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Individualized support for breastfeeding in a case of elevated blood lead levels: A case report

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ABSTRACT

Introduction: Lead is a known toxicant that affects all tissues in the body, most notably the brains of developing children. However, there are limited data on the dynamics of lead transfer via breastmilk and its short-term and long-term consequences. Much of the available data come from areas of the world where numerous sources of lead complicate our understanding of the effects of lead exposure via breast milk.

Case Presentation: We present trends in blood lead levels in a breastfeeding dyad, where the only source of lead exposure identified was prior to pregnancy, without other known ongoing lead exposures for the lactating parent or infant.

Discussion and Conclusions: In this case, all lead exposure in the infant was presumed to come from in utero transmission and breastfeeding; and infant blood lead levels varied significantly with initiation and interruption of breastfeeding. This case is discussed in the context of current models for predicting transfer of lead in breastmilk and highlights pathophysiologic considerations for understanding lead transfer in the breastfeeding dyad.

1. Introduction

Lead exposure and poisoning remain a pervasive issue around the world despite efforts to regulate the use of lead in commercial products and to minimize exposures. Lead poisoning can impact people across the lifespan and can start as early as the gestational and lactational period if the birthing or lactating parent has an elevated blood lead level (BLL). In the United States (US), it is recommended that all pregnant individuals be screened with a lead risk assessment questionnaire, followed by BLL testing for those who are found to be at risk of exposure. Risk factors include being born outside the US, use of imported goods (medications, cosmetics, spices, lead-glazed pottery), pica, exposure to lead dust due to recent home repair or renovation, and occupational or recreational exposures [1].

Lead is a heavy metal that causes negative effects on all tissues in the body. It crosses the placenta and blood brain barrier and can have significant impacts on the growth and development of children. Early risks include fetal loss; survivors may have lower birth weight, shorter length, smaller head circumference, and poor weight gain in the first month of life [2]. Long term consequences include effects on cognition [3], behavioral disorders [4–6], delayed puberty in girls [7], higher blood pressure in adolescent girls [8], and shorter stature, particularly in combination with zinc deficiency [2]. Shekhawat and colleagues found that among umbilical cord blood lead levels (BLLs) in the 5–10.5 μg/dL range, higher umbilical cord blood levels were associated with delayed motor development [9].

Lead is also transferred via breast milk. Among the toxic heavy metals, lead is one of the most likely to be detected in breast milk at levels above the WHO suggested limit, as shown in multiple studies across various regions [10–12]. In a study of breastfeeding dyads in Mexico, Ettinger and colleagues found that breast milk lead accounts for 12 % of variation in infant BLLs, after controlling for umbilical cord BLL, infant weight change, and breast-feeding practice [13]. Cumulative lead exposure seems to play a role in infant BLL, with higher BLL associated with longer duration of breastfeeding [14].

While numerous studies have examined the effects of prenatal lead exposure and transfer in breast milk, no controlled studies comparing breastfeeding versus non-breastfeeding in prenatally exposed infants have been reported, making it difficult to isolate the impact of ongoing lead exposure via breast milk. This case offers additional insight into trends in BLL during the delayed initiation, pause, and resumption of breastfeeding in a prenatally exposed infant without other known

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ongoing exposures.

2. Case presentation

A 37-year-old woman was found to have an elevated BLL of 10 μg/dL at 15 weeks gestation. She had a history of 3 healthy full-term deliveries and no known miscarriages. The source of lead was unclear. She was born in Mexico and moved to the United States 11 years prior. Her home was in good condition and no sources of lead exposure were identified by the Department of Health. The drinking water was not tested. She worked in a restaurant. Her husband worked in plumbing but changed clothes at work and had an undetectable BLL. She reported eating some imported foods about 1 year prior. She denied the use of any additional imported items. She had no history of gunshot wound, pica, or non-nutritive hand/object to mouth behavior. Follow up lead levels were notable for BLL of 20 μg/dL at 22 weeks gestation, 26 μg/dL at 33 weeks gestation, and 24 μg/dL on the day of delivery. The infant was born at 39 weeks, with appropriate for gestational age weight of 3.520 kg and length of 0.508 m. Cord BLL was 28 μg/dL, and infant BLL at 3 days old was 35 μg/dL. The mother wished to breastfeed.

