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AMELIORATIVE EFFECT OF ASCORBIC ACID (VITAMIN C) ON MERCURY INDUCED TEMPORAL LOBE DAMAGE IN RATS

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ABSTRACT

Mercury is a heavy metal of known toxicity, noted for inducing public health disasters in Minamata Bay, Japan and in Iraq. The present study investigated the effect of ascorbic acid on the histology of mercury induced temporal lobe damage in Wistar rats. Twenty five adult Wistar rats (average weight 185 g) were randomly divided into five groups (each n = 5); a control group administered normal saline, mercuric chloride ($HgCl_2$; 49.8 mg/kg), $HgCl_2$ with distilled water, $HgCl_2$ with low dose ascorbic acid (595 mg/kg) and $HgCl_2$ with high dose ascorbic acid (1,190 mg/kg), groups, orally administered daily for three weeks. Results revealed alteration of temporal lobe histoarchitecture; neuronal degeneration, such as necrosis, clumping of cells, neuronal vacuolation, cytoplasmic shrinkage and reduction in the number of cells ($p \leq 0.05$) in $HgCl_2$ intoxicated group. The administration of ascorbic acid remarkably ameliorated $HgCl_2$ induced temporal lobe damage, notably with ascorbic acid 1,190 mg/kg treatment suggesting that ascorbic acid has neuroprotective potentials against $HgCl_2$ induced temporal lobe damage in Wistar rats.

Key words: *Temporal lobe, Histology, Mercuric Chloride, Ascorbic acid, Wistar rats*

INTRODUCTION

Man in his environment has been exposed to potential hazard of heavy metals through bio-accumulation and bio-magnifications, which has been transferred to man via air, water and food chain as a result of anthropogenic activities, mining of mercury, gold and metals including copper, zinc, lead and silver (Ghosh and Sil 2008; Burger et al. 2011). Many populations worldwide have been exposed to doses of mercury through the consumption of fishes and sea foods, and some have experienced neurotoxic effects (Valey et al. 1980; WHO 2003). Some population have experienced subsequent neurotoxic effects, and since the epidemic of mercury poisoning from contaminated fish consumption in Minamata, Japan in the late 1950s, mercury has been one of the most documented examples of bio-accumulation of toxins in the environment, particularly in the aquatic food chain (ATSDR 2011). The toxicity of mercury can also result from vapour inhalation and ingestion or absorption through the skin. However, diagnosis of mercury toxicity can be challenging because the commonly used modalities (blood, urine and hair levels) do not always correlate with total body burden and offer little diagnostically useful information. There are currently no consensus criteria for the diagnosis of mercury overload, nor for overload of other toxic metals (Bernhoft 2012). There is a growing appreciation of the effects that exposure to mercury has on the nervous system, because mercury crosses the

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