Indian J Pediatr (November 2011) 78(11):1420–1423
DOI 10.1007/s12098-011-0445-6

CLINICAL BRIEF

High Prenatal and Postnatal Lead Exposure Associated Lead Encephalopathy in an Infant

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Received: 26 February 2010 / Accepted: 29 April 2011 / Published online: 10 June 2011

Abstract An 11-month-old child presented with persistent seizures requiring ventilator support. The child had global developmental delay, was staying in the premises of battery manufacturing unit, had microcytic and hypochromic anemia with basophilic stripling on peripheral smear, lead line on radiograph of the long bones and BLL of 244 μg/dl. The CT scan of the brain revealed cerebral atrophy. The mother also had high BLL and lead line in the radiograph of the long bones. The child was managed with chelation therapy. Given the continuing lead exposure among occupational and general populations in India, this case study highlights the need for prompt environmental preventive actions as well as nutritional and preventive counseling for occupational populations.

Keywords Lead toxicity · Blood lead level · BLL · Lead poisoning · Lead encephalopathy

Introduction

Lead poisoning is an entirely preventable public health problem. Despite implementation of international environmental regulations pertaining to lead exposure, globally, 40% of children have blood lead levels (BLL) above 5 μg/dL and 20% above exposure risk levels of≥ 10 μg/dL [1]. Although guidelines exist in India, they are not stringently enforced. BLL≥10 μg/dL was found in 54.5% of children in Chennai [2] and 32.2% in Mumbai [3]. The authors report a girl with developmental delay who presented acutely as encephalopathy due to prolonged lead exposure.

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Case Report

An 11-month-old girl presented with persistent seizures and coma since one day. There was no history of antecedent fever, rashes, drug intake, abnormal odor, persistent vomiting or head injury. There was past history of an untreated afebrile convulsion at 7 months of age and delay in acquisition of developmental milestones. Prior to illness she could only sit with support, was unable to reach out, babble only and did not had stranger anxiety. Antenatal and birth history were uneventful. She was the first issue of non-consanguineous parents. The child had been inadequately weaned after exclusive breast feeding for 4 months. The family resided in the premises of a battery manufacturing factory, where the father was an employee. The mother had a previous spontaneous abortion 2 years back.

At presentation child was gasping, vitals were unstable necessitating initiation of immediate ventilatory support. Anthropometry revealed microcephaly, wasting and stunting. Salient neurological findings were a bulging anterior fontanelle, Glasgow Coma Score of

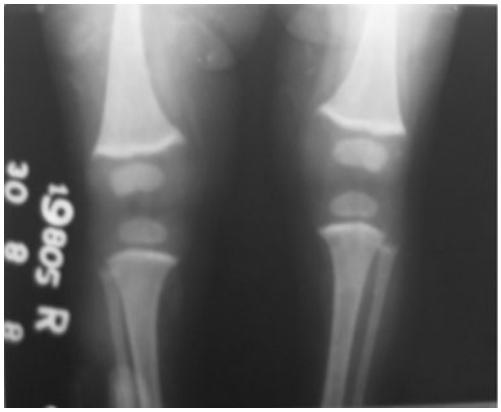

Fig. 1 X-ray of the Knee showing lead line

$E_2M_3V_T$, and decerebrate posturing. The rest of systemic examination was normal.

A provisional diagnosis of acute meningoencephaly was kept. The child was managed in the intensive care unit. Seizures and increased intracranial tension were controlled. Mechanical ventilation was required for 24 h. The patient had hemoglobin of 8.1 g/dL, leukocytosis (TLC 18250/mm^3) normal platelet counts, and a hematocrit of 27% and mean corpuscular volume of 71 fL. The peripheral smear showed microcytic hypochromic erythrocytes with anisocytosis and basophilic stripling. Calcium, electrolytes and blood sugar levels were normal, as were the arterial blood gas, liver and kidney function tests. Radiographs of the wrist and long bones revealed dense metaphyseal lines (Fig. 1). Post stabilization significant CT scan cranium findings were generalized cerebral atrophy and hypo densities in bilateral thalamo-ganglionic region (Fig. 2). The cerebrospinal fluid analysis after 48 h was normal. Blood culture was sterile.. Lead poisoning was suspected considering the residential details, basophilic stippling and lead lines on the radiograph. The BLL was 244 μg/dL. Screening of other family members revealed significantly raised BLL of the father, mother and uncle (55 μg/dL, 49 μg/dL and 44 μg/dL, respectively) and radiographic lead lines.

Chelation was started with a combination of intramuscular British Anti-Lewsite (BAL) and Calcium sodium Edatate (CaNa2EDTA) infusion for 5 days. BLL decreased to 58 μg/dl. Oral chelation with Pencillamine was started with concurrent supplementation with calcium and iron. When discharged after 19 days she was conscious, seizure free but neurologically impaired. The family changed their residence and underwent treatment in the Medicine department.

