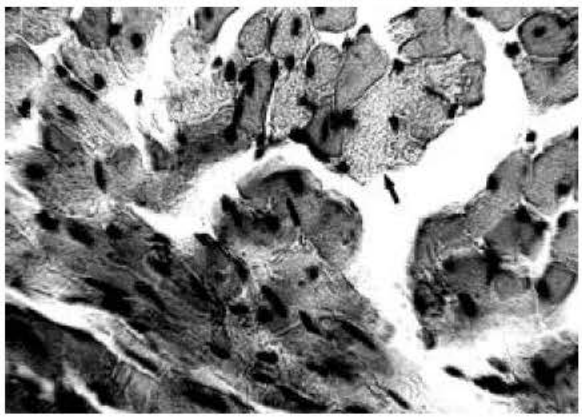

Fig. 4: Myocardium of rat, endosulfan treated group, degeneration of myofibrils (arrow) with granularity (H & E X 400)

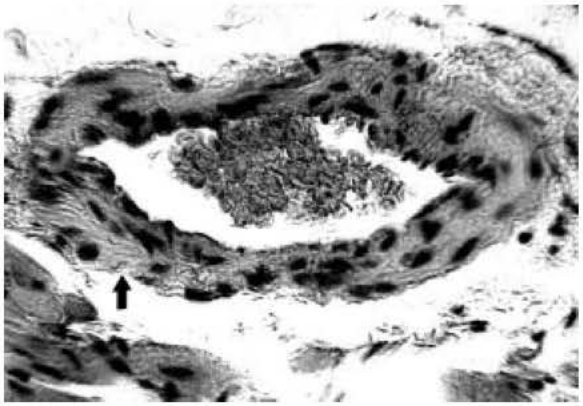

Fig. 5: Myocardium of rat, endosulfan treated group, thickening of the wall of artery (H & E X 400)

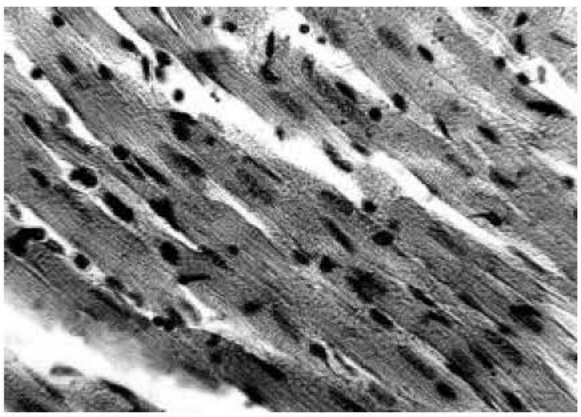

Fig. 6: Myocardium of rat, endosulfan and vitamin E treated group, reduction in the severity of the toxic changes (H & E X 400)

DISCUSSION

The present investigation clearly indicated the myocadiotoxicity of endosulfan which could bring about, circulatory disturbances, degenerative and inflammatory reaction on the heart, it was reported that such changes could be due to the leakage of cellular contents caused by endosulfan-mediated oxidative damage to the sarcolemmal structures handling cellular homeostasis (Ananya *et al*., 2003). Several studies on liver, lungs, adrenocortical cells and testes indicated that endosulfan could cause oxidative stress (Dorval *et al*., 2003; Bebe and Panemangalore, 2003; Naqvi and Vaishnavi, 1993; Saiyed *et al*., 2003). Free radicals generated by endothelial cells of the arterial wall due to the effect of the toxin could be considered in the hyperplastic changes of the arterial wall in this study, experimental studies indicated that dietary supplementation of vitamin E could reduce the atherosclerosis (Meydani, 2001). Vitamin E is broadly considered an antioxidant, preventing biological membranes and plasma lipoprotein from undergoing oxidative stress by quenching free radicals (Traber and Sies, 1996). Some investigators reported that administrating vitamin E, might be useful in controlling the toxic effects of insecticides and chemicals, this fact was clearly brought to light in the present study by the histopathological observations on the heart muscle which showed clear reduction in the degenerative and inflammatory lesions severity, caused by endosulfan, hence one could conclude that administration of vitamin E along with endosulfan would amereolate the toxicity of endosulfan, and vitamin E could be considered as protective agent in myocardiotoxiciy by endosufan. In recent years several such investigations have provided circumstantial evidence for the beneficial effects of vitamin E supplementation on human health in various chronic diseases and acute clinical conditions (Packer, 1992).

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