CASE REPORT

Acute iron overdose associated with ileal perforation in an 11-month-old boy in Rwanda

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East Cent Afr J Surg. 2021;26(2):88-91
https://doi.org/10.4314/ecajs.v26i2.8

Abstract

Iron poisoning is a common and potentially fatal event associated with both local and systemic effects, most commonly in children. Surgical management of patients with iron poisoning primarily focuses on the sequelae of iron intoxication on the gut and liver when nonoperative management has failed. Interventions can include surgical removal of retained iron bezoars, bowel perforation repair, and management of gastrointestinal strictures that may arise weeks after ingestion. The worst cases necessitate liver transplantation.

We present a case of a child with severe bowel ischaemia and distal ileal compromise, successfully treated via bowel resection and primary anastomosis at Rwanda Military Hospital in Kigali, Rwanda.

Keywords: iron toxicity, ileal perforation, paediatric surgery, Rwanda

Introduction

Acute iron poisoning is a common and potentially fatal event in children, causing more than 30% of the deaths in children who ingest drugs unintentionally.[1] Fatal consequences have been associated with both the local and systemic effects of ingested iron.

Iron compounds have been associated with significant gastrointestinal lesions, with the initial injury typically precipitated by a necrotizing haemorrhagic inflammatory reaction.

We present a case of acute iron intoxication in a child with severe bowel ischaemia and distal ileal perforation, which was successfully treated via bowel resection and primary anastomosis. Cases of iron intoxication causing intestinal ischemia and jejunal perforation are rare but have been previously described.[2]-[4] To our knowledge, distal ileal perforation secondary to iron toxicity has not been previously reported in East Africa.

Case presentation

An 11-month-old boy, weighing 7.5 kg, was initially seen by a primary-care paediatrician at Gisenyi District Hospital, Rwanda. He had presented with a history of ingestion of 12 Fercefol (Exphar SA, Belgium) iron supplement tablets (each containing 200 mg of ferrous sulphate and 0.4 mg of folic acid, i.e., about 27 mg/kg). His initial symptoms at presentation included hypersalivation, decreased level of consciousness, abdominal distension, and vomiting.

On evaluation, the patient had a decreased level of consciousness. Initial therapy at Gisenyi District Hospital included intravenous fluid resuscitation and gastric lavage using charcoal. The patient had a transient response with improvement of his level of consciousness, and he was admitted for observation. On admission day 1, the patient developed increased vomiting, diarrhoea, and worsening abdominal distension; these manifestations were presumed to be caused by gastroenteritis, prompting further nonoperative management, including intravenous resuscitation with fluids and oral zinc sulphate administration for 72 hours.

On admission day 4, the severity of the abdominal distention continued to increase, and the patient showed clinical features of partial bowel obstruction, including obstipation for 72 hours associated with bilious vomiting and irritability. He was, therefore, transferred 168 km to Rwanda Military Hospital (RMH) in Kigali (approximately 3 hours and 45 minutes' drive) for further management. The patient was admitted to the RMH paediatric emergency department on 9 November 2019, the fourth day after symptom onset. On clinical evaluation, the patient was haemodynamically stable and alert with an axillary temperature of 37°C, a heart rate of 126 beats per minute, a respiratory rate of 22 cycles

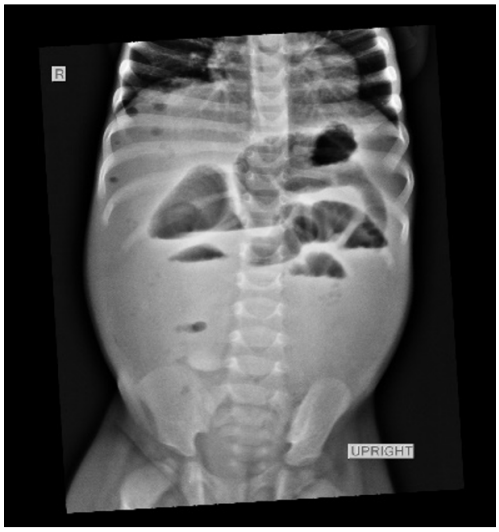

Figure. Preoperative plain abdominal x-ray, showing dilated loops of small bowel, multiple air–fluid levels, no air in the rectum, and no obvious free intraperitoneal air

per minute, and an SpO_2 of 98% on room air. The physical examination was notable for abdominal distension with decreased bowel sounds in all 4 quadrants and mild generalized tenderness to palpation. Initial investigations included plain abdominal x-rays and abdominal ultrasonography, which were inconclusive. The patient was started on intravenous metronidazole (30 mg/kg/day in divided doses 3 times daily) and cefotaxime (150 mg/kg/day in divided doses every 8 hours). Due to the lack of improvement in the patient's clinical status, further investigations, including repeat abdominal ultrasonography, were completed for presumed intussusception, but these yielded no acute findings. The patient was transferred to the RMH paediatric department for medical management of iron intoxication.

