CASE REPORT

Imaging insights in acute iron toxicity

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Abstract

Iron is more dangerous than most prescription medications, even though it is freely available, and is the leading cause of poisoning deaths in young infants. Imaging is important in early diagnosis and management of complications of iron poisoning, acute as well as long term. We report a case of 24-year-old female presenting with deliberate overdose of 30-40 iron (ferrous sulphate) tablets. We have discussed the role of X-ray abdomen and computer tomography in diagnosing iron poisoning. Despite treatment with deferoxamine chelation and hemodialysis, the patient's condition worsened over 72 hours, further progressing to multi-organ failure with refractory shock, culminating in cardiac failure. Despite aggressive supportive care, she succumbed to her illness. Autopsy findings revealed gastrointestinal mucosal erosion. A radiologist's role in diagnosing acute iron poisoning and assessing probable complication is significant.

Keywords: Iron tablets, Overdose, Abdominal radiology, Acute Poisoning, Iron, Adult

Introduction

Iron is more dangerous than most prescription medications, even though it is freely available, and is the leading cause of poisoning deaths in young infants. However, intentional overdoses that produce severe morbidity and mortality in both teenagers and adults indicate that iron poisoning is not limited to the pediatric age group. Only a few instances involving older individuals have been reported in the medical literature. Whether accidental or intentional, iron overdose is a health-related emergency that requires rapid and effective intervention. There is, however, a dearth of research on acute iron poisoning in adults. Therefore, radiologists should know about the significance of iron toxicity [1-4].

Case Report

A 24-year-old female presented to the emergency department with alleged history of consumption of around 30-40 iron (ferrous sulphate) tablets intentionally. Following which she developed diffuse abdominal pain, vomiting and loose stools over the next 4 hours. On examination, the patient was conscious, lethargic, but oriented. Vitals were stable on arrival. Systemic examination revealed diffuse tenderness over abdomen. The nasogastric tube was placed, and gastric lavage was done. Patient was started on intravenous fluids, proton pump inhibitor infusion, antiemetics and was kept nil per oral. After initial stabilization, the patient was shifted to intensive care unit.Her x-ray abdomen revealed multiple round radiopaque densities in the lower abdomen (Figure 1). Apart from leukocytosis (19.28×10^9/L), renal function test, liver function test and random blood sugar levels were within normal limits. Initial serum iron was 63 mcg/dl and arterial blood gas showed reduced bicarbonate

(13.8 mEq/L) which was corrected. The case was further discussed with the toxicology department and was advised for chelation with deferoxamine. Serum iron repeated after 12 hours was elevated (1438 mcg/dL); serum ferritin was also elevated (420.2 ng/mL). Patient underwent one cycle of hemodialysis. Ultrasound examination revealed mild ascites. Non-contrast Computed Tomography (CT) axial and coronal images of the abdomen in soft tissue window showed multiple hyperdense, rounded structures in the stomach, duodenum, ileum and cecum, average sizes approximately 10 × 9 mm, most predominantly in the terminal ileal loops. These hyperdense structures were suggestive of ingested iron tablets (Figures 2a, b and c). Furthermore, the CT showed long segment diffuse circumferential wall thickening with hyperdense mucosa along the ileum and entire colonic loops, with maximum wall thickness measuring approximately 6 mm in the terminal ileum which represents mucosal injury secondary to excess iron intake with mucosal iron deposits/ hemorrhage (Figures 2a, b and c).

Diffuse hypoattenuation of liver parenchyma was seen likely representing acute liver injury (Figure 2c). Colonoscopy revealed medication related colitis with patchy adherent feco-medication particles and colonic lavage was done. Repeat arterial blood gas showed metabolic acidosis. Twenty-four hours post ingestion of iron tablets, patient developed hypotension and was started on inotropes. Repeat liver function test on 2nd day of admission showed elevated serum glutamic oxaloacetic transaminase (565 IU/L) and serum glutamic pyruvic transaminase (644 IU/L). Patient was started on hepatoprotective drugs (N-acetyl cysteine). Patient had developed 1 episode of melena. Investigations were repeated on 3rd day of admission that showed worsening metabolic acidosis, coagulopathy with deranged renal function test (serum creatinine- 2.9 mg/dL) and liver function test (serum glutamic oxaloacetic transaminase- 21990 IU/L; serum glutamic pyruvic transaminase- 15210 IU/L) values. Complete blood count revealed elevated total leucocyte count (30.36×10^9/L) and reduced platelets count (84×10^9/L). The patient was transfused with fresh frozen plasma and single donor platelets. Serial arterial blood gas monitoring revealed severe metabolic acidosis for which the patient was advised to sustain low efficiency dialysis. Patient was treated for acute iron poisoning with multi-organ dysfunction syndrome. After 55 hours, the patient desaturated and was intubated. Further general condition of the patient continued to worsen with refractory shock and multiorgan dysfunction. Patient's attender had been counselled regarding poor prognosis and high risk of mortality. After 72 hours, patient developed bradycardia which led to cardiac failure and succumbed to her illness despite extensive supportive care. Autopsy was done on the same day revealed 400 ml of dark brown liquid in stomach with eroded mucosa. Liver, spleen, and kidney appeared pale.

