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 leucocytes. 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 lipoxygenases 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 Bjelogorie 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 CCl₄ in cattle (Yonezawa et al., 2005) were reported. In this study endosulfan 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 pellet 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 group: 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 endosulfan treated group: In this group E was given along with endosulfan 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>
<thead>
<tr>
<th>Type of lesion</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestion</td>
<td>-</td>
<td>+++</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Haemorrhages</td>
<td>-</td>
<td>+++</td>
<td>-</td>
<td>++</td>
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<tr>
<td>Oedema</td>
<td>-</td>
<td>+++</td>
<td>-</td>
<td>++</td>
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<tr>
<td>Thickening of arterial walls</td>
<td>-</td>
<td>+++</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Leukocytic infiltration</td>
<td>-</td>
<td>++</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Degeneration</td>
<td>-</td>
<td>+++</td>
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</tbody>
</table>

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 (++), Severe lesion (+++)
experimental studies indicated that dietary supplementation of vitamin E could reduce the atherosclerosis (Meydanii, 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 administering 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 ameliorate the toxicity of endosulfan, and vitamin E could be considered as protective agent in myocardioxicity by endosulfan. 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).

REFERENCES


