Volume : 4 | Issue : 8 | Aug 2015 • ISSN No 2277 - 8179

Research Paper

Medical Science

KEYWORDS : Albino rats, Cerebellum, Lead acetate, Neurotoxicity

NeurohistologicalEffects of Lead on Cerebellum of Adult Albimo Rat

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ABSTRACT

LEAD is toxic toevery organ of body, including central nervous system. This study is aimed to observe the histopathological changes in the cerebellum 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% aquous lead acetate orally for 15days., then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Cerebellum was dissected. 10µ thick sections were obtained by usual histological procedure and were stained with Nissl stain. Under light microscope, cerebellum from experimental group revealed loss of Nissl substance in the Purkinje cells and neurons with perineuralspace. It was concluded that lead has toxic effects on the central nervous system including cerebellum which may explain the clinical manifestation of leadneurotoxicity

Introduction

Lead exposure can take place more through inhalation of dust, vapours, fumes or ingestion of contaminated foods and drinks. Plumbism means toxic effects of lead on the body, which on the central nervous system menifests as encephalopathy that is associated with focal cortical necrosis. It's clinical menifeststion includes headache, twitching,convulsions, incoordination, tremor, paralysis, coma and death (1) It can cause toxic effects at any level of exposure. In the brain, it's most severe toxic effects were found on the cerebellum (2). Dendritic alterations of cerebellar Purkinje neurons in postnatally lead-exposed kittens has been reported (3) and decrease in maximum width of the hippocampushave been noted(4). Symmetrirical spongiform changes, bilateraaly, 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 disfunction(8). Lead toxicity is has been shown to cause histological and oxidative damage rat cerebrum and cerebellum (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 cerebellum 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 10rats (5male 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 cerebellum 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

Cerebellum of (control group) shows normal distribution of well defined neurons and in high power(Figure 1). Light microscopic observation,of cerebellum from experimental group revealed degenerative changes with loss of Nissl substance in the Purkinje cells and neurons could be separately identified with perineural space associated with some neurons in high power(Figure2) .

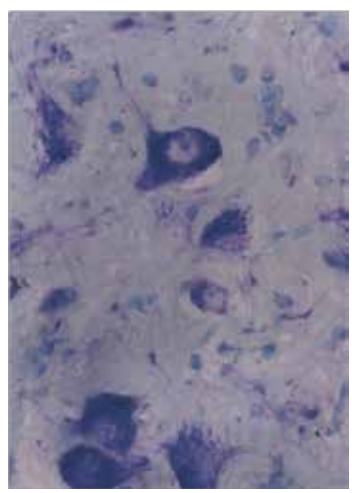

Fig -1 Cerebellum of (control group) shows normal distribution of well defined neurons and in high power (Nissl Stain)

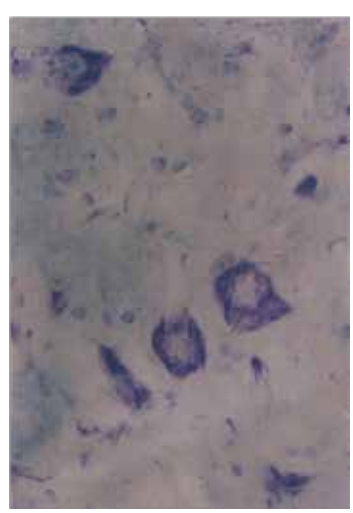

Fig -2Cerebellum from experimental group revealed degenerative changes with loss of Nissl substance in the Purkinje cells and neurons could be separately identified with perineural space associated with some neurons in high power (Nissl Stain

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of lead on the cerebellum. These findings are in partial agreement with other neurohistological

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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 pallets in the forebrain of rat reslted 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 studyhaemorrhagesnoted along withdamage to Purkinge cell layers and oedema in the granule cell layer (14). Lead exposure has also been reported to cause developmental neurotoxicity of the central nervous system (19) The histological findings observed in our study confirmed the cerebellar 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 changes in the cerebellum.

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