JPPT | Clinical Vignette

# Pediatric Lead Chelation Managed During Critical Medication Shortages: Case Report and Literature Review

Mahi K. Singh, MD; Jason M. Kane, MD, MS; and Sana J. Said, PharmD

Lead poisoning in children has the potential for devastating neurodevelopmental consequences. There is significant socioeconomic disparity in children with lead poisoning. Specific lead chelation regimens have been approved for children by the US Food and Drug Administration, however in the United States, there has been a recent national shortage of the primary therapy, edetate calcium disodium (CaNa2 EDTA). This case report presents a 23-month-old child with severe symptomatic lead poisoning during a national shortage of CaNa2 EDTA to highlight the need for advocacy regarding critical medication shortages, especially for antidote therapy. The infant's initial blood lead level was 364 mcg/dL and he received a continuous infusion of CaNa2 EDTA (1000 mg/m$^2$/day), as well as dimercaprol (4 mg/kg intramuscularly every 4 hours). The supply of CaNa2 EDTA was exhausted on day 3 of therapy so he was transitioned to enteral succimer monotherapy. Initial parenteral therapy of 72 hours achieved a lead level of 72 mcg/dL; he then completed his enteral course of succimer along with environmental mitigation. However, elevated blood lead levels persisted and he subsequently required 3 more courses of enteral succimer, and he continues to have detectable blood lead levels 2 years after initial presentation. In the face of medication shortages including CaNa2 EDTA, and now also dimercaprol, clinicians must create and study alternative chelation therapy regimens for pediatric lead toxicity. Furthermore, public policy initiatives, including the development of a national supply stockpile of chelation agents, must be created in order to minimize supply chain disruption and ensure adequate and equitable antidote therapy for lead poisoning outbreaks.

**ABBREVIATIONS** BLL, blood lead level; CaNa2 EDTA, edetate calcium disodium; CDC, US Centers for Disease Control and Prevention; ED, emergency department; FDA, US Food and Drug Administration

**KEYWORDS** chelation therapy; drug shortage; lead poisoning; pediatrics; socioeconomic disparities in health; strategic stockpile

J Pediatr Pharmacol Ther 2024;29(5):544–549

DOI: 10.5863/1551-6776-29.5.544



## Introduction

Lead is a toxic metal that can have serious and devastating short- and long-term health consequences for children. Acute lead intoxication can cause gastrointestinal complaints, encephalopathy, and anemia.[1] Chronic lead exposure can lead to widespread end-organ damage, including learning and behavioral disorders,[2,3] growth failure, renal impairment, and even death.[4] Intellectual development may also be negatively affected as there is a direct correlation between blood lead level (BLL) and decrements in childhood intelligence quotient. In fact, an increase in BLL from 10 to 20 mcg/dL has been associated with a decrease of 2 to 3 IQ points.[2] Despite well-known adverse consequences of lead toxicity in children, sporadic cases of lead poisoning continues to be a challenge to public health in the United States.[5,6] Furthermore, although overall lead toxicity incidence is decreasing in the United States, outbreaks of lead poisoning in children continue to occur. Of additional concern, certain minority groups are disproportionately affected.[7]

There are a number of risk factors that influence the annual number of lead poisoning cases in the United States. Lead-based paint was used in homes and other buildings until the 1970s. When lead-based paint chips or peels, it can release lead dust into the air. Additionally, lead can be found in soil and water. Lead has also been found in a variety of imported products, including in spices, cosmetics, toys, and ceramics.[1,6] As a result of normal exploratory behavior and poor hand-washing, children may ingest lead paint chips, dust, or soil, which can cause elevated serum lead levels and toxicity.[1] Furthermore, many municipalities have lead pipes as the main conduit for the supply of drinking water, which has the potential to cause an epidemic of lead poisoning to an entire community.[5] There is significant socioeconomic disparity in children with lead exposure with lower socioeconomic groups disproportionately affected by lead poisoning.

Fortunately, if severe lead toxicity is recognized before long-standing neurodevelopmental issues have set in, antidote therapy with lead chelation agents can

help remove the toxic heavy metal from the body and perhaps mitigate ongoing short- and long-term toxic effects.[8] Chelation agents contain sulfhydryl groups that bind lead, allowing the resulting complex to be excreted. Succimer (DMSA) and penicillamine are chelation agents that can be given orally, whereas dimercaprol (British Anti Lewisite) and edetate calcium disodium (CaNa2 EDTA) require parenteral administration.[4] If, however, adequate and immediate chelation is not provided, lead atoms can accumulate in solid organs and tissues and become harder to chelate and remove. Lead atoms that accumulate inside the brain can remain there for 1 to 2 years, while lead atoms accumulated within bones may persist for decades.[4] Thus, when indicated, chelation therapy must be initiated urgently to prevent complications related to high lead levels.

