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Fulminant Hepatic Failure Following Intentional Iron Overdose—A Case Report

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ABSTRACT

Intentional iron overdose in adults is a rare but formidable clinical challenge, often complicated by delayed presentation and a concealed history of ingestion. We report the case of a 21-year-old male who presented 4 days after consuming large quantities of ferrous sulfate tablets. The initial nonspecific gastrointestinal symptoms progressed to severe jaundice, encephalopathy, and coagulopathy, and the diagnosis was confounded by the patient's denial of suicidal ingestion. By the time iron toxicity was identified, the therapeutic window for early decontamination or parenteral chelation had been passed. Supportive care, including advanced extracorporeal therapies, facilitated hepatic and renal recovery. This case highlights the importance of high clinical suspicion, thorough history taking, and the potential role of salvage therapies in severe late-presenting iron poisoning.

1 | Introduction

Iron, which is essential for hemoglobin synthesis and cellular metabolism, is a potent cellular toxin at high doses [1]. Ferrous sulfate supplements, although generally safe at therapeutic levels, can cause severe toxicity when ingested in large quantities [2]. Suicidal ingestion in adults is uncommon, but can rapidly progress to fulminant hepatic failure, metabolic derangements, and coagulopathy [3–5]. Early recognition and intervention, ideally gastric lavage or iron chelation, are pivotal [6, 7]. However, in delayed presentations, the therapeutic window closes, forcing clinicians to consider alternative treatment strategies. Additionally, concealed histories, as in this case, can obscure diagnosis until severe hepatic and neurological complications arise. Here, we present the case of a young adult who was covertly overdosed with ferrous sulfate, only disclosing his ingestion after severe organ injury had developed. This report emphasizes vigilance, comprehensive evaluation, and innovative use of extracorporeal therapies for managing complex toxic exposures.

Summary

- Advanced extracorporeal therapies such as plasma exchange and hemodialysis are crucial for managing late-presenting ferrous sulfate poisoning, reversing multiorgan failure, and enabling recovery when early treatments, such as chelation, are no longer effective.

2 | Case Presentation

2.1 | Case History and Examination

A 21-year-old Indian male presented with 4 days of abdominal pain, vomiting, and watery diarrhea. Over the preceding 3 days, he noted progressive jaundice, dark urine, and reduced urine output. Denying recent alcohol intake, he attributed his symptoms to unverified complementary medication. On examination, he was icteric and exhibited prominent scleral icterus (Figure 1A,B), right upper quadrant tenderness, and asterixis, suggestive of hepatic encephalopathy. Vital signs showed BP 115/70 mmHg; pulse, 98 bpm; respiratory rate, 20 breaths/min; and temperature, 37.2°C. His psychiatric history included a suicide attempt 3 years prior, but he initially denied recent toxic ingestion. Table S1 presents a comprehensive, day-by-day timeline of the patients' laboratory trends, clinical interventions, and the corresponding interpretations and correlations with disease progression.

2.2 | Differential Diagnosis and Investigations

Differential diagnoses included acute viral hepatitis, toxic/metabolic liver injury, ischemic hepatitis, and drug-induced or tropical infections. Viral serology and tests for endemic pathogens yielded negative results. Abdominal ultrasonography revealed mild hepatomegaly, altered hepatic echotexture, and moderate ascites. Laboratory evaluations (see Table 1 for admission parameters) revealed markedly elevated transaminase levels (AST > 5000 U/L, ALT > 12,000 U/L), severe hyperbilirubinemia (total bilirubin 6.1 mg/dL, rising rapidly), and profound coagulopathy (INR 5.5 at admission). Elevated ammonia levels (120 μmol/L) correlated with encephalopathy. Metabolic acidosis and elevated lactate (4.2 mmol/L) signified advanced hepatic dysfunction. After persistent questioning, on Day 3 of admission, he admitted ingesting ~30 ferrous sulfate tablets (~3000 mg elemental iron) 4 days before presentation. By this point, conventional interventions like gastric lavage or intravenous deferoxamine were no longer feasible or effective.

FIGURE 1 | Progressive scleral icterus during the course of severe hepatic dysfunction. (A) Initial presentation with prominent jaundice. (B) Worsened scleral icterus observed on Day ~9 post-ingestion, indicative of severe hyperbilirubinemia.

