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Laboratory Findings in a Fatal Case of Iron Toxicity in an Adult: A Case Report

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Background

Acute iron poisoning in adults is an uncommon occurrence, most often associated with intentional overdoses, such as those in suicide attempts. Consuming a significant quantity of iron salts can lead to severe complications, including hemorrhagic shock, multi-organ failure, coagulopathy, and even death. This report is particularly significant as it describes a fatality resulting exclusively from iron toxicity, differing from the more typical cases where overdoses involve multiple substances.

Case Report

Patient Details:

- **Age:** 26 years
- **Gender:** Female
- **Medical History:** No notable prior health issues

Presentation and Clinical Course:

The patient reportedly ingested 30 iron tablets in a deliberate act of self-harm. On examination, she exhibited signs of multi-organ failure, likely a result of overwhelming the normal mucosal barriers responsible for regulating iron absorption. Despite receiving aggressive supportive treatment, including intravenous fluid administration and close monitoring to address the organ dysfunction, the patient succumbed to the toxic effects

of the overdose.

Conclusion

This case underscores the severe health risks posed by acute iron poisoning, emphasizing that ingesting a large quantity of iron can lead to fatal outcomes even in the absence of other substances. It highlights the importance of healthcare professionals being adequately prepared to manage such cases, given the potentially catastrophic effects of iron toxicity.

Keywords: Iron overdose, Acute toxicity, Multi-organ failure, Hemorrhagic shock, Iron poisoning

Background

Iron ranks as the most prevalent trace element in the human body, playing a crucial role in facilitating the operations of various biological systems. (1). The human body contains an estimated total iron content of around 3 to 5 grams, predominantly found within the bloodstream, while the remaining iron is stored in the liver, bone marrow, and muscle tissues in the form of heme (2). Severe iron ingestion in adults is often linked to suicide attempts (1). The concentration of elemental iron varies among different iron supplements. For example, ferrous fumarate consists of 33% elemental iron by weight, while ferrous sulfate provides 20%, and ferrous gluconate contains 12%. (3). Ingesting less than 20 mg/kg of elemental iron is generally non-toxic. Consumption of 20 mg/kg to 60 mg/kg may cause moderate symptoms, while ingesting over 60 mg/kg can lead to severe toxicity, posing significant risks of morbidity and mortality (4). Overdosing on iron leads to acute iron poisoning that can lead to severe gastrointestinal damage, acute liver cell death, haemorrhagic shock, multiple organ failure, coagulopathy, and potentially fatal outcomes (1). The clinical outcome varies based on the amount of iron consumed, the presence of other ingested drugs, and the time elapsed before treatment begins. The literature contains only a limited number of cases that describe clinical and autopsy findings related to acute iron toxicity. Furthermore, most existing studies on iron overdose in adults focus on cases involving multiple-drug overdoses, typically including paracetamol or other hepatotoxic substances. This case is significant as it illustrates a lethal result stemming solely from the toxic properties of iron, independent of any other medications.

Case Presentation

Patient's Demographic Details

On June 23, 2024, a 26-year-old woman arrived at the emergency department after reportedly consuming approximately 30 ferrous sulfate tablets at around 2:30 PM.

Medical History

The patient had no known comorbid conditions and no previous history of self-harm or suicide attempts.

Symptoms and Signs

Upon arrival at the hospital at 6:20 PM, the patient was conscious but lethargic. She reported diffuse abdominal pain, accompanied by 4-5 episodes of vomiting that included undigested tablets and multiple episodes of loose stools. Vital signs indicated hypotension (BP: 100/70 mmHg), with an oxygen saturation of 99% in room air, a capillary blood glucose level of 125 mg/dL, and a temperature of 98.6°F. Physical examination revealed diffuse tenderness in the abdomen.

Treatment or Intervention

A nasogastric tube was inserted, and gastric lavage was performed in the emergency room. The patient was started on intravenous hydration, proton pump inhibitor (PPI) infusion, and kept NPO. After initial stabilization, she was transferred to the ICU. Routine laboratory investigations were conducted, revealing elevated total leukocyte count (TLC) at 19,280. Renal and liver function tests (RFT, LFT) were within normal limits, while a urine pregnancy test was negative. Serum iron levels were initially 89 mcg/dL. ABG analysis indicated

metabolic alkalosis (pH 7.47, HCO_3 13.8). Bicarbonate correction was administered along with other supportive measures. Consultation with the toxicology department recommended intravenous deferoxamine for chelation. In addition, imaging studies including an abdominal ultrasound and CT scan were performed, along with echocardiography, to assess for any complications related to the overdose.