Specific lead chelation regimens have been approved for children by the US Food and Drug Administration (FDA) and promulgated by the US Centers for Disease Control and Prevention (CDC) and the American Association of Pediatrics.[9] For children with BLL of 45 mcg/dL to 70 mcg/dL, the CDC recommends hospital admission and treatment with gastrointestinal decontamination and chelation therapy. Asymptomatic children may not require hospitalization if there is certainty that the exposure has been controlled. Initial treatment for these children includes a succimer regimen of 30 mg/kg/day (or 1050 mg/m$^2$/day) orally in 3 divided doses for 5 days, followed by 20 mg/kg/day (or 700 mg/m$^2$/day) in 2 divided doses for 14 days (maximum dose is 1500 mg/day). If the initial BLL is greater than 70 mcg/dL, the recommended chelating regimen is dimercaprol at 24 mg/kg/day (or 450 mg/m$^2$/day) intramuscularly in 6 divided doses for at least 3 days and also CaNa2 EDTA (1000–1500 mg/m$^2$/day or 50–75 mg/kg/day as a continuous infusion or 2–4 divided doses) for 5 days, followed by a full course of succimer if indicated by subsequent BLL.[10,11] Although there is no safe level of lead in the body, and any BLL greater than 0 is considered toxic, a BLL less than 44 in an asymptomatic child can be closely monitored with serial BLL evaluations and also with a formal living environment investigation by public health officials without initiation of chelation therapy.[5,12]

Recently there have been intermittent national shortages of CaNa2 EDTA, dimercaprol, and succimer in the United States, which has put significant strain on the ability of health care providers to administer recommended and effective chelation therapy to children with lead toxicity.[13] Adequate supply of antidote and chelation therapy is mandatory to adequately address the ongoing outbreaks of lead poisoning in children. Medication shortages including those specifically used as antidote therapy have had a deleterious effect on the treatment of children with lead toxicity since alternative regimens are generally understudied for mitigating short- and long-term health consequences, and are not considered gold-standard, evidence-based therapies. We present a case and review of a child with severe symptomatic lead poisoning in the context of a national shortage of CaNa2 EDTA chelation therapy to highlight the need for further advocacy and a call to action regarding critical national medication shortages.

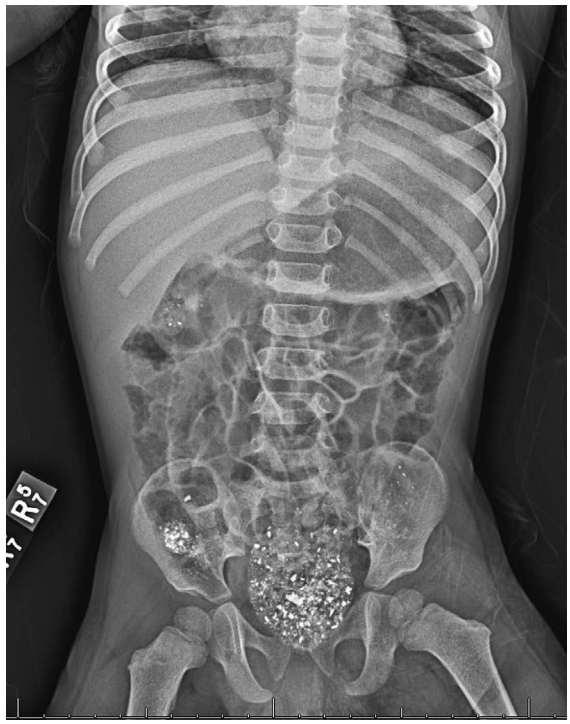
## Case

A 23-month-old male, born full-term and with weight appropriate for gestational age, presented to the emergency department (ED) due to 3 weeks of intractable non-bloody non-bilious emesis. History revealed that the patient was unable to keep any solid foods down despite having a normal appetite. During this time, he continued to have normal daily non-bloody formed stools. There had been no fever noted, and his urine output was appropriate. In the ED, it was noted that the patient's weight was 9.9 kg, which was the fourth percentile for his age. On further clinical history, his mother endorsed that at around 15 months of age, the patient's pediatrician was concerned about possible hearing loss and speech delay. At that visit, the patient received his routine 15-month vaccines and recommended screening laboratory evaluations, which showed a hemoglobin of 11.1 g/dL, hematocrit of 34.2%, and BLL of 3 mcg/dL. He was also referred to audiology by the pediatrician, however was subsequently lost to follow-up until his current presentation. With additional history, the mother endorsed that the patient's speech and language were regressing, and expressed concerns about a delay in his gross motor skills.

