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## **Child Abuse by Intentional Iron Poisoning Presenting as Shock and Persistent Acidosis**

Judy Black and Joseph A. Zenel

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## EXPERIENCE AND REASON—Briefly Recorded

"In Medicine one must pay attention not to plausible theorizing but to experience and reason together. . . . I agree that theorizing is to be approved, provided that it is based on facts, and systematically makes its deductions from what is observed. . . . But conclusions drawn from unaided reason can hardly be serviceable; only those drawn from observed fact." Hippocrates: *Precepts*. (Short communications of factual material are published here. Comments and criticisms appear as letters to the Editor.)

### Child Abuse by Intentional Iron Poisoning Presenting as Shock and Persistent Acidosis

**ABSTRACT.** A case of intentional iron poisoning presented to our hospital. The patient's persistent acidosis and the team's observation of maternal indifference indicated the diagnosis. This case should alert physicians to a potential source for intentional poisoning that is present in most homes with young infants. *Pediatrics* 2003;111:197–199; iron poisoning, intentional poisoning, child abuse.

ABBREVIATION. ED, emergency department.

Intentional poisoning is a less common, yet potentially life-threatening, form of physical child abuse. In the absence of obvious historical or physical clues, this diagnosis may be overlooked until it is too late. Case reports of toxic ingestions suggest that persistent, unexplained metabolic acidosis may be the only signs of a poisoning.<sup>1,2</sup> We present a case of intentional iron poisoning that was initially unsuspected.

#### CASE REPORT

A 7-week-old male infant presented to an outlying emergency department (ED) with the complaint of 1 day of constipation. He appeared healthy on examination, during which time he passed a normal, formed stool. After reassuring the mother, he was discharged home. He presented the next day to the same ED, this time with grunting, tachypnea, and abdominal distention. According to his mother, he had been behaving normally after the previous night's discharge, until about midnight, when he began refusing food. He became fussy and appeared uncomfortable. He had no additional stools after his discharge, and he did not vomit. He had not been febrile. Physical examination in the ED suggested significant dehydration, and he received 2 boluses of intravenous normal saline (20 mL/kg each). Laboratory examination was significant for an arterial blood gas with the following: pH, 7.09; PCO<sub>2</sub>, 32; and base deficit, -19.6. Shortly after intravenous hydration, he passed 4 large, heme-positive stools with mucous. Flat and upright abdominal radiographs suggested bowel obstruction. An orogastric tube was placed and he was transferred to the pediatric intensive care unit at our institution with a working diagnosis of bowel obstruction.

On arrival at our institution, his vital signs were as follows:

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Both authors work at Doernbecher Children's Hospital, Oregon Health and Sciences University, Portland, Oregon.

Address correspondence to Joseph A. Zenel, MD, Oregon Health and Sciences University, Department of Pediatrics, 707 SW Gaines Rd, Mail Code: CDRCP, Portland, OR 97201-2901. E-mail: zenelj@ohsu.edu  
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temperature, 35.6°C (96.1°F); pulse, 172 beats per minute; respirations, 22 beats per minute; and blood pressure, 70/32 mm Hg. Pulse oximetry demonstrated 99% saturation in room air. He was lethargic, but arousable, and had a weak cry. His anterior fontanel was slightly sunken. Abdominal examination revealed a soft, nondistended, nontender abdomen, with active bowel sounds. His hands and feet were cool to the touch, and his overall muscle tone was slightly diminished. Additional history revealed that he had been born at 38 weeks' gestation to a 15-year-old G<sub>1</sub>P<sub>0</sub> mother. Prenatal care began at 4 months' gestation. There were no previous illnesses, and he had not received any immunizations. His mother currently lived with her 19-year-old boyfriend, his cousin, and a teenaged friend. Social services had been involved with the mother and patient since the pregnancy.

