

EFFECT OF CADMIUM CHLORIDE ON HAEMATOPOIETIC ORGAN: THYMUS

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ABSTRACT

Since there is a constant release of trace elements in unnaturally higher concentrations and often in unusual physio-chemical state, the fear of them being hazardous to human health is now an indisputable truth. Once perpetuated in the environment, metals are not readily detoxified by metabolic activity. As a result, they get accumulated contributing to potential environmental hazard. In the present study, the effect of cadmium chloride on thymus of mice has been investigated. For the experiment, adult healthy male Swiss albino mice were fed with aqueous solution of CdCl₂ prepared by dissolving 20mg of CdCl₂ in 1000ml of distilled water, thus giving a concentration of 20ppm and then administered orally in drinking water *ad libitum* continuously till the end of experiment. Animals were autopsied by cervical dislocation at each post-treatment interval of 1, 2, 4, 7, 10, 14 and 28 days. Thymus was taken out after autopsy, weighed and fixed in Bouin's fluid. Then the tissue was dehydrated and embedded in paraffin wax and transverse sections were cut at 5 μ and stained in Harri'shaematoxylin-eosin stain for histopathological studies. After cadmium chloride treatment, thymus showed a significant decline in the organo-somatic index value continuously till day 10, and recovering thereafter. Pathological changes after CdCl₂ treatment include necrosis of thymocytes, fibrous tissue proliferation, Pyknosis, Karyolysis, and distortion of Hassal's corpuscles. Recovery started at a much later interval, and was probably due to the binding of intracellular cadmium to metallothionein which protects the tissue against the cadmium toxicity.

Keywords: Karyolysis, Pyknosis, Metallothionein, Thymocytes, Cadmium Toxicity.

Introduction

Heavy metals are the most toxic, non-biodegradable intrinsic component of earth's crust. With technological advancement and diversification of industries, combined with specialization in all fields, the volume and complexity of metals is also increasing day by day. Due to their toxicity, heavy metals are well known environmental pollutants persisting in the environment, contaminating the food chains and causing different health hazards^[1]. Most of the pollution problems which we face today stem from over exploitation of our natural resources and/or heedless disposal of waste material in the environment. Once perpetuated in the environment, metals are not readily detoxified by metabolic activity. As a result, they get accumulated contributing to potential environment hazard. Cadmium as an industrial pollutant has aroused a great concern due to its toxic effects on various body tissues. Cadmium enters animal tissues via inhalation, ingestion, cigarette smoking or occupational exposures. Tobacco smoking is one of the largest single source of cadmium exposure in humans^[2]. An important route of exposure is the circulatory system and the blood vessels are considered to be the mainstream organ of cadmium toxicity^[3]. In the tissues, concentration of cadmium increases with the increased period of exposure to cadmium^[4]. However, their toxicity depends on several factors including the dose, route of exposure, age, gender and also nutritional status of exposed individuals. They disrupt cellular events including growth, proliferation, differentiation, damage-repairing process and apoptosis. Cadmium also causes genomic instability^[5]. Therefore, an attempt has been made to assess the effect of cadmium chloride on the thymus of Swiss albino mice.

Material and Methods

In the present study, the effect of cadmium chloride on thymus of mice has been investigated. For the experiment, adult healthy male Swiss albino mice were fed with the aqueous solution of the cadmium chloride. For preparing this solution, 20 mg of cadmium chloride was dissolved in 1000 ml of distilled water. This 20ppm cadmium chloride concentration solution was fed orally *ad libitum* in drinking

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water continuously till the last autopsy interval. By cervical dislocation, animals were autopsied after 1, 2, 4, 7, 10, 14 and 28 days of post- treatment. Thymus was taken out after the autopsy, weighed, fixed in Bouin's fluid, dehydrated and embedded in paraffin wax. Transverse sections were cut at 5μ from the middle part of the tissue and stained with Harri'shaematoxylin-eosin stain for histopathological studies.

Results and Discussion

In the present study, animals after cadmium chloride treatment exhibited a significant decline in the organo-somatic index values continuously till day 10, and recovering thereafter. Decrease in the weight of thymus after i.p. or subcutaneous injection of cadmium chloride has also been reported by^[6,7]. Cadmium causes significant decrease in body weight and organ weight [8]. This decrease in the index value may be due to death of lymphocytes at early intervals.

