ORIGINAL RESEARCH

A study on circulating Plasma Lipid Peroxide and Blood Lead Level in Battery workers in Dehradun District of Uttarakhand

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ABSTRACT
Aim: A study on circulating Plasma Lipid Peroxide and Blood Lead Level in Battery workers in Dehradun District of Uttarakhand. **Material and methods:** A total no of 150 subjects (100 battery workers and 50 controls) was participated in the study. Blood samples was collected from the participants by aseptic technique. 5ml blood will be collected and will quickly transferred to test tubes already containing EDTA anticoagulant or plane vials. Blood lead level estimated by lead care blood lead analyzer. The lipid per oxidation was estimated by the method of Okhawa et. al. by measuring the malondialdehyde (MDA) level. **Results:** The mean age of the study group and control group was 42.85±5.85 and 41.85±4.66, respectively. The mean duration of exposure in the study group was 12.52±2.58 years. Individuals who were consistently exposed to lead had significantly greater amounts of lead in their blood compared to the control group that was not exposed to lead, with mean values of 14.25±2.52 mg/dL and 2.01±0.33 mg/dL, respectively ($p < 0.001$). In the study group, the average concentration of Plasma Lipid Peroxide was found to be 23.52±4.85 mg/L, whereas in the control group, it was seen to be 10.25±2.87 mg/L. **Conclusion:** There was a statistically significant positive association seen between blood lead levels and both age and length of exposure in both groups. However, no link was found between blood lead levels and related Plasma Lipid Peroxide.

Keywords: Plasma Lipid Peroxide, Blood Lead, Battery workers

INTRODUCTION

Lead poisoning is also called 'plumbism'. This is because of the ill effect of lead and lead-containing materials on various organs. Lead gets absorbed through lungs, intestine and skin. Blood lead level rise rapidly after a recent exposure. The World Health Organization (WHO) estimates that 1.2 crore people are over exposed to lead and 99% of the most serious cases are in the developing world. The absorption, storage and excretion of lead, modify the blood lead concentration in the body and therefore its effects. It gets accumulated in red blood cells (RBCs) and other organs. Lead enters the fetus from the mothers' blood. Once it gets accumulated in the brain, it cannot be removed. Lead is also stored in bones for a long period[1,2]. This study will be conduct to determine the circulating plasma lipid peroxide, blood lead levels and its association with liver function test in traffic police working in Dehradun & Haridwar city.

Lipid peroxidation in tissues and in tissue fractions represents a degradative process which is the consequence of the production and the propagation of free radical reactions primarily involving membrane polyunsaturated fatty acids (PUFA), especially arachidonic acid. The peroxidative breakdown of PUFA has been implicated in the pathogenesis of many types of liver injury and especially in the hepatic damage induced by several toxic substances. Among these are the halo-alkanes, carbon tetrachloride, trichlorobromomethane, chloroform, 1,2- dibromoethane and halothane; in addition, paracetamol bromobenzene, iron, bipyridyl compounds, allyl alcohol and in some instances,

ethanol have been shown to stimulate lipid peroxidation[3-6].

The stimulation of lipid peroxidation in either artificial membranes of liposomes or in subcellular organelles has been shown to increase membrane rigidity [7]. Such a loss of fluidity seems not to be dependent upon an increase jn the ratio between cholesterol and phospholipids but is rather an effect of the formation of cross-linking between acyl chains and of the depletion of long chain polyenoic fatty acids. In addition to the changes in fluidity, lipid peroxidation causes an increase in the ionic permeability and affects the surface potentials of the membranes. In the liver, the membranes of mitochondria and endoplasmic reticulum vesicles, contain unsaturated fatty acids in high proportion and therefore are vulnerable to peroxidative attack. At the same time they contain enzymes of the electron transport systems which make them capable of producing free radical species The biochemical alterations induced by lipid peroxidation in the endoplasmic reticulum of the liver have been extensively studied. Early reports showed that isolated microsomal vesicles undergo swelling and rough endoplasmic membranes lose bound ribosomes when exposed to peroxidizing agents [8]. Further studies using electron microscopy of negatively stained or freeze-fractured microsomes treated with CC14 have revealed membrane breakages and loss of intramembranous particles [8].

