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Case Report

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Near-fatal ferrous sulfate poisoning: A case report of successful conservative management

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ABSTRACT

Rationale: Acute iron poisoning is commonly seen in a pediatric population caused by accidental ingestion of iron syrups. We describe a case of iron poisoning who presented to the hospital following intentional near-fatal ingestion of ferrous sulfate.

Patient's Concern: A 14-years-old previously healthy female patient presented to the emergency department with a history of an intentional overdose of 80 ferrous sulfate tablets.

Diagnosis: Ferrous sulfate poisoning.

Interventions: The patient developed acute fulminant liver failure 24 h after of the overdose. She was managed conservatively, mainly with deferoxamine and *N*-acetylcysteine while awaiting transfer to a liver transplant facility.

Outcomes: The patient responded well to medical therapy and was discharged on the 9th day of intensive care unit admission.

Lessons: This case highlights the patient's successful recovery with prompt conservative therapy. Severe iron toxicity can be treated with early use of deferoxamine and *N*-acetylcysteine where a liver transplant facility is not available.

KEYWORDS: Iron; Toxicity; *N*-acetylcysteine; Deferoxamine; Liver failure

1. Introduction

Acute unintentional iron poisoning is common and can potentially be fatal in the pediatric age group[1]. Suicidal iron tablet overdose is uncommon in children as well as in adults. Depending on the amount of iron ingested, ingestion along with other drugs, and the timing of intervention, there are various clinical outcomes of an iron overdose. Severe iron overdose can lead to acute hepatic failure, multiorgan failure, and death[2]. Our patient presented with severe iron toxicity with a blood iron of level 2 500 μg/dL resulting in hypotension, severe metabolic acidosis, and multiorgan failure. Despite such high blood iron levels and acute liver failure, she made full recovery with prompt medical management using deferoxamine (DFO) and empirical *N*-acetylcysteine (NAC) therapy. Eventually, she did not require a liver transplant and was discharged in healthy condition.

2. Case report

This case report was approved by the ethical committee of the hospital. The patient's father has given informed consent.

A 14-year-old-female patient, weighing 60 kg, and previously healthy, was presented to the emergency department with a history of an intentional drug overdose. She was brought into the hospital by her father approximately 90 min after ingesting 80 ferrous sulphate tablets (65 mg elemental iron). On admission to the emergency department, she was feeling drowsy with a Glasgow Coma Scale score of 9/15 and having continuous coffee ground vomiting episodes. She was hemodynamically stable with no evidence of end-

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Table 1. Day-wise parameters of ferrous sulfate poisoning.

Parameters	Normal references	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9
Hemoglobin, g/dL	13.0-17.0	15.6	11.7	11.3	11.1	11.4	9.6	9.2	9.5	10.4
WBC, $\times 10^3/\mu L$	3.6-11.0	5.6	8.8	10.8	9.7	8.8	6.6	6.1	7.6	7.6
Platelet, $\times 10^3/\mu L$	150-400	492	281	264	213	222	172	189	202	157
Serum Na, meq/L	136-145	137	142	144	145	139	141	139	141	145
Serum K, meq/L	3.3-4.8	3.5	3.7	3.4	3.2	4.0	3.6	3.6	3.8	3.7
Serum urea, mg/dL	12-40	16	11	15	18	22	15	12	15	20
Serum creatinine, mg/dL	0.7-1.2	0.8	0.9	1.0	0.8	0.6	0.8	0.4	0.7	0.8
Bilirubin, mg/dL	0.8-1.2	0.2	3.1	3.2	2.8	1.7	1.8	1.2	1.4	1.0
ALP, U/L	<187	102	80	89	69	60	66	90	91	91
SGPT, U/L	0-31	16	638	1 549	1 298	745	531	443	339	322
SGOT, U/L	0-32	28	818	1 194	503	242	138	109	56	50
PT, s	12.7-16.1	14.1	23.2	36.9	27.1	20	16.6	15.8	15.1	14.1
APTT, s	33.9-46.1	35	43.9	52.4	45.6	46.4	35.5	38.8	37.2	34.2
INR	0.97-1.30	1.06	2.00	3.59	2.45	1.66	1.31	1.23	1.16	1.08

