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Toxic Effects of Lead Acetate on Liver

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ABSTRACT

Lead is an environmental pollutant and has detrimental effects on human health. Lead can be used in various industries including lead acid batteries production, plumbing, solder, and cosmetics, and gasoline, etc. In this study, the effect of two different sub-toxic doses of lead acetate on liver function and histological modification in liver tissues was evaluated. Male albino rats were divided into four groups, the first group represented the health control animals, while the second, third and fourth groups were ingested orally with sub lethal doses of lead acetate (1/20, 1/40 and 1/60) of the oral LD_{50}, respectively. One dose was ingested every two days during the experimental period (14 weeks) including the adaptation time. Blood was collected and used for all analysis. The results showed that, the ingestion of Pb^{2+} induced significant stimulation in glutamic-pyruvic transaminase (ALT) and glutamic-oxalacetic transaminease (AST) activity. Also, total soluble protein and albumin contents of plasma were significantly decreased, while the content of globulin was changed by the Pb^{2+} treatments. The cholinesterase activity was inhibited, but the activities of alkaline and acid phosphates and lactate dehydrogenase were stimulated, while plasma glucose level was elevated as a result of lead acetate intoxication. In case of blood picture, Pb^{2+} ingestion reduced the contents of hemoglobin and RBCs count of intoxicated rat's blood and the plasma levels of T3, T4 and blood WBCs count were decreased. It can be concluded that lead acetate has harmful effect on experimental male albino rats. Therefore, the present work advises people to prevent exposure to the lead compound to avoid injurious hazard risk.

KEYWORDS: *Lead, Toxicity, ALT, AST, Cholinesterase, T3, T4, Lead acetate, Intoxication, Hemoglobin*

INTRODUCTION

Lead in the environment is considered one of the most dangerous poisons and pollutants [1]. Lead is so toxic element, and occupational exposure to lead defined by Hippocrates and Nikander more than 2000 years ago [2]. **Lead toxicity** appears in multiple forms of neurological and intestinal signs. Hence, depending on the duration and level of exposure to this substance causes both acute toxicity and chronic toxicity [3]. The assessments revealed that the liver of human who are exposed to lead is the biggest recipient compared with the other members. Also it found in treated laboratory animals that lead accumulates in shell of kidney and medulla. As it was found that exposure to lead in the environment increases the toxic influences on different members of the body. Reports in both animals and humans showing that lead has toxic influences on the bone, kidney, liver, lung, blood, heart, finally the testis and brain [4]. It considers paints containing lead are common sources of lead in animals. There is considerable variation in susceptibility for different species to be affected by lead or materials containing lead, which may affect on its toxicity. Hence, lead toxicity varies due to its chemical form. Solid lead sheeting and insoluble lead oxides less toxic than soluble lead acetate. except the bone where the lead is remains an inert form, It is known that lead dose not remain in the tissue for a long time and the form which is freed

from lead in bone later be enough to cause chronic lead poisoning [5]. It is known that people who are exposed to heavy metals suffer from both hepatic and renal toxicity. Previous autopsy studies in the individuals which exposed to lead indicated that the liver is the largest repository of lead 33 %, followed by the kidney cortex and medulla between the rest of the soft tissues

In general, **Acute poisoning** usually occurs with a high dosage of lead acetate. It starts with burning and dryness in the throat, salivation, and intense thirst. Vomiting occurs within 24 h, with colicky pain and a tender abdomen. Constipation is a common feature and urine is scanty. Finally, there may be peripheral circulatory collapse, headache, insomnia, paresthesia, depression, convulsions, exhaustion, and coma leading to death. **Subacute poisoning** occurs from repeated small doses of lead acetate. A blue line is seen on the gums, along with GI symptoms. Urine is scanty and deep red in color. During the later stages, nervous symptoms become prominent, with numbness, cramps, and flaccid paralysis of the lower limbs. Death is rare, but it may occur after convulsions and coma. **Chronic poisoning** with lead compounds may lead to the following:

Facial Pallor: Pallor that is seen especially around the mouth. It is also known as circum oral pallor and is due to the vasospasm of the capillaries and arterioles around the mouth.

***Anemia*:** Hypochromic, microcytic anemia with reticulocytosis and
punctuate basophilia with the presence of marked basophilic stippling in the RBCs. Platelet count decreases. Anemia is probably due to decreased survival time of RBCs and inhibition of heme synthesis by interference with the incorporation of iron into protoporphyrin.

***Burtonian line (lead line)*:** A stippled blue line seen at the junction of the gums, usually nearer to tooth caries, especially in the upper jaw. This is due to the deposition of lead sulfide formed by the action of the combination of lead sulfide formed by the action of the combination of lead with hydrogen sulfide, which evolved from the decomposed food debris in the caries tooth.

Lead colic and constipation: The victim will report severe colicky pain in the abdomen relieved by pressure and bowel irregularities. Abdominal muscles become tense and retracted.

***Lead palsy*:** A typical paralysis affecting the extensor muscles of the fingers and wrist causing wrist drop and claw-shaped hand. Similarly, paralysis may extend to the extensor muscles of the foot, leading to foot drop.

