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IP International Journal of Forensic Medicine and Toxicological Sciences

Journal homepage: https://www.ijfmts.com/

Case Report

# Fatality of health supplements– An autopsy case report on iron tablets poisoning

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## Abstract

Iron tablets are the commonly prescribed and also very frequently used over the counter health supplements. 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 teenage is scant. We report a case of school going boy playfully ingested multiple iron tablets and due to the ignorance of the teacher, presented lately to the medical facility. Because of late presentation and unusual minimal fatal dose, he lost his life, inspite of intensive treatment.

**Keywords:** Iron tablets, Gastrointestinal bleed, Fatal dose, Awareness

**Received:** 02-04-2025; **Accepted:** 06-05-2025; **Available Online**: 07-07-2025



## 1. Introduction

Ferrous sulfate is a commonly prescribed drug for prophylaxis and treatment of anaemia, particularly in women and adolescent girls.[1] It has been given regularly in schools under ANAEMIA MUKT BHARAT programme.[2] However, it's easy availability, potential toxicity at higher doses, and vague clinical presentation make it a drug of concern when evaluating a case of poisoning. In literature, most of the iron poisoning cases reported are of pediatric age group. Most cases of acute iron toxicity occur in children under the age of 5 due to inadvertent intake of iron supplements.[3] Iron poisoning in an adult is uncommon.[4] While adults rarely intentionally overdose on iron supplements, people may falsely believe that iron supplements are harmless and use them as a suicide threat. Here in this article, we present a case of acute iron intoxication in a 15-year-old male who consumed iron tablets in little excess unknowingly and succumbed to an uncommon complication of Iron overdose.

## 2. Case History

A 15 year old apparently healthy male was brought to casuality with alleged history of self-ingestion of ferrous sulphate tablets (Each tablet contains 100mg of elemental iron & 0.5mg of folic acid). It is revealed in the history that he took 10 tablets of ferrous sulphate around 11.00 A.M in his school. Immediately after ingestion, His fellowmates informed their teacher about the incident and they ignored as it was health supplement tablets. Around 8.00 PM on the same day, He was admitted with the symptoms of dark yellow coloured loose stools and brown coloured vomitus and treated as per the protocol including N-acetyl cysteine. Chelation therapy with desferrioxamine was started, and supportive measures were continued. On admission and day 1, all blood parameters (CBC, LFT, RFT) were normal. On 2nd day, liver enzymes were elevated. On 3rd day, patient went into hypovolemic shock and supported with inotropes. Inspite of all the treatment, patient expired at 9.30 A.M on day 4. After police inquest, body was brought for autopsy. Autopsy was done around 3.00 P.M on the same day. The following findings were noted:

Moderately built and moderately nourished male dead body weighed 38kg;

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http://doi.org/10.18231/j.ijfmts.2025.015

*2.1. No external antemortem injuries anywhere on the body*

Postmortem hypostasis seen on the back, ill defined & fixed; Dark red subconjuctival hemorrhages on eyeballs; Nailbeds of finger & toe nails were pale; Oral cavity and genital organs: Intact; Dark red petechial & ecchymotic hemorrhages on the mediastinum, heart and lungs; Larynx and trachea: Empty; Liver, spleen & kidneys: Normal in size and congested; Stomach: Contained 100ml of blackish brown fluid with no definite smell, pale mucosa (**Figure 1**); Small intestine: jejunum part contained thick green mucous; ileal part contained reddish fluid blood and clotted blood; Large intestine: thick fluid blood and intestinal lumen shaped clotted blood for 100cm length(**Figure 2**A & 2B); Dark red petechial hemorrhages seen on the mesentry and mesocolon; Other visceral organs were pale; Scalp, Vault & Base of skull: Intact; Brain: Pale; Ribs, Hyoid bone, Pelvis &Spinal column: Intact;

