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## BLOOD LEAD LEVELS AMONG CHILDREN AND ADULTS IN A POPULATION OF NASSIRIA CITY IN IRAQ.

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### ABSTRACT

Lead is one of the old poisons and continuous exposure considered as a major public health especially in urban areas. Children are affected by lead poisoning more than adults and nervous system is the major system that affected by high levels of lead in children. In this study, lead levels are measured for children volunteers aging between 6 – 13 years and those aging over 13 years. Measurement done by using atomic absorption spectroscopy ( AAS ) for blood samples. Results from AAS are supported by X – ray for long bones and blood analysis for basophilic stippling in order to evaluate the relationship between the blood lead levels (BLLs) and volunteers status. BLLs were between 5 – 25  $\mu\text{g} / \text{dL}$  for both groups

( children and adults ) and the mean for 30 individuals of each was about  $13.9 \pm 4.8 \mu\text{g} / \text{dL}$  for children, and about  $14.4 \pm 3.6 \mu\text{g} / \text{dL}$  for adults and asymptomatic for both groups. After comparison of both sexes ( male and female ), we didn't find any difference in BLLs between two sexes ( $15.6 \pm 3.5$  vs.

$13.9 \pm 3.9$ ) respectively. From this study, we concluded that mean BLLs about 14 – 15  $\mu\text{g} / \text{dL}$  in our city and this level carry no effect on both adults and children, besides; gender has no significant effect on BLL and this is supported by clinical signs and other investigations.

**Keywords:** lead, children, adults, atomic absorption spectroscopy, CNS effects.

### 1. INTRODUCTION

Lead is one of the old poisons and continuous exposure considered as a major public health especially in urban areas ( Tong et al, 2000 ). Human exposure to high levels causing damage to all organ systems particularly central nervous system, kidney, and blood. At low levels, haeme synthesis is affected, psychological and neurofunctions are impaired ( Goldstein, 1992 ). Exposure to lead can be due to one or more of the following causes (Kazantzis, 1989 ):

- a) Food sources
- b) Drinking water for houses with old pipes
- c) Occupational exposure which can occur in those workers working in smelting, painting, plumbing, and printing.

Lead toxicity can be acute or chronic. Acute toxicity is uncommon, but chronic toxicity is common and occur at blood lead levels 40 – 60  $\mu\text{g} / \text{dL}$ . It can be much more severe if not treated in time and is characterized by persistent vomiting, encephalopathy, lethargy, delirium, convulsions and coma (Flora et al, 2006).



## 2. LEAD AND CHILDREN

The potential of adverse effects of lead in children is heightened due to :

- a) Intake of lead per unit body weight is higher for children than for adults.
- b) Young children often place objects in their mouths, resulting in dust and soil being ingested.
- c) Physiological uptake rates of lead in children are higher than those in adults (Mushaq, 1992).

## 3. EFFECTS OF LEAD TOXICITY

- **On the nervous system:**

The nervous system is the most sensitive and chief target of lead toxicity (Cory – Slechta, 1996). Both central and peripheral nervous systems are affected on lead exposure. The effects on the peripheral nervous system are more pronounced in adults while the central nervous system is more prominently affected in children (Brent, 2006; Bellinger, 2004). The proportion of systemically circulating lead gaining access to the brain of children is significantly higher as compared to adults (Needleman et al, 2004). Children may appear inattentive, hyperactive and irritable even at low lead exposure. Children with greater lead levels may be affected with delayed growth, decreased intelligence, short-term memory and hearing loss. At higher levels, lead can cause permanent brain damage and even death (Cleveland et al, 2008). There is evidence suggesting that low level lead exposure significantly affects IQs along with behavior, concentration ability and attentiveness of the child. Effects of lead exposure on the peripheral nervous system appear in the form of peripheral neuropathy, involving reduced motor activity due to loss of myelin sheath which insulates the nerves, thus seriously impairing the transduction of nerve impulses, causing muscular weakness, especially of the exterior muscles, fatigue and lack of muscular coordination (Sanders et al, 2009).

- **On the hematopoietic system :**

Lead inhibits the key enzymes involved in the heme synthesis pathway which are  $\delta$ -aminolevulinic acid dehydratase (ALAD), aminolevulinic acid synthetase (ALAS), and ferrochelatase thus restraining the synthesis of hemoglobin. It also increases the fragility of cell membrane of erythrocytes which decreases their life span causing anemia (Guidotti et al, 2008; Cornelis, 2005). Anemia caused by lead toxicity is of two types : hemolytic anemia, which is associated with acute high level lead exposure, and frank anemia, which is caused only when the blood lead level is significantly elevated for prolonged periods (Vij, 2009).