2.3 | Treatment

Supportive measures included intravenous vitamin K (10 mg daily), repeated FFP transfusions (two units guided by PT/INR), and careful fluid/electrolyte management. NAC was started on Day 8 (150 mg/kg loading dose, then 50 mg/kg over 4 h, followed by 100 mg/kg over 16 h daily) to support hepatic glutathione reserves. Empiric antibiotics were administered briefly and discontinued because no infection was identified. Given the late stages, extracorporeal therapy was considered. Under ultrasound guidance, a double-lumen hemodialysis catheter was placed in the left internal jugular vein (Figure 2). Two PLEX sessions were performed (first on Day ~6 post-ingestion, second on Day ~12), each exchanging about 1.5 L of plasma with donor plasma using citrate anticoagulation. Renal deterioration (creatinine rose from 1.2 to 3.1 mg/dL) necessitated HD on Day ~13 and Day ~19 (each session lasting ~4 h) to manage fluid overload and metabolic derangements. Throughout this period, sedation (e.g., IV midazolam titrated for encephalopathy control) and nutritional optimization were meticulously adjusted. No definitive iron chelation was administered because of the delayed presentation and extensive intracellular iron distribution.

2.4 | Follow-Up

After the second PLEX, clinical stabilization began despite the bilirubin peaking near 37 mg/dL. Ammonia levels declined, coagulopathy gradually improved, and transaminase levels significantly decreased. By Day 15, there was clear clinical improvement; over the next 2 weeks, jaundice subsided, coagulation parameters normalized, and renal function recovered. At about 1 month post-ingestion, liver enzymes and bilirubin levels approached normal, and the patient's mental status returned to baseline. Discharged on Day 30 with psychiatric follow-up, the patient was advised to strictly avoid unsupervised medications. Outpatient evaluations confirmed sustained clinical stability and resolution of the encephalopathy.

3 | Discussion

This case differs from typical iron toxicity scenarios, which generally occur earlier and respond to iron chelation [8, 9]. The delayed presentation and denial of ingestion precluded early intervention, shifting the therapeutic focus to salvage strategies. PLEX and HD, although not standard for iron poisoning, proved instrumental in supporting hepatic and renal recovery when traditional measures were ineffective.

TABLE 1 | Key admission biochemical parameters and clinical interpretations.

Parameter	Patient value at admission	Reference range	Interpretation
Total bilirubin (mg/dL)	6.1	0.1–1.0	Marked hyperbilirubinemia, indicating early severe liver injury
Direct bilirubin (mg/dL)	3.6	0.0–0.3	Predominantly conjugated hyperbilirubinemia, severe hepatocellular dysfunction
Indirect bilirubin (mg/dL)	2.5	0.2–0.8	Elevated unconjugated fraction due to impaired uptake/conjugation
SGOT (AST) (U/L)	5905	5–40	Massive hepatocellular injury (necrosis)
SGPT (ALT) (U/L)	12,357	7–56	Severe hepatocellular injury, consistent with fulminant hepatitis
Alkaline phosphatase (U/L)	210	40–129	Mildly elevated; nonspecific but supports hepatic injury
Gamma-GT (GGT) (U/L)	98	9–48	Elevated, associated with cholestasis or hepatic stress
Prothrombin time (PT) (sec)	66.6	10–13	Severe coagulopathy due to decreased clotting factor synthesis
INR	5.5	≤1.1	Profound coagulopathy, high bleeding risk
Serum creatinine (mg/dL)	1.8	0.6–1.2	Early renal impairment, possibly toxin-related
Serum ammonia (μmol/L)	120	15–45	Elevated, risk of hepatic encephalopathy
Serum lactate (mmol/L)	4.2	0.5–2.2	Elevated, reflecting impaired metabolism and perfusion
Arterial blood pH	7.31	7.35–7.45	Mild metabolic acidosis from hepatic failure
Serum electrolytes (Na/K)	Na 136 mmol/L; K 4.2 mmol/L	Na 135–145; K 3.5–5.1	Near-normal, monitored closely due to fluid shifts and HD

