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Research Paper

Medical Science

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Neurohistopathologicaleffects of Mercury on Cerebellum of Adult Albimo Rat

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ABSTRACT

Mercury is toxic to almost every organ of body, including central nervous system. The aim of present study is to observe the histopathological changes in the cerebellum of rat induced by oral administration of mercuric chloride 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. Cerebellum was dissected out. 10µ thick sections, obtained by usual histological procedure, were stained with H&E. On light microscopic observation, cerebellum from experimental group revealedmarked congestion of the blood capillaries with prominent perivascular fibrosis. Cerebellum of experimental group also showed spongiosis in the molecular layer and markedly increased cellular granular cell layer with pyknotic nucleus. Few purkinje cells could be seen.It was concluded that mercury has toxic effects on the central nervous system including cerebellum which may explain the clinical manifestation of mercury 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), chloralkalifactory 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 cerebellum which may explain the clinical signs and symptoms following mercury intoxication.

Material and Method

A total number of 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 10rats (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 cerebellum 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

On examination, under the light microscope,cerebellum (control group) shows normal 3 layers; Outer molecular, Inner extremely cellular granular cell layer and single layer of Purkinje cells in between the two layers. Haematoxylin and Eosin × 40X(Figure1).The cerebellum of experimental groupshowed marked congestion of the blood capillaries with prominent perivascular fibrosisat 40x (Figure2). Cerebellum of experimental group also showed spongiosis in the molecular layer and markedly increased cellular granular cell layer with pyknotic nucleus. Few purkinje cells can be noted at 40x(Figure 3).

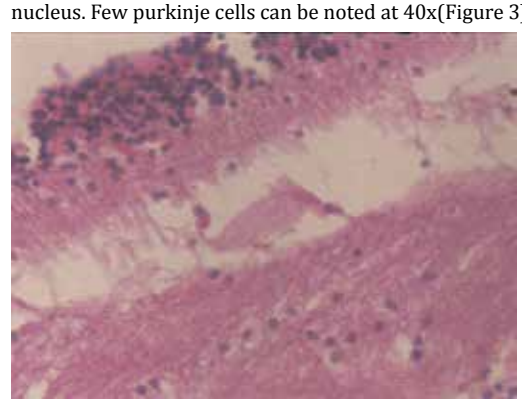

Figure:1. Photomicrograph of cerebellum (control group) shows normal 3 layers; Outer molecular, Inner extremely cellular granular cell layer and single layer of Purkinje cells in between the two layers. Haematoxylin and Eosin × 40X.

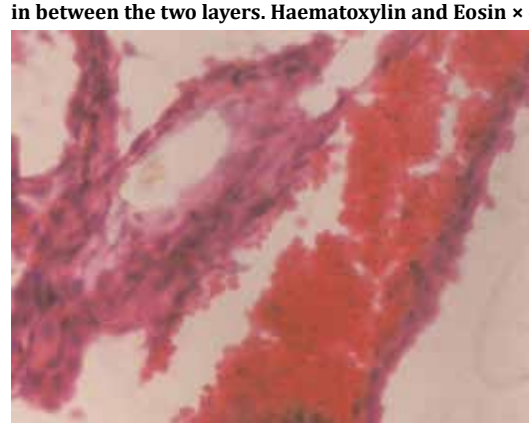

Figure: 2. Photomicrograph of cerebrum (experimental group) shows marked congestion of the blood capillaries with prominent perivascular fibrosis. Haematoxylin and Eosin × 40X.

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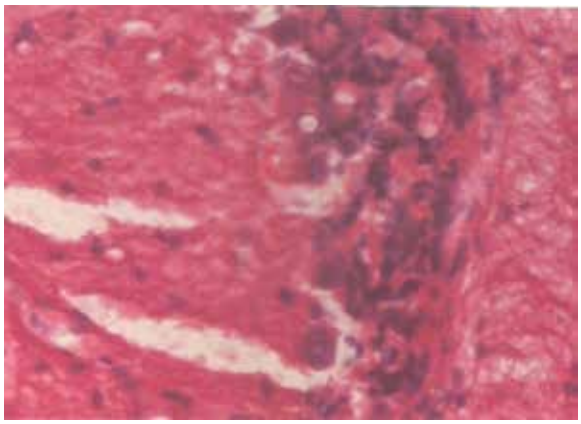

Figure: 3. Photomicrograph of cerebellum (experimental group) shows Spongiosis in the molecular layer and markedly increased cellular granular cell layer with pyknotic Nucleus. Few purkinje cells can be noted. Haematoxylin and Eosin × 40X.

Discussion

In the present study, histological findings were suggestive of neurotoxic and degenerative effects of mercury on the cerebellum. These findings are in conformity with other neurohistological studies. In one of the studies, cerebellum showed pyknosis of nuclei of granular layer cells, edema between granular and molecular layer, degeneration of many purkinje cells (11), these findings were found in the present study also. Earlier, It was also reported thatmercury has neurotoxic effects reflected in it's ability to penetrate and damage Blood Brain Barrier system (12), (13). Another study, reported pathological lesions in brain secondary to mercuric chloride poisoning (14).It has been documented that treatment of rats with mercuric chloride resulted in mild signs of neurotoxicity in the first two weeks of treatment, like congestion of blood vessels perivascular reaction in the cerebrum, cerebellar white matter showed multiple well defined vacuoles with purkinje cells remaining intact while in the third week, pericellular edema with ischemic injury to some neurons and beginning of purkinje cells damage were evident(15). Ithas also been reported that degeneration and necrosis ofpurkinje cells and neuronal shrinkage was due to direct toxic effect of the compound or the role of mercuric chloride as anoxic agent16). 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 histological findings observed in our study confirmed the cerebellar neurotoxicity following mercury poisoning and correlated very well with histological findings of the other studies.

Conclusion

Exposure of rat to mercury for 15 days producesdemonstrable microscopic alterations in the cerebellum.

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