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Acute iron poisoning: a case report

Abstract

In adults, the common cause of iron poisoning is iron overload caused by large excess of iron supplement in take at suicidal attemps. When serum iron level exceeds the iron binding capacity of the body, free radicals occur, leading to lipid peroxidation and cellular membrane damage. In iron poisoning, most affected organs are liver, heart, kidney and lungs. Also hematologic system is affected negatively. Acute iron poisoning can cause serious complications resulting in death. The treatment scheme is determined by the type of iron preparation, time of intake, and the onset of symptoms. Initial treatment approaches mostly consist of supportive care and removal of iron with bowel irrigation.[1–3] Early treatment and close follow-up in intensive care unit are important for acute iron poisoning. In this case report, we examined the approach to a case of acute iron poisoning with moderated oseiron intake for suicide attempt.

Keywords: acute iron poisoning, suicide, intensive care unit

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Introduction

Inadults, the common cause of iron poisoning is iron overload caused by large excess of iron supplement intake at suicidal attemps. Excessive oral iron-induced caustic effect affects the gastrointestinal (GI)tract. This causes massive iron absorption. When serum iron level exceeds their on binding capacity of the body, free radicals occur, leads to lipid peroxidation and cellular membrane damage. In iron poisoning, most affected organsareliver, heart, kidney and lungs. Also hematologic system is affected negatively. Acute iron poisoning can cause serious complications resulting in death.[1,2] The severity of poisoning depends on the amount of iron intake. If intake of elementary iron is below 20mg/kg, the risk of toxicity is low, decontamination and at least 6hours observation is recommended. There is a moderate risk of toxicity between 20 and 40mg/kg. Decontamination and chelation therapy should be considered. Doses above 60mg/kg are at high risk and decontamination with chelation therapy should be started.[3] In this case report, we examined the approach to acute iron poisoning with moderated oseiron intake for suicide attempt.

Case report

A 22years old female-weigh 60kg-consumed 20 tablets of ferrosanolduodenal at home with suicidal attempt. (100mg Fe^{+2} or 567,7mg iron (II)-glycine-sulfate in each tablet). She applied to emergency service after 15-20minutes. Gastric lavage was performed and arterial bloodgas (ABG) resulted as pH: 7,14 pO_2: 53,4mm $HgpCO_2$:46,9mm$HgHCO_3$: 19mmol/L BE: -10mmol/LSO_2: %88. She was admitted to the intensive care unit for metabolic acidosis. There was no property in the patient's history. General condition was good, glas gowcomascale (GCS) was 15 and she was conscious .Vital findings and physical examination were normal. The patient who had vomited twice had no complaints of abdominal pain. Initial ABG was pH: 7,37 pO_2: 57,3mmHg pCO_2: 34,8mm$HgHCO_3$: 20mmol/L BE: -5,1mmol/ LSO_2: %88. Liver function tests for hepatotoxicity, renalfunction tests for nephrotoxicity, complete blood count for leukocytosis, and coagulation parameters for coagulopathy were evaluated in the laboratory and all of them were normal. Electrocardiogram was normal at sinusoidal rhythm. The first serum iron level was 379µg/dL (high), iron binding capacity was 9µg/dL (low), and ferritin was 19,9ng/ mL (normal) after 5hours of drug intake. Deferrioxamine treatment was not given because serum iron level of the patient was not higher than 500µg/dL. ABG was pH: 7,35 pO_2: 88mmHg pCO_2: 32,6mmHg HCO_3: 17,7mmol/L BE: -7,8mmol/L SO_2: %96 after 24hours. Serum ironlevelwas 180µg/dL (normal), ironbindingcapacitywas 136µg/dL (normal), ferritin was 39,7ng/mL (normal) 24hours later. ABG was pH: 7,45 pO_2: 102mmHgpCO_2: 36,3mmHgHCO_3: 24mmol/L BE: -1,5mmol/L SO2: %98 at 48hours. Serum iron level was 69µg/dl (normal), iron binding capacity was 265µg/dl (normal), ferritinwas 44,6ng/ml (normal) 48 hours later. The patient was discharged in good general condition.

Discussion

Acute iron poisoning can cause serious complications resulting in death. These verity of intoxication depends on the amount of iron intake. Iron toxicity can be classified as corrosiveor cellular. Ingested iron can have an extremely corrosive effect on GI mucosa, which can manifest as nausea, vomiting, abdominal pain, hematemesis, and diarrhea; patients may become hypovolemic because of significant fluid and blood loss. Cellular toxicity occurs with the absorption of excessive quantities of ingested iron. Severe over dose causes impaired oxidative phosphorylation and mitochondrial dysfunction, which can result in cellular death. The liver is one of the organs most affected by cellular iron toxicity, but other organs such as the heart, kidneys, lungs, and the hematologic systems may also be impaired.[1,2]

With both corrosive and cellular toxicity, the end result is significant metabolic acidosis, due to several factors. Hypoper fusion due to significant volume loss, vasodilatation, and negative inotropic effect of iron will result in lactic acidosis. Inhibition of oxidative phosphorylation will promote anaerobic metabolism. Individuals demonstrate signs of GI toxicity after ingestion of below 20mg/ kg. Moderate intoxication occurs when ingestion of elemental iron between 20-40mg/kg. Ingestions exceeding 60mg/kg can cause severe toxicity and may be lethal.[3] Our patient also had metabolic acidosis and had a moderate toxic dose of iron uptake (33mg/kg). She had presented with only vomiting.

Clinically, iron toxicity manifests in fivestages. Stage 1/stage of GI toxicity (0-6h since ingestion) causes vomiting, hematemesis, abdominal pain and lethargy; Stage 2/stage of apparent stabilization (12-24h since ingestion) when symptoms subside; Stage 3/stage of mitochondrial toxicity (24-48 h since ingestion) where patients may develop, coagulopathy, acute tubular necrosis, metabolic acidosis and shock. Stage 4 of hepatotoxicity (after 48hours since ingestion) patients who survive this phase go in to Stage 5/stage of gastric carring (2-4weeks since ingestion) characterized by gastrics caring and pyloric stricture. Iron poisoning can lead to cardiovascular collapse, mental status changes, gastro intestinal bleeding, liver and kidney failure.[4] For this reason, it should be diagnosed early, closely followed and treated in intensive care unit.

Treatment modalities includede contamination gastric lavage or whole-bowel irrigation. There are several presentations that may necessitate immediate initiation of deferoxamine therapy. They are: presence of metabolic acidosis, repetitive vomiting, lethargy, hypotension, orsigns of shock. If the serum iron concentration is greater than 500mcg/dL, defroxamine therapy should be initiated. In our case deferrioxamine treatment was not given because serum iron level of the patient was not higher than 500µg/dL.[1,3] The patient whom vital findings were stable was discharged after 48hours.

Conclusion

The physicians should have the knowledge of the signs, symptoms, treatment and prognosis of ironpoisoning. Acute iron poisoning may lead to serious complications that may result in death. Therefore, early treatment and closely follow-up in intensive care unit can reduce mortality.

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Conflict of interest

The author declares no conflict of interest.

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