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A Story of Sorrow: Iron Overdose and a Life Cut Short

[1]Dr Isabella Rita M, Sree Balaji Medical College and Hospital, Chrompet, Chennai-44

[2]Dr Anandan, Sree Balaji Medical College and Hospital, Chrompet, Chennai-44

[3]Dr Saketh Ramineni, Sree Balaji Medical College and Hospital, Chrompet, Chennai-44

Corresponding Author: Dr Isabella Rita M, Sree Balaji Medical College and Hospital, Chrompet, Chennai-44

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Abstract

Iron poisoning is a critical condition that can lead to significant morbidity and mortality if not promptly recognized and treated. This case report discusses a 20-year-old female who presented to the emergency department with severe iron overdose following the consumption of approximately 30 tablets of ferrous sulfate. Despite aggressive medical intervention, the patient developed multi-organ dysfunction and ultimately succumbed to the condition. This report highlights the clinical course, management strategies, and challenges encountered in treating severe iron toxicity.

Keywords: oxidative stress, biphasic pattern, gastrointestinal distress, diarrhea.

Introduction

Iron poisoning is a medical emergency that demands immediate attention and intervention due to its potentially fatal outcomes. This condition occurs when excessive amounts of iron are ingested, leading to toxic effects on multiple organ systems. Iron, while essential for numerous biological functions, becomes highly toxic at elevated levels, primarily due to its ability to generate free radicals and induce oxidative stress. This case report focuses on a tragic instance of acute iron poisoning in a young female, highlighting the severity of the condition, the rapid progression of symptoms, and the multidisciplinary efforts required to manage such a critical situation.

Iron poisoning typically occurs in a biphasic pattern, with an initial phase characterized by gastrointestinal distress followed by a latent phase where systemic toxicity becomes apparent. The initial phase, usually within the first 6 hours of ingestion, involves symptoms such as nausea, vomiting, diarrhea, and abdominal pain due to the direct corrosive effects of iron on the gastrointestinal mucosa. These early symptoms were evident in the case of the 20-year-old patient, who presented with diffuse abdominal pain, multiple episodes of vomiting, and loose stools after consuming a substantial quantity of ferrous sulfate tablets. As the toxic effects of iron progress, systemic involvement becomes more pronounced, leading to metabolic acidosis, hepatic failure, coagulopathy, and multi-organ dysfunction. This progression underscores the importance of early intervention and aggressive management. The patient in this case was stabilized through initial measures such as gastric lavage to prevent further iron absorption and was subsequently admitted to the Intensive Care Unit (ICU)

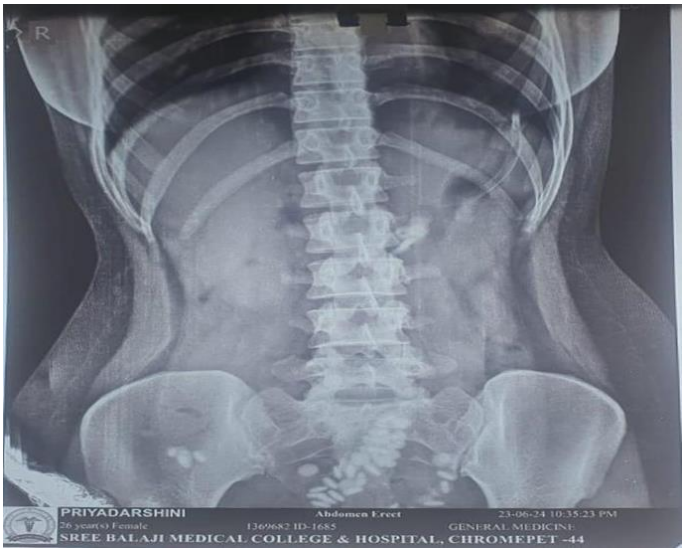
for closer monitoring and treatment. The use of deferoxamine, an iron-chelating agent, is a cornerstone in the management of severe iron poisoning. Deferoxamine binds free iron, facilitating its excretion and thereby reducing its toxic effects. In this case, despite the administration of deferoxamine, the patient's serum iron levels continued to rise, indicating ongoing iron absorption and highlighting the challenges in managing severe cases of iron toxicity. The patient's condition necessitated further interventions, including hemodialysis and continuous renal replacement therapy (CRRT), reflecting the complexity of care required in such scenarios. The involvement of multiple specialties, including toxicology, nephrology, hepatology, and gastroenterology, underscores the multidisciplinary nature of managing severe iron poisoning. This case report details the collaborative efforts and the rapid escalation of care as the patient's condition deteriorated. Despite these efforts, the patient ultimately succumbed to multi-organ dysfunction, illustrating the severe and often fatal nature of acute iron poisoning. Iron poisoning remains a significant cause of morbidity and mortality, particularly in young adults and children who may accidentally or intentionally ingest large quantities of iron supplements. This case highlights the critical need for prompt recognition, early decontamination, and aggressive chelation therapy. Furthermore, it underscores the importance of public health measures to prevent iron poisoning, such as childproof packaging and education on the safe storage of iron-containing medications.

