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Effects of Lead on the Olfactory Bulb of the Adult Albino Rats

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Abstract

Lead is virtually toxic to every organ of body including central nervous system where it may manifest as encephalopathy and hyposmia yet the exact mechanism of these clinical manifestations remains inconclusive. The present study was aimed to see the microscopic changes in the olfactory bulb of rat induced by oral administration of a lead compound in adult albino rats. A total number of 12 adult albino rats of either sex were included in the present study consisting of equal numbers in both control and experimental groups. Experimental group received 4% aquous lead acetate orally for a period of 3 weeks then animals of both groups were euthanized with overdose of general anaesthesia and perfused with 10% formalin. Olfactory bulbs were dissected out and processed for paraffin embedding. 10 µ-thick sections were stained with H&E and observed under light microscope. On gross examination brains from the experimental group revealed generalized edema and petechial haemorrhages. Histopathology of the olfactory bulbs revealed edema and congestion with vacuoles of variable sizes almost throughout. Distortion of glomeruli, clumping of periglomerular cells and increasing number of pyknotic cells were also noticed. It was concluded that lead has toxic effects on the central nervous system including olfactory bulb in the form of edema, microscopic hemorrhages and neuronal loss which may explain the clinical manifestations of lead toxicity.

Key words: Albino rats, Olfactory bulb, Lead acetate, Neurotoxicity, Edema, Haemorrhage

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Introduction

Exposure of lead can take place either through inhalation of dust, fumes, vapours, or ingestion of contaminated foods or drinks. Because of its cumulative property it is capable of exerting toxic effects at any level of exposure. Toxic effect of lead on the body is known as Plumbism which on the central nervous system manifests as encephalopathy that is accompanied by areas of focal cortical necrosis. Its clinical manifestation includes headache, incoordination, tremor, twitching, convulsion, paralysis, coma and death [1]. In the brain, cerebellum was found to be most severely affected [2]. Significant decrease in spine density [3] and reduction in the maximum width of the hippocampus [4] have also been reported. Bilaterally symmetrical spongiform changes in the roof nuclei of cerebellum [5] was also reported in dogs exposed to orally fed lead while bilaterally symmetrical areas of vacuole formation were observed at the tips of cortical gyri [6]. Other heavy metals like Cadmium dust induced anosmia [7] and in another study it was reported that inhalation of cadmium affected olfactory bulb and contributed to olfactory dysfunction [8]. Zinc gluconate trihydrate induced cellular and tissue damages to olfactory neuroepithelium and to mitral cells in rat olfactory bulb [9]. Exposure to high levels of mercury (a heavy metal) has also been thought to cause olfactory loss [10]. The present study was aimed to see the effect of lead on the histology of the olfactory bulb which may explain the olfactory dysfunction in the individuals exposed to lead.

Material and Method

A total number of 12 adult albino rats (6 male & 6 female) weighing 120g (±10g) were used in the present study. 6 rats with equal number of either sex were treated with 4% lead acetate, while the other 6 (3 male and 3 female) served as control did not receive any active compound. The concentration of lead acetate was ascertained after a careful trial in order to find maximum survival of 15 to 20 days. After this period, rats were anaesthetized with ether and perfused with buffered 10% formalin. Both olfactory bulbs were dissected out from superior aspect and separated from the brain. Olfactory bulbs were cut transversely into two parts and processed for paraffin embedding. From each blocks 10 µ thick sections were cut with rotary microtome. Haematoxylin and eosin stained sections were used for light microscopic observations.

Observations

On histological examination of olfactory bulb of treated group, it was observed that as compared to control (Fig. 1A, B) there was generalized edema and congestion in almost all layers of olfactory bulb. Capillaries appeared dilated and congested. Distortion of glomerular contour was obvious. Periglomerular cells were hyperchromatic and showed clumping. Multiple vacuoles of variable sizes were noticed in the outer plexiform layer (Fig. 1C and 1D). Granule cell layer showed loss of cells. Dark and pyknotic nuclcei were also present. No such types of abnormalities were found in control group of rats.

