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Lead Toxicity and Evaluation of Oxidative Stress in Humans

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Abstract
Lead toxicity and oxidative stress caused by lead poisoning were studied in control and lead exposure subjects in the Faisalabad population. The study was conducted to investigate the relationship between total oxidant status and serum glucose, triglyceride, LDL-cholesterol, globulin, HDL-cholesterol and total protein, alanine aminotransferase, aspartate aminotransferase and triiodothyronine (T3). In the present study, 25 control and 25 lead exposed subjects were studied. Serum samples were collected from control and lead exposed workers to determine glucose, triglycerides, LDL-cholesterol, globulin, HDL-cholesterol, total protein, alanine aminotransferase, aspartate aminotransferase, Thyroxine (T3) and serum total oxidant status by using respective kits and biochemical methods. The results showed that serum total oxidation status (TOS) was closely related to glucose, triglyceride, LDL-cholesterol and negatively correlated with globulin, HDL-cholesterol and total protein. There was also a positive correlation between total oxidative stress (TOS) and serum alanine aminotransferase and aspartate aminotransferase. There was no positive or negative correlation between total oxidative stress (TOS) and triiodothyronine (T3). Lead concentration was determined by Atomic Absorption Spectrometer, a relatively low concentration of lead was found in control subjects than lead exposed workers. It was concluded that total oxidant status (TOS) was positively correlated with some serum components and negatively correlated with others. But there was no positive or negative association of total oxidative stress (TOS) with Triiodothyronine.
Keywords: Lead, intoxication, oxidative stress, LDL-cholesterol.

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INTRODUCTION

Lead is health hazardous metal and can be a cause of many chronic diseases in our body. There are many resources of lead which are most commonly gasoline, paints, pipes of different kinds and canned foods. Distribution of lead in blood, bones and soft tissues occur after its absorption by different means. Percentage of lead that binds to blood is about 99 % and our plasma also contains 1 % lead (Hryhorczuk *et al.,* 1985). There are many toxic effects of lead from which oxidative stress has main importance (Gurer *et al.,* 1998; Sharma *et al.,* 2014). Imbalance in the generation and removal of reactive oxygen species are the main mechanisms of lead induced oxidative stress (Nita and Grzybowski, 2016). These mechanisms can cause DNA damage, proteins and membranes damage (Halliwell, 1989). DNA oxidation and membrane lipoprotein oxidation are also effects of oxidative stress which can cause tissue damage. There is a great link of polymorphism of ALAD gene with the accumulation of Lead in different body parts like blood, bones and organs. Neurotoxic effects of workers that always exposed to lead have also been reported (Keleda *et al.,* 2001; Sanders *et al.,* 2009; Gidlow, 2015).

The most important clinical indicators of lead toxicity are mainly zinc protoporphyrin levels, high level of lead in blood and impaired or low activity of delta aminolevulinic acid dehydaratase (Gurer-Orhan and Ozgünes, 2004). Individuals with a high lead concentration showed an increase in MDA concentration in red blood cells of about 91% and were due to a decrease in GPx activity and an increase in GR activity in red blood cells (Kasperczyk *et al.,* 2004). In this study, we evaluated oxidative stress due to lead poisoning and its association with various risk factors.

MATERIALS AND METHODS

Theme

There were total 50 subjects in this case control study including 25 numbers of volunteers as control subjects from all over the city of Faisalabad and 25 subjects who were exposed to lead daily and worked in Happilac paint industry, Faisalabad. This study was approved by University ethics committee, informed consent was given to all subjects involved in this study. A volunteer Physician performed the physical examination of all subjects. Body mass index of subjects was checked by following formula
BMI=Weight (kg)/(kg/m^2).

Hypertension of subjects was also checked with systolic ≥90 mmHG and diastolic pressure ≥ 140 mmHg. Medical conditions of all subjects were recorded carefully.

Design

There were a number of risk factors considered for lead toxicity which included HDL, BMI, Total Cholesterol, total oxidant status, Glucose, total proteins, ALT, AST and Triiodothyronines. Subjects are considered to have high levels of cholesterol if they have a value of 5.69 mmol/L or greater than it. A manual sphygmomanometer was used to check the systolic and diastolic blood pressure for lead exposed and control subjects. All cutoff values for the subjects were clinical. A questionnaire was filled by each subject that included some questions about medical history, vomiting, medication, demographic data, immune status and nutritional status. All of them did not take any medication and they were normal in all cases. All the subjects belonged to urban areas and they did not get interacted with any kind of pesticides and herbicides. Blood samples were collected (5ml from each subject) were collected in clot activator tubes. After that they were centrifuged to get the serum at 4000 rpm for 10 minutes. Serum was then stored at -20˚C for further analysis.

Analytical method

A commercial kit (Biocon) was used to perform serum glucose test by spectrophotometer. Another kit of Randox Laboratories Ltd was used to determine total cholesterol and triglycerides. Then a commercially available kit from Bio Ray Company was used to find LDL-cholesterol, serum total proteins including Albumin and Globulin and HDL-cholesterol. The reagent colorimetric measurements were used to check total oxidant status. At the end serum triiodothyronine was checked by Kits from Biocheck Inc., USA. All the reagents that had been used were supplied by the proper manufacture companies. Atomic absorption spectrometry was performed with each lead absorbed serum sample and control subjects in High Tech Laboratory to check the concentration of lead.

Statistical Analysis

The collected data was analyzed to calculate mean ± SEM. To assess the relationship between blood characteristics and total oxidant status, a comparison of the correlation coefficients provides a viable estimate of the intensity of the association between different anthropometric and biochemical curves.

