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Case Report

Lead toxicity in a new-born due to in utero exposure

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ABSTRACT

Lead poisoning in a neonate is poorly defined, and limited data exists on appropriate follow-up and treatment of such infants. We are presenting the case of a newborn infant, who had a lead level of 63 mcg/dL. Treatment involved five days of intravenous chelation therapy. At discharge, no clinical sequelae of lead toxicity were found. However, due to the chronic nature of in utero exposure the infant requires close follow-up, in particular neurologic and developmental sequelae. Lead toxicity has many complications. Long-term complications include delays in growth and development. Furthermore, these complications may develop in children with minimal toxicity, let alone those with grossly abnormal values. Due to lack of data, perhaps it is worthwhile to screen those women of child-bearing age, who are of "high risk", for elevated blood lead levels to reduce the risk of in utero exposure.

Keywords: In utero exposure, Lead, Neonatology

INTRODUCTION

Lead is a metal that exists in 4 isotopic forms and all forms are considered toxic. The threshold level of lead that causes clinical, subclinical, and biochemical changes remains to be determined. Lead has multiple effects on cells as it affects several enzymes diminishing function. It prevents the development of tertiary brain structures by inhibiting the normal neuronal pruning process, resulting in poor neurodevelopment, cognitive outcomes, and behavioral disorders.[1]

No safe BLLs have been identified in children.[2] The current value of 5 mcg/dL is used by clinical and public health care providers for children.

There is also limited evidence on the treatment of lead poisoning in the neonate. Chelation therapy with assistance from an expert in the field remains the mainstay of management. There are no specific guidelines for which chelating agents to use, duration of treatment, and dose of agents for neonatal plumbism. Ultimately, the result of treatment should stop further damage and reverse any prior lead effects.[1]

CASE REPORT

A full-term female infant was born to a 28-year-old via spontaneous vaginal delivery at 37 6/7 weeks of gestation; rupture of membranes was less than 12 hours prior to delivery. APGARs were 9, 9 at 1 and 5 minutes respectively. Her prenatal course was unremarkable until 33 3/7 weeks of gestation when a blood lead level (BLL) was 44.6mcg/dL.

Upon further questioning, regarding exposure and her eating habits, she admitted to having eaten cheese and chocolate brought in from her native country, Ecuador, as well as Mexican candies. She also admitted to ingesting "ice from her freezer" throughout her pregnancy, suggestive of exposure through her diet and PICA. A home visit performed by the New York City Department of Health (NYC-DOH) found elevated lead levels in the windows, doors, and peeling paint. Upon consultation

with lead poisoning experts, she underwent 5 days of chelation therapy with IV Edetate disodium calcium (CaNa2EDTA) and immediately post-chelation, a BLL was 5 mcg/dL.

At the time of delivery, which was 4 weeks post-maternal chelation, mother and baby's BLL was 33 and 63 mcg/dL, respectively. The neonate underwent chelation therapy with CaNa2EDTA for a total of 5 days. Baseline and sequential complete blood count (CBC), comprehensive metabolic panel (CMP), urinalysis, and urine output were monitored and found to be normal. An X-ray of the knees was obtained to look for any chronic changes; also reported as normal. A repeat pre-chelation lead level drawn on day 2 of life, was 46 mcg/dL. We attributed the discrepancy in baby's BLL pre-chelation to be the result of haemoconcentration; as the initial haematocrit was found to be elevated.

On post chelation days 3 and 6, BLLs were 19 and 10 mcg/dL, respectively. Baby was discharged and a follow up BLL at 21 days post chelation showed a moderate increase to 18 mcg/dL.

DISCUSSION

Our case report identified a neonate exposed to in utero high levels of lead, presumably early in gestation, since the maternal lead level was already 45mcg/dL at 33 weeks of gestation. During pregnancy, a foetus can be exposed endogenously from lead accumulated in the mother's blood or bones due to past exposure; we believe the latter occurred in our case.