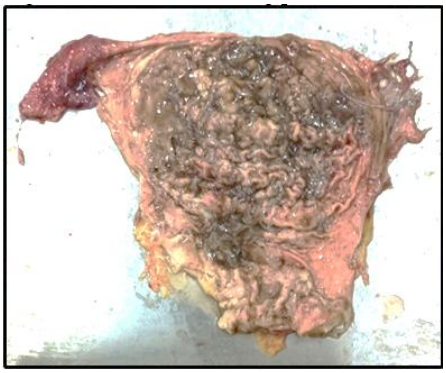

**Figure 1:** Stomach showing pale mucosa

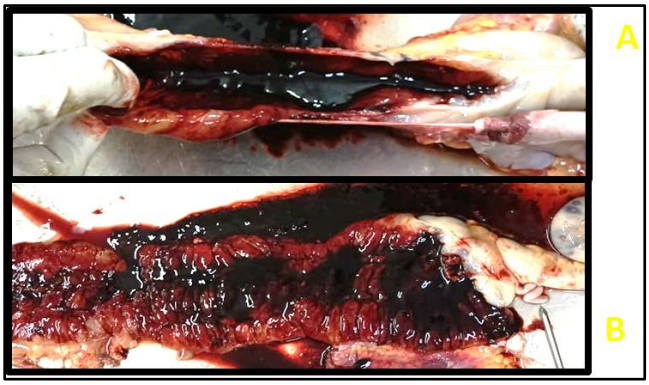


**Figure 2 A:** Small intestine shows reddish fluid blood & clotted blood and **B:** Large intestine shows thick fluid blood.

Viscera sent for chemical analysis: Tissue bits sent for histopathological examination: Blood sent for biochemistry examination;

*2.1. Viscera report*

Negative for poison or alcohol;

*2.2. Biochemical report*

B.Urea: 107 mg/dl, S.Creatinine: 3.34 mg/dl, Total Bilirubin: 2.89, Total Protein: 4.6, SGOT: 4670 U/L, SGPT: 3105 U/L, ALP: 321 U/L.

S. Ferritin: 493 microgram/dl, UIBC: 492.2 microgram/dl, Iron: 19.3 microgram/dl.

*2.3. Histopathological examination (HPE)*

1. Myocardium and coronaries: Normal
2. Lungs: Interstitial congestion and diffuse intra-alveolar hemorrhage with focal emphysematous changes;
3. Liver: Congestion
4. Kidneys: Congestion with acute tubular necrosis
5. Cerebrum and cerebellum: Normal
6. Stomach: Ulcerated mucosa and congested
7. Small intestine: Inflamed, ulcerated mucosa with congestion (**Figure 3** A)
8. Large Intestine: Ulcerated, congested, inflamed mucosa with submucosal edema (**Figure 3** B)
9. Spleen: Congestion

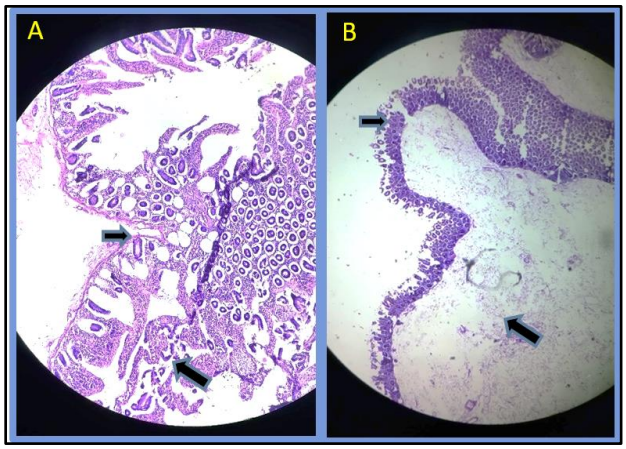


**Figure 3 A:** HPE of Small intestine shows inflamed & ulcerated mucosa and B: HPE of large intestine shows inflamed mucosa with submucosa edema

*2.4. Final opinion*

On perusal of history of the case, clinical diagnosis of cause of death, hospital treatment records, chemical analysis of viscera, histopathological examination reports and biochemistry reports, the deceased would appear to have died due to effects of Ferrous sulphate poisoning.

## 3. Discussion

Iron is the most abundant trace element in the body and is essential in most biological systems but can be toxic in excessive amounts.[5] Iron is an intracellular poison, and it acts on mitochondria by shunting electrons away from the electron transport chain, uncoupling the oxidative phosphorylation, which leads to anaerobic metabolism and toxic effects.[6] Clinical outcomes vary depending on the amount of elemental iron consumed, other medications taken,

and the length of time between diagnosis and treatment.[7] Understanding these factors enables a forensic pathologist to arrive an opinion on the cause of death and guides the physician on treatment and precautions.