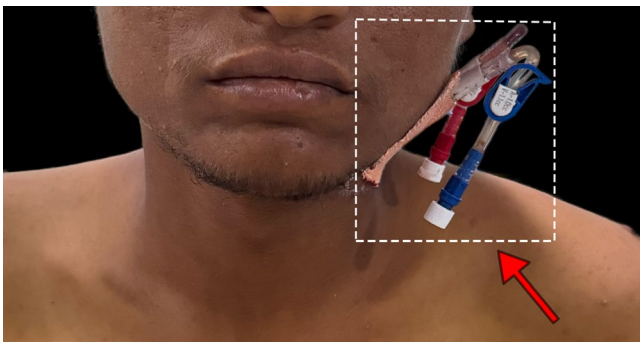

FIGURE 2 | Clinical image showing the patient during advanced extracorporeal therapy. The inset and red arrow highlights the placement of a double-lumen hemodialysis catheter in the left internal jugular vein, used for plasma exchange and hemodialysis sessions.

The timing and severity highlight the challenges of late-stage iron toxicity. When iron saturates binding proteins, it inflicts oxidative damage on hepatocytes and endothelium, leading to fulminant hepatic failure and coagulopathy [10–12]. At this advanced stage, removing circulating mediators and supporting failing organs can tip the balance toward recovery [13]. Although evidence for extracorporeal therapies for iron overdose is limited, their successful use suggests a role in selected cases.

Future research directions include early detection tools (rapid bedside assays for iron levels), improved prognostication models, and next-generation iron-binding agents. Multidisciplinary collaboration remains essential. While early recognition and treatment are paramount, cases like this demonstrate the necessity of broadening the therapeutic arsenal. Psychiatric intervention and preventive measures are vital given the psychiatric history and deliberate nature of the overdose.

4 | Conclusion

Late-presenting ferrous sulfate overdose can cause life-threatening fulminant hepatic failure and coagulopathy, which are resistant to standard interventions. Full recovery is achievable with vigilant supportive care and advanced extracorporeal therapies (PLEX and HD). High clinical suspicion, thorough psychiatric evaluation, and willingness to employ innovative treatments can turn the tide into severe, delayed iron toxicity.

Author Contributions

Hrithik Dakssesh Putta Nagarajan: conceptualization, writing – original draft, writing – review and editing. **Ram Vivek Ramamoorthy:** conceptualization, writing – original draft, writing

– review and editing. **Naveen Vishwanath:** writing – original draft, writing – review and editing. **Miruthula Murugan:** writing – original draft, writing – review and editing. **Balakrishnan Kamaraj:** conceptualization, writing – original draft, writing – review and editing. **Shiva Jeyaprakash Narayan Balamurugan:** conceptualization, writing – original draft, writing – review and editing. **Shubham Kumar:** writing – original draft, writing – review and editing. **Shilpa Gaidhane:** writing – original draft, writing – review and editing. **Sanjit Sah:** writing – original draft, writing – review and editing. **Prakasini Satapathy:** writing – original draft, writing – review and editing. **Rachana Mehta:** writing – original draft, writing – review and editing. **Amogh Verma:** conceptualization, supervision, project administration, validation, writing – original draft, writing – review and editing.

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Disclosure

Guarantor: Amogh Verma. Generative artificial intelligence (AI) tools, including Paperpal and ChatGPT-4o, were utilized solely for language refinement, grammar enhancement, and stylistic refinement. These tools had no role in the conceptualization, data analysis, interpretation of results, or substantive content development of this manuscript. All intellectual contrivutions, data analysis, and scientific interpretations remain the sole work of the authors. The final content was critically reviewed and edited to ensure accuracy and originality. The authors take full responsibility for the accuracy, originality, and integrity of the work presented.

Ethics Statement

No formal ethical approval was required for this case report. All identification details were de-identified to maintain confidentiality.

Consent

Written informed consent for publication of this case report, including associated images and data, was obtained from both the patient and his legal guardian in accordance with our institution's guidelines for cases involving psychiatric evaluation.

Data Availability Statement

All relevant data, including figures, tables, and Supporting Information, are provided within the manuscript and accompanying files. Any further details can be obtained by contacting the corresponding author.

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