Case Report

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Lead Ingestion in a Child: A Case Report

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Abstract

Lead is a heavy metal capable of causing significant toxic effects in children during acute exposure. Accidental ingestion of lead-containing objects is a common cause of acute poisoning in the pediatric population. Acute lead exposure can manifest with a broad clinical spectrum, ranging from neurological and gastrointestinal symptoms to life-threatening complications. This report presents the case of a 6-year-old boy who accidentally ingested a lead object. The patient presented to the emergency department approximately 20 minutes after the incident. His vital signs were stable, physical examination findings were unremarkable, and laboratory results were normal. An upright abdominal X-ray revealed an oval-shaped radiopaque object located in the duodenum. On the fourth day, the blood lead level was measured as 14 µg/dL, and no symptoms were observed. In conclusion, the rapid passage of the lead object through the gastrointestinal tract may have played a role in limiting toxicity. This case contributes to the literature regarding the management and clinical course of acute lead poisoning in children.

Keywords: Acute lead, childhood lead toxication, lead ingestion poisoning

Introduction

Lead is a toxic heavy metal with rapid effects during acute exposure, posing serious health risks, particularly in children. Accidental ingestion of lead-containing objects constitutes a significant proportion of acute poisoning cases in the pediatric population. Lead is readily absorbed through the gastrointestinal tract, entering systemic circulation within a short period and potentially causing systemic toxicity. Acute lead poisoning can present with a diverse clinical spectrum, including neurological symptoms, gastrointestinal distress, hematological abnormalities, and life-threatening complications. Early diagnosis and intervention are critical to mitigating potential fatal outcomes (1,2).

The common sources of acute lead exposure in children include ingestion of toys, batteries, bullets, or other objects containing lead. Such incidents are more frequent among younger children due to their natural curiosity. The movement of lead objects through the gastrointestinal tract is a critical determinant of the severity of toxic effects. The duration of exposure and the time the object remains in the gastrointestinal system are key factors influencing toxicity (3). While objects that pass rapidly through the stomach may have limited toxic effects, those that remain for prolonged periods are associated with higher absorption rates and increased toxicity risk. In this context, the management of acute lead exposure requires close monitoring of clinical symptoms and, when necessary, planning for endoscopic or surgical intervention within a multidisciplinary approach. (4). In this report, we aim to contribute to the literature by presenting the case of a 6-year-old boy who accidentally ingested a lead object.

Case Report

A 6-year-old boy was brought to the emergency department of a secondary care hospital with a complaint of accidental ingestion of a lead object approximately 20 minutes prior. The patient had no significant medical history, regular medication use, or history of previous surgeries. At presentation, his vital signs were as follows: blood pressure 100/70 mmHg, heart rate 70 bpm, body temperature 36 °C, and oxygen saturation 99%. Physical examination findings were unremarkable. Laboratory tests, including complete blood count, liver and renal function tests, and INR, were within normal limits.

An upright abdominal X-ray (Figure-1) revealed an ovalshaped radiopaque object located in the duodenal region. The patientdid not receive activated charcoal treatment. He was referred to a tertiary care center for further evaluation of potential toxic effects and, if deemed necessary, endoscopic removal of the object. At the tertiary center,

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Figure 1. Lead X-ray Image.

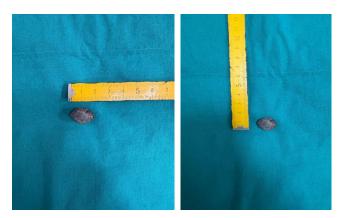


Figure 2. Extracted Lead Material

no endoscopic procedure was performed, and the patient was monitored without any intervention. At 32 hours postexposure, the object was reported to have passed through the gastrointestinal tract and was excreted in the stool (Figure-2). The retrieved lead object weighed 19 grams, with dimensions of 17 mm, 12 mm, and 6 mm.

On the fourth day, follow-up assessments revealed normal complete blood count, liver and renal function tests, and electrolyte levels. Blood lead levels, measured using the AAS/ICP-MS method, were found to be 14.1 μ g/dL. During this period, the patient remained asymptomatic, and no abnormalities were detected on diagnostic evaluations. The patient was scheduled for follow-up at the hematology outpatient clinic to monitor for potential long-term effects.

Discussion

Lead poisoning has been recognized as an occupational disease since ancient times. Hippocrates first documented this condition in a lead worker presenting with abdominal pain (5). Today, lead exposure occurs through diverse routes, including occupational exposure, accidental ingestion, or environmental contamination. Lead exposure may occur via inhalation, dermal contact, or gastrointestinal absorption.

In the presented case, exposure occurred via accidental ingestion, a common scenario also reported in cases involving indoor shooting ranges, leaded wine pitchers, jewelry manufacturing, and consumption of contaminated food o rmedications (6-9).

While the diagnosis in this case was straight forward, the underlying etiology in patients presenting with acute abdominal symptoms can be challenging to determine (10). In our case, the lead object was promptly identified. Despite the absence of symptoms, acute lead exposure has been associated with a wide range of clinical manifestations, including acute abdomen, anemia, encephalopathy, brain herniation, and fatal outcomes (11-13). The lack of symptoms in this case may be explained by the rapid gastric emptying of the object, which minimized the time it remained in the gastrointestinal system.

A study by Offor et al. in 2017 demonstrated that activated charcoal reduced liver enzymes, oxidative stres markers, and pro-inflammatory cytokines in male albino rats exposed to lead acetate, suggesting protective effects on liver and kidney tissues (14). However, human studies indicate that activated charcoal does not bind to lead, and its use in acute lead poisoning is not recommended (15). Furthermore, it is well-documented that children's gastrointestinal mucosa is more sensitive to lead absorption, with higher absorption rates compared to adults. (11)

In our case, the rapid transit of the ingested lead object through the gastrointestinal tract and it sexcretion in stool likely played a critical role in preventing toxic effects. Nevertheless, the measured blood lead level of 14 μ g/dL is note worthy despite the absence of acute symptoms. Similarly, in the case reported by Gopinath et al., no symptoms were observed in a patient with a blood lead level of 14 μ g/dL. Although asymptomatic in the acute phase, this level may pose long-term risks for cognitive and neurological development.

Conclusion

The pediatric population is particularly vulnerable to lead poisoning. In this case, despite the absence of activated charcoal administration, the blood lead level of 14 μ g/dL did not result in acute symptoms. This case highlights the importance of prompt recognition and management of lead exposure in children, emphasizing the need for long-term monitoring for potential cognitive and neurological effects.

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