Trends in maternal and infant blood level are shown in Fig. 1. Maternal and infant BLLs were checked each month. Levels were checked more frequently if the BLL was noted to rise unexpectedly. There was one missed infant level between 19 and 27 weeks postpartum. The BLLs from 6 weeks prior to delivery through 18 weeks postpartum consisted of a venous sample drawn in a lead-free tube and measured using atomic absorption spectroscopy at our center; the remainder of the labs were measured by inductively coupled plasma mass spectrometry at a commercial lab.

Initiation of breastfeeding was delayed, and the mother pumped and discarded milk to maintain supply. The infant's BLL decreased from birth to 4 weeks. At 4 weeks postpartum, maternal BLL was 30 μg/dL and infant BLL was 22 μg/dL, and breastfeeding was initiated with close monitoring. At 8 weeks postpartum, the infant's BLL increased to 28 μg/dL as mother's BLL began to decline, and breastfeeding was paused for two weeks. After this pause in breastfeeding from 8 to 10 weeks old, infant BLL declined to 25 μg/dL and breastfeeding was resumed. Thereafter, maternal and infant BLL continued to decline in typical fashion, with decline slowing as BLL reached lower levels. Maternal BLL initially declined more rapidly than infant BLL. Breastfeeding was stopped per maternal choice, and complementary foods were started around 6 months old.

Throughout the pregnancy and postpartum period, dietary supplements included prenatal vitamins and ferrous sulfate 325 mg (65 mg elemental) daily. Calcium carbonate supplement of 1250 mg (500 mg elemental calcium) was prescribed at 10 weeks postpartum. Laboratory studies reflecting maternal calcium metabolism and iron status are shown in Tables 1 and 2, respectively. Table 1 summarizes the findings of vitamin D deficiency without biochemical signs of increased bone turnover, which were measured at 8 weeks when the infant's BLL increased. Table 2 shows evidence of improving iron deficiency anemia from the third trimester to 4 and 8 weeks postpartum.

Tables 3 and 4 show the infant's laboratory studies. Table 3 shows the infant's routine lab studies. At 4 and 8 weeks old, the infant had normal hemoglobin, ferritin, and alkaline phosphatase levels for age. Table 4 shows trends in the infant's zinc protoporphyrin (ZPP), which is a marker of defective heme metabolism and is often followed in chronic

Table 1
Maternal Laboratory Studies, Calcium Metabolism.

	8 weeks postpartum	10 weeks postpartum	Reference Range*
Calcium (mmol/L)	2.4	-	2.1–2.6
Phosphorus (mmol/L)	-	1.3	0.81–1.5
Alkaline Phosphatase (units/L)	68	-	<130
Parathyroid hormone (pmol/L)	5.58	6.21	2.10–8.40
25-hydroxy-Vitamin D (nmol/L)	66.6	64.1	74.7–149

* Reference ranges as provided by the clinical laboratory, not specific to the pregnancy/postpartum period.

