Acute Lead Poisoning In an Infant

M. Madhusudhanan,[1] S.B. Lall [2]

Abstract

A case of acute lead poisoning in an infant without overt clinical manifestations of encephalopathy is reported for the first time in Oman. The case was diagnosed at Rustaq Hospital on the basis of (i) history by the mother of giving the child a traditional remedy for treating constipation (ii) X-ray of abdomen showing radio-opaque speckles and (iii) detection of high blood lead levels (83.3 μg/dL) at the toxicology laboratory of the poison control centre. The source of lead was confirmed by high content of inorganic lead (20.2%) found in the sample of the traditional remedy (bint al dahab). The blood lead levels significantly decreased, after the intravenous calcium edetate (EDTA) therapy was given to the baby. The case highlights that early detection and treatment of acute lead poisoning in children can prevent morbidity and sequelae associated with encephalopathy. It also indicated the need for awareness and prevention programme for parents on this issue.

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From [1]Laboratory Medicine, Rustaq hospital, South Batinah Region; [2]Poison Control Centre, Directorate of Environmental Health and Malaria Eradication, Ministry of Health.
Address correspondence and reprint request to: S.B. Lall. [2]Poison Control Centre, Directorate of Environmental Health and Malaria Eradication, Ministry of Health.
E-mail: shyamlall@hotmail.com

Introduction

Acute lead poisoning in children is uncommon and is generally observed in patients who have been exposed to high concentration of lead from folk remedies, lead containing paint chips or foreign bodies or from intrauterine exposure.[1-4] Acute lead encephalopathy is most severe presentation of lead poisoning and has been reported to occur after use of lead containing eye cosmetic, kohl on the umbilical stump of newborns.[5, 6] Bint al dahab, a traditional remedy containing 20-93% lead, commonly used in this part of the world for treating minor ailments in children, is also reported to cause lead encephalopathy in infants.[7, 8] Chronic environmental exposure, however, is more common in children and depending upon the degree and duration of exposure, the toxic manifestations range from subtle cognitive deficits and neurobehavioral abnormalities to overt neurotoxicity, hematologic effects and nephrotoxicity.[9] The clinical manifestations of lead poisoning correlate well with blood lead levels, 70-100 μg/dL, and are usually associated with encephalopathy, persistent vomiting and pallor.[10] However, we report a case of acute lead toxicity in a two month Omani male infant who presented to Rustaq Hospital with a history of constipation without any overt symptoms of encephalopathy at blood lead levels of 83.3 μg/dL. The cause of such high blood lead level was found to be Omani traditional medicine (bint al dahab), which was administered to the baby for the complaint of constipation. The traditional medicine sample was also found to contain high lead content. This was the first case of lead poisoning investigated from our hospital and analysis was done in the Toxicology laboratory of Poison Control Centre, Ministry of Health in Muscat.

Case Report

A two month Omani male infant was referred from a Polyclinic to Rustaq hospital with one month history of constipation. The baby passed stools only after glycerine suppository. The infant was exclusively breast fed. The mother gave a history of giving the baby Omani traditional medicine for constipation. Examination revealed mild distension of the abdomen. Plain X-ray of abdomen revealed scattered radio opaque speckles and abdomen loaded with stools. A routine full blood count showed a hemoglobin of 10.2 μg/dL, white blood count of 10,000/uL and platelet count of 490,000/uL. Peripheral blood smear revealed a normocytic normochromic blood picture with basophilic stippling of red blood cells. Renal function tests and urine analysis were within normal limits. Stool analysis did not show any ova or cyst.

His initial blood lead level was 83.3 μg/dL (reference range = less than 10 μg/dL). The traditional Omani medicine was also analyzed and found to contain high content of lead (20.2%). After a 5day course of intravenous calcium edetate (EDTA) This blood lead levels decreased to 53 μg/dL. The parents were advised by the poison control centre not to use such traditional Omani medicines. The infant was given one more course of calcium edetate and blood lead levels after one month was 49 μg/dL. A rectal biopsy was done which showed presence of normal ganglion cells and nerve fibres ruling out Hirschprung disease.