- **On the renal system :**

Renal dysfunction occurs mostly at high levels of lead exposure ( $>60 \mu\text{g/dL}$ ) but damage at lower levels has also been reported ( $\sim 10 \mu\text{g/dL}$ ) (Grant, 2008). Renal functional abnormality can be of two types: acute nephropathy and chronic nephropathy. Acute nephropathy is characterized functionally by an impaired tubular transport mechanism and morphologically by the appearance of degenerative changes in the tubular epithelium along with the occurrence of nuclear inclusion bodies containing lead protein complexes. It does not cause protein to appear in the urine but can give rise to abnormal excretion of glucose, phosphates and amino acids, a combination referred to as *Fanconi's syndrome*. Chronic nephropathy is much more severe and can lead to irreversible functional and morphological changes. It is characterized by glomerular and tubulointerstitial changes, resulting in renal breakdown, hypertension and hyperuricemia (Rastogi, 2008).

- **On bone :**

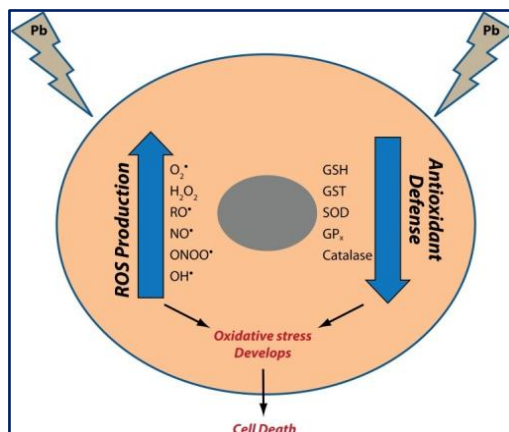
Bone is the primary site of lead storage in human body (Renner, 2010; Silbergeld et al, 1993). Lead can be stored at the surface of the bone and deeper in the cortical bone. Lead can enter blood easily from the surface of the bone but lead in the deeper zone move firstly to the surface before entering blood (Patrick, 2006). About 85 % - 95 % of lead in adults is stored in bones; while in children lead in bones accounts about 70 % resulting in higher concentration of lead in soft tissue in children.

## 4. MECHANISM OF LEAD TOXICITY

Oxidative stress has been reported as a major mechanism of lead toxicity. There are two pathways that operate simultaneously :

- ❖ The generation of ROS, like hydroperoxides ( $\text{HO}_2^\bullet$ ), singlet oxygen and hydrogen peroxide ( $\text{H}_2\text{O}_2$ )
- ❖ The antioxidant reserves glutathione become depleted (Flora et al, 2002). Lead forms a covalent bonds with the sulfhydryl group of the antioxidant enzyme which are the most susceptible target of lead and are eventually inactivated. Lead inactivates glutathione by binding to sulfhydryl groups present in it. This results in synthesis of GSH from cysteine via the  $\gamma$ -glutamyl cycle, which is usually not effective in replenishing the supply of GSH (Hultberg et al, 2001).

**Figure 1. Mechanism underlying the development of oxidative stress in a cell on lead exposure.**



## 5. IONIC MECHANISM OF LEAD TOXICITY

This mechanism arises due to its ability to substitute other bivalent cations like  $\text{Ca}^{2+}$ ,  $\text{Mg}^{2+}$ , and  $\text{Fe}^{2+}$  (Lidsky & Schneider, 2003). Significant effects have been found on various fundamental cellular processes like intra and intercellular signaling, cell adhesion, protein folding and maturation, apoptosis, ionic transportation, enzyme regulation, release of neurotransmitters (Garza et al, 2006). The ionic mechanism contributes principally to neurological deficits, as lead, after replacing calcium ions, becomes competent to cross the blood brain barrier (BBB) at an appreciable rate. After crossing the BBB, lead accumulates in astroglial cells (containing lead binding proteins). Toxic effects of lead are more pronounced in the developing nervous system comprising immature astroglial cells that lack lead binding proteins. Lead easily damages the immature astroglial cells and obstructs the formation of myelin sheath, both factors involved in the development of BBB.

## 6. PREVENTION OF LEAD INDUCED TOXICITY

Preventive measures are preferred over the treatment regimens, considering the toxic effects of lead. This is due to the fact that once lead enters the body, it is almost impossible to remove it completely or to reverse its damaging effects on the body. Guidotti and Ragain (2007) suggested a three-way measure as preliminary preventive approach towards lead toxicity, it includes :

- a) Individual intervention
- b) Preventive medicine strategy
- c) Public health strategy.