A repeat serum iron test showed a significant increase to 1,438 mcg/dL and ferritin at 420 ng/mL. Nephrology was consulted for hemodialysis, and one cycle was performed. Gastroenterology advised sigmoidoscopy and colonoscopy, which revealed medication-related colitis with visible medication particles; colonic lavage was performed (Fig.1).

Outcomes

Despite intensive management, the patient's condition deteriorated. She developed hypotension and required inotropic support. Laboratory results showed worsening LFT, with SGOT rising to 15,210 IU/L and SGPT to 21,990 IU/L. On June 25, 2024, her renal function worsened, with serum creatinine at 2.9 mg/dL and further elevated LFTs.

A repeat serum iron test on June 28 indicated 89 mcg/dL, but ferritin surged to 2,835 ng/mL. FFP and IVIG were administered. Serial monitoring showed continued deterioration in her clinical status, leading to recommendations for sustained renal replacement therapy (SLED) and the addition of intravenous steroids.

As her condition worsened with refractory shock and multi-organ dysfunction, a Do Not Resuscitate (DNR) consent was obtained. On June 26, 2024, the patient experienced bradycardia and cardiac arrest, succumbing to her illness at 3:20 PM.

Other Significant Details

Throughout her treatment, the patient's family was continuously updated regarding her poor prognosis and high mortality risk. Despite aggressive medical intervention, the outcome remained unfavorable, highlighting the severe impact of acute iron toxicity.

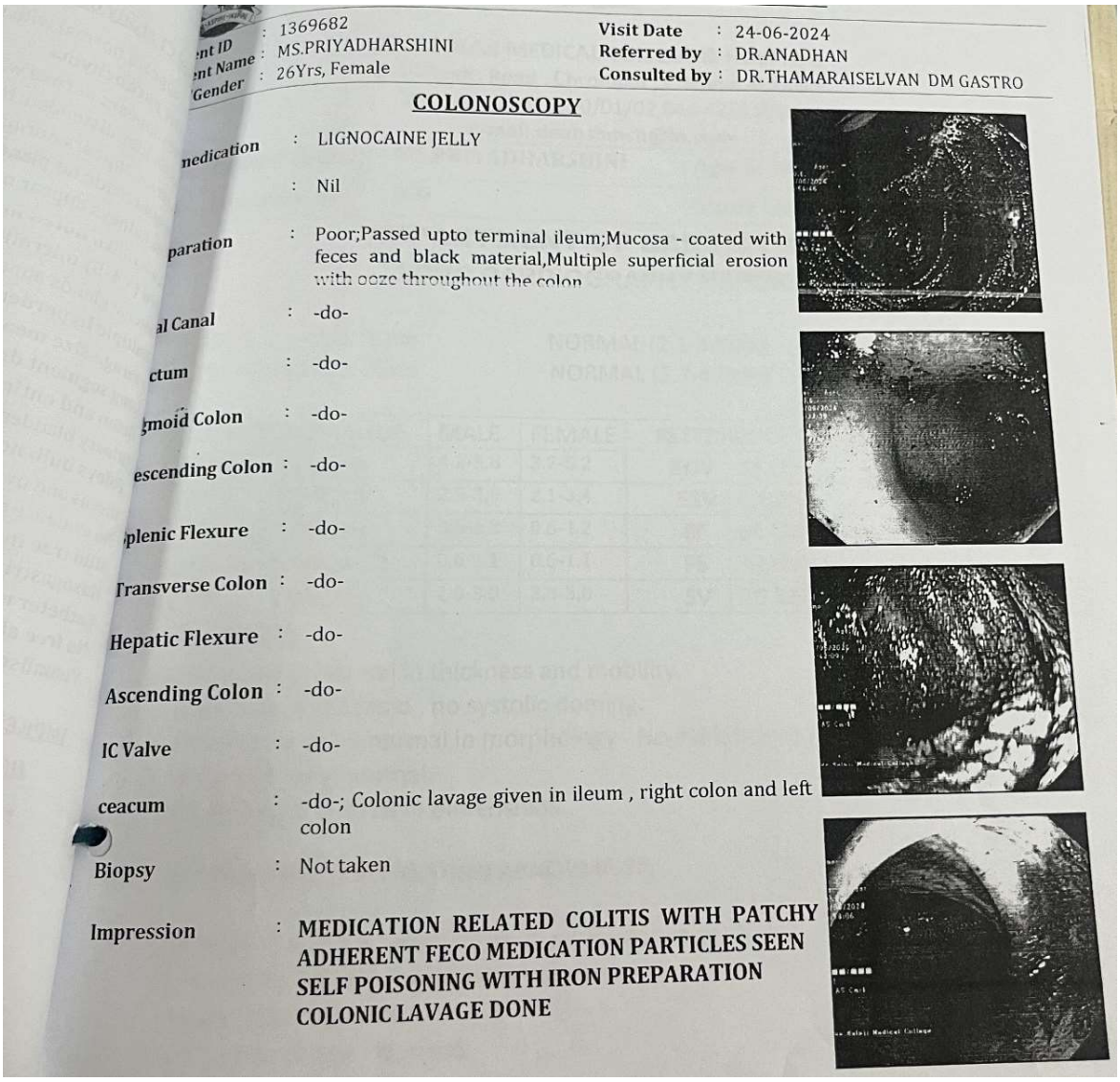
Discussion:

