

Case Report

A CASE OF IRON POISONING-CASE REPORT

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ABSTRACT

We report a case of acute iron poisoning in a 28 year old female presented to casualty with alleged history of consumption of 90 tablets of iron tablets-polyfol forte(100mg of elemental iron + folic acid 0.5mg) with suicidal intention. She developed initially haemetesis followed by shock and classical iron poisoning presentation. We discuss here the management of acute iron poisoning in ICU.

Keywords: *Acute Iron Poisoning, Adults, Desferrioxamine*

INTRODUCTION

Iron preparations are available in the form of oral supplements, intramuscular injections, and intravenous injections. Iron preparations are used in the treatment of milder forms of anemias. Iron poisoning is most commonly seen in the children, but rarely in adults.

CASES

A 28 year old female presented to casualty with alleged history of consumption of 90 tablets iron tablets-polyfol forte (100mg elemental iron + folic acid 0.5mg) with suicidal intention. She initially developed sudden onset of vomiting. Later, she developed frank hematemesis. Patient lost around 250ml of blood. On examination, her pulse rate was 100bpm, blood pressure 120/70mmhg, and respiratory rate was 18/min and saturation was 96-98% on room air and other systemic examinations were normal. Whole bowel irrigation was administered and started on PPI infusion, anti emetic and inj. Desferrioxamine at a dose of 15 mg/kg/hr and was shifted to ICU for further management. Lab investigations reports showed hb-8.7g/dl,pcv-31.9%,total counts-8840 cells, peripheral smear-microcytic hypochromic anemia. Platelets-2.6 lakhs, pt-23.4, aptt-113.2, inr-1.69, bun11.7, s.creatinine-0.61, uric acid-4, s. electrolytes were normal. Serum iron->2130 TIBC - 2146.8, LFT – total bilirubin 0.82, direct bilirubin 0.29, total protein 7.4, serum albumin 4.4, a:g 1.5, AST 45, alt 20, alp 62, ABG ph 7.29 pco2 30.3 po2 78.6 hco3 14.3 (metabolic acidosis). USG abdomen and pelvis and x-ray were normal. In the ICU, 2 units of prbc's and 4 units plasma were transfused. Patient continued to have hematuria and hematemesis. On 2nd day, after 48hrs, hemoglobin dropped to 6.6, pt, APTT, INR were 28.9, 53.6, 2.149 respectively. 2 units of prbc's and 4 ffp's were transfused, desferrioxamine and pantoprazole infusions were continued and later vitamin k 10mg was added. On 3rd day, patient gradually deteriorated and started tachycardia(125/min), b.p. 100/60mmhg, tachypnea (35-40/min), ABG showed metabolic acidosis, pt 87.9, INR 7.62, APTT 71.6, serum creatinine 1.54, bun 25.7, ldh > 2500, total ck 313, serum uric acid 7.3, LFT – total bilirubin 7.5, direct bilirubin 6.3, albumin 3.8, AST 7522, alt 6735, alp 172, electrolytes were normal. For coagulation derangement, 8 units plasma were transfused. Gastroenterology opinion was taken. Despite fluid administration (crystalloids and colloids) patient developed hypovolaemic shock, which was treated with nor-adrenaline? Later, patient went into respiratory distress (Olenmark *et al.*, 1987) and was intubated and put on ventilator support. 2 units packed RBCS and plasma was transfused. On day4th inotrope supports were increased in view of persistent hypotension (noradrenaline, dopamine @ maximum doses) ABG showed severe metabolic acidosis (ph 7.193 pco2 20 po2 140 hco3 7.5). In differential diagnosis, sepsis was also considered and started with broad spectrum antibiotics (carbapenems) serum pct (procalcitonin) was sent ant it was 2.5, hb. 8.8, inr 8.34, aptt 91.8, pt 94.8, t.billirubin 7.5, direct bilirubin 3.29, ast 8383, alp 272, alt 7032, total protein 5.2, serum creatinine 3.64. 2d echonormal. For acute liver failure, gastroenterologist's added terlipressin and explained the requirement of liver transplantation to the patient relatives. On day 5th, patient could not be revived and succumbed to death and our final diagnosis was

Case Report

acute liver failure secondary to iron overload complicated with multiorgan failure and coagulopathy. Lft – total bilirubin 8.21, direct bilirubin 3.06, total protein 5.2, albumin 3.0, a:g 1.4, alt > 7000, ast > 7000, alp 242.

