CASE PRESENTATION

Lead Encephalopathy in a Bahraini Infant

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ABSTRACT

To increase awareness about lead poisoning generally and lead encephalopathy specifically, we report a case of infantile lead encephalopathy resulting from the use of Kohl (Surma) on the umbilical cord. There is a need for public education about the danger of this practice. Strict regulations about importation and distribution of such products are needed.

Lead poisoning in children is the most common and serious environmental hazard[1]. Children can breathe it, swallow it via drink and food, and absorb it through skin or mucus membranes[2]. Pica (eating non-food items such as leaded paint flakes) and use of leaded cosmetics such as Kohl (Surma) puts infants and toddlers at great risk of lead poisoning[3,4].

Acute lead encephalopathy is the most serious and dramatic presentation of lead poisoning. It results from massive cerebral vasculopathy and brain edema that leads to increased intracranial pressure and culminates in brain stem herniation and death. If the child survives the ordeal of the acute stage, he or she is still at great risk of long-term sequel of permanent brain damage. These can range from delayed developmental milestones to cerebral palsy.

In this report, I present a case of acute lead encephalopathy in a Bahraini infant.

THE CASE

Six weeks old male infant was admitted to the Paediatric service at Salmaniya Medical Centre (SMC) with a 5 day history of vomiting, irritability, constipation, and 2 episodes of generalised tonic-clonic convulsions; each episode lasting for about 10 minutes. The last episode was terminated in the emergency room after IV valium infusion.

Vomiting was forceful, non-bilious and contained milk curds. Bowel had not opened for one week. Urine was dark yellow. He was seen 3 times in a local health centre and given antiemetic and glycerine suppositories. There was no history of fever or head trauma. Pregnancy, labour, delivery and neonatal period were uneventful. Birth weight was 4.2kg.

Prior to this illness, his sleeping, feeding and activity were all normal. He was exclusively breast fed. Family history was unremarkable.

Physical examination showed a chubby, pale and drowsy infant with high pitched cry. Both pupils were equal and sluggishly reactive to light. Anterior fontanelle

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was full and pulsating. Muscle tone was increased with brisk deep tendon reflexes.

After controlling seizures and stabilising the infant's condition, lumber puncture was performed which yielded clear fluid under high pressure. CSF total white blood cell count was 20 with a predominance of lymphocytes and occasional RBCS. CSF chemistry showed protein of 300 mg/dl, glucose of 60mg/dl, and lactic acid of 28 mmol/L. Arterial blood gases, serum electrolytes, glucose, BUN, creatinine, magnesium and phosphorus were all within normal limits. Serum calcium however, was 7.1 mg/dl. The infant was anaemic with a haemoglobin of 6.0 g/dl and a reticulocyte count of 12%. Peripheral smear showed microcytosis and hypochromia with distorted and crenated red blood cells. Total WBC count was 21.6 x 10^9/L and platelet count was 398 x 10^9/L.

Liver function tests revealed a total bilirubin level of 2.6 mg/dl with an indirect bilirubin value of 2.0 mg/dl (NR < 1 mg/dL). SGPT 60 iu/L (NR 50–136 U/L), and alkaline phosphatase 580 iu/L (NR 50–136 U/L). Cultures of blood, urine and CSF were sterile.

Chest x-ray and skull ultrasound were normal. Haemoglobin electrophoresis and G6-PD activity were normal. EEG was abnormal and consistent with encephalopathy. Parathyroid hormone level was within normal limits.

Based on the clinical picture and laboratory findings, lead poisoning was suspected. Blood lead level was 109 microgram/dL. More detailed history was obtained from his mother who admitted using surma (kohl) on the umbilical cord stump for about 15-20 days after birth and in both eyes until admission to hospital. She brought a sample of gray surma that she bought from the local market. The family lives in a new house in Hamad Town with no recent renovation or paint removal.