Initial laboratory examination in the pediatric intensive care unit was significant for the following: bicarbonate, 8 mmol/L; and anion gap, 35. Blood urea nitrogen and creatinine levels were normal for age. Serum glucose was 51 mg/dL, and serum acetone was negative. A complete blood count showed a white blood count of 30.9 × 10<sup>3</sup>/mm<sup>3</sup>, with 20% neutrophils, and 8% bands. Analysis of a urine specimen obtained by catheter showed 3+ protein, 1+ heme, and 0 to 5 red blood cells per high-power field, with no white blood cells or bacteria. After additional intravenous hydration, a venous blood gas showed the following: pH, 7.24; PCO<sub>2</sub>, 39; and base deficit, -9.5. Upper and lower gastrointestinal barium stools ruled out bowel obstruction. Stool rotametry was negative. After blood, urine, and stool cultures were collected, the patient was started on intravenous ampicillin and gentamicin for presumed sepsis.

By the next day, the patient appeared clinically stable, and he was transferred to the general ward. He was awake and eating well. No diarrhea or vomiting occurred after admission. Antibiotic administration was discontinued when stool, urine, and blood cultures demonstrated no growth by 48 hours after admission. Repeat laboratory examination at 48 hours after admission revealed the following: serum bicarbonate, 17 mmol/L; anion gap, 15; blood urea nitrogen, 33 mg/dL; creatinine, 1.2 mg/dL; alkaline phosphatase, 691 U/L; lactate dehydrogenase, 4394 U/L; aspartate aminotransferase, 1603 U/L; alanine aminotransferase, 1600 U/L; and creatine phosphokinase, 1771 U/L. A renal ultrasound was normal. The team felt that these findings could be explained by initial dehydration leading to secondary acute tubular necrosis and hepatitis. Consults with nephrology and gastroenterology supported this theory.

On hospital day 4, despite daily sodium bicarbonate supplementation in his intravenous fluids, and an adequate infant formula intake, the patient's serum bicarbonate level was 13 mmol/dL. Serum creatinine remained stable at 1.1 mg/dL, and liver function tests decreased to the following levels: aspartate aminotransferase, 1188 U/L; alanine aminotransferase, 1555 U/L; alkaline phosphatase, 551 U/L; and lactate dehydrogenase, 2916 U/L. A repeat urinalysis continued to show blood, with 0 to 2 red blood cells per high-power field on microscopic examination. Metabolic services was requested. They recommended a full work-up for metabolic disorders, however, they also suggested looking for possible toxic ingestion.<sup>2</sup> Remaining pooled serum from the first 36 hours of hospitalization was tested for iron, salicylates, ethylene glycol, and alcohol.

In addition, by hospital day 4, both the nursing staff and resident pediatricians had noted that the patient's caretakers were infrequent visitors and that, when present, the mother needed to be actively encouraged to care for her child. She slept through her child's cries for food and did not inquire about her child's care or current state of health. Based on these concerns, an evaluation for

possible physical abuse was initiated. A skeletal survey showed a possible posterolateral rib fracture, confirmed by bone scan. Head magnetic resonance imaging and ophthalmologic examination were unremarkable. Local child protective services and law enforcement were notified. During an interview with the police, the mother confessed to "picking him up and squeezing him hard." She also admitted that she had squirted Formula 409 (Clorox Company, Pleasanton, CA) (active ingredient: 0.3% alkyl dimethyl benzyl ammonium chloride) in his mouth, and had crushed a few prenatal iron tablets and fed them to the child. After complete recovery, the patient was discharged to foster care. Several days later, the iron level on the collected serum from the first 36 hours of hospitalization was reported at 308  $\mu\text{g}/\text{dL}$  (normal: 50–170  $\mu\text{g}/\text{dL}$ ; or 55  $\mu\text{mol}/\text{L}$  with normal 9–30  $\mu\text{mol}/\text{L}$ ). No serum evidence for ethylene glycol, alcohol, or salicylates was found.