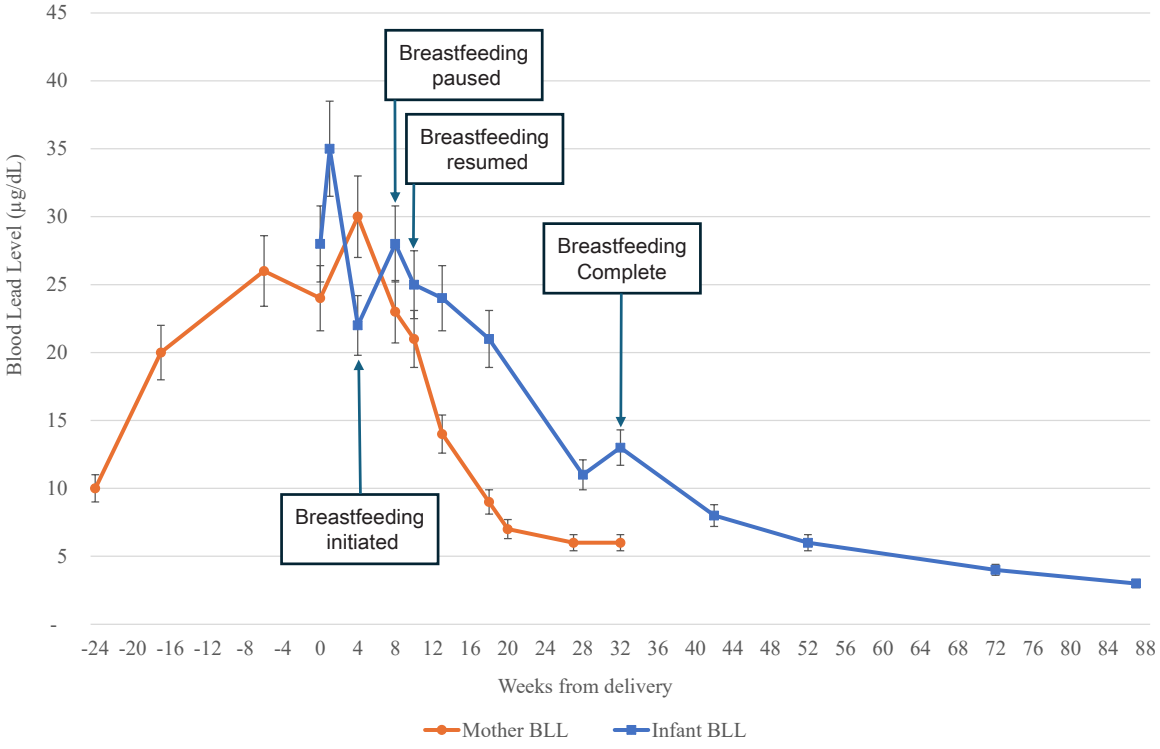

Fig. 1. Mother and Infant BLLs During Pregnancy and Postpartum. Error bars represent lab measurement variability of +/- 10 %.

Table 2
Maternal Laboratory Studies, Iron Status.

	33 weeks gestation (-6 weeks)	4 weeks postpartum	8 weeks postpartum	Reference Range*
Hemoglobin (g/L)	86	109	115	122–153
Hematocrit (proportion)	0.29	0.36	0.37	0.36–0.45
Mean Corpuscular Volume (fL)	77.0	78.3	79.6	80.0–96.0
Mean Corpuscular Hemoglobin Concentration (g/L)	299	299	314	330–360
RDW (proportion)	0.16	0.22	0.21	0.12–0.17
Ferritin (pmol/L)	15	42.2	15	10.0–150.0

* Reference ranges as provided by the clinical laboratory, not specific to the pregnancy/postpartum period.

Table 3
Infant Laboratory Studies, Routine Labs.

	3 days old	4 weeks old	8 weeks old	Reference Range*
Hemoglobin (g/L)	172	106	100	90–140
Mean Corpuscular Volume (fL)	96.8	97.5	90.0	77.0–115.0
Mean Corpuscular Hemoglobin Concentration (g/L)	361	337	338	200–360
RDW (proportion)	0.14	0.13	0.13	0.12–0.16
Ferritin (pmol/L)	-	643.1	454.4	10.0–150.0
Carbon dioxide (mmol/L)	-	24	21	20–30
Calcium (mmol/L)	-	2.53	2.60	2.13–2.63
Alkaline Phosphatase (units/L)	-	351	341	<500

* Reference ranges as provided by the clinical laboratory, not all are age-specific.

lead poisoning. The infant's ZPP increased between 4 and 8 weeks and remained above the upper limit of quantification until 32 weeks postpartum.