Computed tomography of the head was performed and revealed no intracranial pathology. Repeat laboratory examinations including a complete blood count and BLL were performed and revealed a microcytic anemia with hemoglobin of 8.1 g/dL and hematocrit of 24.2%. Abdominal plain radiographs were performed with results as shown (Image). His BLL was found to be 364 mcg/dL with a zinc protoporphyrin concentration of greater than 600 mcg/dL (normal less than 40 mcg/dL). He was admitted to the hospital and parenteral chelation therapy was initiated, as well as laxatives to aid in the excretion of the existing paint chips. The local Poison Control Center was consulted and treatment decisions outlined below were made based on their recommendations.

Due to the severely toxic BLL, he was started on a continuous infusion of CaNa2 EDTA (1000 mg/m$^2$/day), as well as dimercaprol (4 mg/kg intramuscularly every 4 hours) for a planned 5 days of therapy. Topical lidocaine was used to mitigate the injection site pain associated with dimercaprol injections. On day 3 of CaNa2 EDTA therapy, the hospital pharmacy was made aware of a national shortage of CaNa2 EDTA. Attempts at procuring additional supply from local and regional medical centers were unsuccessful. As such, with the

**Image.** Plain radiograph of the abdomen revealing radiopaque foreign bodies scattered throughout the gastrointestinal tract, consistent with lead paint chip ingestion.



supply of CaNa2 EDTA exhausted, the medication regimen was transitioned to enteral succimer monotherapy on hospital day number 4. The decision to stop parenteral therapy in lieu of enteral therapy was made based on published recommendations in consultation with regional poison control and also based on family preference to avoid additional injections. He remained in the hospital for 2 additional days to ensure continued improvement in his BLL and to allow time for home delivery of enteral succimer. The patient was discharged home with a venous BLL of 64 mcg/dL and a planned 19-day total course of enteral succimer regimen of 100 mg 3 times a day for 5 days, followed by 100 mg twice a day for 14 days.

The local Department of Public Health was contacted to conduct home inspections in both the primary home and other extended family members' living environments where the patient also spent significant time. Paint chips containing lead were found in both homes. An alternative temporary living arrangement was established, and all siblings were referred for lead testing. Lead was subsequently mitigated in both homes, and follow-up home inspections found no lead.

Repeat measurement of his BLL 2 months after initial presentation was 73 mcg/dL, and remained elevated at 4 months (56 mcg/dL), and 5 months (53 mcg/dL). The patient remained asymptomatic without any evidence of nausea, vomiting, or encephalopathy. However, due to the persistently elevated BLL, he was prescribed a second 19-day course of enteral succimer. The patient's BLL continued to trend down over the next few months; at 9 months after initial presentation, the BLL was 43 mcg/dL, and at 10 months was 38 mcg/dL. Given the persistent elevated BLL, a third course of enteral succimer was prescribed however with a higher dosing regimen to account for patient growth. The patient received enteral succimer 200 mg 3 times a day for 5 days, followed by 200 mg twice a day for 14 days, with a end-of-therapy BLL of 24 mcg/dL.

Two years after initial presentation, the patient's BLL increased to 32 mcg/dL. Repeat abdominal x-ray was negative for any evidence of new lead ingestion. Parents endorsed that the patient did occasionally chew on his toys or other objects around the house, but they have been vigilant about keeping him from doing so, and deny any chipping or peeling paint on his toys or other objects in the house. Additionally, they denied purchasing any candies or other food items from overseas. Given the persistently elevated BLL and recognizing that current guidelines do not recommend additional chelation at this BLL, extensive discussions with the family and medical toxicologist regarding the utility of additional therapy occurred, and ultimately the patient was prescribed a fourth course of enteral succimer. He remains neurodevelopmentally delayed, but with otherwise appropriate end-organ function. His BLL continues to be monitored in the outpatient setting, and he continues to receive developmental therapies.

