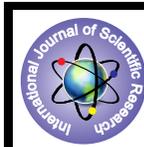


Neurohistological Effects of Lead on Cerebrum of Adult Albino Rat



Medical Science

KEYWORDS : Albino rats, Cerebrum, Lead acetate, Neurotoxicity

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ABSTRACT

LEAD is virtually toxic to almost every organ of body, including central nervous system. The present study is aimed to observe the histopathological changes in the cerebrum of rat induced by oral administration of lead compound in adult albino rats. A 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 period of 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Cerebrum was dissected. 10 μ thick sections were obtained by usual histological procedure and were stained with Nissl stain. On light microscopic observation, cerebrum from experimental group revealed multipolar neurons having shrunken soma. It was concluded that lead has toxic effects on the central nervous system including cerebrum which may explain the clinical manifestation of lead 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). Plumbism means toxic effects of lead on the body, 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 hippocampal area have been noted (4). Symmetrical spongiform changes, bilateral 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 gyri (6). Cadmium, another heavy metal has been reported to induce anosmia (7). In another study effect of chronic lead administration has been shown to cause aggressive behaviour among Male Albino Wistar Rats (8). Zinc gluconate trihydrate was reported to induce cellular and tissue damages to olfactory neuroepithelium and to olfactory bulb mitral cells in rats (9). Higher level of mercury (heavy metal) exposure has been thought to cause olfactory loss (10). The aim of present study was to see the effect of lead on the histology of cerebrum 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 sliced pieces were cut from cerebrum 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

Cerebrum of (control group) shows normal distribution of the multipolar neurons and the different cells in low power (Figure 1) and in high power (Figure 2). On examination, under the light microscope, the cerebrum of the treated group showed neurons having shrunken soma and in low power (Figure 3). Cerebral cortex of experimental group was suggestive of degenerative changes with multipolar neurons having shrunken soma in high power (Figure 4)

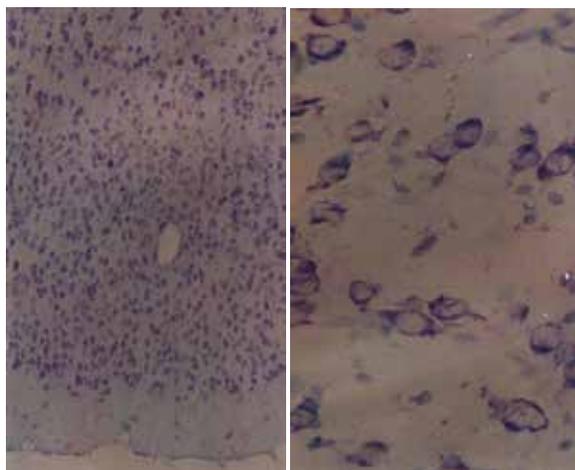


Fig -1 Cerebrum (control group) shows normal distribution of the neurons and darkly stained glial cells in low power (Nissl Stain)

Fig -2 Cerebrum (control group) shows normal distribution of the well defined multipolar neurons and glial cells in high power (Nissl Stain)

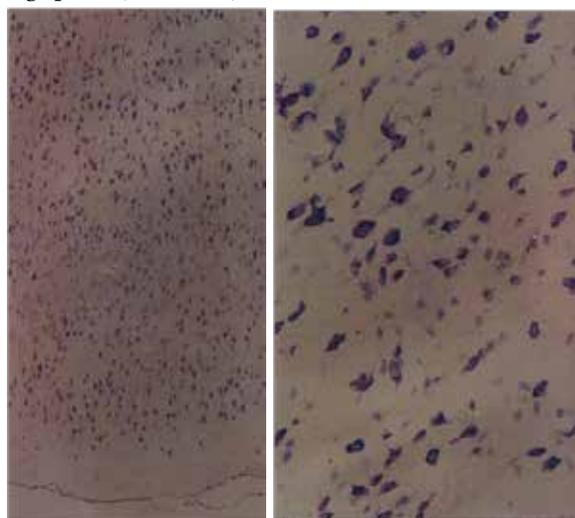


Fig -3 Cerebrum (experimental group) showed shrunken soma of the neurons in low power (Nissl Stain)

Fig -4 Cerebral cortex (experimental group) showed degenerative changes with shrunken soma of the neurons in high power (Nissl Stain)

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of lead on the cerebrum. These findings are in partial agreement with other neurohistological studies. In one of the studies, vascular changes in addition to encephalopathic, were 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 vascular 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 haemorrhages noted along with damage to Purkinje cell layers and oedema in the granule cell layer (14). It has also been shown that Lead exposure causes developmental neurotoxicity of the central nervous system (21). The histological findings observed in our study confirmed the cerebral neurotoxicity 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 cerebrum.

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