



Neurohistological Effects of Lead on Pons of Adult Albino Rat

Dr S M Dawar
Husain

MS (Anatomy), Associate Professor, Department of Anatomy, J N M C,
A M U Aligarh, U.P., INDIA

ABSTRACT

LEAD is toxic to every organ of body, including central nervous system. Present study is aimed to observe the histopathological changes in pons 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. Pons was dissected. 10 μ thick sections were obtained and were stained with Glees Silver stain. Under light microscope, pons from experimental group revealed disorganization of pontine cytoarchitecture with loss of direction of fibers, swollen tracts and disappearance of almost all of the pontine nuclei. It was concluded that lead has toxic effects on the central nervous system including pons which may explain the clinical manifestation of lead neurotoxicity

KEYWORDS : Albino rats, Pons, 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). 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 post-natally lead-exposed kittens has been reported (3) and decrease in maximum width of the hippocampus (4) have been noted. 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 gyri (6). Cadmium, another heavy metal has been reported to induce anosmia (7). Effect of Chronic Administration of Lead (Pb) on Aggressive Behaviour among Male Albino Wistar Rats has been reported (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 pons 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 cerebrum and processed for paraffin embedding. Then, 10 μ thick sections were cut with rotary microtome. These sections were stained with Glees stain and observed under the light microscope.

Observations

Pons of (control group) shows continuity of the well defined fibers, tracts and conspicuous pontine nuclei in high power (Figure 1). On examination, under the light microscope, the pons of the treated group showed almost total disorganization of pontine cytoarchitecture with breaking and loss of direction of darkly stained fibers, swelling of tracts and disappearance of almost all of the pontine nuclei in high power (Figure 2)

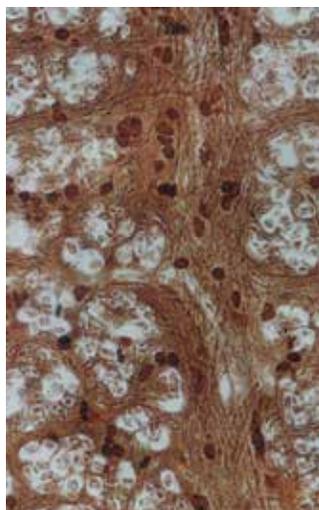


Fig -1 Pons of (control group) shows continuity of the well defined fibers, tracts and conspicuous pontine nuclei in high power (Glees Silver Stain)

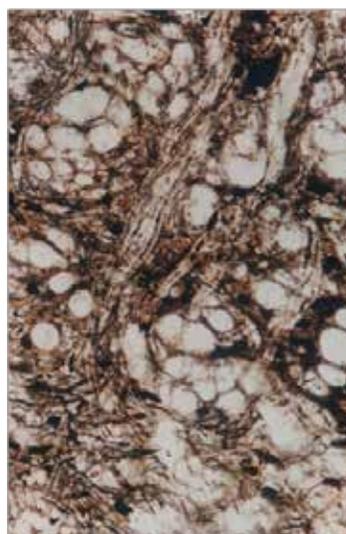


Fig -2 Pons of (experimental group) showed almost total disorganization of pontine cytoarchitecture with breaking and loss of direction

of darkly stained fibers, swelling of tracts and disappearance of almost all of the pontine nuclei in high power (Glees Silver Stain)

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of lead on the pons. 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 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). Inflammation-like glial response in lead-exposed immature rat brain has been shown (20) The histological findings observed in our study confirmed the pontine 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 degenerative microscopic alterations in the pons.

REFERENCES

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. 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. | 9. Carboni AA; Oral administration of zinc gluconate trihydrate. *Am J Rhinology*. 2006; 20, 262-268 | 10. Upadhya U; Olfactory loss as a result of toxic exposure. *Otolaryngo 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. Pizzol M, Thomsen M, and Andersen MS. Long-term human exposure to lead from different media and intake pathways. *Sci Total Environ*, 2010; 408(22), 5478-5488. | 18. Sidhu P and Nehru B Lead intoxication: Histological and oxidative damage in rat cerebrum and cerebellum. *J Trace Elem Exp Med*, 2004; 17, 45-53. | 19. Sanders T, Liu Y, Buchner V, and Sulkowski G. Neurotoxic effects and biomarkers of lead exposure: a review. *Rev Environ Health*, 2009; 24(1), 15-45. | 20. Struzynska L, Dabrowska-Bouta B, Koza K, and Sulkowski G. Inflammation-like glial response in lead-exposed immature rat brain. *Toxicol Sci*, 2007; 95(1), 156-162. | 21. Jean R; Harmful effects of cadmium on olfactory system in Mice. *Inhalation toxicology*. 2008; 20, 1169-1177 | 22. Jack H and Elva D, Lead and developmental neurotoxicity of the central nervous system, *Current Neurobiology* 2011; 2(1), 35-42 |