## Discussion

Lead poisoning in children has the potential for life-long devastating consequences to neurodevelopmental outcomes. At present, the CDC estimates that approximately 500,000 children in the United States have BLLs at or above 3.5 mcg/dL.[5] As there is no safe level of lead in humans, a number of medical and governmental organizations in partnership with public health organizations have worked to address the problem of lead exposure in children, and have created treatment guidelines to address therapy for lead toxicity.[14] Unfortunately, sporadic outbreaks of lead poisoning continue to occur in the United States. Children who have BLL higher than 70 mcg/dL require hospitalization for parenteral therapy with CaNa2 EDTA. The current national shortage of CaNa2 EDTA has impaired the delivery of gold-standard chelation therapy. Critical shortages of heavy metal chelators have also encouraged the import of products with variable dosing and use guidance.[15] In the event of suboptimal initial chelation with CaNa2 EDTA, increased lead accumulation can deposit in solid tissues, with a persistent slow leaching of lead from these tissues into the blood, contributing to persistently elevated BLL.

In our case, the national shortage of CaNa2 EDTA required the creation of an alternative chelation regimen, which initially reduced BLL. As symptoms improved in the patient and BLL continued to trend downward towards the 70 mcg/dL threshold, transition to an enteral succimer monotherapy was selected in the absence of available parenteral chelator. However, only limited evidence is available regarding the dosing regimen of enteral succimer in pediatric patients. Dosage recommendations suggest either 10 mg/kg/dose or 350 mg/m$^2$/dose every 8 hours for 5 days followed by 10 mg/kg/dose, or 350 mg/m$^2$/dose every 12 hours for 14 days.[16] Dose adjustments are rounded to the available capsule strength and counseling on opening capsules and mixing with soft foods is imperative as the palatability of enteral succimer can present a distinct challenge for medication administration, especially for young children.

Drug shortages are not unique to chelating agents. In a national survey of pharmacists conducted in 2019, all respondents reported experiencing drug shortages in the previous year and more than half responded that the number of medications on shortage had exceeded 50. The list of medication categories experiencing resource interruption was wide ranging, including analgesics, cardiac medications, and even intravenous fluids. The vast majority of respondents reported receiving less than 1 month's notice for most shortages, making management strategy planning challenging.[17]

CaNa2 EDTA has been in shortage in the United States since December of 2020 with variable and intermittent availability.[18] In the event of no available CaNa2 EDTA for acute lead poisoning, the American Society of Health-System Pharmacists recommends initiating succimer 10 mg/kg (350 mg/m$^2$) orally every 8 hours for the first 5 days, then every 12 hours for 14 days (treatment duration of 19 days and maximum daily dose of 500 mg). It is unclear whether this regimen will be as efficacious as parenteral therapy, particularly for severe toxicities as was the case in the patient presented above.[19] Additionally, there are no long-term neurodevelopmental data supporting an alternative to the current standard of parenteral CaNa2 EDTA therapy. The risk to pediatric health from lead toxicity associated with recurring interruptions in CaNa2 EDTA availability has increased in light of the discontinuation of the manufacturing of dimercaprol in February 2023.[18]

Lead poisoning disproportionately affects children in lower socioeconomic classes and thus, represents a form of health care inequity.[20–22] Children in lower socioeconomic households in the United States are more likely to live in homes with lead-based paint, have less access to lead-free tap water or home-based filtration, and have lower frequency of follow-up with primary care pediatricians for recommended BLL screening tests.[23,24] In developing nations, industrial causes of lead toxicity including battery manufacturing plants and disposal sites tend to cluster around lower socioeconomic neighborhoods.[25–28] Despite public health campaigns, anticipatory guidance, universal screening recommendations, and other mitigation strategies, there continue to be lead poisoning outbreaks in children in the United States at an unacceptable rate.[4] It is important to prioritize lead poisoning prevention and intervention efforts in an effort to improve social equity and protect all children from this potentially devastating and yet preventable health hazard.

