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Neonatal Plumbism Secondary to Maternal Chronic Lead Poisoning and PICA: A Case Report

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Abstract

Plumbism, (lead toxicity), has gained increasing attention secondary to its ubiquitous distribution in the environment and its potentially serious medical complications, particularly in children. Although the incidence of lead poisoning has decreased since the 1970's, it is estimated that 310,000 children in the United States are at risk for exposure to harmful lead levels. As lead easily crosses the placenta, fetuses are readily susceptible to lead intoxication from maternal lead exposure. A case of neonatal plumbism secondary to maternal chronic lead toxicity is presented herein. Upon delivery of the infant, the infant was noted to have secondary lead toxicity from its mother- evident from blood lead levels as well as clinically. The infant was treated for its lead poisoning.

Background

Plumbism, (lead toxicity), has gained increasing attention secondary to its ubiquitous distribution in the environment and its potentially serious medical complications, particularly in children. Although the incidence of lead poisoning has decreased since the 1970's, it is estimated that 310,000 children in the United States are at risk for exposure to harmful lead levels. Secondary to the use of lead paint in non-renovated city housing such as Detroit, Michigan, housing and the presence of lead pipes in old plumbing systems, lead poisoning is ever-present at high proportions. As lead easily crosses the placenta, fetuses are readily susceptible to lead intoxication from maternal lead exposure. A case of neonatal plumbism secondary to maternal chronic lead toxicity is presented herein [1].

History of Present Illness

A 17 year-old African American G1P1 at $40^{3/7}$ weeks presents to labor and delivery an inner city hospital. The patient gives a history of probable lead poisoning since age two or three, likely secondary to water polluted from household lead pipes. She also admits to a history of PICA in which she eats dirt and stones as well as silk clothing. During her pregnancy, the patient had a prior admission to a nearby hospital for lead intoxication in which her lead level was equal to 50 mcg per deciliter. During this hospital admission, toxicology was consulted and did not suggest chelation therapy at that time as the patient was asymptomatic. It was recommended that her diet be supplemented with iron, calcium, thiamine, and folate, that she avoids eating non-food items, and that she be followed up in the Medical Center's "lead clinic." The patient's home was inspected the following month and was cleared.

Prenatal care included an official ultrasound four months prior to labor that showed a normal amniotic fluid index with normal fetal weight and anatomy. During the patient's admission for her labor, toxicology was consulted to rule out neonatal plumbism [2, 3].

Past medical history

Chronic plumbism, head injury as a child.

Past surgical history

None

Medications

None

Allergies

NKDA

Social history

Patient is adopted. Denies tobacco, ETOH, illicit drugs.

Family history

Chronic Plumbism

Hospital course

Delivery: The patient is admitted to labor and delivery, and the toxicology attending is at bedside during her delivery. Patient

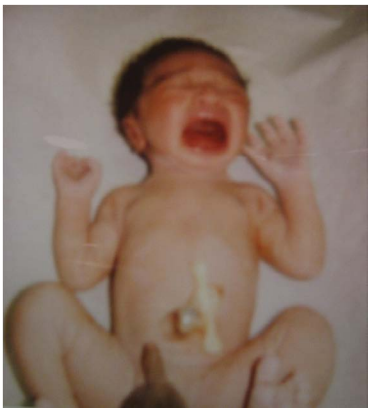

Figure 1: Infant one minute post-delivery.

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undergoes spontaneous vaginal delivery of a viable male infant with APGARS of 9^1 9^5 (Figure 1). The infant was immediately assessed by pediatrics as alert, awake and in no apparent distress. The infant did not show signs of encephalopathy. Infant was not apenic, and he had good tone in all extremities. The infant's head circumference was measured as small for gestational age. At that time, the toxicologists did not feel it was necessary for an emergent exchange transfusion for the infant. The infant and mother's blood was drawn to assess lead levels. Additionally, radiographs were ordered to rule out metaphaseal lead lines.

Post-partum day one

The mother's lead level was 28 (normal <10) and infant's lead level was 28. In their note, toxicology concluded that the infant had significant exposure to lead throughout gestation. It was also noted by nursing that the infant had some "minor issues in feeding." The toxicologists indicated that any elevation of lead in the infant is considered toxic. The chief physician of the *Lead Clinic* was consulted, and he recommended that the infant should undergo chelation immediately while the lead was available in the blood to prevent sequestration into the soft tissues and bone. He recommended DMSA/ Succimer and IV $CaNa_2EDTA$ therapy with concurrent multivitamin and iron supplementation for five days. Additionally, a 24-hour urine over five days was collected. It was planned to re-check the infant's lead levels two weeks post chelation therapy. Also, it was recommended that the infant not breastfeed.

