LEAD POISONING AND ALCOHOL INGESTION

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Keywords: *metabolism and toxicity of lead, experimental evidences, lead poisoning, alcohol ingestion*

Abstract: *Lead exposure is one of the primary concerns in most of the industrial developing countries. Metabolism and toxicity of lead may be influenced by alcoholism. Experimental evidences suggest that ethanol could enhance the absorption of lead in the body and alcoholics may be more susceptible to lead poisoning. There are a number of clinical evidences which suggest alcoholism as one of the possible factors responsible for determining individual susceptibility to lead poisoning. In time, the number of recent investigations clearly suggested the modulatory role of ethanol on lead toxicity. As alcoholism is common among the industry workers and the general population who may be exposed to lead, the present article discusses about the influence of alcohol ingestion on the metabolism and toxicity of lead.*

Cuvinte cheie: *metabolismul şi toxicitatea plumbului, dovezi experimentale, intoxicaţia cronică cu plumb, consumul cronic de alcool*

Rezumat: *Expunerea cronică la plumb este una dintre principalele preocupări în cele mai multe ţări industrializate sau în curs de dezvoltare. Metabolismul şi toxicitatea plumbului poate fi influenţată de alcoolism. Dovezi experimentale sugerează că etanolul ar putea spori absorbţia plumbului în organism şi că cei care consumă cronic alcool pot fi mai sensibili la intoxicaţia cronică cu plumb anorganic. Există un număr de dovezi clinice care sugerează că alcoolismul este unul dintre factorii posibili responsabili pentru determinarea susceptibilităţii individuale la intoxicaţia cu plumb. În timp, numărul de investigaţii care sugerează în mod clar rolul etanolului cu privire la toxicitatea plumbului au crescut considerabil. Dramatic este că alcoolism este un viciu comun în rândul muncitorilor, dar şi în rândul populaţiei generale care poate fi expusă la plumb, de aceea prezentul articol abordează aspecte despre influenţa consumului cronic de alcool asupra metabolismului şi toxicologiei plumbului.*

INTRODUCTION

It has been known for a long time that the blood lead level is high in alcoholics. In 1967, Gounelle (1) found that the EDTA body burden chelatate lead was higher in alcoholics than in the control batches. He related the findings to the high lead contents of several samples of urine in which the lead concentration reached 230 µg/l. Different factors contributed to these high values: fermentation of vats, brittle capsules, contamination of the grapes by dust containing lead. Subsequent studies confirmed this finding.(2,3)

Vives (4) found that in alcoholics, the blood lead level can be as high as 69 µg/100 ml, a value considered above the tolerable range by the occupational physician. A decrease was observed after a period of abstinence. On the other hand, the blood lead level was in the normal range in the patients with non-alcoholic hepatic disease, indicating that the high blood lead level seemed to be related to alcoholism and not to liver disease. In a group of 1052 residents near Copenhagen, a significant correlation was found between blood lead level and tobacco or alcohol habit.(5) One ethanol unit (1.35 cl) raised the blood lead level by 1 µg/100 ml and ethanol influenced the blood lead level more than tobacco.

Delta-aminolevulinic acid dehydratase (ALAD), a sensitive indicator of lead exposure was found to be lower in alcoholics and this drop was correlated to the blood lead level but abnormal values were found only in the patients suffering from liver disease.(6)

Dally et al (7) studied 161 alcoholics who had a daily intake of ethanol higher than 1 g/kg/day. The mean blood lead level was 28 µg/100 ml and was as high as 72,5 µg/100 ml in one subject. Wine drinkers had a higher blood lead level (29,8 µg/100 ml) than beer or spirit drinkers (23,8 µg/100 ml). A significant correlation was found between the blood lead level and systolic and diastolic arterial blood pressure and this relation was independent of sex, weight or age. Shaper (8) also found a correlation between blood lead level and alcohol consumption. However, most of the subjects were beer drinkers and the lead in the beer was as low as 20 µq/l, a very low value compared to the acceptable lead intake of 3 mg a week, as proposed by the WHO. The high blood lead level found in these heavy drinkers was possibly related to other mechanisms like hepatic disturbances.