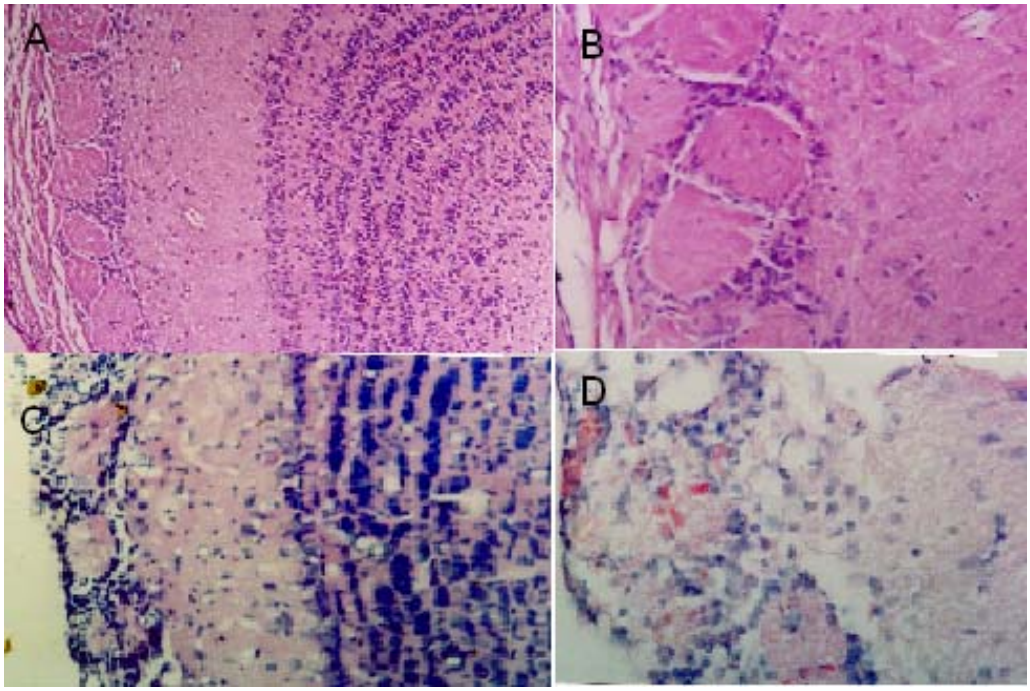

Figure 1. *Photomicrographs from olfactory bulb of control rat (A and B) showing typical laminar pattern without edema, congestion or vacuolation while those from experimental group (C and D) show edema, vacuolation, congestion, loss of glomerular contour and clumping of periglmerular cells.*
H &E stain, X100 (A &C). X400 (B&D).

Discussion

The layers of the olfactory bulb which show damage mainly include the lamina glomerulosa, outer plexiform layer and the granule cell layer. Gross damage in the region of olfactory bulb was also seen in present study in the form of petechial haemorrhage which might have been due to capillary dilatation. These findings are in partial agreement with those reported by certain workers [11] who exposed adult guinea pig to Lead carbonate and reported vascular changes in addition to encephalopathic effects of lead mediated directly at the neuronal level. Some other workers [12] have demonstrated hypertrophy of vascular pericytes. Lead pellets implantation in the rat forebrain produced vascular changes in addition to parenchymal necrosis and spongiosis in the hypothalamas [13]. Histological study of many parts of brain e.g. cerebral cortex , corpus striatum, choroid plexus and cerebellum after lead exposure revealed cerebellum to be most severely damaged [2]. In addition in this study [2] hemorrhages noticed along with damage to molecular and Purkinje cell layers and edema in the granule cell layer which correlated very well with the findings of the present study.

Histopathological findings of olfactory bulb on neuron and neuropil in the present study are to a great extent in agreement with those reporting degeneration of cells in the cerebral cortex [4] and reduced number of Purkinje and granule cells [14] of cerebellum and of hippocampal neurons on lead exposure [15] as well as vacuolations after incubation of guinea pig hippocampus in a lead containing medium [16] which was more pronounced in outer plexiform layer of olfactory bulb. The vascular changes observed in the present study are in agreement with those reported after exposure of lead in dogs [5] which indicates that irrespective of animal species, olfactory bulb is vulnerable to lead acetate toxicity.

Conclusion

From the above study it was concluded that olfactory bulb is vulnerable to toxicity of lead similar to the other parts of brain and that histopathological changes mainly included edema, vacuolation and congestion, glomerular distortion and pyknotic periglomerular cells.

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