Post-partum day two

The infant underwent naso-gastric tube inserted secondary to vomiting, and a PICC line was placed so chelation therapy could begin. According to the toxicology evaluation of the right leg, a metaphaseal lead line was noted at the distil femur. Yet, per the official radiology report, no lead line was noted. Abdominal x-ray did not show lead flecks.

Post-partum day three

On examination, it was noted that the infant had a small amount of subcutaneous tissue and minimal crying. *Mees lines* were also noted in the infant's nails on examination (Figure 2). On blood smear, no basophilic stippling or anemia was noted. On hearing screen, both right and left ears failed.

Infant's clinical manifestations of plumbism [4]

Increased blood lead level

Mees lines on nails

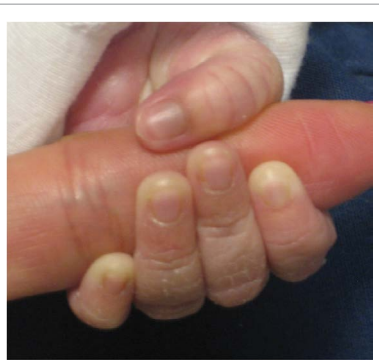

Figure 2: Mee's lines in infant's nails.

Bilateral failed hearing exam

Questionable metaphaseal lead line

Discussion

Lead freely crosses the placenta. Consequently, gestational lead poisoning is both harmful to the mother, but also to the developing fetus, invariably producing congenital lead poisoning. Lead poisoning is diagnosed when the blood lead level is greater than 10 mcg/dL [5].

Lead poisoning in children presents as developmental delay, loss of milestones, and encephalopathy. In addition to cognitive and behavioral problems, lead poisoning can cause hearing loss, peripheral neuropathy, and decreased nerve conduction velocity. The hearing loss occurs primarily in the higher frequencies and may contribute to learning and behavioral problems. Other signs and symptoms of lead poisoning include irritability, hyperactivity or lethargy, sleeplessness, poor appetite, headaches, vomiting, constipation, abdominal pain, ataxia, somnolence, and seizures [6, 7].

Children, particularly between 12 and 36 months old, are more susceptible to the toxic effects of lead because they have an incomplete blood-brain barrier that permits the entry of lead into the developing nervous system. These children also have a greater prevalence of iron deficiency which may allow for increased absorption of lead from the gastrointestinal tract.

Lead is renally excreted which helps individuals to avoid persistently high free blood lead levels. Yet, during the first days of life, newborns may suffer from a functional renal insuffiency. The half-life of lead varies depending on the body compartment. The half-life of lead in the blood is 28-36 days, in the soft tissue is 40 days, and in mineralizing tissues (bones and teeth) is greater than 25 years. More than 70 percent of the total body burden of lead in children is contained in the mineralized tissues. Therefore, the blood lead level is not a good reflection of the total body lead burden. In the minieralizing tissues, the lead accumulates in two sub-compartments: a labile compartment that readily exchanges lead with the blood, and an inert pool of lead that can be mobilized during periods of physiologic stress (pregnancy, lactation, fractures, chronic disease, etc). The inert pool can cause the blood lead level to remain elevated long after exogenous exposure. Additionally, as the body can accumulate lead over a lifetime and releases it slowly, lead toxicity may occur without a major acute exposure.

As lead can be consumed from contaminated water, it can also gain access to the human body by PICA behavior. PICA, (which is Latin for *magpie*, a bird notorious for eating almost anything), is defined as the practice of craving substances with little or no nutritional value. Most PICA cravings include non-food items such as dirt, chalk, pottery, stones, paint etc. PICA may be seen in 25-30% of children. Although there is no identified cause of PICA, it has been associated with iron deficiency. Some individuals have speculated that PICA is the body's attempt to obtain vitamins or minerals that are missing in an individual's diet. Yet, some cravings may be related to mental illness [8, 9].

It is agreed that reducing neonatal lead burden as quickly as possible reduces the risk for developmental delays. Parenteral chelation and exchange transfusion have been used to reduce elevated blood lead levels [10].

Although immediate sequela of plumbism has been noticed in this

neonate's first day's of life, further is yet to be revealed and the neonate develops and matures in childhood.

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