Original Paper

Effect of Lambda Cyhalothrin on Rats: An Acute Toxicity Study

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ABSTRACT

The objective of the study was to evaluate the acute toxicity of lambda cyhalothrin in rat.

Male albino rats were exposed to 80 mg/kg body weight of lambda cyhalothrin by means of gavage.

The toxicological manifestations include myocardial hyalinisation, lysis of reticular framework in spleen, and spermatogonial cell degeneration in testes.

Key Words: Lambda cyhalothrin; Histopathology

Introduction

Lambda cyhalothrin, a pyrethroid insecticide, is effective against a wide range of pests. Today, pyrethroids are used preferably over organochlorines and organophosphates due to their high effectiveness, low toxicity to non-target organisms, and easy biodegrability.[1] They act by disrupting the normal functioning of the nervous system in an organism resulting in paralysis or death. Lambda cyhalothrin is a stomach and contact insecticide, and is used in vector control such as mosquito control by direct sprayingover water bodies.[2] It shows adulticidal, ovicidal and, particularly, larvicidal activity against insects belonging to Lepidoptera, Hemiptera, Diptera, and Coleoptera.[3,4] It is effective against cockroaches, mosquitoes, ticks and flies, which often act as disease vectors.[5] Though pyrethroids are not as toxic as some other insecticides, they do exhibit some degree of toxicity to mammals.[6] The present investigation was undertaken to find out the acute toxic effects of lambda cyhalothrin in laboratory animals (albino rats).

Materials and Methods

Acute toxicity of lambda cyhalothrin (5% emulsifiable concentrate) in male albino rats was determined by approximate Lethal Dose method.[7] For this study, two male rats were fasted overnight before treatment. One rat served as control and was orally administered distilled water only by means of gavage. The other rat was administered lambda cyhalothrin diluted in distilled water at a dose of 80 mg/kg body weight, in dose volume of 10 ml/kg. Depending on the result, the dose was decreased or increased by a factor of 1.5 for another rat in further experimentation.

All the animals were kept under constant observation for recording the toxic manifestations. After gaining the approximate lethal dose of lamda cyhalothrin, the experiment was repeated 5 times to evaluate reproducibility. At the end of the study, gross pathology and histopathology of the sacrificed rats (test and control) were examined. Histopathology was undertaken after haematoxylin and eosin (H&E) staining.[8] Statistical analysis was done by using one way ANOVA, and post-test analysis by using Tukey t-test.

Results

Apart from agricultural uses, cyhalothrin also has public and animal health applications by way of control of a broad spectrum of insects including cockroaches, flies,

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mosquitoes, and ticks. Lambda cyhalothrin-treated rats (80 mg/kg body weight) succumbed after 8 hours following exposure. Toxic manifestations included profuse salivation, mild fur erection, exophthalmia, abnormal gait and posture, hyperaesthesia, tremors, respiratory distress and death.

Rats administered lambda cyhalothrin at a dose of 53.33 mg/kg body weight (reducing the dose rate by dividing by the factor 1.5) showed similar symptoms, but there was no fatality. Therefore, approximate lethal dose of acute lambda cyhalothrin toxicity in rats was estimated as 80 mg/kg body weight. Approximate lethal dose was again administered to one rat with its duplicate for reducibility of the data (n=5). Death of treated rats in each experiment was observed between 8-15 hours with similar clinical signs as above.

Histopathology revealed noticeable changes in heart, spleen and testes of rats exposed to 80 mg/kg of lambda cyhalothrin. Hyalinization of cardiac muscle, interspacing of fibres leading to necrosis and myolysis (**Fig 1 & 2**) were observed. Interspacing between muscle fibres, congestion, haemorrhages and oedema in the myocardium were also observed. There was severe depletion of lymphocytes in the Malpighian corpuscles of the spleen (**Fig 3**). Lymphocyte deficiency was mainly seen in periarterial spaces, with lysis of reticular framework. Testes revealed degeneration of spermatogonial cells, detachment from basement membrane, and coagulation of spermatozoa in luminal spaces of testicular lobules (**Fig 4**).

Discussion

Available data indicate that lambda cyhalothrin is moderately toxic via the oral route in test animals. Reported oral LD_{50} values are 79 mg/kg and 56 mg/kg for male and female rats, respectively.[9,10] Similar clinical signs with slight variations have been observed in rats subjected to fenvalerate exposure.[11,12] The LD_{50} values for technical grade lambda cyhalothrin and lambda cyhalothrin 2.5% EC for male rats were reported to be 79 mg/kg and 398 mg/kg body weight respectively.[10,13]

The lesions observed in the heart, spleen and testes can be reasonably attributed to the cytotoxic effects of cyhalothrin. Pulmonary oedema has been reported rarely following pyrethroid ingestion, usually in association with severe neurological complications and may contribute to a fatal outcome.[14] Toxic irritant substance brought to the heart via blood circulation can exert direct toxic effect on cardiac muscles, or may cause anoxia due to congestion and reduction in blood circulation. Thus, the changes in the cardiac muscles and spleen could be the direct effect of lambda cyhalothrin and/or the result of anoxia due to congestion. Increased venous hydrostatic pressure in acute toxicity may be responsible for cardiac oedema.[15] Lindane has been reported to cause immune dysfunction.[16] This may explain the depletion of lymphocytes in the spleen of rats.