## DISCUSSION

This patient's differential diagnosis evolved during the course of his hospitalization. The final diagnosis depended on reevaluation of the possible causes of persistent metabolic acidosis such as methanol ingestion, uremia, diabetes mellitus, paraldehyde, iron, ethanol, ethylene glycol or salicylate ingestion ("MUDPIES"). On initial presentation, his condition appeared to be attributable to sepsis, gastrointestinal obstruction, and/or hypovolemia from stool losses and poor oral intake. Because of his young age, other possible differential diagnoses, particularly toxic ingestions, were not thoroughly considered by the team until the initial working diagnoses were excluded. His rapid clinical recovery after rehydration, together with the negative urine, stool, and blood cultures, suggested hypovolemic shock as the underlying cause for presentation. Although both the subsequent acute tubular necrosis and hepatitis could be explained by severe dehydration, the prolonged, persistent acidosis and the initial cause of his bloody diarrhea remained unexplained. Reconsideration of the causes of persistent metabolic acidosis and the lack of maternal concern, in addition to the patient's presentation of hematochezia, finally indicated the underlying diagnosis of iron toxicity from intentional poisoning. Signs and symptoms included: 1) bloody diarrhea; 2) shock; 3) positive anion gap metabolic acidosis; 4) hepatic dysfunction with elevated aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and creatine phosphokinase; 5) leukocytosis; and 6) acute tubular necrosis and myoglobinuria.

Iron toxicity generally progresses through 5 stages. The first stage presents with predominately gastrointestinal symptoms within 6 hours after ingestion. Iron is directly corrosive to gastric mucosa, and ingestion leads to diarrhea, nausea, vomiting, and gastrointestinal bleeding. During the second stage of iron toxicity, also known as the latent stage, symptoms are continued metabolic acidosis, mild hyperventilation, and oliguria.<sup>3</sup> Hyperglycemia and leukocytosis may also appear during this stage.<sup>4</sup> The second stage generally occurs 6 to 24 hours after toxic ingestion. Supportive care and chelation therapy with deferoxamine at this point may help prevent additional toxicity. Without supportive care, the latent stage effects culminate in the third stage, characterized by systemic shock and hepatic and renal failure.<sup>4</sup> The fourth stage is the period of clinical

recovery, and the fifth stage is the development of gastrointestinal strictures, which may follow the first 4 stages within 2 to 8 weeks.

Metabolic acidosis attributable to iron toxicity results from several mechanisms. After absorption in the gut, iron is converted from the ferrous to the ferric state, thus releasing hydrogen ions into the serum.<sup>4</sup> Hepatocyte absorption of iron leads to generation of free radicals, which can uncouple oxidative phosphorylation, causing anaerobic metabolism and the production of lactic acid. Free iron in the serum also has some effect on blood vessels, causing increased capillary permeability and loss of venous tone.<sup>5</sup> Additional fluid losses in the gut, from hemorrhage and diarrhea, and bradycardia from the cardiac toxicity of free iron accentuate the hypoperfusion, thereby exacerbating the lactic acidosis.<sup>3</sup>

Our patient most likely presented soon after his ingestion. Gastrointestinal effects of iron ingestion typically occur within 6 hours after exposure, and the patient had bloody diarrhea only after presentation to the ED. He also had an initial radiograph suggestive of bowel obstruction. In a study by Staple and McAlister,<sup>6</sup> air fluid levels appeared within 1 hour and disappeared within 6 hours after ingestion. The patient's dehydration from poor oral intake over the course of the preceding day also contributed to the acuity of his presentation. Both hypovolemic shock and direct iron toxicity probably caused acute tubular necrosis and intensified hepatic injury.

The case presented here has several ambiguities. The serum iron level was a composite of several samples obtained during the first 36 hours of hospitalization and may reflect a rising serum iron level from hepatic damage secondary to systemic shock. However, the mother's confession and the clinical presentation of bloody stools, leukocytosis, persistent metabolic acidosis, and hepatic and renal damage strongly indicate iron ingestion. Although no iron tablets were visible on radiographic examination, the mother admitted to grinding the tablets, and the degree of radiopacity of individual iron pills varies.<sup>6</sup> The ingestion of 0.3% alkyl dimethyl benzyl ammonium chloride, the active ingredient in the household cleaning product Formula 409, may have added to the gastrointestinal symptoms, but the concentration and small amount ingested would not have caused systemic effects. Finally, other unaccounted acute or chronic poisoning with iron or other agents may have caused acute tubular necrosis and hepatic injury.

Iron ingestion is one of the most common causes of pediatric poisonings in the United States.<sup>5</sup> Almost all reported cases are accidental ingestions by a toddler or young child. We believe this is the only reported case of nonaccidental iron poisoning in the literature, as determined by a computer search of English language literature from 1966–2001, using the keywords iron toxicity, iron poisoning, child abuse, and deferoxamine. According to Dine and McGovern's<sup>7</sup> review of nonaccidental poisoning, the most common presenting cases involved excessive salt ingestion and water intoxication. Other common poisonings included medications belonging to the parent. In our

case, the mother had her prenatal iron tablets readily available and the package of pills was clearly labeled as harmful to children <6 years of age.