The municipality of Rochester, New York addressed lead toxicity by enacting municipal code that helped to eliminate lead hazards in high-risk rental housing in the early 2000s. These building codes in Rochester not only significantly reduced the prevalence of elevated BLL in children in the community, but also reduced environmental health disparities.[28] More recent outbreaks of lead poisoning in impoverished communities continue to demonstrate the failure of US public policy to adequately address the health inequities related to lead toxicity; one recent example includes the failures of public policies related to ensuring the safe water supply in Flint, Michigan.[29] Overall, the failure to adequately screen for and also the inability to appropriately treat children with lead poisoning may lead to a large cohort of children negatively impacted by the toxic effects of lead.[2,30]

There are a number of ethical considerations related to health care equity that justify the mandate for adequate antidote supply for lead poisoning. First, as lead poisoning disproportionately affects lower socioeconomic communities, access to essential medications is critical for improving and maintaining health equity. When underserved populations have access to the medications they need, it helps to improve their health outcomes and close the gap in health disparities. Second, access to essential medications including antidote therapy is essential for protecting public health. Given the known medical complications associated with lead poisoning, and the frequency with which "outbreaks" of lead toxicity occur, particularly in underrepresented communities, an adequate supply of antidote therapy is requisite to protect entire communities from the consequences of untreated or undertreated poisonings. Third, access to antidote therapy can help to reduce long-term health care costs related to unmitigated or undertreated lead toxicity. Ensuring access to and a robust supply of antidote therapy for lead poisoning can help to prevent the long-term neurodevelopmental and chronic medical complications, which will reduce the overall cost of health care.

In order to mitigate the ongoing chelation shortage, consideration should be given to augment the United States' Strategic National Stockpile (SNS) given the high consequence of inadequate antidote therapy to public health. The SNS is a national repository of medical supplies and equipment that can be used to respond to public health emergencies, such as bioterrorism

attacks, natural disasters, and pandemics. Managed by the US Department of Health and Human Services (HHS), the SNS supplies can be accessed by state and local authorities in order to distribute SNS medications and other medical supplies to local communities as quickly as possible. The SNS is an important part of the US public health preparedness system and should play a role in addressing critical medication shortages, and specifically develop a supply of antidote and chelation therapies. By having a stockpile of these essential medications, the federal government can provide a safety net for patients who need access to these medications in the event of a public health emergency.[31] As lead poisoning continues to affect children across the United States, and disproportionately children of lower socioeconomic status, there is an urgent need for adequate supply of antidote and chelation therapies.

## Conclusions

In the face of medication shortages including CaNa2 EDTA, it is critical for clinicians to create and study alternative standards for chelation therapy regimens for lead poisoning and intoxication. As highlighted in this case, we present an alternative treatment regimen using succimer; however, given the persistently elevated BLL in our patient, further work is required to elucidate the best alternative standards. Furthermore, public policy initiatives, including the development of a national supply stockpile of chelation agents, must be created in order to minimize supply chain disruption and ensure adequate reserve of available chelation therapy agents to support public health, especially for children.

## Article Information

**Affiliations.** Department of Pediatrics (MKS, JMK), University of Chicago and Comer Children's Hospital, Chicago, IL; Department of Pediatrics (JMK), Endeavor Health, Evanston, IL; Department of Pharmacy (SJS), UChicago Medicine, Chicago, IL.

**Correspondence.** Jason M. Kane, MD, MS; jasonk@uchicago.edu

**Disclosure.** The authors declare no conflicts or financial interest in any product or service mentioned in the manuscript, including grants, equipment, medications, employment, gifts, and honoraria. The authors had full access to all patient information in this report and take full responsibility for the integrity and accuracy of the report.

**Ethical Approval and Informed Consent.** The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation. The University of Chicago does not consider a single case report to be research and was thus exempt from IRB review.

**Submitted.** November 30, 2023

**Accepted.** February 25, 2024

**Editor's Note.** This case report highlights yet again the profound, preventable negative impact lead has on the infants and children in our society and the longstanding impotence of our public policy efforts. Interested readers are directed to the eloquent editorial by Sample J (*JPPT* 2024;29(3):212–214) and the work of Whitledge JD, et al (*JPPT* 2024;29(3):306–315) expounding on these and other important challenges confronting practitioners treating lead exposed/intoxicated patients.