DISCUSSION

Iron poisoning is most commonly seen in the children but less commonly seen in adults. In literature, iron poisoning is mentioned starting from early 1950's. There are only few case reports of acute iron poisoning in adults. Most common cause is suicidal attempts and iron over dosage during pregnancy (Baranwal and Singhi, 2003). Iron over dosage leads to corrosion of gastric mucosa and leads to acute haemorrhagic gastritis, massive fluid shifts, bleeding and shock (Bentur *et al.*, 1991). However, the severity of the symptoms don't always correlate with the serum iron level (Black and Zenel, 2003). Intracellularly, iron exerts toxic effects on the mitochondria, uncoupling oxidative phosphorylation. It leads to anaerobic metabolism and subsequently metabolic acidosis. Iron also causes increased capillary permeability, coagulopathy and arteriolar dilatation leading to severe acidosis and shock (Chen *et al.*, 1993). Even our patient had severe metabolic acidosis and shock. Oxygen free radicals cause myocardial damage and can induce cardiac failure. It can cause pulmonary damage, renal damage, pancreatitis and peri-portal hepatic necrosis (Daram and Hayashi, 2005). Our patient went into respiratory distress (Dorota and Magdalena, 2006) and was intubated and put on ventilator support. Iron is an essential element for normal cell metabolism, but in excess is a known hepatotoxin. Following iron overdose, the mortality rate is as high as 50% or more once hepatotoxicity occurs. Hepatotoxicity is second only to cardiovascular collapse as the cause of death. This patient developed multiple organ failure secondary to acute liver failure. This could be explained by the various patterns of hepatic injury due to acute iron poisoning. In our case patient consumed ferrous sulphate, with a fatal effect on the cardiovascular, liver and renal system. Review of Milton Tenenbein literature and of experimental animal studies demonstrates that hepatotoxicity is a known sequela of acute iron poisoning, it occurs early in the clinical course and has a relatively high mortality. The lowest acute serum iron concentration

Associated with hepatotoxicity was 1700 µg/dl (Ioannides and Panisello, 2000). The oldest case report, published in 1987, reports fatal hepatotoxicity in a 30-year-old pregnant woman who took 5,000 mg of elemental iron. Her highest serum iron concentration was measured at 492 µg/dl, 15 hours after ingestion. She died two weeks later after progressing into a hepatic coma (Olenmark *et al.*, 1987). Our patient had a serum iron concentration of 2130 µg/dl which is highest noticed ever and TIBC of 2146.8 (Siff *et al.*, 1999).

Treatment modalities include decontamination gastric lavage or whole-bowel irrigation (Singhi *et al.*, 2003; Skoczynska *et al.*, 2007) after abdominal radiograph has confirmed the presence of radio-opaque tablets before or after the pylorus. As gastric lavage is not without risk, it is usually confined to those who present within one hour of ingestion and those children who have ingested more than 20 mg/kg. Activated charcoal has been used to adsorb ingested iron and is likely to be effective in ferrous sulphate. Desferrioxamine (Tenenbein, 2001) is the only approved iron chelator available and administered as a continuous intravenous infusion at 15 mg/kg/hr. This drug can evoke hypotension as a side effect. Disappearance of the 'vin rose' discoloration has often been taken as an endpoint to iron chelation, but normal urine color has also noted. N-acetyl cysteine was used when hepatotoxicity was evident in a pure ferrous sulphate overdose. In our case, kidney function got deteriorated, rising serum creatinine and falling urine output nephrology opinion was sought was taken and suggested conservative management with iv fluids and n – acetylcysteine 600 mg. although it is unknown whether this aided avoidance of liver transplantation. Liver transplant should be considered in all cases of hepatotoxicity.

Conclusion

The treating physicians should have the knowledge of the signs, symptoms and prognosis of iron toxicity. Early identification and resuscitation can reduce mortality. Iron toxicity leads to hepatotoxicity. Serum iron levels do not appear to correlate well with the risk of hepatotoxicity. Early chelation therapy reduces mortality in iron toxicity but the occurrence of acute liver failure is associated with a high mortality.

Case Report

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