Despite this clinical association between lead poisoning and anhydrous alcohol ingestion, there are still relatively few studies available on the direct effects of ethanol on lead induced changes. Despite these interesting clinical studies, there was not much evidence provided in these studies about the possible source of lead contamination/uptake or even most of the clinical lead sensitive variables were not studied in these reports, which could have provided a definite role of ethanol ingestion in modifying lead intoxication or suggesting the exact mode of interaction between the two toxins.

It is clearly evident from the above studies that alcohol use among humans is associated with elevated blood lead concentration.

Mahaffey et al (9) first studied the influence of ethanol on tissue contents of lead and measures of lead toxicity in experimental animals given controlled diet/ nutrients. Combined exposure to ethanol (10% in drinking water) with lead (200 µg in 10% ethanol) for 10 weeks did not result in substantially higher lead levels in the kidneys, however, there was an increase in blood, liver and bone lead contents. As the study was conducted under controlled nutrient intake, not much conclusion was drawn about the mechanism of interaction or even whether

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ACTA MEDICA TRANSILVANICA March 2010; 2(1):207-209

ethanol interacts with lead, directly or due to certain other factors.

But the study definitely provided the first experimental evidence about the interaction between alcohol consumption and lead poisoning. Experiments with rats fed isocaloric diets and controlled nutritional contents suggest that clinically suspected synergism between ethanol consumption and lead intoxication observed among industry workers is more likely due to nutritional factors than the mutual enhancement of the two toxins.(9) In addition to studies showing that combined exposure to ethanol and lead augment disturbances in hepatic function.(10,11,12) and vasoregulation,(13) investigation of neurotoxicity has shown that co-exposure causes significant increase in dopamine (DA) contents of rat striatum, as well as an increase in 5 hydroxytryptamine (5-HT) content of the hypothalamus. It is clear that ethanol alters the effect of lead poisoning, it is equally apparent that the reverse is also true.

Flora and Tandon (10) provided experimental evidence that interactive effects between lead and ethanol appear to be on variables that are primarily related to lead toxicity, as well as on those more characteristic of ethanol toxicity, for example combined lead ethanol exposure produced more pronounced alterations in intermediary metabolism enzymes of ethanol, namely alcohol and aldehyde dehydrogenase.

Thus, it was suggested that ethanol exposure may potentiate the toxic effects of lead, as well as the vice versa.

Also, further reports showed that chronic alcohol consumption produces a depletion of body calcium and magnesium in experimental animals. However, coexposure to ethanol and lead produced a more pronounced depletion of these essential metals and this could be the likely cause of enhanced lead toxicity.(14,15,16,17)

There was another study on the effects of chronic lead exposure on some hematopoietic and hepatic biochemical indices and urine, faeces and tissue essential metal concentration in the rats pre-exposed to different doses of ethanol. At the same time, the chronic pre-ethanol ingestion produces the inhibition of blood delta- aminolevulinic acid dehydratrase (ALAD), the decrease in hepatic glutathione concentration and the elevation in hepatic lipid peroxidation level. Toxic effects of ethanol on blood ALAD activity were the result of its conversion into acetaldehyde which seemed to be the true inhibitor of ALAD. (18) Administration of ethanol produced a significant depletion of blood Ca, hepatic Ca and Mg and pronounced elevation of urinary Ca and faecal Ca and Mg particularly at the higher dose. (19) The finding also confirmed our earlier observation that depletion of blood Ca and Mg is the likely cause of suspected synergism between lead poisoning and alcohol consumption. These results were further confirmed by Dhawan et at (12) where lead exposed animals pre-exposed to ethanol showed comparatively more severe signs of plumbism along with serious depletion of body Ca and Mg contents.(20)

Evis et al (21) demonstrated the effect of chronic (3 or 10 months) administration of lead and ethanol altered the susceptibility of heart to arrhythmias induced, either by coronary artery occlusion, or noradrenalin infusion in pentobarbitone anesthetized rats. It is worthwhile to indicate that chronic exposure to high level of ethanol may result in cardiac arrhythmias in man (22) and indeed it may be a contributory factor in sudden cardiac death.(23)

A constant monitoring of metal industrial workers is also recommended, particularly in reference to their dietary habits. More detailed epidemiological studies are still lacking particularly when general population is constantly exposed to environmental lead pollution.

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