Congenital Lead Poisoning: An Unusual Presentation

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Abstract Lead poisoning presents a common acquired as well as congenital environmental threat to children's health today. An unusual case of severe lead poisoning in breast fed male infant is presented here. The objective of the study is to describe a patient who developed clinical lead intoxication with an uncommon source of poisoning. A 6 months old male baby presented with gradual loss of weight, not feeding well and persistent vomiting. Laboratory investigation revealed that he was having anemia (Hb level 5.4 gm/dl), abnormal liver enzymes (including elevated transaminase activity) and high blood lead value (83 µg/dl). RBC morphology showed basophilic stippling with cabot ring, suggestive of a case of lead poisoning. A course of chelation treatment using calcium versenate (EDTACaNa₂) was prescribed following which a radical solution for mobilization of lead from his systems was observed.

Keywords Lead poisoning · Anemia · Basophilic stippling

Introduction

Good environmental condition is an essentiality for holistic lifestyle, but probably the greatest danger mankind faces today is from environmental pollution. Apart from

producing various acquired disorders, heavy metal poisoning from polluted environment is also perceived as a great threat for various congenital disorders. Lead poisoning remains an important threat for children as well as adults. Inhalation and ingestion are generally two potential routes of lead absorption in adults, after which it accumulates in blood, soft tissues and bone [1]. This accumulated heavy metal is in turn transferred to the unborn child through feto maternal circulation, causing several congenital disorders due to lead intoxication. Breast milk consumed after birth is another route by which the heavy metal accumulates in an infant's system from a mother suffering from chronic poisoning herself. It affects the hemopoietic system, central and peripheral nerves, renal function, vascular system and the gastrointestinal tract causing anemia, kidney impairment, nephropathy and gastrointestinal symptoms including impairment of liver function [1].

Congenital lead poisoning may go undiagnosed for some time, giving rise to confusion in diagnosis and development of complications. We present a case in which a delay in diagnosis occurred and few months passed before the exact cause of the poisoning could be determined.

Case Report

A 6 months old male infant was presented in our clinic on January 2013, with loss of weight, progressive pallor, low feeding and persistent vomiting since the last month. According to the mother of the baby, he had no symptoms 1 month back but developed intermittent episodes of persistent vomiting, fever and progressive pallor over last 1 month. No significant birth history was obtained,

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delivery being normal and post natal period uneventful. Baby was exclusively breast fed, without any weaning, and other developmental milestones, such as social smile, head control (grade IV), side to side turning, and social recognition were within normal limits.

Physical examination revealed a grossly undernourished male baby with body weight 4.7 kg (<5th percentile), head circumference 36 cm. Pallor was severe, cyanosis nil, edema nil, frontal bossing and maxillary prominence was seen, and anterior fontanelle was open normally. Abdominal examination revealed nontender, 2 cm/1 finger palpable, firm liver and 2 cm/1 finger firm and palpable spleen. Examination of cardiovascular system showed irregular febrile pulse, rate 92/min, slight systolic murmur and shifting of the apex beat towards left suggesting cardiomegaly with left heart involvement, and features of systemic hypo dynamic circulation. In other systems no abnormalities were detected upon examination.

