The Effects of Inorganic Mercury Intoxication on Atherosclerosis in Rats

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Abstract—Today many populations are exposed to multiple species of mercury. The reports indicate that inorganic mercury may have intoxication effect on heart. This study was exerted to determine the effect of inorganic mercury intoxication on atherosclerosis in rats. In this laboratory experimental study, rats were exposed to 5mg/kg, 10mg/kg, 15mg/kg and 20 mg/kg of mercury. After 28 days, tissues samples placed on lam and checked with microscope. The data was analyzed using ANOVA. Our data show that measure of atherosclerosis increased significantly when exposed to 15 mg/kg and 20 mg/kg of inorganic mercury (P<0.01). Other doses of inorganic mercury had no significant effect on intoxication in atherosclerosis in rats. Our findings show that high dose of inorganic mercury has intoxication effects on atherosclerosis in rats.

Index Terms— Inorganic mercury, intoxication, atherosclerosis.

I. INTRODUCTION

Many population are exposed to multiple species of mercury [1] Inorganic mercury compounds exist in two oxidative states (mercuric, Hg⁺; mercuric, Hg²⁺), which are generally in solid states as mercurous or mercuric salts and mercury compounds with chlorine, sulfur, or oxygen [2]. The known intoxications are mainly occupational (mining, agriculture, incineration) or related to the use of dental amalgams or the consumption of contaminated fish and shellfish [3]. Human toxicity varies with the form of mercury, the dose and the rate of exposure. The target organ for inhaled mercury vapor is primarily the brain. Mercurous and mercuric salts chiefly damage the gut lining and kidney, while methyl mercury is widely distributed throughout the body. Toxicity varies with dosage: large acute exposures to elemental mercury vapor induce severe pneumonitis, which in extreme cases can be fatal. Low-grade chronic exposure to elemental or other forms of mercury induces subtler symptoms and clinical findings, as discussed hereinafter [4]. Atherosclerosis is hypercholesterolemia, as excess cholesterol initiates foam cell formation in the arterial wall. Subsequently, inflammatory processes including recruitment of innate and adaptive immune cells contribute to further development of the plaque, which may eventually lead to plaque rupture and/or vessel occlusion [5] Most of the damage occurs when plaques become fragile and rupture. Plaques that rupture cause the formation of blood clots that can block blood flow or break off and travel to another part of the body. In either of these cases, if a clot blocks a blood vessel that feeds the heart, it causes a heart attack. If it blocks a blood vessel that feeds the brain, it causes a stroke. If blood supply to the arms or legs is reduced or blocked, it can cause difficulty walking and eventually gangrene. Atherothrombotic stroke is the most common stroke. It occurs when a blood clot forms on a atherosclerotic plaque within a blood vessel in the brain and blocks blood flow to that part of the brain [6].

Studies show that Human exposure to mercury has a very long history. Several thousand years ago in China, an inorganic mercury compound (mercury sulfide) was used to prepare red dye pigment vermilion, which is a brilliant red ore, namely cinnabar. Inorganic mercury compounds have also been used in medical applications since ancient Egypt. [7] On the other hand researches show that some doses of inorganic mercury have intoxication effect on heart diseases [8]-[14]. This study was exerted to determine the effect of inorganic mercury intoxication on atherosclerosis in rats.

II. MATERIAL AND METHODS

In this laboratory experimental study, white male rats was prepared from Pasteur research complex and different doses of mercury (5mg/kg, 10mg/kg, 15mg/kg and 20mg/kg) were used in our study.

Briefly, the procedure was continued and carried out in the following steps:

Mercury was injected to rats until 28 days. After this 28days period, that rats were anesthetized with chloroform and blood samples were taken by syringe 10ml from the right atrium animal’s heart and samples transferred to container. After washing samples, heart and aorta were placed in 10% formalin in samples container and then samples are cut for molding and were placed within liquid paraffin and after samples molding, samples are ready for tissues cutting. After removal of the samples, they are cut by microtome devices and placed into hot water and after this stage, samples placed on lam and coloring by eosin _ Haematoxylin color and checked our tissue with microscope.

Statistical significance was evaluated b one-way analysis variance (ANOVA) using SPSS 16 . Differences with P<0.01 were considered significant.

III. RESULTS

Figure I shows the atherosclerosis in response to different doses of inorganic mercury. Our finding show that atherosclerosis increases significantly in animals receiving 10
mg/kg (P<0.05), 15 or 20 mg/kg (P<0.01) of mercury compared with control group.

![Graph showing atherosclerosis in different groups of rats](image)

IV. DISCUSSION

We have shown that high dose of inorganic mercury intoxication has increasing effect on atherosclerosis. In line with our finding there are also other records indicating that inorganic mercury may have atherosclerosis effects. Studies also suggest that Patients with Minamata disease and hair mercury levels above 100 μg per gram did not have a higher rate of death from heart disease than controls, nor did they have a higher degree of arteriosclerosis [15-16]. However, it has been shown that Guallar et al. (Nov. 28 issue) report that a toenail mercury level as low as 0.11 to 0.66 μg per gram (estimated hair level, 0.34 to 2.03 μg per gram) was directly associated with a doubling of the risk of myocardial infarction [12].

The cardiovascular effects of cadmium have been observed in vitro studies and in experimental animal models [17-18]. Mercury-induced oxidative damage has been observed both in vivo and in vitro, including myocardial tissues. The mechanisms by which mercury exerts its cardiovascular effects are not fully understood. However, exposure to mercury can lead to oxidative stress induction [19]. The combination of high mercury and low selenium was not associated with higher CVD risk [20]. Toxic effects of mercury also induce a number of stress proteins which include heat shock proteins and glucose-regulated proteins that have also been implicated in cardiovascular pathophysiology [21].

V. CONCLUSION

We have shown that high dose of inorganic mercury has intoxication effect on atherosclerosis in rats.

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REFERENCES