Iron overdose, especially at such high levels, is known to cause systemic toxicity, affecting multiple organs, notably the liver and GI tract (5). Initially, serum iron levels were elevated, peaking at a critical 1,438 mcg/dL, far exceeding normal limits (usually 60-170 mcg/dL). The significant initial rise in serum iron levels, followed by a decrease back to 89 mcg/dL on subsequent testing, suggests redistribution of iron from the bloodstream into body tissues, which is common after the acute phase of iron toxicity. This pattern reflects the dual impact of iron's corrosive effect on the gastrointestinal lining and the harmful effects of unbound iron circulating in the body (6). The regulation of free unbound iron in the bloodstream occurs through its binding to transferrin, while the hormone hepcidin plays a crucial role in controlling this process by diminishing iron absorption and the release of iron from storage sites (7). Additionally, surplus iron is sequestered in ferritin to avert potential toxicity. In cases of acute iron poisoning, the regulatory mechanisms are significantly impaired, particularly the mucosal barrier (8). Ferritin levels also surged significantly, reaching 2,835 ng/mL, consistent with iron overload and acute-phase response. These findings correlate with hepatotoxicity and the massive hepatic enzyme release, with SGOT at 15,210 IU/L and SGPT at 21,990 IU/L, indicating hepatocellular injury or necrosis. A study conducted in 2005 involving 70 patients suffering from iron toxicity revealed that 13 individuals experienced hepatotoxicity, with nine of these patients exhibiting severe toxicity characterized by SGPT levels exceeding 1,000 U/L (9). The liver is particularly susceptible to iron-induced oxidative stress, which can lead to severe hepatocellular damage, sometimes progressing to fulminant hepatic failure. The patient exhibited declining renal function, evidenced by an elevated serum creatinine level of 2.9 mg/dL, likely secondary to either direct iron toxicity or secondary effects from systemic inflammatory responses and

circulatory compromise. Acute kidney injury (AKI) is not uncommon in severe iron overdose cases due to direct nephrotoxic effects and potential shock.

The clinical presentation of iron toxicity unfolds in four distinct phases. Stage I, the gastrointestinal toxicity phase (0-6 hours post-ingestion), results in symptoms such as vomiting, hematemesis, abdominal distress, and lethargy; Stage II, the phase of seemingly stable condition (6-12 hours post-ingestion), is marked by a reduction in symptoms; Stage III, the mitochondrial toxicity and hepatic damage phase (12-48 hours post-ingestion), can lead to acute liver failure, coagulopathy, acute tubular necrosis, metabolic acidosis, and shock. Those who manage to survive this stage enter Stage IV, the gastric scarring phase (4-6 weeks post-ingestion), which is defined by gastric scarring and pyloric stricture. (9).

Imaging revealed numerous hyperdense, rounded structures in the stomach, duodenum, ileum, and cecum, suggestive of ingested iron tablets that are still present in the GI tract. The patchy colitis observed, with adherent fecal and medication particles, reflects mucosal irritation and injury from iron deposition. Additionally, there is long segmental wall thickening and hyperdense mucosa along the ileum and colon, pointing toward mucosal injury, hemorrhage, or iron deposition within the GI walls. This correlates with the characteristic findings of iron toxicity, where iron deposits can cause localized necrosis and bleeding (10). A CECT abdomen was recommended to exclude the possibility of bowel ischemia, which is a concern in iron toxicity due to the corrosive nature of iron on the GI mucosa. Bowel ischemia or perforation, though rare, could be a severe complication of iron overdose (11). The mild ascites noted may be secondary to hepatic impairment or inflammation within the peritoneal cavity. The absence of pneumoperitoneum rules out any acute perforation, which is reassuring in this context. Cardiovascular findings, including the ascending aorta and pulmonary arteries, appeared normal, with IAS/IVS and no evidence of clot, vegetation, or pericardial effusion, which suggests no acute cardiovascular compromise. Colonic lavage was performed. The colonoscopy revealed medication-related colitis characterized by patchy areas of inflammation and adherent fecal particles, consistent with self-poisoning due to iron ingestion. Colonic lavage was performed to clear the affected areas. The findings indicated significant mucosal injury likely resulting from excessive iron intake, contributing to the observed colitis (12). In summary, this case demonstrates the multi-organ impact of iron overdose, with predominant hepatotoxicity, nephrotoxicity, and GI mucosal injury.



