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Evaluation of the effect of lead exposure on the liver in Egyptian lead tank welders


With 2 tables

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Chronic kidney disease and arteriosclerosis in lead workers of many years standing was believed by the early observers to be a direct though delayed consequence of their occupational exposure (4). In experimental lead poisoning Yung Han Hsu (46) suggested that lead poisoning would cause hypertension before arteriosclerosis. Arteriosclerosis was most apparent in heart and brain, then spleen and adrenal glands. However, a group of 50 workers who were occupationally exposed to lead for 5 years showed no clinical abnormalities or serum lipid values which would indicate the premature development of atherosclerosis (32). Certain reports have been published also on the effect of lead on the liver, however, the opinions of authors vary greatly, while some of them cast doubt on the hepatotoxicity of lead in industrially exposed workers (30, 40).

Others reported their investigations showing a toxic effect of lead upon the liver (6). These differences in opinions stimulated us to study the changes in serum lipids and some of the liver function tests which may elucidate the effect of lead on the liver in a group of Egyptian lead tank welders who were exposed to lead fumes for periods up to 22 years.

Materials and methods

The material of the present study is composed of a group of 16 lead tank welders who were admitted to our Institute for periodical medical examination. Their age ranged between 28 and 50 years and they were exposed to lead fumes for periods up to 22 years. They were clinically free from any sign or symptom of lead poisoning and also free from any parasitic infestation. Their medical reports revealed no present or past history of liver disease. A control group of 10 healthy workers who have never been exposed to lead and are of the same social class were similarly investigated.

The following investigations were carried out on both exposed and control groups.

Determination of whole blood lead as an index of lead exposure and absorption, blood haemoglobin level and urinary delta amino levulinic acid (ALA).

Determination of serum glutamic oxaloacetic transaminase (GOT) serum glutamic pyruvic transaminase (GPT), alkaline phosphatase (Alk phosph) and lactic dehydrogenase (LDH), as well as total bilirubin, total protein and albumin fraction.
Serum total lipid, triglycerides, cholesterol, phospholipid and lipoprotein pattern were also determined.

The method of Keenan et al. (19) for the determination of lead in blood has been applied in the present work. Urinary ALA determination was carried out according to the method of Grabeeki et al. (12). The acid haematin method of Sahli (35) was used for determination of blood haemoglobin. Concerning the serum enzymes, the method of Reitman and Frankel (33) was used for determination of serum glutamic pyruvic and glutamic oxalacetic transaminases. Serum alkaline phosphatase was estimated by the method of King and Armstrong (20). The method of Wroblewski (45) was used to determine serum lactic dehydrogenase activity. Total serum proteins were determined by the biuret method and electrophoretic separation of serum proteins was done according to King and Wootton (21).

Lipids were extracted from plasma according to the method of Folch et al. (9). The phase containing lipid was evaporated to dryness. The lipid was redissolved in chloroform and aliquots were used for further analysis. Triglycerides were determined by the method of Van Handel and Zilversmit (41). An aliquot of the extract was taken for phospholipid analysis. Inorganic phosphate was determined by the method of Morrison (31). Total cholesterol in plasma was measured by the method of Bloor (3). And total lipid was measured according to the method of Swahn (36).

Results and discussion

Lead poisoning is a syndrome caused by the toxic action of lead which may be seen in people whose tissues contain higher than normal amounts of lead. Lead poisoning may occur by ingestion or by inhalation of lead dust or fumes. The metabolism of lead follows closely that of calcium particularly with regard to its deposition in and mobilization from bone (4). A relatively high lead content was also found in the liver of patients suffering from acute manifestations of lead poisoning (39).

Lead in common with other heavy metals has a variety of toxic actions on protoplasm; the most precisely described are on certain enzyme systems and on cell membrane (5).

In rats poisoned with lead acetate ultrastructural changes in liver cells indicated a disequilibration of metabolic processes. Mitochondria and cytoplasm were mainly affected with marked myelinic degeneration of cytoplasm (13).

In the present work the absence of clinical abnormalities in the exposed group shows that the exposure has not reached a dangerous level. However, the danger of continual absorption of lead in amounts which do not of themselves cause clinical symptoms and signs of poisoning is that a point may be reached when the “threshold level” for potential poisoning can be exceeded and/or intercurrent factors causing lead mobilization from bone may cause a sudden outbreak of clinical symptoms of lead poisoning (4). Estimation of the level at which lead absorption has become potentially dangerous usually depends upon examination of the blood or anaemia, for a raised lead content and the urine for increased excretion of delta amino levulinic acid (ALA). It was shown that the blood lead level is probably the most valuable indication of excessive lead absorption (28).

It is shown in table 1 that the lead level in blood of the present exposed group was significantly increased and the blood haemoglobin level was significantly more decreased than the control values.