The patient improved clinically on medical management as described above and was started on oral feeding; he was discharged on 14 November 2019, which was his sixth day at RMH and the ninth day from the onset of symptoms. The next day, however, before the family had left the hospital, the patient acutely developed severe bilious vomiting with associated abdominal distension. The patient was severely dehydrated, with a decreased level of consciousness and weak peripheral pulses. Abdominal examination revealed severe distension in conjunction with absent bowel sounds and diffuse tenderness to palpation consistent with peritonitis. A plain abdominal x-ray done revealed dilated loops of small bowel, multiple air–fluid levels, no air in the rectum, and no obvious free intraperitoneal air (Figure).

Management, outcome, and follow-up

Given the patient's clinical status, the decision was made to proceed with an exploratory laparotomy. Upon entering the abdomen through a supraumbilical transverse incision, free purulent fluid and stool was found throughout the abdominal cavity with associated fibrinous tissue and adhesions between the small bowel and bladder, which were carefully dissected. The small intestine was thickened and dilated with a 10-cm necrotic segment with a longitudinal perforation involving the distal ileum. The surrounding bowel and mesentery were viable and well perfused, and there were no other visible injuries. The decision was made to perform a resection of the necrotic ileal segment with primary end-to-end handsewn anastomosis. The entire abdomen was washed out and closed in 2 layers. The patient received 100 cc of packed red blood cells intraoperatively.

Postoperatively, the patient developed a paralytic ileus, which resolved with nonoperative management on postoperative day 3. The patient then tolerated a soft diet with return of bowel function and was discharged home on postoperative day 10 (25 November 2019).

The patient returned for a 3-month follow-up visit on 29 February 2020. At that time, the patient and his caretaker denied any complaints; he was eating, passing stools normally, and voiding well, with no abdominal distension or pain, and his skin incision had completely healed.

Discussion

Ingested iron causes both a direct corrosive effect on tissue and impaired cellular metabolism affecting the gastrointestinal tract, heart, liver, and nervous system.[2]-[4] Initially, direct caustic injury to the gastrointestinal mucosa results in vomiting, abdominal pain, diarrhoea, haematemesis, and melaena, leading to hypovolaemia.[2],[3] Retained iron in the gastrointestinal tract can cause bowel inflammation, infarction, and even perforation.[5],[6] Serum iron levels tend to drop dramatically 3 to 5 hours after the ingestion of iron or 6 to 8 hours after the ingestion of sustained-release capsules.[2],[3] Determining serum iron levels before this drop is a priority, since the maximum iron level is the most useful laboratory test for determining prognosis.[3]

Ingested iron is deposited in the liver, which damages hepatocytes and can eventually lead to liver failure with associated coagulopathy.[3] Free iron can also directly damage myocardial cells, leading to decreased contractility and heart failure. Additionally, free iron causes direct vasodilation, damaging the capillaries, leading to fluid third-spacing, worsening hypovolaemia, and hypotension.[7] Poor tissue perfusion can cause acute renal failure, alter mental status, and aggravate the metabolic acidosis from anaerobic metabolism. The presentation of iron toxicity can be divided into 4 stages, with cellular damage starting approximately 12 hours after ingestion (Table).[4],[6],[8],[9]

The diagnosis of iron toxicity is based on the patient's history and clinical presentation. Suspected toxicity warrants investigation of iron levels upon admission and every 1 to 2 hours thereafter.[2]-[4] A plain abdominal x-ray should also be obtained, as it may reveal radiopaque iron tablets in the gastrointestinal tract and guide management, including whether gastric lavage or whole-bowel irrigation may be helpful. If a patient presents within 1 hour of ingestion and has ingested more than 20 mg/kg of iron, gastric lavage using tap water or normal saline may be of limited benefit.[7] Ac-

Table. Clinical stages of iron toxicity[6]