3. Discussion

The woman in the case was screened with a BLL due to her country of origin, Mexico. Based on 2022 data from the New York City Department of Health, 87 % of pregnant individuals with elevated BLLs were born outside of the US, and the most common country of origin was Mexico (58 %) [15]. Sources of lead exposure vary by country of origin. The most common sources of lead exposure in Mexico overall include glazed ceramics, lead-contaminated utensils, and lead-contaminated water [16]. Studies of pregnant individuals in Mexico have found that the main predictors of higher BLL for this population are occupational exposure, use of lead-glazed pottery, and consumption of contaminated soil [17]. For the woman in this case, consumption of imported food items prepared using lead-contaminated water, cookware, or utensils may have contributed to lead poisoning.

The typical U-shaped curve for BLL during pregnancy was not observed, as the maternal BLLs continued to uptrend in the second and third trimesters and immediately postpartum. Typically, there will be a trough in the second trimester, followed by a peak at or near delivery due to increased mobilization of lead stores in bone as fetal calcium demand increases. This curve may vary depending on the chronicity or acuity of lead exposure. Postpartum BLL continues to rise, particularly in lactating individuals [18], suggesting a greater bone lead mobilization during lactation. In this case, the upward trend of the mother's BLL in the second trimester suggests ongoing exposure, such as pica or continued ingestion of imported foods, which are often underreported. Other potential explanations include maternal calcium deficiency leading to accelerated bone resorption or missed visualization of the typical trend due to assessment of first level at 15 weeks gestation.

As noted in the case presentation, initiation of breastfeeding was delayed due to informed projections of maternal BLL. The lactating parent's BLL is used to approximate the amount of lead transferred via breast milk, as there are numerous technical challenges in measuring breast milk lead directly [19]. Based on data from a 2004 study by Ettinger and colleagues on the transfer of lead via breast milk in breastfeeding dyads in Mexico City [13], the CDC provides a model for predicting the degree of increase in infant blood lead level based on lactating parent BLL. For example, when a lactating parent's BLL is 1 μg/dL, the expected increase in the infant's BLL at 1 month old is 0.12 μg/dL. Likewise, when a lactating parent's BLL is 30 μg/dL, the expected increase in the infant's BLL at 1 month old is 3.7 μg/dL. At a lactating parent BLL higher than 30 μg/dL, the data is extrapolated. Based on this information, CDC guidance recommends that breastfeeding should not be initiated when maternal BLL is greater than 40 μg/dL and that serial infant BLL should be monitored closely if breastfeeding is initiated at maternal BLL 20–39 μg/dL [1]. In this case, although cord BLL was only 28 μg/dL at delivery, infant BLL was 35 μg/dL on day of life 3. Based on projections from the data presented by the CDC, the infant's BLL would be at risk of nearing the chelation threshold (≥45 μg/dL) if breastfeeding was initiated at birth, and an individualized decision was made to defer breastfeeding at birth.

The difference in cord BLL of 28 μg/dL and infant BLL of 35 μg/dL on day of life 3 may reflect hemodilution of the cord BLL and hemoconcentration of the day of life 3 BLL. Many fluid shifts occur in the early newborn period, and newborn weight reaches a nadir at 2–3 days of life due to diuresis. The actual BLL at birth likely falls between 28 and 35 μg/dL. The decrease in BLL at 4 weeks of life may also reflect hemodilution, as the blood volume rises from about 85 mL/kg at birth to about 105 mL/kg at 1 month of life [20]. Based on calculations, this change in blood volume alone can account for the decrease observed. However, the decrease of 6 μg/dL from cord BLL to 1-month BLL appears to be high compared to existing studies of breastfeeding dyads, which may raise concern for lab error. In breastfeeding dyads from Mexico, the average umbilical cord lead level was 6.9+/-3.9 μg/dL, and the 1-month infant BLL was 5.6+/-3.0 μg/dL [13]. However, the cohort from Mexico was different because there were additional environmental lead exposures for both mothers and infants, lower maternal BLLs at delivery, and ongoing breastfeeding as a source of lead for the infants. For this case, the lack of ongoing lead sources and the above explanation of hemodilution makes the more drastic decrease in BLL plausible.