Case Presentation

A 20-year-old female presented to the emergency department after allegedly ingesting approximately 30 tablets of ferrous sulfate at 2:30 PM due to family-related stress. She reported diffuse abdominal pain, vomiting (4-5 episodes), and multiple episodes of loose stools. Upon arrival, the patient was conscious but lethargic and oriented. Her vital signs were stable. Initial management included gastric lavage and total bowel irrigation to reduce further absorption of iron. The patient was stabilized and transferred to the Intensive Care Unit (ICU) for close monitoring. Laboratory investigations revealed a serum iron level of 39 mg/dL, and arterial blood gas (ABG) analysis showed metabolic acidosis, which was promptly corrected. Following a consultation with the toxicology department, the patient was started on deferoxamine, an iron-chelating agent, due to the significant iron overdose. A repeat serum iron level increased to 1438 mg/dL, and ferritin levels were elevated at 480 ng/mL. This alarming increase necessitated further intervention. The patient underwent a colonoscopy, which revealed medicine-related colitis, a complication of the iron overdose. Serial monitoring of serum iron levels showed a continued increasing trend, indicating worsening iron toxicity. The prognosis was communicated to the patient and her family. Serial liver function tests (LFTs) demonstrated a three-fold increase in serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT) levels, prompting a hepatology consultation. The hepatology team recommended a liver transplant due to the severe hepatic injury. As the patient's condition deteriorated, she required inotropic support to maintain hemodynamic stability. Serial increases in the international normalized ratio (INR) and liver enzymes were observed, indicating progressive liver failure. Worsening renal function was also noted, with increasing creatinine levels, leading to a nephrology consultation and the initiation of sustained low-efficiency dialysis (SLED) Serum ammonia levels were significantly elevated at 122 µg/dL. The medical team, including gastroenterologists, added vitamin K to the treatment regimen to address coagulopathy along

with colonic lavage. Despite these interventions, the patient's condition continued to worsen. Continuous renal replacement therapy (CRRT) was initiated. Ultimately, the patient developed multi-organ dysfunction and succumbed to her condition on the third day of admission.

Figure 1:



Discussion

Iron poisoning, particularly due to acute overdose, represents a significant clinical challenge. This case of a 20-year-old female who ingested a large quantity of ferrous sulfate tablets underscores the severity and rapid progression of iron toxicity, as well as the complexities involved in its management.

Pathophysiology of Iron Poisoning

Iron is a vital nutrient involved in various physiological processes, including oxygen transport, DNA synthesis, and cellular respiration. However, in excessive amounts, iron becomes highly toxic due to its ability to catalyze the formation of free radicals, leading to oxidative stress and cellular damage. The toxicity of iron affects multiple organ systems, with the gastrointestinal tract, liver, cardiovascular system, and central nervous system being the most impacted.

The clinical course of iron poisoning typically progresses through five stages. The first stage, occurring within the initial 6 hours post-ingestion, is characterized by gastrointestinal symptoms such as nausea, vomiting, diarrhea, and abdominal pain. These symptoms result from direct irritation of the gastrointestinal mucosa by iron. In this case, the patient presented with diffuse abdominal pain, vomiting, and multiple episodes of loose stools, consistent with this initial stage of iron toxicity.