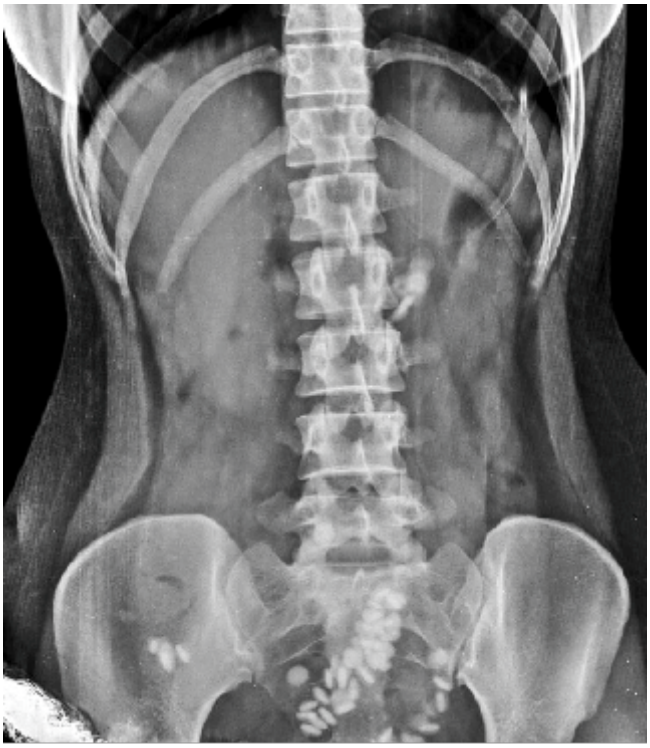

Figure 1: X-ray Abdomen AP erect, shows multiple (~40) radio opaque ingested iron tablets noted in hypogastric and right iliac fossa region