RESULTS

The general and anthropometric characteristics of the subjects are shown in Table 1. BMI in lead exposed men were significantly higher than in control subjects. The overall mean systolic blood pressure increased in lead exposed men while diastolic blood pressure decreased. Serum biochemical values are shown in Table 2, in which the mean serum glucose concentration in control subjects was significantly lower and the average serum glucose concentration in subjects exposed to lead was higher; this meant that the toxicity of lead increased the blood glucose level. Mean serum cholesterol and triglycerides were also significantly higher in individuals exposed to lead. But the total protein, HDL cholesterol levels were low in lead exposed individuals. The mean serum concentrations of alanine aminotransferase, aspartate aminotransferase, and total oxidant status were greater in lead exposed individuals than in control. But there was no increase in the value of triiodothyronine in individuals exposed to lead. The serum lead concentration in the lead-exposed subjects was higher than that in the control (Table 3).

Table 1. Physiological parameters of control and lead exposed subjects

Parameters	Control subjects (n=25)	Lead exposed subjects (n=25)
Body mass index (BMI:kg/m2)	23.32	25.21
Systolic blood pressure (mm/Hg)	128.56±1.93	129.21±1.72
Diastolic blood pressure (mm/Hg)	77.33±3.32	75.32±2.31

* Data are normally distributed and presented as mean ± SEM.

Table 2. Serum biochemistry and biomarkers of control and lead exposed subjects.

Parameters	Control subjects	Lead-exposed subjects
Glucose (mg/dl)	91.30 ± 1.58	117.04 ± 1.49
Cholesterol (mg/dl)	86.90 ± 2.69	125.43 ± 2.71
Triglycerides (mg/dl)	155.10 ± 2.09	211.64 ± 3.10
HDL-cholesterol (mg/dl)	38.00 ± 1.51	33.84 ± 1.82
LDL-cholesterol	117.10 ± 2.66	177.56 ± 4.72
Total protein (g/dl)	7.120 ± 0.106	6.900 ± 0.129
Albumin (g/dl)	3.740 ± 0.177	4.6280 ± 0.0871
Globulin (g/dl)	3.380 ± 0.220	2.272 ± 0.129
Alanine transaminase (ALT;U/l)	39.340 ± 0.853	49.68 ± 1.03
Aspartate transaminase (AST;U/l)	30.830 ± 0.930	42.596 ± 0.780
Triiodothyronine (T3) (T_3; ng/ml)	2.43±0.19	2.41±0.17
Total oxidant status (TOS; µmol H_2O_2 Equiv./L)	9.91±0.43	17.46±0.98

* Data are normally distributed and presented as mean ± SEM.

Table 3. Determination of lead concentration

Concentration	Control subjects	Lead exposed subjects
Concentration of lead(µg/dL)	6.0 µg/dL	11µg/dL

DISCUSSION

Lead is a harmful and toxic metal and can cause many health hazardous diseases. Many different kinds of biochemical, physiological and behavioral dysfunctions can occur due to this metal so its clinical importance is increasing day by day. The main problem that might arise in our body is the capability of lead to be interacted with proteins by mimicking the actions of calcium. Cell mediated cell injury can also be the cause of lead toxicity (Muntner *et al.,* 2005).

In the present study, glucose, albumin, total oxidant status, aspartate aminotransferase (AST), alanine aminotransferase (ALT), LDL-cholesterol were shown to be high levels in lead exposed subjects. Khanam et al. (2016) demonstrated that Lead acetate alters the serum AST and ALT levels by affecting the liver. A previous study reported mean serum value for both AST and ALT in HCV positive group was non-significantly higher than those of the control group (Abdel-Hamid *et al.,* 2016). Tandon et al. (1997) reported liver function damage due to inhalation of lead.

Our results showed that the levels of total protein, globulin, and HDL-cholesterol in the lead exposed subjects have decreased compared to the control. According to Bashandy, (2006) the demonstration of inhaled lead increased LDL levels and decreased plasma HDL. Glueck et al. (1986) provided evidence that elevated cholesterol and LDL levels lead to coronary heart disease. Previous studies have shown an increase in glucose levels in lead-exposed subjects and this study has also shown an increase in blood glucose levels in subjects exposed to lead. Kopp and Tow, (1988) have indicated levels of serum cholesterol in subjects having great exposure towards lead and a dose response relationship was found between lead exposure and serum cholesterol levels. The current research has also shown the similar results and gave high serum cholesterol level. Badiei et al. (2009) supported the reduction in the level of triiodothyronine (T3) in sheep exposed to lead with different doses of lead. In contrast, this study showed no significant differences in T3 values between lead exposure and control subjects. The next parameter of interest is the total oxidizer state (TOS), which showed a high total oxidant status in subjects exposed to lead and agreed to previous studies (Gurer *et al.,* 1998), which also provides evidence of the overall effect of lead oxidantive state. The oxidative stress mediated toxicity of heavy metals involves damage primarily to liver (hepatotoxicity), kidney (nephrotoxicity), central nervous system (neurotoxicity), and DNA (genotoxicity) in animals and humans (Flora *et al.,* 2012; Sharma *et al.,* 2014).

CONCLUSION

The study provided a positive correlation between total oxidant status and triglycerides, LDL-cholesterol, BMI, systolic blood pressure, cholesterol, ALT, AST, albumin, but negatively correlated with globulin. Thus, an increase in the total oxidant state due to lead poisoning causes a large amount of interference in the biological system of the body.

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CONFLICT OF INTEREST

The authors declare that they don't have any conflicts of interest and are also not interested in competing with anyone.

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