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The Efficacy of Oral Chelator in Acute Iron Poisoning

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Abstract **Case Report**

Iron intoxication can occur accidentally in children or intentionally by adults as a suicide attempt. Serious iron toxicity depends upon the amount of elemental iron ingested, the peak serum iron concentration measured before 6 hours after ingestion, and the presence of clinical manifestations of toxicity. The aim of this case report is to demonstrate that the clinical manifestations may be not related to the amount of elemental iron ingested and that oral iron chelator can be used as an antidote for an iron poisoning.

Keywords: Oral Chelator Acute Iron Poisoning.

INTRODUCTION

Iron is found in many multivitamins. Its intoxication can occur accidentally in children or intentionally in adolescents and adults as a suicide. Although it is more frequent in children. 34 cases of iron exposure were reported between January 2001 and November 2006 [1].

The amount of elemental iron ingested depends on the drug preparation. Ferrous sulfate contains 20%, ferrous gluconate contains 12% and ferrous fumarate contains 33%.

The exposure of less than 20 mg/kg of elemental iron is nontoxic. Ingestion of 20 mg/kg to 60 mg/kg results in moderate symptoms. Ingestion of more than 60 mg/kg can result in severe toxicity and lead to severe morbidity and mortality.

CASE REPORT

A 29-year-old pregnant woman in her first trimester with no medical history, was admitted to the emergency department five hours after attempting suicide by ingesting 30 Tablets of Tardyferon 50 mg, equivalent of 18.54 mg/Kg of elemental iron.

On admission she was stable, a Glasgow coma scale of 15. She had no complaints except for vomiting and abdominal pain. Her vital signs were a heart rate of 90 beats/min, a blood pressure of 120/63 mmHg.

Arterial blood gas analysis showed a pH of 7.47, a pCO_2 of 26.4 mmHg, a bicarbonate level of 19 mmol/l, and a pO_2 of 105 mmHg and a serum lactate level of 0.65 mmol/l.

Her serum iron level at her admission (after 5 hours of ingestion) was 452µg/dl. Laboratory tests did not show neither any abnormalities in her liver enzymes (alanine transaminase, aspartate transaminase, gamma-glutamyl transpeptidase, alkaline phosphatase, and lactate dehydrogenase were normal), nor a kidney injury; her creatinine level was normal. She did not develop haemolysis or rhabdomyolysis. Echocardiography didn't show any abnormalities, or hypovolemia.

Deferoxamine wasn't available; the patient received 15mg/Kg of iron oral chelator (Deferasirox) for a persistent symptomatology. Her serum iron level was decreasing, 355µg/dl after 10 hours of poisoning, 220µg/dl after 18 hours and 179µg/dl after 20 hours.

Her laboratory test after 20 hours showed a normal Ferritin level (110ug/l), total iron-binding capacity at 52% and unsaturated iron-binding capacity at 1.67 mg/l.

After 48 hours of admission in the intensive care unit, the patient didn't show any other symptoms. After her discharge, she underwent a psychiatric evaluation.

DISCUSSION

Iron is an essential element for normal cell metabolism; however, it is highly cytotoxic and even

lethal in excess quantities. An adult human contains approximately 3–5 g of iron, 60% of it is incorporated into hemoglobin and the rest is stored in hepatocytes and reticulo-endothelial macrophages [2]. It is absorbed by duodenum enterocytes in the form of ferrous iron (peak between 3 hours and 5 hours).

Serious iron toxicity depends upon the amount of elemental iron ingested, the peak SIC measured before 6 hours after ingestion, and the presence of clinical manifestations of toxicity [3].

Iron poisoning cause a multiphasic syndrome. Few hours after ingestion, the first phase is characterized by gastrointestinal symptoms (vomiting and diarrhea), it is caused because of the corrosive effects of iron on the gastrointestinal tract.

The second phase is a silent one, the symptoms disappear and it lasts for 24 hours or so. However, in severe cases, this resolution of symptoms might not be observed [4, 5], multiple organs dysfunctions can occur secondary to the free iron in the blood that disrupts cellular function, which is characteristic of the third phase that typically becomes apparent 48 – 96 hours after ingestion. The patient develops renal and liver failure, lactic acidosis, coma and convulsions.

Four to six weeks after ingestion, patients who survived severe iron poisoning develop scarring on the gastrointestinal tract which is characteristic of the fourth phase [6].

Measurement of serum iron concentration should be done at its peak (between 3 and 5 hours after ingestion). A serum iron level of more than 350 µg/dl correlates with a significant intoxication and levels more than 500µg/dl suggest a danger of acute liver failure [7]. Although our patient ingested only 18.54 mg/kg of elemental iron, her serum iron level was more than 350 µg/dl, which correlates with a significant intoxication.

In the study conducted by Robertson *et al*. [8], the hepatotoxicity is related to the fact that the free serum iron is absorbed by Kupffer cells and hepatocytes, exceeding the storage capacity of ferritin and causing oxidative damage. Pathologic changes include cloudy swelling, peri-portal hepatic necrosis, and elevated transaminase levels.

The therapeutic agent for significant iron intoxication is intravenous Deferoxamine. It is a chelating agent that can remove iron from tissues and free iron from plasma and is indicated in patients with systemic toxicity, metabolic acidosis, worsening symptoms, or a serum iron level of more than 500µg/dl [9]. It is administered as a continuous infusion at 15 mg/kg/hr for up to 24 hours with a maximum dose of 80 mg/kg per day.

For our case, unfortunately, Deforoxamine was not available. The patient received 15mg/Kg of iron oral chelator (Deferasirox) for a persistent gastrointestinal symptomatology. Griffith *et al*. [10] conducted a study to determine the effect of orally administered deferasirox in 8 healthy human adults after ingesting 5 mg/kg of elemental iron. The use of Deferasirox significantly reduced serum iron levels.

Mohamed Yassin *et al*. [11] reported a case of a 27-year-old female nurse presented with acute intravenous iron intoxication (60 mg/kg). Her serum iron level was 1116µg/dl, she accepted only oral therapy and was started on deferasirox at a dose of 30 mg /kg daily. This oral chelation proved to be effective in clearing her hepatic iron overload after six months.

Other than iron chelator, exchange transfusion can be used in cases with iron poisoning. In a case reported by Carlsson *et al*. [12], an 18-month-old girl ingested 442mg/kg of elemental iron, after 2 hours of standard therapy; her serum iron had risen threefold to 1362 µg/dl. The child was treated with exchange transfusion and serum iron fell to 134 µg/dl.

CONCLUSION

The severity of iron poisoning depends on the amount of the ingested iron; a dose greater than 40mg/kg is seriously toxic and greater than 60mg/kg is often fatal. It causes gastric irritation, renal, hepatic and cardiac failure. The treatment is based essentially on intravenous Deferoxamine. Oral iron chelator can be effective in acute iron intoxication.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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