WBC: White blood cell; ALP: Alkaline phosphatase; SGPT: Serum glutamic-pyruvic transaminase; SGOT: Serum glutamic oxaloacetictransaminase; PT: Prothrombin time; APTT: Activated thromboplastin time; INR: International normalized ratio.

organ damage. She was electively intubated for airway protection. Gastric lavage was done by using saline. Urgent blood investigations did not reveal any abnormality (Table 1). An electrocardiogram showed normal sinus rhythm. Chest and abdominal X-ray did not show any abnormal opaque material. The first serum iron level, which was taken approximately 2 h after ingestion, was 2 500 μg/dL (normal: 37-145 μg/dL). Whole bowel irrigation was administered as the toxicologist advised till the effluent was clear. In case of worsening condition in the next 24 h, the patient was transferred to intensive care unit (ICU) for further management. She was started on DFO infusion at the rate of 5-15 mg/kg/h for chelation of iron and her serum iron level was measured frequently (Figure 1). As expected, her hemodynamic and liver function started to deteriorate 24 h after admission. She developed severe metabolic acidosis and hemodynamic instability along with deranged liver function tests. All supportive ICU care was continued and the decision for referral to a liver transplant facility was considered. NAC infusion was started because of acute liver failure while waiting for transfer to a liver transplant facility. Her liver function started to improve in 24 to 48 h with an improvement in hemodynamics. Since there was a dramatic improvement in liver function tests and hemodynamics after NAC infusion, the planning of the liver transplant was kept on hold. She was eventually extubated 6 d after ICU admission and discharged to the ward with stable vitals.

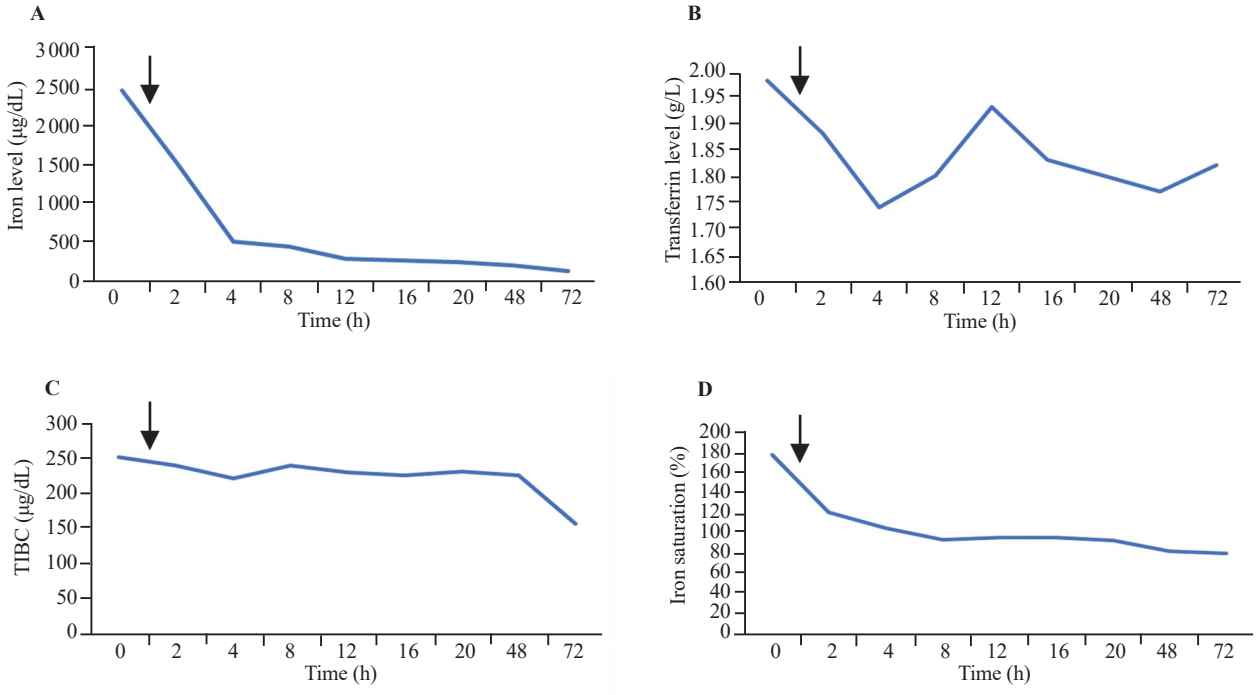

Figure 1. Trends of (A) iron, (B) transferrin, (C) TIBC, and (D) iron saturation levels of a 14-year-old female patient with ferrous sulfate poisoning over a 72 h period. Deferoxamine was given at the time indicated by the arrows. TIBC: total iron-binding capacity.