## References

1. Chandran L, Cataldo R. Lead poisoning: basics and new developments. *Pediatr Rev.* 2010;31(10):399–406.
2. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect*. 2005;113(7):894–899.
3. Ruff HA, Bijur PE, Markowitz M, et al. Declining blood lead levels and cognitive changes in moderately lead-poisoned children. *JAMA*. 1993;269(13):1641–1646.
4. Markowitz M. Lead poisoning: an update. *Pediatr Rev.* 2021;42(6):302–315.
5. Centers for Disease Control and Prevention. Lead poisoning prevention. Accessed November 1, 2023. https://www.cdc.gov/nceh/lead/prevention/default.htm
6. Roberts DJ, Bradberry SM, Butcher F, Busby A. Lead exposure in children. *BMJ* 2022;377:e063950.
7. Jain RB. Trends and variability in blood lead concentrations among US children and adolescents. *Environ Sci Pollut Res Int*. 2016;23(8):7880–7889.
8. de Souza ID, de Andrade AS, Dalmolin RJ. Lead-interacting proteins and their implication in lead poisoning. *Crit Rev Toxicol*. 2018;48(5):375–386.
9. Ettinger AS, Leonard ML, Mason J. CDC's lead poisoning prevention program: a long-standing responsibility and commitment to protect children from lead exposure. *J Public Health Manag Pract*. 2019;25(suppl 1 LEAD POISONING PREVENTION):S5–S12.
10. Calello DP, Henretig FM. Lead. In: Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. *Goldfrank's Toxicologic Emergencies, 11e*. McGraw-Hill Education; 2019. Accessed August 08, 2024. https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=2569&sectionid=210261271 2019.
11. Woolf AD, Goldman R, Bellinger DC. Update on the clinical management of childhood lead poisoning. *Pediatr Clin North Am*. 2007;54(2):271–294, viii.
12. Rogan WJ, Dietrich KN, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med.* 2001;344:1421–1426.
13. Mazer-Amirshahi M, Fox ER, Routsolias JC, et al. How can we "get the lead out" without chelators? *J Med Toxicol*. 2021;17(4):330–332.
14. Committee on Drugs. Treatment guidelines for lead exposure in children. *Pediatrics.* 1995;96(1):155–159.
15. Food and Drug Administration Web site. Accessed January 19, 2024. https://www.fda.gov/media/163578/download

16. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. *Pediatrics*. 2005;116(4):1036–1046.
17. Hantel A, Siegler M, Hlubocky F, et al. Prevalence and severity of rationing during drug shortages: a national survey of health system pharmacists. *JAMA Intern Med*. 2019;179(5):710–711.
18. Wheeler M. American Society of Health-System Pharmacists (ASHP). Calcium Disodium Versenate Injection. Bethesda, MD; 2023. Accessed November 1, 2023. https://www.ashp.org/drug-shortages/current-shortages
19. Bradberry S, Vale A. A comparison of sodium calcium edetate (edetate calcium disodium) and succimer (DMSA) in the treatment of inorganic lead poisoning. *Clin Toxicol*. 2009;47(9):841–858.
20. Rio MD, Gibson JM. Factors influencing inequities in lead exposure in United States children: a systematic review. *Int Public Health J*. 2023;15(3):263–281.
21. Oberle MW. Lead poisoning: a preventable childhood disease of the slums. *Science*. 1969;165(3897):991–992.
22. Whitehead LS, Buchanan SD. Childhood lead poisoning: a perpetual environmental justice issue? *J Public Health Manag Pract*. 2019;25:S115–S120.
23. Hauptman M, Rogers ML, Scarpaci M, et al. Neighborhood disparities and the burden of lead poisoning. *Pediatr Res*. 2023;94:826–836.
24. Council on Environmental Health. Prevention of childhood lead toxicity. *Pediatrics*. 2016;138(1):e20161493.
25. Mani MS, Nayak DG, Dsouza HS. Challenges in diagnosing lead poisoning: a review of occupationally and nonoccupationally exposed cases reported in India. *Toxicol Ind Health*. 2020;36(5):346–355.
26. Al Sukaiti WS, Al Shuhoumi MA, Al Balushi HI, et al. Lead poisoning epidemiology, challenges and opportunities: first systematic review and expert consensus of the MENA region. *Environ Adv*. 2023;100387.
27. Obeng-Gyasi E. Sources of lead exposure in various countries. *Rev Environ Health*. 2019;34(1):25–34.
28. Korfmacher KS, Ayoob M, Morley R. Rochester's lead law: evaluation of a local environmental health policy innovation. *Environ Health Perspect*. 2012;120:309–315.
29. Bellinger DC. Lead contamination in Flint—an abject failure to protect public health. *N Engl J Med*. 2016;374:1101–1103.
30. Naranjo VI, Hendricks M, Jones KS. Lead toxicity in children: an unremitting public health problem. *Pediatr Neurol*. 2020;113:51–55.
31. Giovachino M, Calhoun T, Carey N, et al. Optimizing a District of Columbia strategic national stockpile dispensing center. *J Public Health Manag Pract*. 2005;11(4):282–290.