The lethal dose and minimum toxic doses of iron poisoning are not clearly established. Ingestion of more than 60 mg/kg elemental iron can be associated with serious toxicity. A wide range of iron doses (from 60 to 300 mg/kg of elemental iron) has been reported in lethal cases[8]. However in our case, it is only half of the documented lethal dose (1000mg of elemental iron / 26mg/kg) which is rare and unusual.

Adult iron poisoning is uncommon and often results in significant clinical management challenges due to its nonspecific presentation and potential for rapid deterioration. It is reported that patients will have better outcome and survival if treated early and managed with desferrioxamine, bowel irrigation, and other supportive treatment.[9] But In this case, there was a delay of 8-9 hours for medical attention which could have fatal outcome. This delay in medical attention can be attributed to lack of awareness related to iron poisoning.

The common symptoms of iron poisoning include vomiting, abdominal pain, diarrhoea, and in severe cases, metabolic acidosis, hypotension, hypoglycemia, dehydration, liver and kidney injury, as well as injuries to the cardiovascular system and the central nervous system.[10] Of course, victims also suffer damage to the gastrointestinal tract, which is due to the direct corrosive effects of iron compounds on the gastrointestinal mucosa.[11] Though the fatal GI bleed is rare, this case succumbed to this rare effects of Iron supplements;

The clinical presentations of acute iron poisoning have been well documented in the literature and are typically divided into five stages.[12-14]

1. Stage I: (Gastrointestinal phase) occurs 30 min to 6 h after ingestion with the major gastrointestinal manifestations including abdominal pain, vomiting, explosive diarrhea, gastrointestinal bleeding, and hematemesis. Death caused by hypovolemic shock can also occur at this stage in cases with severe toxicity.

2. Stage II: (Latent phase) typically occurs from 6 to 24 h after ingestion and is a quiescent stage. There is recovery of gastrointestinal symptoms and redistribution of iron to the reticuloendothelial system.

3. Stage III: (shock and metabolic acidosis) usually begins about 6– 72 h after iron ingestion. In addition to hypovolemic shock due to blood loss or distributive shock resulting from increased capillary permeability and third spacing, cardiogenic shock can also occur due to the depressant effect of iron on the cardiomyocytes. Metabolic acidosis can be caused as the result of tissue hypoperfusion and free radical-related mitochondrial dysfunction.

4. Stage IV: (Hepatotoxicity) can occur 12– 96 h after ingestion. Changes that happens are acute hepatic necrosis, hepatic failure, and resultant coagulopathy. Excessive free radical production can also cause acute lung and renal injuries.

5. Stage V: (bowel obstruction and other complications) can occur 2– 8 weeks after ingestion and may manifest as gastrointestinal scarring and obstruction, hepatic cirrhosis, and cerebral damage

Gastrointestinal hemorrhage with shock caused by the corrosive action of the iron was the main mechanism of death in this case examined. However the presentation is late when compared to other studies. Abhilash et al. reported GI hemorrhage and metabolic acidosis as causes of death within 24 h from ingestion in two adults.[5] This correlates with the gastrointestinal and latent stage of iron toxicity. Overlapping of features of different phase is more common as described by Dongfang Yu MD, PhD Mark A. Giffen Jr. DO.[15] Crofton et al. showed GI symptoms to be the most common presenting symptoms and liver failure to be the most common fatal complication.[16] Though the presenting symptoms in our case is GI symptoms, the cause of death is related to intestinal haemorrhage which is a rare entity.

## 4. Conclusion

This case adds to the limited literature on intestinal haemorrhage due to iron poisoning and serves as a reminder of the potential fatal complication of iron poisoning. It calls for stringent regulation of iron supplement distribution and public education on the risks associated with overdose.

## 5. Source of Funding

None.

## 6. Conflict of Interest

None.

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**Cite this article:** Saravanan S[1], Muthukumar G, Veeravijayan A. Fatality of health supplements– An autopsy case report on iron tablets poisoning. *Int J Fotren Med Toxicol Sci.* 2025;10(2):54-57.