3. Discussion

The present case discusses the diagnostic presentation and the importance of prompt conservative treatment in iron toxicity. Acute iron poisoning can cause serious complications leading to death in absence of early treatment. Iron toxicity follows five stages, although these stages usually have an overlapping presentation[3]. Stage 1 (0 to 6 h) presents with vomiting, diarrhea, haematemesis, melena, and abdominal pain. Significant fluid losses may lead to hypovolemic shock. At stage 2 (6 to 12 h), gastrointestinal symptoms wane and the patient appears to be getting better. During this time iron shifts intracellularly from circulation. At stage 3 (12 to 48 h), cellular toxicity begins to manifest as a vasodilative shock, third-spacing, high anion gap metabolic acidosis, and hepatorenal failure. Acute hepatic failure happens at stage 4 (2 to 5 d) and has high mortality rate. At stage 5 (2 to 6 weeks), chronic sequelae occur in survivor's cirrhosis and gastrointestinal scarring and strictures. The stages of the clinical course may not be seen in all patients and cases of massive overdose patients may present in a shocking state. For this reason, when iron toxicity is diagnosed, it should be followed closely and treated in the ICU. Treatment modalities include and are not limited to gastric lavage, whole bowel irrigation, and DFO as an iron-chelating agent. Immediate initiation of DFO therapy is warranted in presence of serum iron concentration of more than 500 μg/dL, severe metabolic acidosis, repetitive vomiting, lethargy, and signs of shock[3]. The standard recommended infusion rate is 15 mg/kg/h, though some authors advise starting at a lower dose and titrating up to avoid hypotension[4,5]. In our case infusion was started at 1 mg/kg/h and was titrated to 15 mg/kg/h slowly. After starting DFO infusion, urine color changed to orange which indicated active chelation of iron. DFO is ideally administered early while the majority of iron is accessible for chelation in serum, for a short duration, and to avoid side effects of prolonged infusion. DFO infusion was stopped after 10 h once urine color returned to normal. Despite DFO infusion, iron toxicity may progress to hepatotoxicity which is associated with 50% of mortality[6]. NAC has been shown to have a beneficial effect in non-acetaminophen acute liver failure[7]. NAC is a thiol-containing agent that acts as a free radical scavenger and replenishes mitochondrial and cellular glutathione stores. It can also serve as a source of glutathione surrogate that combines directly with reactive metabolites or serve as a source of sulfate, thus preventing liver damage[8]. Various trials have proven the anti-inflammatory, antioxidant, inotropic, and vasodilating effects of NAC[9]. Based on the evidences above, NAC was used with other conventional treatments for the benefit of patients as a bridging modality where a transplantation facility was not available. NAC was given in a loading dose of 150 mg/kg/h over 1 h followed by 12.5 mg/kg/h for 4 h then a continuous infusion of 6.25 mg/kg/h for the remaining 67 h. To the best of our knowledge, only four prospective studies have reported some benefits of NAC used in non-acetaminophen acute liver failure patients[10]. We were compelled to use NAC due to worsening liver failure and unavailability of a liver transplant facility at our center and the relative safety profile of NAC. Fortunately, the patient's liver function started to improve once NAC was started. The patient was further stabilized over 5 d and then discharged from ICU after having stable vitals.

Acute iron poisoning in humans has not been adequately studied because of the infrequent and sporadic occurrence of cases. Reduction in the incidence of iron poisoning over the years due to preventive measures instituted by concerned authorities has resulted in clinicians' inexperience regarding evaluation and management of cases, particularly DFO and NAC. This case highlights successful management of severe iron toxicity with early use of DFO and NAC. Early consultation with toxicologists can help physicians to manage complicated cases of iron toxicity, especially in the context of a reduction in the frequency of such cases. Early treatment and close follow-up in ICU can reduce mortality significantly.

Conflict of interest statement

The authors report no conflict of interest.

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Authors' contributions

WSS.: concept and design of article, acquisition of data, drafting the article; AS and ZA: acquisition of data, revising the article critically for important intellectual content; SS and MAB: revising the article critically for important intellectual content.

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