

# Fatal overdose of iron tablets in adults

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## Abstract

Acute iron toxicity is usually seen in children with accidental ingestion of iron-containing syrups. However, the literature on acute iron toxicity with suicidal intent in adults is scant. We report, the first instance of two adults with fatal ingestion of a single drug overdose with iron tablets from India. Two young adults developed severe gastro-intestinal bleeding and fulminant hepatic failure 48 h after deliberate consumption of large doses of iron tablets. Serum iron levels measured 36 h after ingestion were normal presumably due to the redistribution of iron to the intracellular compartment. Despite aggressive supportive management in medical intensive care unit of a tertiary care hospital, the patients succumbed to the toxic doses of iron.

**Keywords:** Adults, fatal, iron tablets, overdose

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## Introduction

Intentional iron tablets overdose in adults is uncommon. The majority of acute iron toxicity cases occur in children less than 5 years of age who present with accidental ingestion of iron supplements.<sup>[1]</sup> Clinical outcome is variable and depends on the quantity of iron ingested, other drugs ingested and the delay to treatment. Severe iron overdose can cause acute hepatic necrosis and lead to multi-organ failure and death.<sup>[2,3]</sup> Most of the literature on iron overdose in adults is on multiple-drug overdose usually with paracetamol or other hepatotoxic drugs. This report is unique in that our patients allegedly consumed only iron tablets and both died as a result of direct toxicity of iron alone.

## Case Reports

### Case 1

A 25-year-old lady allegedly consumed 200 tablets of ferrous sulphate with suicidal intent. The total dose amounted to 13.5 g of elemental iron (270 mg/kg). She had abdominal pain with vomiting and was given gastric lavage along with supportive care at a primary

health center. She was referred to our hospital after 24 h of ingestion for further management. At presentation, she was hemodynamically stable with a normal pulse rate and blood pressure. Complete blood count (CBC) showed hemoglobin-14.1 g/dl, total white blood cell (WBC) count-19,300/cu mm (90% neutrophils) and platelet count-283,000/cu mm. Serum iron level as measured by spectrophotometric analysis was 73 µg/dl (normal range 40-145 µg/dl) and the total iron binding capacity was 350 g/dl (normal range 250-350 µg/dl). Liver function tests (LFT) showed marked elevation in liver enzymes (serum glutamic oxaloacetic transaminase [SGOT]-11,000 U/L and serum glutamic pyruvic transaminase [SGPT]-11,420 U/L), total bilirubin (2.6 mg%), direct bilirubin (1.1 mg%), protein (4.6 g%), albumin (2.8 g%) and alkaline phosphatase (161 U/L). Serum creatinine was 1.9 mg%, bicarbonate was 15 mmol/l and blood sugar level was 81 mg/dl. Chelation therapy with desferrioxamine was started as an intravenous infusion of 15 mg/kg/h and increased to 25 mg/kg/h. 24 h after admission, she developed severe gastro-intestinal (GI) bleeding and went into a state of circulatory shock. Hemoglobin dropped from 14.1 to 7 gm%. Activated partial thromboplastin time (aPTT) was 71.3 s and prothrombin time (PT) was 24.5 with an international normalized ratio (INR) of 1.9. Arterial blood gas analysis showed severe metabolic acidosis with a pH of 6.96. She became progressively drowsy with un-recordable blood pressure, for which she was intubated and ventilated and supported with multiple

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inotropes. Aggressive blood product supports were given and hemodialysis was initiated for metabolic acidosis. Despite these measures, she continued to deteriorate and died within 36 h of admission due to multi-organ failure.