MATERIAL AND METHODS

This prospective study was conducted in the department of Biochemistry, Government Doon Medical college & Hospital Dehradun, Uttarakhand. A total no of 150 subjects (100 battery workers and 50 controls) was participated in the study. The subjects shall be taken in 22 to 60 years of age group and consent was obtained from all the participants. They were asked to fill a questionnaire which was included the details regarding their education, dietary habits, drinking water supply, type of housing, residing locality, medical history including use of over the counter or any other medication especially ayurvedic/herbal medications. Blood samples was collected from the participants by aseptic technique. 5ml blood will be collected and will quickly transferred to test tubes already containing EDTA anticoagulant or plane vials. Blood lead level estimated by lead care blood lead analyzer (Magellan Diagnostics USA, the lead care II system). The lipid per oxidation was estimated by the method of Okhawa et. al. by measuring the malondialdehyde (MDA) level.

RESULTS

Table 1 illustrates that the majority of the participants consisted of males, accounting for 80% of the total, while females constituted 20%. The majority of participants fell within the age range of 30-40 years, including 36.60% of the total sample. This was followed by those aged 40-50 years, accounting for 26.67% of participants. Those below the age of 30 constituted 19.33% of the sample, while individuals beyond the age of 50 included 17.33%. The majority of the participants have achieved a level of up to 10.A total of 82.67% of the participants were identified as residing in rural areas. The demographic profiles did not provide statistically meaningful results.

Table 1: Demographic profile of the participants

	Number =150	Percentage	P value
Gender			0.63
Male	120	80	
Female	30	20	
Age			0.47
below 30	29	19.33	
30-40	55	36.6	
40-50	40	26.67	
above 50	26	17.33	
Education status			0.41
up to 10^{th}	77	51.33	
up to 12^{th}	51	34	
Graduate or above	22	14.67	
Residential area			0.41
Rural	124	82.67	
Urban	26	17.33	
Co morbidity			0.19
Yes	67	44.67	
No	83	55.33	

Table 2 presents the lipid peroxide levels observed in plasma, as well as the amounts of lead, and the ages and duration of exposure for the participants who were exposed. The mean age of the study group and

control group was 42.85±5.85 and 41.85±4.66, respectively. The mean duration of exposure in the study group was 12.52±2.58 years. Individuals who were consistently exposed to lead had significantly greater amounts of lead in their blood compared to the control group that was not exposed to lead, with mean values of 14.25±2.52 mg/dL and 2.01±0.33 mg/dL, respectively ($p < 0.001$). In the study group, the average concentration of Plasma Lipid Peroxide was found to be 23.52±4.85 mg/L, whereas in the control group, it was seen to be 10.25±2.87 mg/L. The observed disparity between these two cohorts did not provide a statistically meaningful outcome. The present study examines the associations between lipid peroxide levels and blood lead levels, taking into account age and length of exposure.

Table 2: Blood lead and Plasma Lipid Peroxide

	Study group	Control group	P value
Age	42.85±5.85	41.85±4.66	0.25
Exposure years	12.52±2.58	--	0.61
Blood lead (pbB-µg/dl	14.25±2.52	2.01±0.33	0.36
Plasma Lipid Peroxide	23.52±4.85	10.25±2.87	0.41