Discussion

Clinical manifestations of lead poisoning are myriad, including anemia, short stature, acute encephalopathy and chronic neurological problems like cognitive regression, poor school performance, and hyperactivity [4] Children are more susceptible due to immature organ systems, pica, and increased prevalence of concurrent iron and calcium deficiencies that enhance gastrointestinal lead absorption [5]. During pregnancy lead is mobilized from the maternal bone stores, crosses the placenta and affects the fetus. Umbilical cord BLL reflects maternal levels [6]. High antenatal BLL is a risk factor for global developmental delay [7]. Even greater

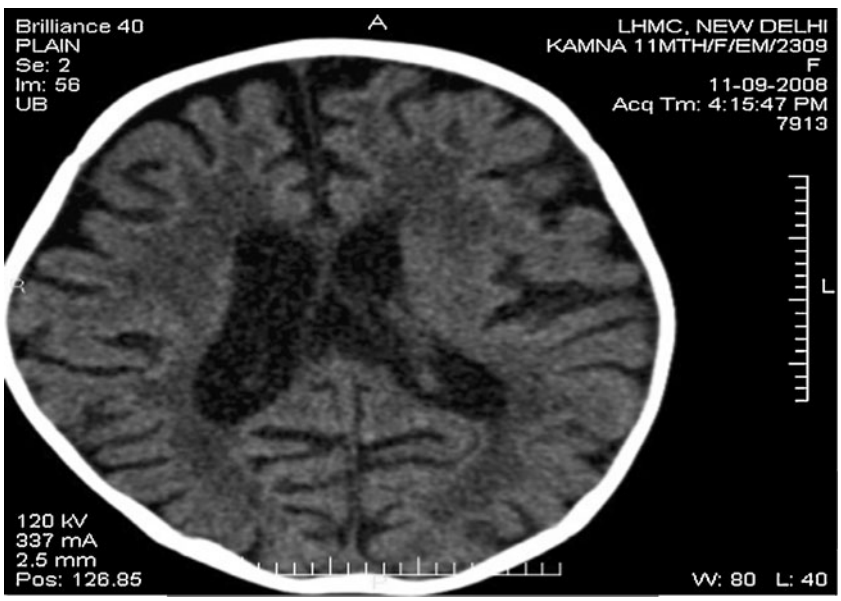

Fig. 2 CT Cranium Showing cerebral atrophy

mobilization of maternal lead occurs during lactation. The milk to plasma lead ratio increases with increase in BLL of the mother [8]. In the present case, high maternal BLL and radiographic lead lines signify prolonged exposure in the mother. Although the child's presentation was suggestive of acute encephalopathy, the presence of concurrent developmental delay, cerebral atrophy and radiographic lead lines indicate chronic exposure. This probably reflects a combination of both pre and continuing post natal exposure. Chelation therapy was effective in reducing BLL and reversing encephalopathy but not the neurological sequelae.

Acute lead encephalopathy should be considered in children with convulsions or coma when history of lead exposure exists. Radio-dense distal metaphyseal lines in long bones signifying chronic exposure are not pathognomic and may be absent in acute poisoning. BLL is considered the gold standard for establishing the diagnosis. It is also the level at which AAP recommends community level risk management [9]. But at individual level there is no known threshold for the effects of lead on the nervous system and most of the IQ deficit takes place <10 μg/dl [10]. A value ≥10 μg/dL requires reconfirmation and initiation of management (Fig. 3). All sources of lead exposure should be eliminated after identification. BLL≥45 μg/dL requires prompt chelation therapy, which decreases BLL, reversing hematologic abnormalities and enhances urinary excretion. Since lead deposits are released slowly from the bone, the efficacy of chelation is sub-optimal and prolonged. Follow-up requires repeated BLL. In chronically exposed mothers, calcium

Adapted from American Academy of Pediatrics: Lead exposure in children: Prevention, defection, a management Pediatrics 2005; 116:1036–1046.

EP, erythrocyte protoporphyrin; ZPP, zinc protoporphyrin, 2,3-dimercaptosuccinic acid (DMSA [succimer]) , CaNa2EDTA Calcium sodium ethylenediaminetetraacetic acid (versenate), British antilewisite (BAL), and penicillamine.

Fig. 3 The treatment flow chart for lead Poisoning

supplements during pregnancy and lactation reduce lead mobilization from the bone and may reduce neurological damage to the child.

Contributions SKK reviewed the literature, wrote the manuscript and revised the final manuscript. VK also reviewed the manuscript.

Conflict of Interest None

Role of Funding Source None

References

1. Canfield RL, Henderson Jr CR, Cory-Slechta DA, et al. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. N Engl J Med. 2003;348:1517–26.
2. Roy A, Pallaniappan K, Hu H, Wright R, Balakrishnan K. Predictors of blood lead among 3-7-year-old children in Chennai, India. Epidemiology. 2007;18:S175.
3. Nichani V, Li WI, Smith MA, et al. Blood lead levels in children after phase-out of leaded gasoline in Bombay, India. Sci Total Environ. 2006;363:95–106.
4. Dietrich KN, Ware JH, Salganik M, et al. Effect of chelation therapy on the neuropsychological and behavioral development of lead-exposed children after school entry. Treatment of Lead-Exposed Children Clinical Trial Group. Pediatrics. 2004;11:19–26.
5. Lanphear BP, Winter NL, Apetz L, et al. Randomized trial of the effect of dust control on children's blood lead levels. Pediatrics. 1996;98:35–40.
6. Patel AB, Prabhu AS. Determinants of lead levels in umbilical cord blood. Indian Pediatr. 2009;46:791–3.
7. Ahmed G, Howard Hu, David B, et al. Maternal bone lead as an independent risk factor for fetal neurotoxicity: a prospective study. Pediatrics. 2002;110:110–18.
8. Gulson BL, Mahaffey KR, Jameson CW, et al. Mobilization of lead from the skeleton during the postnatal period is larger than during pregnancy. J Lab Clin Med. 1998;131:324–9.
9. Committee on Environmental Health Policy statement of American Academy of Pediatrics, Lead exposure in children: prevention, detection, and management. Pediatrics. 2005;116:1036–46
10. Lanphear BP, Hornung R, Khoury J, Yolton K, Kim N, Lanphear D. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect. 2005;113:894–9.