### Case 2

A 17-year-old lady allegedly consumed 300 tablets of ferrous sulfate amounting to a total dose of 20 g of elemental iron (400 mg/kg). She was initially taken to a local hospital with abdominal pain and vomiting and gastric lavage given. Further treatment details are unknown. After 48 h, she developed multiple episodes of hematemesis and melena and progressively became oliguric and drowsy. She was intubated and referred to our hospital 48 h after ingestion. At presentation, pulse and blood pressure were not recordable and resuscitated with colloids. CBC profile showed hemoglobin-2.8 g/dl, total WBC count-4,500/cu mm, platelet count-52,000/cu mm. LFT showed marked elevation in liver enzymes (SGOT-4,250 U/L and SGPT-3,908 U/L), total bilirubin (0.7 mg%), direct bilirubin (0.5 mg%), protein (2.2 g%), albumin (0.7 g%) and alkaline phosphatase (72 U/L). Serum iron levels were not measured as the patient presented more than 48 hours after ingestion. Serum creatinine was 2.4 mg%, bicarbonate was 10 mmol/l, blood sugar level was 27 mg/dl, aPTT was >3 min and PT was >2 min with an INR of >10. Aggressive supportive management along with desferrioxamine as a 15 mg/kg/h infusion was initiated. Hemodialysis was planned, but could not be instituted as the patient had refractory hypotension and had a cardiac arrest within 2 hours of presentation. Despite ventilator and inotropic supports, she continued to deteriorate with circulatory collapse and could not be resuscitated. She died within 3 h of presentation to our hospital.

### Discussion

Iron is the most abundant trace element in the body and is essential in most biological systems. Acute iron poisoning causes GI, cardiovascular, metabolic, hepatic and central nervous system toxicity. This is due to a direct caustic effect of iron on the GI mucosa and the toxicity of free unbound iron in the circulation. Clinically, iron toxicity manifests in four stages. Stage I/stage of GI toxicity (0-6 h since ingestion) causes vomiting, hematemesis, abdominal pain and lethargy; Stage II/stage of apparent stabilization (6-12 h since ingestion) when symptoms subside; Stage III/stage of mitochondrial toxicity and hepatic necrosis (12-48 h since ingestion) where patients may develop acute liver failure, coagulopathy, acute tubular necrosis, metabolic acidosis and shock. Patients who survive this phase go

into Stage IV/stage of gastric scarring (4-6 weeks since ingestion) characterized by gastric scarring and pyloric stricture.<sup>[2]</sup>

Acute liver failure and cardiovascular collapse are the main causes of death due to iron overdose. In 2005, a review of 70 patients with iron toxicity showed hepatotoxicity in 13 patients with severe toxicity (alanine transaminase/SGPT >1,000 U/L) in nine patients. Ten of these patients (all <18 years) died with one of them requiring liver transplantation.<sup>[3]</sup> Kozaki *et al.* reported a patient with massive iron ingestion successfully treated with liver transplantation.<sup>[4]</sup> A literature search for adult cases of iron poisoning associated with hepatotoxicity yielded only three cases that were older than 15 years of age.<sup>[5]</sup> The only reported adult death directly attributed to iron toxicity without ingestion of other drugs was that of a 30-year-old pregnant lady who consumed 70 mg/kg of elemental iron.<sup>[6]</sup> She died of fulminant hepatic failure 2 weeks later. Ingestion of more than 60 mg/kg is associated with high toxicity and the lethal dose of elemental iron is said to be 200-250 mg/kg.<sup>[7]</sup>

A serum iron level of more than 350 µg/dl between 2 and 6 h post-ingestion is supposed to indicate a significant intoxication and levels more than 500 µg/dl suggest serious risk of acute liver failure. However, serum iron levels as measured by spectrophotometric analysis may not be reliable in diagnosis or prognostication if the patient presents late as iron is redistributed to the intracellular compartment within 6-12 h.<sup>[2,8]</sup> There is also a possibility of idiosyncratic reaction to iron. Our patients presented more than 24 h after ingestion of the tablets, which explains normal serum iron levels with severe toxicity.

Treatment of acute iron poisoning includes early decontamination of the gut, chelation with parenteral desferrioxamine and intensive supportive therapy. Administration of desferrioxamine after acute intoxication may color the urine a pinkish red, a phenomenon termed "*vin rose* urine." Whole bowel irrigation (WBI) may be useful in children if abdominal X-ray reveals radio opaque iron tablets beyond the pylorus or throughout the GI tract.<sup>[7]</sup> X-ray abdomen in both our patients did not show any radio-opaque pills and WBI was not done.

### Conclusion

In conclusion, though iron tablets overdose with suicidal intent is uncommon, delay in diagnosis and administration of chelation therapy may rapidly allow

progression of toxicity to multi-organ failure and death even in adults. Serum iron levels may not be reliable for diagnosis and prognostication if the patient presents late.

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