Thernational	Research Paper	Medical Science
	Neurohistopathologicaleffectsofmercury on Hippocampus of Adult Albimo Rat	
Brajesh Ranjan	Department of Anatomy, JN Medical College, Aligarh Muslim University, Aligarh,	
S. M. Dawar Husain	Department of Anatomy, JN Medical College, Aligarh Muslim University, Aligarh,	
S. M. Yunus	Department of Anatomy, JN Medical College, Aliga Aligarh,	arh Muslim University,
ABSTRACT Merce	ury is toxic to almost every organ of body, including central nervous s	ystem. The aim of present study is to

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 mercuric chloride in distilled water for aperiod of 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Hippocampus was dissected out. 10µ thick sections, obtained by usual histological procedure, were stained with H&E. On light microscopic observation, hippocampus from experimental group revealed increased cellularity of neurons with marked pyknosis of nuclei, increased cellularity with highly pleomorphic neuronal cells and markedly hyperchromatic nuclei in the molecular cell layer It was concluded that mercury has toxic effects on the central nervous system including hippocampus which may explain the clinical manifestation of mercury neurotoxicity.

KEYWORDS : Albino rats, hippocampus, mercuric chloride, neurotoxicity

Introduction

Mercury intoxication can occur more through inhalation rather than ingestion of contaminated foods and drinks.Deposition of mercury rich industrial waste in Japan, drew the attention of the scientific society when it causedtriggering signs and symptoms in the local population like ataxia, speech impairment, visual field constriction, sensory disturbance, deafness, blindness, tremors, involuntary movements, mental retardation, coma and death (1). Mercury is an occupational hazard for dental staff (2), chloralkali factory workers (3), goldminers (4) etc. Mothers consuming diet containing mercury pass the toxicant to foetus (5) and to infants through breast milk (6). Subtle neurological disorders in children over mercury exposure have been widely reported (7). Disruption of attention, fine motor function and verbal memory due to exposure to low mercury levels in fish eating adults, has been documented (8). Reports showed vacuoles in the white matter of cerebellum, pericellular as well as periventricular edema and congestion of blood vessels after treatment of rats with mercury (9). In another study, oral and subcutaneous administration of mercuric chloride for eleven weeks, caused degenerative changes in cerebellum of rat (10). The aim of present studywas to see the effect of mercury on the histology of hippocampus which may explain the clinical signs and symptoms following mercury intoxication.

Material and Method

20 adult albino rats (10 male and 10 female) weighing approximately 120g were used in the present study. 10 rats with equal number of either sex were treated with mercuric chloride (0.330 mg /kg body weight while the remaining 10 rats (5male and 5 female) served as control group and were given distilled water. Freshly prepared sterile solution of mercuric chloride in distilled water was used for oral administration as drinking water. This concentration was ascertained after a careful trial in order to find maximum survival days, which were 15 days. Then, rats were anaesthetized with ether and perfused with buffered 10% formalin. Brain was dissected out. 3mm thick coronally sliced pieces of hippocampus wereremoved and processed for paraffin embedding. Then, 10 μ thick sections were cut with rotary microtome. These sections were stained with H & E and observed under the light microscope.

Observations

Hjippocapus of control group shows the Normal distribution of the molecular and granular cell layer at 10x (Figure 1). Under the light microscope, the hippocampus of the treated group shows increased cellularity of neurons with marked pyknosis of nuclei in the molecu-

lar cell layer at 10x(Figure2). At higher magnification, hippocampus of experimental group shows increased cellularity and highly pleomorphicneuronal cells with markedly hyperchromatic nuclei in the molecular cell layer(Figure 3).



Figure:1. Photomicrograph of hjippocapus (control group) shows the Normal distribution of the molecular and granular cell layer. Haematoxylin and Eosin \times 10X.



Figure:2. Photomicrograph f hippocampus (experimental group) shows increased cellularity of Neurons with marked pyknosis of nuclei in the molecular cell layer. Haematoxylin and Eosin × 10X



Figure:3. Photomicrograph of hippocampus (experimental group) shows increased cellularity with highly pleomorphic neuronal cells with markedly hyperchromatic nuclei in the molecular cell layer. Haematoxylin and Eosin × 40 X.

Discussion

Histological findings in the present study were suggestive of neurotoxic and degenerative effects of mercury on the hippocampus. In one of the studies, cerebellum showed pyknosis of nuclei of granular layer cells, edema between granular and molecular layer, degeneration of many purkinie cells (11). Earlier, it was also reported thatmercury has neurotoxic effects reflected in its ability to penetrate and damage Blood Brain Barrier system (12), (13). Another study, reported pathological lesions in brain secondary to mercuric chloride poisoning (14). Thinning and depletion of granular layer of the cerebellum, after mercury toxicity, has been reported (15,16). Reports are available, showing degeneration and necrosis of purkinje cells and shrinkage of neurons in cerebellum secondary to mercury poisoning (17). Mercury compounds can induce cerebellar degeneration, sensorimotor and gating disturbances, tremor, ataxia and depression (18,19,20). The histopathological changes in the hippocampus were observed in our study indicating the neurotoxic effects of mercury poisoning and correlated very well with the histological findings of the other studies on the brain in general, though almost negligible material is available specifically in relation to histopathological changes of hippocampus secondary to mercury poisoning.

Conclusion

It is concluded that exposure of rat to mercury for 15 days producesdemonstrable microscopic alterations in the hippocampus.

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