DISCUSSION

Currently, lead poisoning is emerging as the predominant illness of environmental aetiology and is seeing a significant surge in prevalence within developing nations [9]. Lead exposure studies have been extensively explored in economically advanced nations, however there is a scarcity of published findings about lead poisoning and its related consequences in underdeveloped countries such as Nigeria [9,10]. Membrane damage often plays a role in the pathogenesis of several human illnesses, affecting particular organs or tissues. The aforementioned state leads to the occurrence of lipid peroxidation inside the membrane, hence expediting its deterioration in terms of both structure and function. Once the concentration of lipid peroxides in tissues above a certain threshold, they are released into the circulation, hence elevating the levels of lipid peroxides in the serum or plasma. The presence of elevated levels of lipid peroxides in the circulation cannot be excreted by urine. Instead, they persist in the circulatory system until they are broken down by enzyme antioxidants such as glutathione reductase, glutathione-S-transferase, superoxide dismutase (SOD), and catalase [11]. Hence, the regulation of lipid peroxide levels in the bloodstream or tissues is determined by the rates at which they are formed and decomposed inside the human body. An elevation in lipid peroxide concentration might arise from either foreign factors or endogenous factors.[12,13] Given the existing evidence on the association between lead and lipid peroxidation, our objective was to investigate if prolonged exposure to lead, particularly in those occupationally exposed to this metal, may potentially generate oxidative stress. Consequently, a good cohort of male individuals was chosen to assess their levels of MDA, which serves as a biomarker for a significant series of biochemical processes that result in the oxidation of polyunsaturated fatty acids, such as linoleic acid [14]. The blood lead amounts observed in employees who were exposed were found to be below the suggested health limit of 50 µg/dL for males, as reported in a previous study [15]. The present research observed that the mean age of the study group and control group was 42.85±5.85 and 41.85±4.66, respectively. The mean duration of exposure in the study group was 12.52±2.58 years. Individuals who were consistently exposed to lead had significantly greater amounts of lead in their blood compared to the control group that had not been exposed to lead, with mean blood lead levels of 14.25±2.52 mg/dL and 2.01±0.33 mg/dL, respectively ($p < 0.001$). In the study group, the average concentration of Plasma Lipid Peroxide was found to be 23.52±4.85 mg/L, whereas in the control group, it was seen to be 10.25±2.87 mg/L. The observed disparity between these two cohorts did not provide a statistically meaningful outcome. In a study conducted by Tenchova et al., the authors examined the level of lipid peroxidation in the plasma of 46 individuals employed in lead-storage battery industry. This was assessed by monitoring the concentration of MDA [16]. The researchers observed significantly elevated levels of MDA concentrations (4.61 ± 0.6 mol/L) among individuals who were exposed to the substance, in contrast to a control group of workers who were not exposed to it. The statistical analysis revealed a strong association between lipid peroxidation and the blood lead levels seen in our study participants. However, it is worth noting that the strength of this correlation was not as pronounced as the findings published by Tenchova et al.Two other research teams have conducted investigations on the status of lipid peroxidation among employees who have been exposed to lead. One study demonstrated a favourable association between the concentration of lead in the bloodstream and significant elevations in both MDA levels and SOD activity [17]. In a separate study, it was shown that there was a significant correlation between lipid peroxidation and blood lead levels above 35 µg/dL [12]. There is a hypothesis suggesting that the decreased activity levels of antioxidants, such as SOD, catalase, and glutathione peroxidase, might potentially contribute to the heightened peroxidation of membrane lipids. In accordance with this concept, Sugawara et al. conducted a study to examine lipid peroxidation by assessing the activity of antioxidants in a group of

employees with an average blood lead content of 57.1 g/dL. The magnitude of this level is thrice greater than that seen in our study participants. The levels of SOD and catalase enzymes in the red blood cells of individuals who were occupationally exposed to lead were found to be considerably reduced compared to a control group. In the conducted in vitro studies, erythrocytes were subjected to incubation with lead at a temperature of 37ºC for a duration of 24 hours. The results indicated that there were no observable alterations in the contents of glutathione or lipoperoxide. However, there was a notable suppression of the activities of SOD, catalase, and glutathione peroxidase [13]. Lead may act against this enzyme by reducing the level or availability of these metals. This may explain why high lead levels cause a decrease in the activity of SOD but not of catalase and glutathione peroxidase. Missiry injected lead acetate in rats for 7 d, observing that it resulted in hepatic deficiency of copper and zinc, accompanied by a significant elevation of the lead concentrations in both plasma and liver [18]. Also, hepatic lipid peroxidation was elevated and enzymatic antioxidants decreased coincident with the elevation of lead concentration.

CONCLUSION

There was a statistically significant positive association seen between blood lead levels and both age and length of exposure in both groups. However, no link was found between blood lead levels and related Plasma Lipid Peroxide. The findings given in this research suggest a potential correlation between the concentration of lead, age, and period of exposure with the observed elevation in lipid peroxide levels and lead levels in employees who have been exposed to lead.

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