Intentional poisonings occur for many different reasons, involve a variety of toxic substances, and have no classic clinical presentation.<sup>8</sup> As in all cases of suspected physical child abuse, signs and symptoms that remain unexplained by history should raise the possibility of poisoning.<sup>9</sup> In our case, the prolonged acidosis was the first clue to arouse suspicion that the clinical presentation was not simply attributable to dehydration. Review of the patient's presentation, attention to the mother's behavior and a radiologic search for skeletal fractures confirmed the suspicion that the patient had been abused. Although iron poisoning led to our patient's admission to the hospital, the discovery of the rib fracture was the catalyst for placing him in protective custody. The high pooled serum level of iron confirmed the diagnosis of intentional poisoning, but that evidence arrived days after he was ready for discharge. Had the work-up for physical abuse not been performed, the infant might have been discharged to his mother's care. Our patient may be fortunate that he did return to his household. In a review of 48 cases by Dine and McGovern,<sup>7</sup> the mortality rate of nonaccidental poisoning was 17%. Furthermore, nonaccidental poisoning in a household tends to be recurrent, and our patient was removed from a caretaker who may be untreatable.<sup>10</sup>

In 1997, the Food and Drug Administration required the packaging of iron supplements containing >30 mg of iron per tablet as individual doses to reduce the risk of toxic overdose.<sup>5</sup> Although this packaging may decrease the incidence of accidental iron ingestion, toxic iron ingestions still occur. Iron has been implicated as the second most common agent in overdoses during pregnancy.<sup>11</sup> Given the reality of postpartum depression and psychosis, it is important to remember that a mother with "no medications" at home may still have easy access to a toxic substance.

### CONCLUSION

This case is a reminder that the acute onset of bloody stools and shock in an infant or child followed by persistent metabolic acidosis and elevated liver function tests may be attributable to iron intoxication, even in children who are incapable of ingesting the iron on their own. If there is no history of accidental ingestion and the household environment is suspect, then a thorough search for abuse, including radiographic studies to look for skeletal fractures, should proceed, as the rate of coincident battering has been found to be as high as 20%.<sup>7</sup> Iron preparations, now labeled as harmful to children <6 years of age, are likely to be present in most households with infants.

JUDY BLACK, MD  
JOSEPH A. ZENEL, MD  
Oregon Health and Sciences University  
Department of Pediatrics  
Portland, OR 97201-2901

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## Cervical Spine Involvement in Larsen's Syndrome: A Case Illustration

**ABSTRACT.** We present the progressive clinical course of a child with myelopathy attributable to cervical spine abnormalities associated with Larsen's syndrome. After anterior and posterior cervical fusion, his preoperative symptoms of weakness, gait dysfunction, and hyperreflexia have improved at 9-month follow-up. The progressive course and importance of early referral and intervention should be of interest to the general pediatric community. *Pediatrics* 2003;111:199-201; *Larsen's syndrome, myelopathy, cervical spine, pediatric.*

ABBREVIATION. MRI, magnetic resonance imaging.

Larsen's syndrome is a rare inherited defect of connective tissue formation that is transmitted in both an autosomal dominant and recessive pattern. First described by Larsen in 1950, its cardinal findings consist of multiple congenital joint dislocations usually of the hips, knees and elbows, frontal bossing, a depressed nasal bridge, hypertelorism, flat facies, distinctive deformities of the hand and calcaneus, and spinal anomalies that may lead to major spinal instability and spinal cord injury.<sup>1-10</sup> Outside of the orthopaedic literature, few references to its identification and treatment exist. Therefore, to stress the importance of a thorough evaluation of the spine in children with Larsen's syndrome, we present the

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Address correspondence to John C. Wellons III, MD, Children's Hospital of Alabama, 1600 7th Ave, S, ACC 400, Birmingham, AL 35233. E-mail: [jay.wellons@ccc.uab.edu](mailto:jay.wellons@ccc.uab.edu)

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