Preventive medicine strategy mainly aims at screening the blood levels of children that are at a high risk of lead exposure. If lead is detected in blood, medical intervention is carried out with the aim to control undesirable outcomes of poisoning and prevent further accumulation of lead. Public health strategy has a larger influence and acts at a population level with a target to reduce the risk of lead exposure in habitable regions.

Nutrition also plays an important role in prevention of lead induced toxicity. Studies have shown that uptake of certain nutrients like mineral elements, flavonoids and vitamins can provide protection from the environmental lead as well as from the lead already present in the body. These nutrients play a pivotal role in restoring the imbalanced pro-oxidant/oxidant ratio that arises due to oxidative stress. (Hsu and Guo, 2002).

Role of antioxidants in protecting lead induced oxidative stress :

Antioxidants can prevent lead toxicity in three ways (Garcia & Gonzalez, 2008) :

- a) By inactivating the generated ROS at molecular level, thereby terminating the radical chain reaction (chain breaking)
- b) By chelating the lead ion and preventing further formation of ROS.
- c) By chelating lead and maintaining it in a redox state, which leads to its incompetency to reduce molecular oxygen.

## 7. AIM AND METHOD :

The aim behind this study is to measure the mean of lead in the society of Nassiryah city. Professionals in our poison control center decided to determine the mean of lead levels to make it as a reference in comparison high blood lead levels. This is accomplished by :



- a) Calling both children ( below 13 years old ) and adults as volunteers . Individuals with chronic or acute disease must be excluded to prevent any abnormal results. Results of each group ( children and adults ) must be estimated separately.
- b) Getting samples of blood from each volunteer which is used to measure the blood lead level ( BLL ) by atomic absorption spectroscopy (AAS), and for making complete blood picture to detect the presence or absence of basophilic stippling.
- c) An X - rays for wrist and long bones were done to exclude chronic exposure to lead that causing metaphyseal lines occurring at the end of bones.

## 8. RESULTS :

After measurement of BLL by means of AAS ( Atomic Absorption Spectroscopy ), the results as shown in table 1 for both children and adults groups :

Table 1 : Blood lead levels for both children and adults		
Age group	No.	Mean $\pm$ SD
Children	30	13.9 $\pm$ 4.8
Adults	30	14.4 $\pm$ 3.6

There is no significant difference between mean of blood lead level for children and adults.

As a comparison between the BLLs for children below six years with levels for those above six years, it is shown in table 2 :

Table 2 : Blood lead levels for children below and above six years		
Age group	No.	Mean $\pm$ SD
<6 years	8	14.5 $\pm$ 6.2
>6 years	8	12.9 $\pm$ 4.9

We didn't show any significant difference in mean BLLs between these groups.

After comparison of mean BLLs between males and females, there is no significant difference between both groups as in table 3:

Table 3 : Blood lead levels between males and females		
Sex group	No.	Mean $\pm$ SD
Male	30	15.6 $\pm$ 3.5
Female	30	13.9 $\pm$ 3.9

Hemoglobin levels were estimated and compared with BLLs for each individual, but; there is no significant difference between BLLs and hemoglobin levels.

Table 4 : Means for hemoglobin levels with different BLLs.

Group	No.	Mean $\pm$ SD
BLL $\leq$ 15	27	13.4 $\pm$ 1.5
BLL > 15	27	13.7 $\pm$ 1.5

Blood samples were tested by hematologist to detect presence or absence basophilic stippling. For all groups, there is no basophilic stippling

An X – ray was done for wrist and long bones of each volunteer to show lead deposition in these regions. There is no metaphyseal lines in these bones.



## 9. DISCUSSION

According to this study, we noticed that BLLs are the same for all age groups. No significant difference in BLLs between children and adults ( $13.9 \pm 4.8$  vs  $14.4 \pm 3.6$  respectively), males and females ( $15.6 \pm 3.5$  vs  $13.9 \pm 3.9$  respectively). These results indicate that there is no association between age and BLLs; besides, there is no relationship between BLLs and gender. Presence of any source of exposure to lead is the most important factor in this setting. These results reflect the environment, where there is no big manufactures to be as a source of exposure. As shown from this study, blood lead levels are more than 10 in all groups and all volunteers are asymptomatic, this means that these levels are tolerated by children and adults.

## 10. CONCLUSION

We concluded from this study that BLLs in Al – Nassyria city approximately 14 – 15 mcg / dl and this level can be tolerated for both children and adults where all individuals recruited in this study are asymptomatic. Gender has no significant effect on BLLs

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