Review of Literature

Lead is the most ubiquitous toxic material and is detectable in practically all phases of environment. The most susceptible populations are children particularly toddlers, infants in the neonatal period and the fetus.[9] The toxicity of lead is related to its multiple biochemical effects. It has high affinity for sulfhydryl groups and inhibits numerous enzymatic, receptor and structural proteins,

including enzymes involved in heme biosynthesis. It competes with calcium ions and interferes with nerve transmission. It inhibits membrane associated enzymes and metabolic pathways leading to heamolysis, renal damage and hypertension. It interferes with metabolism of vitamin D. It causes testicular injury and infertility in men and interferes with implantation of fertilized ovum in the uterus in women.[11] Lead is stored in bones and it can be released in blood during pregnancy when there is calcium deficiency. This is an important source of exposure to fetus as it freely crosses the placenta.[12]

Environmental sources of lead include leaded paint, water pipes with lead joints, lead glazed ceramics, certain canned foodstuff, air/dust/soil, traditional remedies and eye cosmetics, fishing weights and snooker chalks.[9] Occupational sources include lead and silver smelting, painting and home remodeling, manufacture of batteries, ceramics, stained glassware, jewelry and bronze. Environmental Protection Agency (EPA) Guidelines for lead in air is 1.5 $\mu g/m^3$, WHO guidelines for water and urban soil are 10 $\mu g/L$ and 400 ppm respectively.[9] Lead, is absorbed by inhalation (50-70%) or ingestion (10-15%). In children the absorption through ingestion is 40-50%. Absorption of lead increases if there is concomitant iron or calcium deficiency. 95% of absorbed lead accumulates in bones or teeth. Half life of lead in soft tissue pool is 40 to 50 days whereas it is 10 to 20 years in skeletal pool. Excretion of lead is 90% through kidney and 10% through bile. If intake exceeds 3.5 $\mu g/kg/day$ accumulation in the body occurs.[11] Clinical manifestations are diverse and involve multiple body systems. Glycosuria, aminoaciduria and phosphaturia occur due to reversible renal tubular dysfunction. Renal impairment secondary to renal interstitial fibrosis is irreversible. Anemia in lead poisoning may be microcytic or normocytic hypochromic with red cells showing punctate basophilia. Haemolytic anemia is seen in severe lead poisoning. Lead lines in gums are due to blue colouration of the margins from lead sulphite deposits. Peripheral neuropathy leads to foot and wrist drop.[10] Several studies carried out in the past two decades have proven that fetal and early childhood exposure to low doses of lead results in lower IQ, learning, reading and hearing deficits and physical growth retardation. A 2 to 4 point IQ deficit occurs for each microgram/deciliter increase in blood lead levels beyond the reference level (10 $\mu g/dL$.).[13, 14] Recent evidences indicate that blood levels between 5-10 $\mu g/dL$ can also cause cognitive deficits in children,[15] and thus no levels in blood are safe. Laboratory diagnosis includes increased blood lead levels; raised delta-amino levulinic acid in blood and urine, increased free red cell Zn protoporphyrin, and urinary corproporphyrinogen.[11] However, whole blood lead estimation is definitive of diagnosis of lead poisoning. Two milliliters of blood must be collected in EDTA (1.8 mg/ml) and sent without separation to the laboratory for blood lead levels. The various methods of blood lead estimation include Anodic stripping voltammetry, Atomic absorption spectrophotometry and inductively coupled plasma mass spectrometry. In Anodic stripping method whole blood is added to the Reagent solution and lead is released from the blood components. Released lead is concentrated onto a thin film electrode.

Table 1: CDC guidelines for management in children are as below:[9]

Class	Blood lead levels	Management
I	≤9 $\mu g/dL$	No action. Class I child is not considered to be lead poisoned.
IIA	10-14 $\mu g/dL$	Nutritional and educational intervention and remove from source. Community wide screening and prevention programme. Repeat blood test after one month. Repeat lead test after 3 months.
IIB	15-19 $\mu g/dL$	Remove from source. Repeat blood test after one month
III	20-44 $\mu g/dL$	Remove from source. Give DMSA only if blood Lead remains high.
IV	45-69 $\mu g/dL$	Remove from source. Oral Chelation therapy (DMSA) recommended
V	> 70 $\mu g/dL$	Remove from source, Medical emergency. BAL and EDTA Chelation therapy

The plated lead is removed from the electrode by applying a stripping current. The amount of lead is measured by integration of the current released during the rapid electrochemical step. This is the method followed in the poison control center to estimate blood lead levels.

Treatment for lead poisoning includes supportive measures and chelating agents. The chelating agents are intravenous calcium disodium edetate (EDTA), oral dimercaptosuccinic acid (DMSA) and intramuscular dimercaprol (BAL). BAL is used only when there is severe toxicity as in lead encephalopathy.

Conclusion

This case is presented to highlight that infants and children are

vulnerable to lead toxicity in Oman, and health education programs in pediatric clinics are required for advising the parents not to use illogically any traditional medicine, which may have high lead content. Furthermore, the case indicates the usefulness of the blood lead analysis in diagnosis of acute lead toxicity in absence of overt manifestations of encephalopathy and availability of this facility in the country at poison control centre.

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