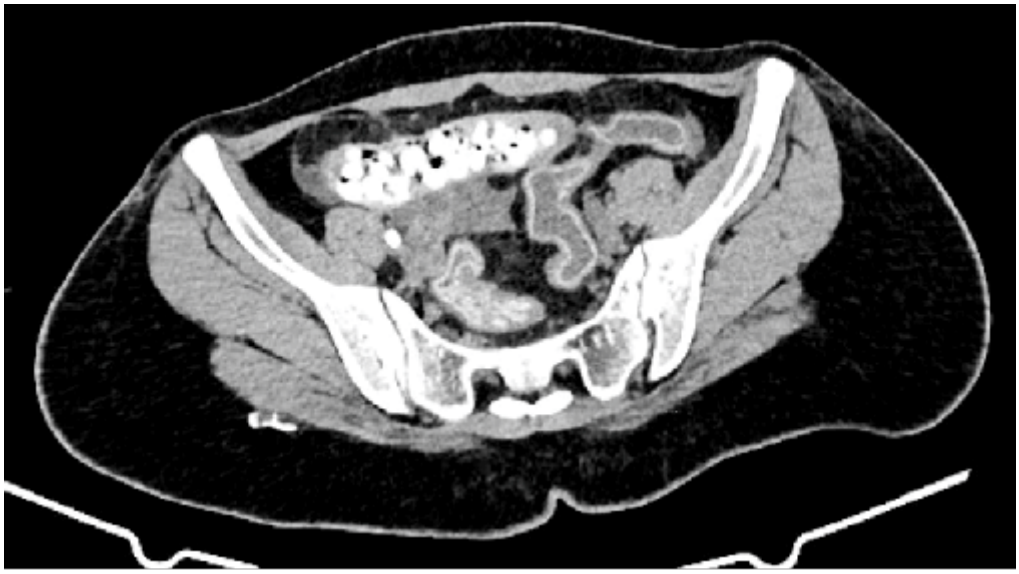

Figure 2a: Axial CT abdomen at lower abdomen

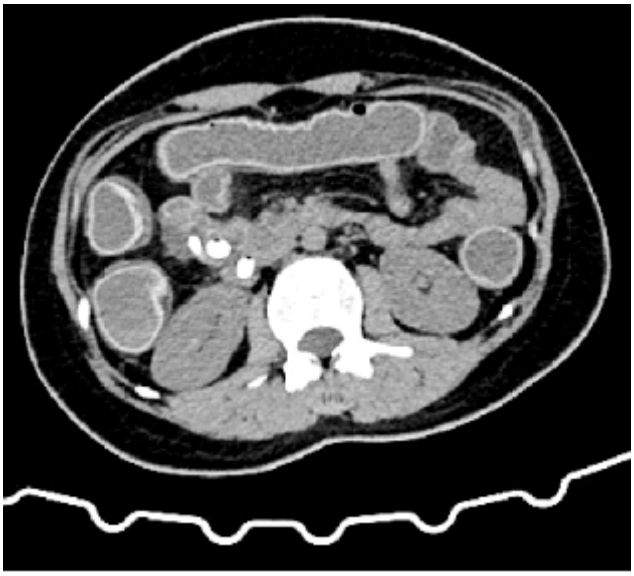

Figure 2b: Axial CT abdomen at mid abdomen

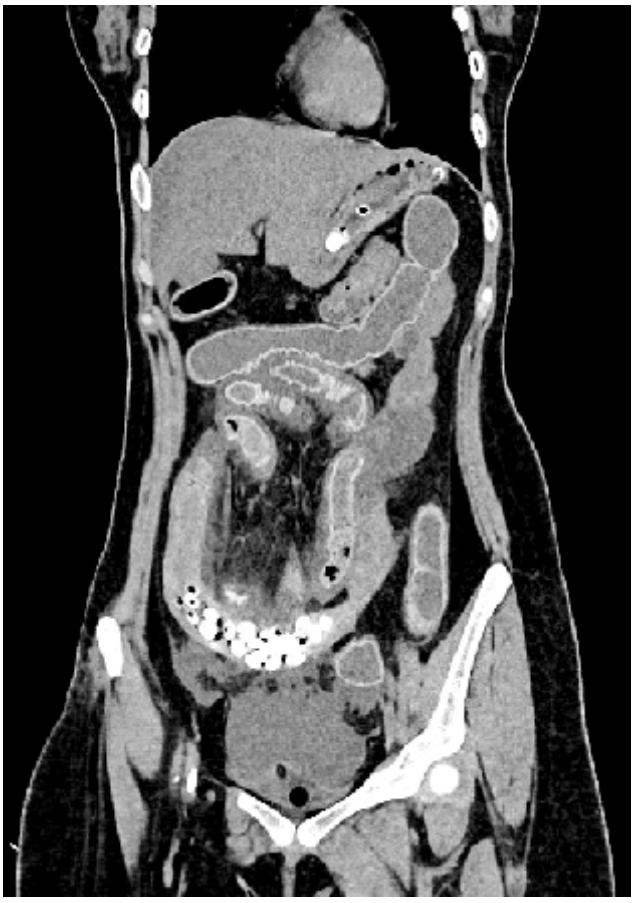

Figure 2c: Coronal CT abdomen

Figure 2 a-c: a- Axial CT image at mid abdomen, b-Axial CT image at lower abdomen and c-Coronal CT image. Axial and Coronal CT image showing multiple hyperdense rounded structures in the stomach, duodenum, ileum, and cecum with long segment diffuse circumferential wall thickening with hyperdense mucosa along the ileum and entire colonic loops. Coronal image also shows diffuse hypoattenuation of liver parenchyma.