Lead encephalopathy: Mostly seen in infants presenting with severe ataxia, vomiting, lethargy, stupor, convulsion, and coma; cerebral psychic effects may be present.

***Cardiorenal manifestations*:** Elevated blood pressure and arteriosclerotic changes are observed. Urine contains albumin and an abnormal quantity of lead, coproporphyrin III, and delta amino laevulinic acid. Interstitial nephritis may occur.

Sterility/infertility: Both may be observed.

General manifestations: These include weakness, anorexia, metallic taste in the mouth, dyspepsia, and foul breath.

DISCUSSION

In animals Acute lead exposure can lead to renal toxicity. The acute intraperitoneal LD_{50} for lead acetate in rodents is 100–200 mg kg^{-1}. Lead acetate is considerably less toxic by the oral route (LD_{50} 44 g kg^{-1} in rats). The acute oral LD_{50} of tetraethyl lead in rodents is 10–100 mg kg^{-1}. Acute organo-lead exposure can sensitize dopaminergic neurotransmission in the central nervous system.

In humans Anorexia, vomiting, malaise, and convulsions (caused by increased intracranial pressure) are most commonly seen in children. Sources of childhood exposures are typically environmental such as to paint chips, pottery, drinking water, and dust. Acute exposure in adults may cause gastrointestinal effects, pain in arms and legs, and hypertension. Exposure to very high levels may cause tremor, memory loss, confusion, stupor, renal failure, convulsions, and coma.

Animal experiments have shown a tumorigenic effect of lead (Silbergeld *et al.*, 2000). Hence, soluble lead salts, such as lead acetate and subacetate, have produced kidney and brain tumors, and lead phosphate kidney tumors, in rodents after oral or parenteral administration. Synergistic effects exist for the development of cancer between lead acetate and oxide, on the one hand, and some organic carcinogens, such as benzo(a)pyrene and nitrosamines, on the other.

In a series of epidemiological studies, lead workers had increased risks of total, kidney, lung, or stomach cancers [6] Also, there is limited support for a synergism with other carcinogens .The studied cohorts have had a high exposure

(mean B-Pb >3 µmol/L), at least in most of the studies [7].

Animal experiments have shown a tumorigenic effect of lead [8]. Hence, soluble lead salts, such as lead acetate and subacetate, have induced kidney and brain tumors, and lead phosphate has induced kidney tumors, in rodents after oral or parenteral administration. There are synergistic effects on the development of cancer between lead acetate and oxide, on the one hand, and some organic carcinogens, such as benzo(a)pyrene and nitrosamines, on the other. In a series of epidemiological studies, lead workers had increased risks of total, kidney, lung, or stomach cancers [9]. Cohorts have had a high exposure levels (mean B-Pb > 3 µmol/L), at least in most of the studies .In a recent, large case-referent study of renal cell carcinomas in the Czech Republic, Poland, Romania, and Russia, there was an increased adjusted risk associated with occupational lead exposure, as assessed by industrial hygienists on basis of a questionnaire (highest exposure category: OR 2.25, 95% CI 1.21-4.19) [10].

However, the pattern of carcinogenesis in humans is not consistent [11]. Furthermore, there are major problems in terms of confounding (e.g. regarding coexposure to arsenic and cadmium in the occupational cohorts). There may also be selection bias. Hence, lead workers may differ in many ways besides lead exposure. In particular, confounding by smoking is a problem that has only occasionally been tackled. There may also be differences in physical fitness and diet.

RESULTS

It is possible that *ALAD* polymorphism affects the risk of renal cell cancer. Thus, the risk associated with occupational lead exposure was highest among subjects who had heterogenous or homogenous variants of the *ALAD1* polymorphism (van Bemmel et al., 2011a). In a case-referent study, there were some indications that the risk of meningiomas increased with rising estimated (questionnaire data) occupational lead exposure (µg/m^3 × years) in *ALAD2* carriers; for gliomas, there was no interaction [12]. It has been claimed that polymorphisms in genes encoding enzymes that protect against oxidative stress modify the risk of brain tumors [6].

CONCLUSION

The liver histopathological changes in mice which treated with 18mg/kg of lead acetate shows mild degeneration, lobular disarrangement with congestion and mild chronic inflammation for one week from treated while the result for two weeks from treated shows mild congestion, lobular disarrangement and mild-moderate chronic inflammation. Also, shows lobular disarrangement, congestion, moderate portal and lobular hepatitis, these changes in liver mice treated with LA for three weeks compared with the normal state in control group reported that the acute treatment with lead for one week led to severe damage in liver cells, absence of arrangement of the hepatic cells and expansion of blood sinusoids.

Chronic exposure to lead leads to appearance of inflammatory cells in liver tissue, perhaps as a result to interact lead with enzymes and proteins of liver tissue, interfering with the mechanism of antioxidant defense to produce a traditional inflammatory response as a result of the generation of reactive oxygen species(ROS). Pathological changes in the liver tissue could be as a result to lead action on the content of DNA, liver glycogen and portability of lead to convert amino acids into proteins. Recent studies have suggested that the reason of the pathophysiology changes in the liver may due to oxidative stress, or programmed cell death (apoptosis).[12]

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