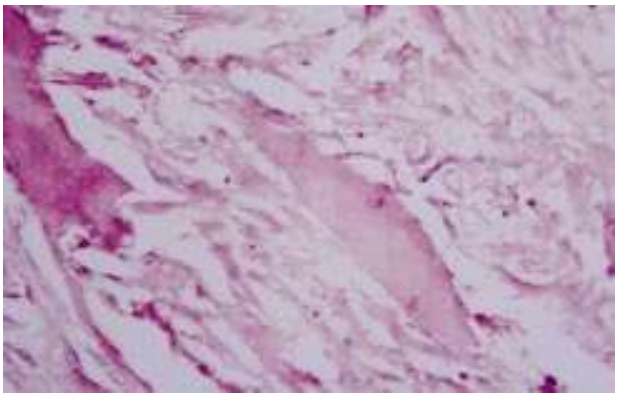

Fig. 1 Hyalinization of cardiac muscle, interspacing of fibres leading to necrosis and myolysis

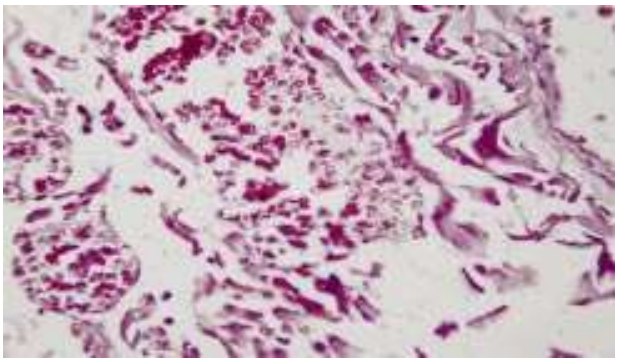

Fig. 2 Interspacing between muscle fibres, congestion and haemorrhages in myocardium

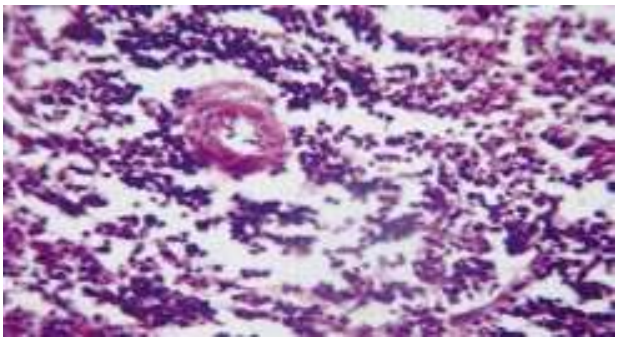

Fig. 3 Mid Lymphocytes deficiency in periarterial space and lysis of reticular framework in spleen

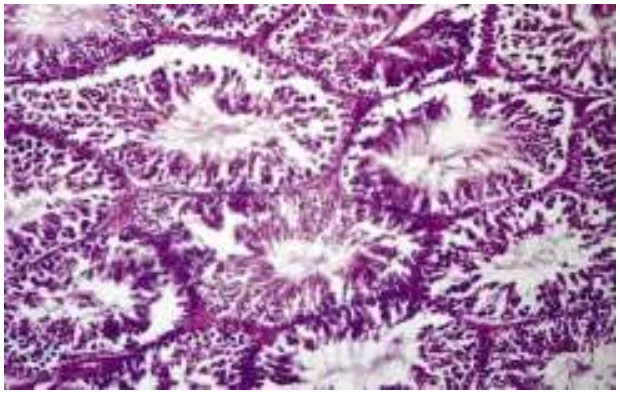

Fig. 4 Degeneration of spermatogonial cells, detachment from basement membrane, coagulation of spermatozoa in luminal space of the testicular lobule.

The direct effect of the pesticide coupled with interference of oxygen uptake and depression of brain cell respiration might be responsible for the damage observed in the brain tissues.[17] Severe changes in testes might have been caused by direct toxic effect of lambda cyhalothrin. There was reduction in the number of germinal layers as reported in some earlier studies.[18] Several pesticides are known to cause problems related to male fertility.[19] The prenatal effects of cyhalothrin exposure on the physical and behavioral development of infant and adult rats have been demonstrated in earlier studies.[20] Drug-induced sexual dysfunction is mediated by multiple mechanisms, mainly toxicity, stress, sedation and possibly via GABA and dopaminergic systems. Previous reports have shown reduced numbers of viable offspring at doses of 50 mg/kg/day in the second and third generations, in three-generational rat studies.[10,20]

Thus it can be concluded that the approximate acute toxic dose of lambda cyhalothrin in rats is 80 mg/kg body weight, and that it causes cardiac hyalinisation, mild lymphocyte deficiency in the spleen, and testicular degeneration.

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