Discussion

Acute iron toxicity, especially in the context of deliberate overdose, is a potentially life-threatening condition that requires early diagnosis and aggressive management. The patient in this case ingested approximately 30-40 iron tablets, leading to systemic toxicity, which manifested as gastrointestinal symptoms (abdominal pain, vomiting, and diarrhea) within hours of ingestion and further developed liver failure and acute kidney injury leading to multiorgan dysfunction syndrome and death. [4, 5]. Iron is crucial for various biological functions and maintaining its concentration within a narrow normal range–typically 60–170 mg/dL in males–is vital for health. One of iron's primary roles is its involvement in oxidation-reduction (redox) reactions, particularly those that occur during electron transfer in the mitochondrial respiratory chain [6]. Total Iron Binding Capacity (TIBC) and serum ferritin are commonly used to evaluate iron levels in the body. Ferritin serves as a marker of stored iron, whereas TIBC reflects the capacity of transferrin to carry iron through the bloodstream. Transferrin saturation offers a more accurate indication of iron status, as it is less affected by external factors compared to other tests. It also shows a strong correlation with serum ferritin, making it a reliable measure of the body's iron

reserves. Transferrin saturation measures the percentage of transferrin that is occupied by iron. In normal individuals, this percentage typically falls between 20% and 50%. A saturation level exceeding 50% often points to iron overload [7].

There are several stages of iron toxicity, each of which is clearly represented by clinical features:

The first stage is the gastrointestinal phase, which begins within 6 hours of consumption. Symptoms are vomiting, diarrhea, abdominal pain, and gastrointestinal hemorrhage. Hypovolemic shock and metabolic acidosis with a positive anion gap can develop at this stage and lead to mortality. Blood tests may demonstrate leukocytosis and hyperglycemia, which are associated with hepatic damage caused by iron absorption.

The second stage is the latent phase, which typically occurs 6 to 24 hours after intake. Symptoms appear to have resolved at this point, most likely due to free circulation iron redistribution to the intracellular compartment.

The third stage occurs 24 to 48 hours after intake and is characterized by shock (distributive, hypovolemic, or cardiogenic) and metabolic acidosis. Once iron enters the intracellular compartment and causes cellular malfunction, treatment is ineffective, and the prognosis worsens.

The fourth stage is defined by hepatotoxicity, which results in liver failure 48 to 96 hours after intake. This is the second leading cause of death from iron poisoning.

The final stage occur 2-8 weeks after ingestion, is associated with late sequela of gastrointestinal mucosa scarring, resulting in bowel obstruction [8].

Radiologists play a crucial role in diagnosis, assessment of severity, and management. Initial diagnosis like in this case, where history of oral tablets ingestion is present, a simple X-ray abdomen can confirm the diagnosis as evident by radio dense opacities visualized in bowel loops [9].

It is most useful in the first 2–6 hours after ingestion. Also useful in assessing complications such as intestinal obstruction or perforation, which may occur because of iron toxicity causing tissue necrosis. In cases of bowel perforation, pneumoperitoneum can be detected, which may call for emergency surgical intervention.

Ultrasound is not the primary modality for diagnosing acute iron poisoning. But sometimes, signs of inflammation are seen in the form of bowel wall thickening and intraperitoneal free fluid collection.

For further evaluation of the severity, CT is helpful. It detect bowel wall thickening, pneumatosis intestinalis secondary to bowel ischemia and detection of minimal pneumoperitoneum. In later stages, gastrointestinal mucosal scarring can lead to bowel obstruction, detected by dilated bowel loops with multiple air fluid levels. This is critical to ensure proper recovery and management of any long-term sequelae [8].

In some severe cases, interventional radiologists may be consulted for procedures such as placement of central venous catheters for chelation therapy (e.g. deferoxamine) in patients with significant iron poisoning.

Heart failure is the main cause of death in chronic iron toxicity. T2*-weighted MRI is the method of choice evaluating iron-content in myocardial cells, which have a high correlation with tissue biopsy [10].

In summary, while the primary treatment of acute iron poisoning involves gastrointestinal decontamination, supportive care, and chelation, radio-

logists are essential in diagnosing, assessing severity, and identifying complications through various imaging modalities.

Conclusion

Acute iron poisoning, although rare in adults, poses high risk of morbidity and mortality. The combination of clinical history, imaging findings, and laboratory results constitutes the broad symptom structure of iron poisoning. It also helps with forming a treatment plan custom fitted to the patient's particular requirements. Observing and close follow up is essential for surveying the patient's reaction to treatment and the progression of toxicity, for proper intervention to reduce complications. Imaging modalities ensure that clinicians can accurately analyse and treat the underlying complication, resulting in better outcome.

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