

Neurohistological Effects of Lead on Hippocampus of Adult Albino Rat

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ABSTRACT

LEAD is toxic to every organ of body, including central nervous system. This study is aimed to observe the histopathological changes in the hippocampus of rat induced by oral administration of lead compound in adult albino rats. Total number of 20 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% aqueous lead acetate orally for 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Hippocampus was dissected. 10 μ thick sections were obtained by usual histological procedure and were stained with Nissl stain. On light microscopic observation, hippocampus from experimental group revealed shrunken neurons, shrinkage of the layers associated with area of vacuolation. It was concluded that lead has toxic effects on the central nervous system including hippocampus which may explain the clinical manifestation of lead neurotoxicity

KEYWORDS : Albino rats, Hippocampus, Lead acetate , Neurotoxicity

Introduction

Exposure of lead can take place more through inhalation of dust, vapours, fumes or ingestion of contaminated foods and drinks. It is capable of causing toxic effects at any level of exposure. In the brain, its most severe toxic effects were found to be on cerebellum (1) Toxic effects of Lead on the body have been given the name as Plumbism, which on the central nervous system manifests as encephalopathy that is associated with focal cortical necrosis. Its clinical manifestation includes headache, twitching, convulsions, incoordination, tremor, paralysis, coma and death (2). Dendritic alterations of cerebellar Purkinje neurons in postnatally lead-exposed kittens has been reported (3) and decrease in maximum width of the hippocampus have been noted (4). Symmetrical spongiform changes, bilaterally, in the roof nuclei of cerebellum have also been reported in the dogs exposed to orally fed lead (5) as well as bilaterally symmetrical areas of vacuole formation were noted at the tips of the cortical gyrii (6). Cadmium, another heavy metal has been reported to induce anosmia (7) and in still another study cadmium inhalation was reported to affect olfactory bulb and contribute to olfactory dysfunction (8). Zinc gluconate trihydrate was reported to induce cellular and tissue damages to olfactory neuroepithelium and to olfactory bulb mitral cells in rats (9). Developmental exposure to lead causes inherent changes on voltage-gated sodium channels in rat hippocampal CA1 neurons (10). The aim of present study was to see the effect of lead on the histology of hippocampus which may explain the clinical signs and symptoms following lead intoxication.

Material and Method

Total number of 20 adult albino rats (10 male and 10 female) weighing approximately 120 g were used in the present study. 10 rats with equal number of either sex were treated with 4% lead acetate while the remaining 10 rats (5 male and 5 female) served as control group and were given distilled water and did not receive any active compound. The concentration of lead acetate was ascertained after a careful trial so as to find a maximum survival to 15 to 20 days. Then, rats were anaesthetized with ether and perfused with buffered 10% formalin. Brain was dissected out, meninges removed and 3 mm thick coronally sliced pieces were cut from hippocampus and processed for paraffin embedding. Then, 10 μ thick sections were cut with rotary microtome. These sections were stained with Nissl stain and observed under the light microscope.

Observations

Hippocampus of (control group) reveals normal neuronal cell layers and characteristic large multipolar neurons in low power (Figure 1) and in high power (Figure 2). On examination, under the light microscope, the hippocampus of the treated group showed shrunken neurons and also showed shrinkage of all the layers associated with area of vacuolation deep in low power (Figure 3) as well as in high power (Figure 4).

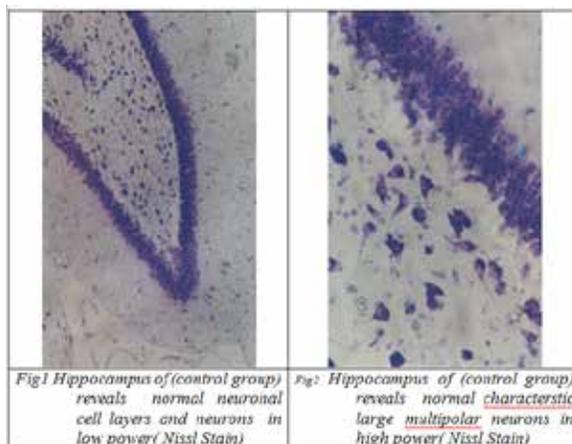


Fig 1 Hippocampus of (control group) reveals normal neuronal cell layers and neurons in low power (Nissl Stain)

Fig 2 Hippocampus of (control group) reveals normal characteristic large multipolar neurons in high power (Nissl Stain)

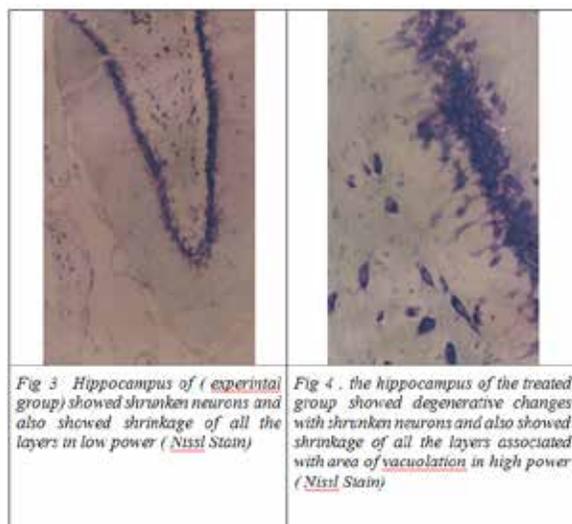


Fig 3 Hippocampus of (experimental group) showed shrunken neurons and also showed shrinkage of all the layers in low power (Nissl Stain)

Fig 4. the hippocampus of the treated group showed degenerative changes with shrunken neurons and also showed shrinkage of all the layers associated with area of vacuolation in high power (Nissl Stain)

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of lead on the hippocampus. These findings are in partial agreement with other neurohistological studies. In one of the studies, vascular changes in addition to encephalopathic effects of lead mediated directly at the neuronal level, was reported, when adult guinea pig was exposed to Lead carbonate (11). Some other workers have reported hypertrophy of vascular pericytes (12). Implantation of lead pellets in the forebrain of rat resulted in vaso-

lar changes in addition to parenchymal necrosis and spongiosis in the hypothalamus(13). Histological study of many parts of the brain e.g. cerebral cortex, cerebellum, choroid plexus and corpus striatum after toxic lead exposure showed cerebellum to be most severely affected(14). Additionally in this study(14) haemorrhages noted along with damage to Purkinje cell layers and oedema in the granule cell layer. Chronic Lead exposure is also shown related to muscarinic cholinergic modulation of synaptic transmission and plasticity in rat hippocampus (18). The histological findings observed in our study confirmed the hippocampus toxicity following lead poisoning and correlated very well with the histological findings of the other studies.

Conclusion

Exposure of rat to lead for 15 days produces demonstrable microscopic alterations in the hippocampus.

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