

Neurohistological Effects of Lead on Corpus Striatum of Adult Albino Rat



Neuroanatomy

KEYWORDS : Albino rats, Corpus striatum, Lead acetate, Neurotoxicity

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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 corpus striatum 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 this 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. Corpus striatum was dissected. 10 μ thick sections were obtained by usual histological procedure and were stained with Glee's Silver stain. Under light microscope, corpus striatum from experimental group revealed shrunken neurons and well defined fiber bundles surrounded by the glial cells. It was concluded that lead is toxic to central nervous system including corpus striatum which may be correlated to and 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 hippocampus have been noted (4). Symmetrical spongiform changes, bilaterally, in the roof nuclei of cerebellum(5) have also been reported in the dogs exposed to orally fed lead 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) and in still another study cadmium inhalation was reported to affect olfactory bulb and contribute to olfactory dysfunction(8). Past adult lead exposure is reported to be linked to neurodegeneration measured by brain MRI (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 corpus striatum 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 corpus striatum and processed for paraffin embedding. Then, 10 μ thick sections were cut with rotary microtome. These sections were stained with Glee's Silver stain and observed under the light microscope.

Observations

Corpus striatum of (control group) shows normal distribution of the well stained large neurons and the fiber bundle areas are poorly defined in high power (Figure 1). On examination, under the light microscope, the corpus striatum of the treated group showed shrunken neurons and also showed well pronounced fiber bundle areas surrounded by glial cells (Figure 2)

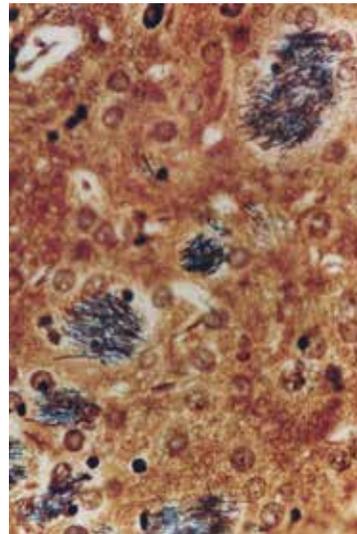


Fig -1 *Corpus striatum of (control group) shows normal distribution of the well stained large neurons and the fiber bundle areas are poorly defined in high power(Glee's Silver Stain)*

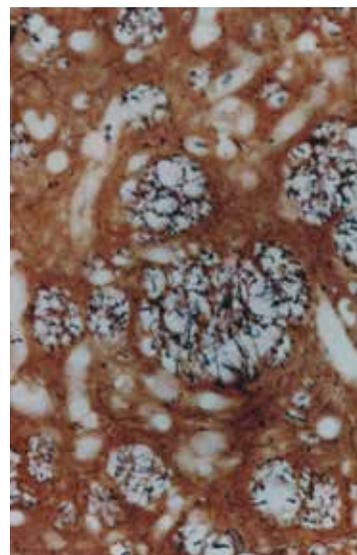


Fig -2 *Corpus striatum of (experimental group) showed shrunken neurons and also showed well pronounced fiber bundle areas surrounded by glial cells in high power (Glee's Silver Stain)*

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of lead on the corpus striatum. These findings are in partial agreement with other neuro-histological 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 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(14) haemorrhages noted along with damage to Purkinje cell layers and oedema in the granule cell layer. Oxidative stress was reported in mouse brain exposed to Lead (20) The histological findings observed in our study confirmed the neurotoxicity of corpus striatum 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 corpus striatum.

REFERENCE

1. Press, MF a) Lead encephalopathy in neonatal Long-Evans rats: morphologic studies. *J Neuropathol Exp Neurol*, Jan 1977; 36, 169-93; | 2. Balbus-Komfeld JM, Stewart W, Bolla KI, Schwartz BS; Cumulative exposure to inorganic lead and neuro-behavioral test performance in adults: an epidemiological review *Journal: Occup Environ Med* 1995; 52, 2-12 | 3. Patrick GW, Anderson WJ; Dendritic alterations of cerebellar Purkinje neurons in postnatally lead-exposed kittens. *Dev Neurosci* 2000; 22, 320-8 | 4. Bansal MR, Kausiial N, Banejee UC; Effect of oral lead acetate administration on the mouse brain. *J Trace Elem Exp Med* 1990, 3, 235-246 | 5. Hamir AN, Sullivan ND, Handson PD; Neuropathological lesions in experimental lead toxicosis of dogs. *Neurobiol Aging* 1984 Winter 1984; 5, 297-307 | 6. Stowe HD, Vandeveld M; Lead-induced encephalopathy in dogs. *J Neuropathol Exp Neurol*; 1979; 38, 463-74 | 7. Adam and Crabtree. Anosmia in alkaline battery workers. *Br J Ind Med*. 1961; 18, 216-221. | 8. Jean Robert; Harmful effects of cadmium on olfactory system in Mice. *Inhalation toxicology*. 2008; 20, 1169-1177 | 9. Stewart WF, Schwartz BS, Davatzikos C, Shen D, Liu D, Wu X, et al. Past adult lead exposure is linked to neurodegeneration measured by brain MRI. *Neurology*, 2006. 66(10): p. 1476-1484 | 10. Upadhyaya U; Olfactory loss as a result of toxic exposure. *Otolaryngology Clinics of North America*; 2004; 37, 1185- 1207. | 11. Bouldin and Krigman; Acute lead encephalopathy in the guinea pig. *Acta Neuropathologica*, August 1975; 33, 185-190 | 12. Markov DV and Dimova RN; Ultrastructural alterations of rat brain microglial cells and pericytes after chronic lead poisoning. *Acta Neuropathol* 1974; 25-35 | 13. Hirano A and Kochen JA; Some effects of intracerebral lead implantation in the rat. *Acta Neuropathol (Berl)* 1975; 30, 307-15 | 14. McConnell P, Berry M; The effects of postnatal lead exposure on Purkinje cell dendritic development in the rat. *Am. J. Dis. Child* 1979; 133, 786-90 | 15. Zook BC, London WT, Wilpizeski CR, Sever JL; Experimental lead paint poisoning in nonhuman primates. Pathologic findings. *Brain Res* 1980; 189, 369-76. | 16. Brink U, Wechsler W; Microscopic examination of hippocampal slices alters short-term lead exposure in vitro. *Neurotoxicol Teratol* 1985; 11, 539-43 | 17. Sanders T, Liu Y, Buchner V, and Tchounwou PB, Neurotoxic effects and biomarkers of lead exposure: a review. *Rev Environ Health* 2009; 24(1), p. 15-45. | 18. Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, Altaye M, Egelhoff JC, et al. Decreased brain volume in adults with childhood lead exposure. *PLoS Med* 2008; 5(5), e112. | 19. Carboni AA; Oral administration of zinc gluconate trihydrate. *Am J Rhinology*. 2006; 20, 262-268 | 20. Wang J, Wu J, and Zhang Z. Oxidative stress in mouse brain exposed to lead. *Ann Occup Hyg*. 2006. 50(4), 405-409. | 21. Balogun, S. K. Effect of Chronic Administration of Lead (Pb) on Aggressive Behaviour among Male Albino Wistar Rats. *Br J Arts and Social Sci*, 2012; 4, (2) , 150- 163. |