The routine evaluation revealed haemoglobin (Hb): 5.4 gm/dl; total leukocytes count (TLC): 4100/cumm; differential leukocyte count (DLC): 60 % polymorphs, 25 % lymphocytes, 1 % basophil and 3 % eosinophil; platelets: 1.8 lakh/cumm; ESR: 30. Peripheral blood smear revealed polychromasia, predominantly microcytic hypochromic picture with predominance of blast cells. The reticulocyte count was: 3 %. Urine analysis was normal. Biochemical parameters suggested abnormal liver function with serum bilirubin: 3.5 mg/dl (direct bilirubin: 2.0 mg/dl), aspartate aminotransferase (AST): 191 IU/l, alanine aminotransferase (ALT): 133 IU/l, alkaline phosphatase (ALP): 838 IU/l and γ -glutamyl transferase (GGT): 193 IU/l. Calcium level was low (7.6 mg/dl). Ferritin level was high: 936 mg/dl. The triglyceride value was high (448 mg/dl) as patient was in non-fasting state, maintained exclusively on mother's milk. In view of the anaemia, increased polychromasia, raised bilirubin and transaminases, the patient was further evaluated. Other biochemical parameters were normal. CLIA serology for hepatitis viral markers and HIV were negative. Eye examination including fundus was inconclusive. Chest X-ray was normal. Other X-ray evidence showed transverse bands of increased density at the ends of growing bones. A film of the abdomen revealed opaque particles, especially in the recto sigmoid area. Endoscopy of upper GIT did not reveal any abnormality. The cause of anaemia and hyperbilirubinemia remained obscure. Hb-electrophoresis and Coomb's test, osmotic fragility, serum Vitamin B₁₂ and folate levels did not reveal any abnormality and a repeat peripheral blood smear showed microcytic hypochromic picture with increased polychromasia. RBC morphology showed basophilic stippling with Cabot ring. Reticulocyte count was gradually rising, a repeat test showing count as 8 %. A decline in Hb percentage with increasing

reticulocytosis and hyperbilirubinemia suggested haemolysis. Basophilic stippling with Cabot ring in the RBC's raised doubt of lead poisoning as a cause.

Blood lead concentration by Atomic Absorption Spectrophotometry was found to be 83 μ g/dl. Neurological and renal function evaluation did not reveal any abnormality.

Based on these findings patient was diagnosed as a case of chronic lead poisoning, complicated with severe anaemia. He was started chelation therapy with calcium versenate (EDTACaNa₂) as an intravenous (IV) infusion over 5 days and three such courses were given over 3 months. Over the period his blood lead concentrations gradually fell to 18 μ g/dl.

Patient has recovered now and is asymptomatic. His hemogram and liver functions are normal. He is on regular follow-up.

Discussion

Healthy children need healthy environment. A healthy baby is born of a healthy mother. Women who once lived in a lead rich environment may pass on the toxin to their unborn offspring. Lead poisoning generally presents as an occupational hazard in populations exposed to lead particles. It has also been reported due to traditional medicine [2], from the use of cosmetics like surma [3], due to vegetables [4], fishes [5] and street food [6]. The presentation of lead toxicity is variable.

Pregnant women with high blood lead levels could pass on the lead to the foetus 'in utero' through umbilical cord in fetomaternal circulation and thereafter while breast feeding the child. The stored lead in the bones of the mother passes to the developing baby. Prenatal exposure to lead produces toxic effects on the human foetus including pre-maturity, intra uterine growth retardation, and arrested mental development.

The situation gets worse when the mother maintains a poor diet. Deficiency of iron, calcium and zinc increases lead absorption. To compensate for the deficiency of calcium and minerals, the mother's body will start breaking down bone and releasing lead. The amount of lead present in the placenta indicates long-term exposure of the mother to the metal.

One of the worst effects of lead poisoning is its interference with heme synthesis, which leads to hematological damage. The overall effect is the disruption of the synthesis of hemoglobin as well as respiratory pigments, such as cytochromes, which require heme for their synthesis. Lead inhibits the enzyme δ -ALA synthetase; the key or rate-limiting enzyme for heme synthesis; so the synthesis cannot proceed beyond δ -ALA. The resultant anemia directly affects the cardiovascular system, causing LVF (left

ventricular failure) over time, due to chronic hypodynamic circulation.

Finally, lead does not permit utilization of oxygen for life sustaining energy production. Increased P450 dependent monooxygenase activity; and ALT and AST activities, which is associated with impaired membrane function [7] may cause impaired liver function test. Increase in AST and γ -GT suggests abnormal macrophage function, which could be related theoretically to increased lead consumption [7]. The impaired liver function test, coupled with severe hemolysis activates the reticulo endothelial system producing hepato and splenomegaly; also causing hyperbilirubinemia. However, it is worthwhile to review the whole picture when the baby would be taken off from the mother's milk and start weaning.

Environmental lead pollution is an indirect but serious threat to the health of newborn babies and infants. Therefore a knowledge of clinico-pathological features of congenital lead poisoning is essential to make early diagnosis and provide timely therapy and save these children from impending morbidity and mortality.

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