The increase in infant BLL after initiation of breastfeeding and the subsequent decrease in infant BLL after a 2-week break from breastfeeding without other interventions suggests that the source of lead was transfer via breast milk. The CDC projections are based on data between birth to 1 month, but if this model is applied to this slightly later timeframe, a maternal BLL of 30 μg/dL predicts that the infant BLL would increase by 3.7 μg/dL in a month. In this case, the infant BLL increased by 6 μg/dL in a month. Assuming sample measurement variability of

Table 4
Infant Laboratory studies, Zinc Protoporphyrin (ZPP).

	4 weeks old	8 weeks old	10 weeks old	13 weeks old	18 weeks old	32 weeks old	Reference Range*
ZPP (μg/dL)	89	>120	>120	>120	>120	79	<100

* Reference range as defined for adults with occupational exposure

+/-10 %, the actual increase in BLL from 4 to 8 weeks may range from 1 to 11 μg/dL. Since the predicted increase of 3.7 μg/dL falls within this range, it would be reasonable to say that this increase in BLL was solely caused by breastfeeding. However, there are limited data to support the application of this model to this later time frame, and other contributors to increasing BLL should be considered. Though this case is a single example, it highlights other factors that may contribute to increases in infant BLL in a breastfeeding dyad.

Some degree of hemoconcentration may help explain the increase from 4 weeks to 8 weeks, as the blood volume per kilogram is expected to decrease gradually after the peak at 1 month old [20]. Though the trend in hemoglobin does not reflect hemoconcentration, trend in hemoglobin may not be an accurate proxy for hemoconcentration in this age range due to newborn hemoglobin trending towards physiologic nadir around 3–4 months of age.

The timing of the maternal BLL peak may also contribute to the degree of increase in infant BLL. Though the recorded peak in maternal BLL was 30 μg/dL at 4 weeks postpartum, the postpartum/lactational peak may have occurred after this measurement, contributing to higher infant BLL than expected based on the CDC model.

Changes in lead content of breast milk over time may also contribute. Lead content in breast milk has also been found to vary with breast milk fat content and the changes in breast milk that occur over time, though the timing is highly variable. Several studies demonstrated a trend toward higher lead concentrations in colostrum and lower concentrations as lactation progresses [21–23], though lactating parent BLLs were not available for correlation in these studies. Other studies have attempted to quantify a milk/blood ratio or milk/plasma ratio for lead [19,24], which is quite variable and not widely available, likely due to technical challenges in breast milk lead measurement, differences in lead content in colostrum versus mature milk, environmental contamination, and differences in the rate of transfer to breast milk. In this case, measurements of breast milk lead were not available.

The potential for maternal calcium deficiency at the time of breastfeeding initiation should also be considered, as a lactating individual's diet can also impact trends in breast milk lead and infant BLL. Adequate calcium intake in lactating individuals has been shown to mitigate the impact on infant lead levels [9,25,26], so it is recommended that lead-exposed lactating individuals consume 2000 mg/day of calcium through diet or supplementation [1,27]. Sufficient vitamin D is essential for efficient calcium absorption. In this case, the mother's reported diet and supplementation reflected adequate calcium intake. Her lab results also reflected adequate calcium intake, with normal total calcium, phosphorus, and parathyroid hormone levels, despite mild biochemical vitamin D deficiency measured at 8 weeks postpartum when the increase in infant BLL was observed (Table 1).