Ca EDTA was given intramuscularly for 5 days. Subsequently lead level gradually dropped to 47 microgram/dl then to 27 microgram/dl.

In the last follow up, at the age of 2 years, he showed no fine or gross motor developmental delay. However, his speech is delayed with no sentences yet at 2 years of age. He is also described as an "overactive" child.

DISCUSSION

In many countries, acute lead encephalopathy is considered a problem of the past, but this does not seem to apply to many other societies. Several recent reports have described acute lead encephalopathy in infants and young children mainly due to the use of cosmetics, Bakhoor and Farouk[4-10].

Kohl (surma) is an eyeliner used by women in the Middle-East, Africa and Asia. Kohl has also been used as an astringent applied on the umbilical cord of newborns. And applied in the eyelids and conjunctive for beauty and to prevent ocular diseases respectively.

The original Kohl contains antimony sulfide and trisulfide as it's main constituents. Its source is a black stone known in Arabic as "ithmed", which translates to antimony in English and Surma in Urdu. Due to the scarcity of ithmed many manufacturers resorted to the use of galena (lead sulfide) as a cheaper and more easily available substitute[11]. This lead to the saturation of the market with poisonous and poorly controlled preparations.

In a study of lead level in Kohl used in Bahrain, the concentration of lead varied greatly and ranged from 0.07 to 150 mg/g, with an average value of 18.5 mg/g. Of interest, is the finding that the colour of the sample reflected the concentration of lead. The gray coloured specimens contained far more lead than the dark black samples, 38 mg/g vs 6.9 mg/g (P value < 0.001)[12].

Samples of Kohl available in Saudi Arabia (1/3 of Kohl available in Bahrain market is imported from Saudi Arabia) were studied for their microbial content and found to be heavily contaminated with several species of Bacilli and a number of fungi[13].

Obviously, the most serious aspect of Kohl use is lead poisoning. Acute lead encephalopathy has been well documented after liberal use of Kohl in the eyes and its application on umbilical stump. Shaltout et al in a study of 20 infants in Kuwait with lead encephalopathy found Kohl to be the source in at least 11 of these cases[5]. Abdulla and Yaish et al reported six and ten Saudi children respectively with lead encephalopathy due mainly to the use of a lead containing powder to rub over the gums to stimulate teething. In one case, the source of lead was inhalation of Bakhoor, intended to chase the devils and cure several illnesses[6-9].

Acute lead encephalopathy in infancy manifests as irritability, refusal of feeds, vomiting and seizures that may culminate in coma and death. Other symptoms include constipation, nausea and anorexia. Hypochromic – microcytic anaemia is very common and results from

reduced heme synthesis, altered globin synthesis and reduced red cells survival due to increased fragility and resultant haemolysis. Our patient exhibited all the aforementioned manifestations. In addition acute lead poisoning can lead to renal tubular damage and Fanconi-like syndrome.

With the decline in cases of acute lead poisoning, attention has been directed to chronic sub-clinical lead poisoning. Chronic low dose exposure to lead is far more common and results in subtle and non-specific signs and symptoms. Of considerable concern is the negative impact of low dose chronic lead poisoning on mental abilities and behaviour of children[14-16]. Several studies have documented IQ deficit and disturbed adaptive behaviour among children with a lead level > 10 microgram/dl[17]. The findings of these and other studies prompted the United States Centre for Disease Control (Atlanta, GA) to change the definition of lead toxicity from 25 microgram/dl to 10 microgram/dl[18].

The average blood level among Bahraini children is 15microgram/dl with a range of 7.5 microgram - 27.5 microgram/dl[19]. By international standard, this is a toxic level and of considerable concern.

Although, acute lead toxicity is not as common as chronic lead toxicity, it still exists in our society and merits our attention. We need to maintain a high index of suspicion whenever faced with encephalopathy in an infant. More importantly this tragic event is preventable by simple measures such as the avoidance of child's exposure to Kohl, Bakhoor, Farouk and other sources of lead.

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