Stage	Timing	Presentation
1	0-6 h	Common gastrointestinal complaints (e.g., nausea, vomiting, diarrhoea)
2	6-12 h	Mild hypovolaemia, hypoperfusion, and acidosis
3	12-24 h	Progression to hepatic, renal, and/or cardiac failure
4	2-6 wk	Gastrointestinal scarring from mucosal healing can create strictures and lead to bowel obstruction

tivated charcoal can also be used soon after presentation if it is ingested concurrently with other medications, which was not the case for our patient.[10] A x-ray is recommended after gastric lavage to evaluate for any retained tablet fragments.[5],[11]

Whole-bowel irrigation is contraindicated for patients with bowel obstruction, perforation, ileus, haemodynamic instability, or compromised airways, primarily owing to concerns about perforation or aspiration with respiratory failure.[10],[12] Whole-bowel irrigation has been used to successfully remove toxicants from the gastrointestinal tract in some patients; however, no clinical studies have confirmed associated improved outcomes.[8],[13]

Iron chelation therapy should be completed using intravenous deferoxamine to bind with free iron, creating a ferrioxamine complex excreted by the kidneys.[2]-[4],[13] Infusion should start at 15 mg/kg/hour, with a maximum daily dose of 360 mg/kg/day and a total limit of 6 g.[3],[12] Medication side effects include hypotension if the infusion is too fast and acute respiratory distress syndrome if deferoxamine is used for more than 3 days.[3] In severe cases for which deferoxamine is insufficient, exchange transfusion can remove free iron from the blood.[8] In Rwanda, however, deferoxamine is not available; instead, bicarbonate can play a role in the treatment of acute iron toxicity. Bicarbonate oxidizes ferrous iron to ferric iron and can be used to treat the metabolic acidosis associated with poisoning as well as to enhance the renal excretion of iron.[14]

The surgical care of these patients primarily focuses on the sequelae of iron intoxication on the gut and liver. Surgical management can include removal of retained iron bezoars, bowel perforation repair, follow-up to detect stricture formation, and interventions for strictures that do arise. The worst cases necessitate liver transplantation.[15]

Finally, if children manage to survive treatment, it is important to remember that strictures may form 2 weeks or later after ingestion.[3],[6],[7],[9] These patients will require monitoring for obstructive symptoms and possibly operative resection or strictureplasty if they become symptomatic.[2],[8]

For our patient, we started with nonoperative management, including intravenous fluid resuscitation and activated charcoal. No iron chelation was done due to the lack of availability of this type of management at our hospital and in Rwanda in general. Exploratory laparotomy was chosen after failed medical and nonoperative management, and it involved resection of the gangrenous perforated ileal segment and primary end-to-end ileal anastomosis. No bowel lavage was done. To our knowledge, only 2 other cases of bowel perforation secondary to iron toxicity have been previously described in the literature, including a perforated jejunal diverticulum[16] and multiple punctate perforations of the small bowel,[9] for both of which resection and primary anastomosis were performed. The latter case was associated with significant organ failure caused by the systemic effects of iron overdose.

Our case report exemplifies the successful operative management of a life-threatening ileal perforation. A persistently high index of suspicion for perforation is crucial when managing patients with iron toxicity, particularly when treating young children, for whom verbal communication of symptoms is difficult, and when imaging is inconclusive.

Conclusions

Acute iron toxicity is a common and potentially fatal event among children. Primary treatment focuses on eliminating iron through gastric lavage, whole-bowel irrigation, deferoxamine chelation, or exchange transfusion while medically stabilizing the patient.

Surgical treatment may be necessary to remove retained iron bezoars, repair bowel perforations as in the presented case, and to manage stricture formation.

Although it is not standard therapy, surgical irrigation to remove iron from the bowel lumen could also provide additional benefit if instituted soon after patient presentation, as it can limit the total amount of iron absorbed.

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Peer Reviewed

Competing Interests: None declared

Received: 8 Apr 2020 • **Revised:** 2 Jul 2020

Accepted: 16 Jul 2020 • **Published Online**: 8 Jan 2021

Cite this article as: Gasana G, Vilchez V, Lule J, Costas-Chavarri A. Acute iron overdose associated with ileal perforation in an 11-month-old boy in Rwanda. *East Cent Afr J Surg*. 2021;26(2):88-91. doi:10.4314/ecajs.v26i2.8