Histogram-1

Changes in the Values of Organo-Somatic Index of Thymus (mg/100gm Body Weight) of Mice

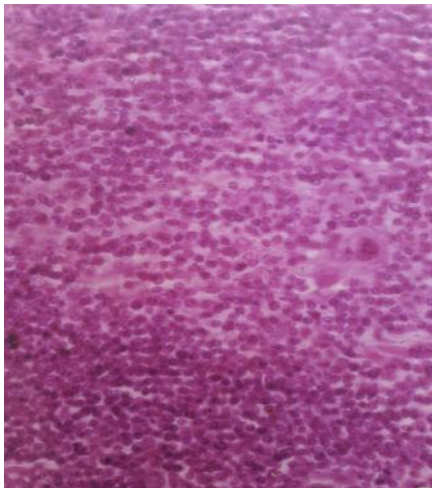
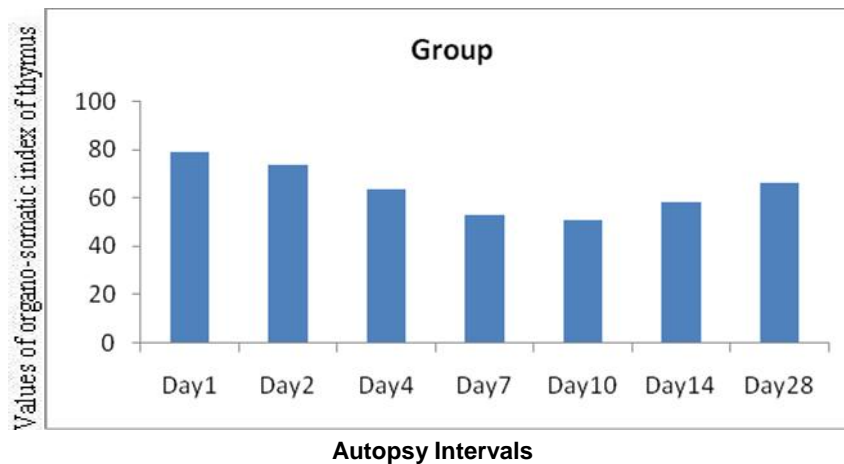


Fig: 1 Photomicrograph of thymus of albino mice after 2 days of $CdCl_2$ treatment showing destruction of lymphocytes. Cortex filled with nuclear debris and a large number of necrotic cells.

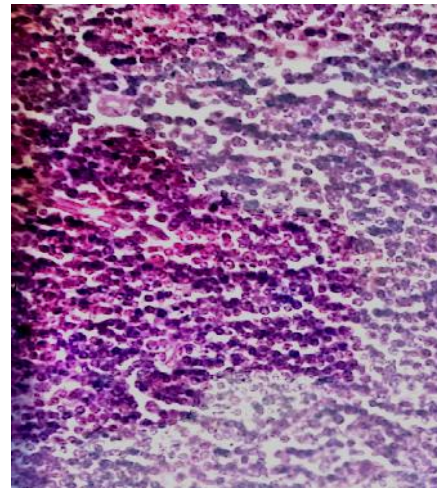


Fig: 2 Photomicrograph of thymus of albino mice after 10 days of $CdCl_2$ treatment showing a large number of dividing cells both in cortex and medulla. Hassal's corpuscles do not show normal structure.

Exposure to cadmium produces a wide variety of effects involving many vital organs and systems. Concentration of cadmium in the tissues and blood of fish exposed to higher doses of cadmium increases with the duration of exposure [4]. Acute cadmium administration induces histological changes[9] and apoptosis in rat thymus [10]. In the present study pathological changes after cadmium chloride treatment include pyknosis, necrosis of thymocytes, karyolysis, fibrous tissue proliferation and

distortion of Hassal's corpuscles. Few mitotic figures were observed during the early intervals but were not normal and resulted in mitotic death. In chicks, cadmium induces thymic atrophy, decreased lymphocytes in cortex, gaps at certain places, degeneration of reticular cells and inflammatory edema in medullary region [11]. Recovery started at much later interval. Although the mechanism of cadmium induced toxicity is not well understood but may be probably due to the binding of intracellular cadmium to metallothionein which protects the tissue against the cadmium toxicity. If the exposure to cadmium is extensive, the cadmium binding sites in the organism becomes progressively saturated and despite continuous exposure, the cadmium concentration in the tissue tends to stabilize. Once this point is reached, the cadmium that is still absorbed cannot be further retained and is rapidly excreted [12]. The results of the present study confer the above explanation as thymus showed a progressive recovery even after the continuous exposure to cadmium chloride.

Conclusion

From the present investigation following can be deduced:

- Decrease in organo-somatic index values of thymus during the early intervals was probably due to the death of lymphocytes and their removal from the tissue by active phagocytosis.
- Histopathological studies showed recovery of the tissue at much later intervals, which might probably due to the binding of intracellular cadmium to metallothionein which protects tissue against the cadmium toxicity.

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