: 1369682
nt ID : MS.PRIYADHARSHINI
nt Name : 26Yrs, Female
Gender

Visit Date : 24-06-2024
Referred by : DR.ANADHAN
Consulted by : DR.THAMARAISELVAN DM GASTRO

COLONOSCOPY

nedication : LIGNOCAINE JELLY

: Nil

paration : Poor;Passed upto terminal ileum;Mucosa - coated with feces and black material,Multiple superficial erosion with ooze throughout the colon

al Canal : -do-

ctum : -do-

gmoid Colon : -do-

escending Colon : -do-

plenic Flexure : -do-

Transverse Colon : -do-

Hepatic Flexure : -do-

Ascending Colon : -do-

IC Valve : -do-

ceacum : -do-; Colonic lavage given in ileum , right colon and left colon

Biopsy : Not taken

Impression : **MEDICATION RELATED COLITIS WITH PATCHY ADHERENT FECO MEDICATION PARTICLES SEEN SELF POISONING WITH IRON PREPARATION COLONIC LAVAGE DONE**

Conclusion:

In summary, this case reflects acute iron toxicity from an intentional overdose, with systemic effects including severe hepatic injury, acute kidney impairment, and gastrointestinal mucosal damage. Elevated liver enzymes, ferritin levels, and serum creatinine indicate significant liver and renal stress. Imaging and colonoscopy findings suggest extensive mucosal injury and inflammation due to iron deposition, with adherent iron-containing particles observed in the colon. Prompt intervention, including supportive care and chelation therapy, is essential to mitigate the potential for severe complications.

List of Abbreviations

ABG - Arterial Blood Gas
AKI - Acute Kidney Injury
CECT - Contrast-Enhanced Computed Tomography Scan
CT - Scan - Computed Tomography
DNR - Do Not Resuscitate

FFP - Fresh Frozen Plasma
GI – Gastrointestinal
IVIG - Intravenous Immunoglobulin
LFT - Liver function test
RFT – Renal function test
SGOT - Serum glutamic oxaloacetic transaminase
SGPT - Serum glutamate pyruvate transaminase
SLED - Sustained Renal Replacement Therapy
TLC - Total Leukocyte Count

References

1. Sane MR, Malukani K, Kulkarni R, Varun A. Fatal Iron Toxicity in an Adult: Clinical Profile and Review. Indian J Crit Care Med. 2018 Nov;22(11):801-803. doi: 10.4103/ijccm.IJCCM_188_18. PMID: 30598567; PMCID: PMC6259445.
2. Abhilash KP, Arul JJ, Bala D. Fatal overdose of iron tablets in adults. Indian J Crit Care Med 2013;17:311-3.
3. Manoguerra AS, Erdman AR, Booze LL, Christianson G, Wax PM, Scharman EJ, et al. Iron ingestion: an evidence-based consensus guideline for out-of-hospital management. Clin Toxicol(Phila) 2005;43:553-70.
4. Yuen HW, Becker W. Iron Toxicity. 2023 Jun 26. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 29083637.
5. Sohal, Aalam, and Kris V. Kowdley. "A Review of New Concepts in Iron Overload." *Gastroenterology & Hepatology* 20.2 (2024): 98.
6. Goyer, Robert A. "Toxicity of metals." (1990).
7. Rochette, Luc, et al. "The iron-regulatory hormone hepcidin: a possible therapeutic target?." *Pharmacology & therapeutics* 146 (2015): 35-52.
8. Gulec, Sukru, Gregory J. Anderson, and James F. Collins. "Mechanistic and regulatory aspects of intestinal iron absorption." *American Journal of Physiology-Gastrointestinal and Liver Physiology* 307.4 (2014): G397-G409.
9. Robertson A, Tenenbein M. Hepatotoxicity in acute iron poisoning. Hum Exp Toxicol.2005;24:559-62.
10. Siegelman, Evan S., Donald G. Mitchell, and Richard C. Semelka. "Abdominal iron deposition: metabolism, MR findings, and clinical importance." *Radiology* 199.1 (1996): 13-22.
11. Mueller, Paula D., and Neal L. Benowitz. "Toxicologic causes of acute abdominal disorders." *Emergency Medicine Clinics of North America* 7.3 (1989): 667-682.
12. Chua, Anita CG, et al. "Iron: an emerging factor in colorectal carcinogenesis." *World journal of gastroenterology: WJG* 16.6 (2010): 663.