The second stage, occurring between 6 to 24 hours post-ingestion, may present as a deceptive period of apparent recovery where symptoms may temporarily improve. However, systemic toxicity continues to progress. During this stage, the absorption of excess iron can lead to metabolic acidosis, which was observed in our patient and corrected early in the management process.

The third stage, occurring 12 to 48 hours post-ingestion, involves systemic toxicity manifesting as shock, acidosis, coagulopathy, hepatic failure, and renal impairment. Our patient exhibited a severe escalation in serum iron levels, metabolic acidosis, and increasing liver enzyme levels, indicating significant systemic toxicity.

The fourth stage, occurring 2 to 5 days post-ingestion, is characterized by hepatic failure and coagulopathy. In this case, the patient’s liver function tests showed a three-fold increase in SGOT and SGPT, indicative of severe hepatic injury. Additionally, increasing INR levels highlighted the development of coagulopathy.

The fifth stage, occurring weeks after ingestion, may involve gastrointestinal scarring and strictures, although our patient did not survive long enough to reach this stage due to multi-organ dysfunction and death on the third day of admission.

Clinical Management

The management of iron poisoning involves several critical steps: stabilization of the patient, decontamination

to prevent further absorption of iron, administration of antidotes, and supportive care for affected organ systems. In this case, the patient underwent gastric lavage shortly after presentation to reduce further iron absorption. While the efficacy of gastric lavage is debated, it is considered when the patient presents within a few hours of ingestion.

The cornerstone of medical management for iron poisoning is the administration of deferoxamine, an iron-chelating agent. Deferoxamine binds free iron, forming a complex that is excreted in the urine, thus reducing the toxic effects of iron. The patient in this case was started on deferoxamine following consultation with the toxicology department, leading to a transient improvement. However, subsequent iron levels showed a significant increase, indicating ongoing iron absorption and toxicity.

Complications and Multidisciplinary Interventions

Severe iron poisoning often necessitates a multidisciplinary approach due to the involvement of multiple organ systems. In this case, the patient's condition deteriorated rapidly, requiring consultations with nephrology, hepatology, and gastroenterology.

Nephrology was consulted when the patient's creatinine levels increased, indicating renal impairment. Sustained low-efficiency dialysis (SLED) was recommended and initiated to manage the renal complications and facilitate the removal of excess iron. Additionally, continuous renal replacement therapy (CRRT) was later required as the patient's condition worsened.

Hepatology was consulted due to the significant hepatic injury evidenced by a three-fold increase in liver enzymes and an elevated INR. The hepatology team recommended a liver transplant, highlighting the severity of hepatic failure. Unfortunately, the rapid progression of multi-organ dysfunction precluded the possibility of transplant.

Gastroenterology played a role in managing the coagulopathy associated with iron poisoning. The team recommended the administration of vitamin K to address the elevated INR, although this intervention was ultimately insufficient to reverse the patient's deteriorating condition.

Prognostic Factors and Challenges

Several factors influence the prognosis of patients with iron poisoning. These include the amount of iron ingested, the timing of presentation and intervention, the effectiveness of decontamination and chelation therapy, and the presence of complications such as hepatic failure and renal impairment. In this case, the large quantity of ferrous sulfate ingested and the rapid progression of systemic toxicity contributed to the poor prognosis.

One of the significant challenges in managing severe iron poisoning is the dynamic nature of iron levels in the body. Initial serum iron levels may not accurately predict the severity of poisoning or the eventual outcome. Serial monitoring is crucial to assess the effectiveness of treatment and guide further interventions. In this case, serial iron monitoring showed an increasing trend despite aggressive treatment, indicating worsening toxicity and poor prognosis.

Conclusion

Iron poisoning is a medical emergency that requires prompt recognition and aggressive management. This case highlights the severity and rapid progression of iron toxicity, the need for a multidisciplinary approach, and the challenges faced in treating such cases. Despite timely and comprehensive interventions, the patient developed multi-organ dysfunction and succumbed to the condition. This underscores the importance of early detection, effective decontamination, and the critical role

of iron-chelating therapy in managing severe iron poisoning. Future research should focus on improving diagnostic and therapeutic strategies to enhance outcomes for patients with iron toxicity.

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