Lead poisoning can occur in utero since lead crosses the placenta by passive diffusion, accumulating in fetal bones, brain, and other organs. In the present case, we found a rebound lead level of 18 mcg/dL at 21 days post-chelation, reflecting lead levels in the baby's tissue. The Center for Disease Control and Prevention (CDC) as well as The American Congress of Obstetricians and Gynecologists (ACOG) do not recommend routine screening of all pregnant women for BLL. Rather, only those women who are considered to be high risk should be screened. These include, but are not limited to, women that are exposed to lead via lifestyle habits, which includes cookware, cosmetics, contaminated water, and so forth.[3] In the present case, the mother was screened because of a spouse working in the construction industry, as well as the mother admitting to eating foods brought in from abroad.

Lead can potentially affect any system or organs in the body. Elevated BLLs in pregnancy has been associated with hypertension, low birth weight, spontaneous abortions, and impaired neurodevelopmental outcomes of the fetus.[3]

A recent cohort study in New Zealand of children exposed to lead level of 10.9 mcg/dL, showed a decline in IQ and socioeconomic status during adulthood after being corrected for maternal IQ, childhood IQ, and childhood socioeconomic status, suggesting that lead toxicity persists into adulthood.[4] We did not observe any neurologic sequelae during the hospital course of our patient. However, the long-term outcome remains to be seen.

In the hematologic system, lead can inhibit several enzymes in heme synthesis, leading to injury of the surface of erythrocytes and decreasing their lifespan.[5] In our case, this effect was manifested as prolonged hyperbilirubinemia requiring phototherapy with a peak bilirubin level of 11.1 mg/dL at 49 hours of life, plotted on the 95th percentile, which remained above the 95th percentile for several days.

Nephrotoxicity includes proximal tubular nephropathy, glomerular sclerosis, and interstitial fibrosis and related functional deficits, proteinuria, impaired transport of organic anions and glucose, and depressed glomerular filtration rate. Furthermore, in addition to the direct effects on the kidney, lead affects the metabolism of Vitamin D and calcium.[5] In our patient we did not observed any renal or calcium metabolism effect as demonstrated by normal urinalysis and renal function tests, as well as normal serum calcium levels.

The American Academy of Pediatrics (AAP) recommends exclusive breastfeeding for the first 6 months of life. Limited data exists suggesting that maternal breast milk has a limited impact in increasing BLLs of infants. Nevertheless, in those infants exposed in utero to high lead levels, breast milk may increase their BLL over and above recommended safe levels. Women exposed to lead are encouraged to breastfeed as long as BLL are <40mcg/dL. Women with higher BLLs should be advised to not breastfeed and instead pump and discard their milk until their BLL is <40mcg/dL. In infant's whose BLL rises to >5mcg/dL, breastfeeding should be discontinued until a source can be identified and exposure terminated. Women with levels <20mcg/dL are unlikely to cause significant increases in infant BLL; however, it is recommended that these infants undergo testing of their BLL to determine a baseline value. In women with BLL between 20-39mcg/dL, no data exists regarding the benefits of breastfeeding and the risk of exposure. Therefore, the CDC recommends that these mothers be advised cautiously, with follow-up beginning at 2 weeks postpartum and subsequently at 1-3-month intervals.[1] In the present case, we advised the mother to not breastfeed as the infant's BLL was >5mcg/dL and the source for the infant was clearly the mother who had a BLL of 33mcg/dL at the time of birth.

Four pharmacologic agents are used in the US for neonatal plumbism - CaNa2 EDTA, DMSA (2,3 Dimercaptosuccinic acid), BAL (Dimercaprol), and PCA (Penicillamine). All of these agents increase the excretion of lead, primarily via kidneys.[1] PCA and DMSA are

given orally so can be used in the outpatient or inpatient setting. Dimercaprol and Calcium Edetate are given parenterally in a hospital setting. Calcium Edetate is given for 5 days intravenously and is the most studied chelator.

In the present case we treated with CaNa2EDTA because it has been the most studied chelating agent. We did not observe any short-term side effects related to treatment; renal function and cardiac side effects were not observed.

CONCLUSION

In the United States, despite public health education and environmental policies on the toxic effects of lead; lead exposure still exists. Identification and eliminating exposure among pregnant and women of childbearing age improves maternal and infant outcomes. Perhaps advising women of childbearing age, who are considered to be "high-risk", to have BLLs performed, may potentially reduce in utero exposure.

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