The contribution of maternal iron status may be considered. Increased iron consumption by the lactating individual has also been associated with lower breast milk lead content, though this relationship may reflect decreased ongoing lead absorption in the GI tract [28]. In this case, the mother did have evidence of iron deficiency anemia, which was improved but not resolved at the initiation of breastfeeding. The data was not robust to support maternal iron deficiency as a significant factor in this case.

The increase in infant BLL may also represent an environmental source, but no such history was elicited in this case. It may also represent the feeding of breastmilk expressed in the first month postpartum, but this milk was reported to have been discarded. Although the mother may have potentially ingested lead-contaminated food during this period, a corresponding increase in maternal BLL was not observed.

Between two months and six months postpartum, the infant's BLL declined appropriately but more slowly than maternal BLL, which may reflect ongoing exposure to lead via breast milk but may also reflect the slower elimination of lead by children as described in the literature [29]. There is limited literature correlating the BLL of lactating individuals and their infants throughout the lactation period, likely due to heterogeneity in feeding patterns and numerous variables which make generalization difficult. However, the slower decline in infant versus maternal BLL is interesting to note in this case, where the only identified source of lead exposure was breast milk.

4. Conclusion

Current models provide an excellent foundation for appropriate decision-making about breastfeeding in the case of maternal lead poisoning, but ongoing research is needed to better understand the complexities of lead transfer in lactation. This case highlights important pathophysiologic considerations for applying existing guidelines to lead-poisoned breastfeeding dyads at different points in their breastfeeding journey.

Glossary

- Blood lead level: Blood lead level refers to measurement of lead concentration in whole blood sample. In clinical laboratory settings, this level is measured by atomic absorption spectroscopy or inductively coupled plasma mass spectrometry.
- Atomic absorption spectroscopy Atomic absorption spectroscopy is a technique for measuring lead, in which the sample is atomized via flame or electrothermal device, and characteristic wavelengths of light are absorbed by lead atoms in the sample. The amount of absorption is linearly related to the concentration of lead in the sample. [30]
- Inductively coupled plasma mass spectrometry: Inductively coupled plasma mass spectrometry is a technique for measuring lead, in which an inductively couple plasma is used to dissociate the sample into atoms or ions, which are then passed through a mass spectrometer.
- Lead exposure: Lead exposure refers to lead being present in a person's environment. Lead exposure may or may not be accompanied by detectable blood lead levels.
- Lead poisoning: Lead poisoning refers to the presence of a detectable blood lead level.
- Zinc Protoporphyrin: Lead poisoning, particularly at BLL >20 μg/dL, is known to impair the function of ferrochelatase, the enzyme responsible for the insertion of iron into protoporphyrin [31]. When iron is not properly inserted into protoporphyrin, increased levels of zinc protoporphyrin are formed. Therefore, zinc protoporphyrin is often followed in the case of chronic lead poisoning as a marker of lead's impact on heme metabolism.

List of Abbreviations

BLL- blood lead level
CDC- Centers for Disease Control and Prevention
US- United States
WHO- World Health Organization
ZPP- Zinc Protoporphyrin

Ethics approval and consent to participate

Not Applicable

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Authors' information

AW, JM, and MM work at the Lead Poisoning Prevention and Treatment and Program at The Children's Hospital at Montefiore. Founded in 1969, this program is the oldest and most comprehensive of its kind. The Program is recognized both nationally and internationally

as a model for others focused on the prevention and management of lead poisoning in children and pregnant and lactating women.

CRediT authorship contribution statement

Morri Markowitz: Writing – review & editing, Supervision. **Joann Mercedes:** Writing – review & editing, Investigation. **Ashley Wallace Wu:** Writing – original draft, Visualization, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Consent for publication

Written consent for the publication of this case report was obtained from the patient.

Data Availability

Data will be made available on request.

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