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Case Report

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Long-Term Follow-Up of Overdose of Iron Tablets in Adult Successfully Managed Without Liver Transplant: A Case Report

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Keywords:

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Abbreviations:

LFT: Liver function test; SGOT: serum glutamic oxaloacetic transaminase; SGPT: Serum glutamic pyruvic transaminase; PAD: Post admission days; TB: Total Bilirubin; INR: International Normalization Ratio

1. Abstract

Iron toxicity is common in children because of accidental ingestion. However, there are very less reports of adults with suicidal attempts. We report a case of 19 years old who developed acute liver injury 48 hrs after the deliberate consumption of a large dose of iron tablets. Laboratory tests for other hepatotoxins were negative. Although the peak iron level was lower 197 µg/dl than the hepatotoxic level, there was significant acute liver injury reflected by rise in Total bilirubin (10 mg %), Serum glutamic pyruvic transaminase (3014 U/L), and International normalization ratio (4.5). Treatment with deferoxamine was given along with other symptomatic treatments. This case was managed conservatively without a liver transplant. We also report the longest follow-up period showing clinical, biochemical and radiological changes in this case report.

2. Introduction

The majority of acute iron poisoning is reported in children less than 5 years which is mostly accidental. However, iron poisoning with suicidal intent in an adult is rare and those reported show variable outcomes [1-5]. While some showed despite aggressive medical management patients who succumbed to toxic doses of iron [1-3], others reported remarkable recovery [4, 5].

We report here a case of an acute iron overdose in an adult with severe hepatotoxicity despite relatively low serum iron levels. We also report the change in the clinical pattern of this patient managed conservatively without liver transplantation.

3. Case Report

A 19-year-old lady allegedly consumed 30 tablets of ferrous sulfate with suicidal intent. The total dose amounted to 1959 mg of elemental iron. She had abdominal pain, vomiting, and black-colored stool and was given gastric lavage along with supportive care at a primary health care center. She was then referred to our hospital after 48 h of ingestion for further management and a plan for liver transplantation. At presentation, she was hemodynamically stable with a normal pulse rate and blood pressure. On physical examination, she was conscious and alert. She had mild icterus and mild epigastric tenderness with nausea. Liver Function Tests (LFT) showed marked elevation in liver enzymes (serum glutamic oxaloacetic transaminase (SGOT) 218 U/L and serum glutamic pyruvic transaminase (SGPT) 3014 U/L, total bilirubin (10 mg %), direct bilirubin (6.5 mg%). Serum iron level was 197 (normal range 50-170µg/dl) and the total iron-binding capacity (TIBC) was 188(normal range 250-425µg/dl), serum ferritin 640 (normal range 10-120 ng/mL), and transferrin 105 (15.2- 49.3 %). She was negative for viral markers as well as autoimmune markers. 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. Contrast-enhanced computed tomography (CECT) scan showed multiple hyperdense lesions in both livers which showed en-

hancement in arterial and portal phase without washout in venous phase and becomes isodense in the delayed phase suggestive of acute liver injury. (Figure 2A). Over the next 7 days, the patient's clinical condition, including the epigastric tenderness and nausea, improved. Her liver enzymes, prothrombin time, and bilirubin also improved as well as the liver enzymes (Figure 1A, B, C). She was discharged on Post-Admission Day (PAD) 8 in a stable condition. On follow up she came with fever and tachycardia with rising in TB (Figure 1 B). She was readmitted and managed with Intravenous antibiotics, desferrioxamine, and other supportive care. CECT was repeated which showed multiple hyperdense siderotic nodules scattered in both lobes of the liver with heterogeneous parenchymal enhancement with periportal edema, enlargement of caudate, and left lobe secondary to volume redistribution with mild ascites suggesting drug-induced liver injury secondary to iron deposit (Figure 2B). Her Serum iron level was 132, Total iron binding capacity (TIBC) was 187, serum ferritin 294, and transferrin 134. The patient improved clinically with a slow downtrend in TB. She was discharged because of financial conditions on PAD 19. She was readmitted again on PAD 30 with a complaint of itching rashes along with rising in SGOT (Fig 1 C). Her CECT revealed multiple hyperdense nodules and hyperdense lobulated lesions representing regenerative nodules. Large areas of hyperenhancement in the right posterior segment and a few in the left lobe along with periportal edema represent areas of drug-induced liver injury (Figure 2C). She was conservatively managed and her skin rashes improved along with her liver function. She was discharged on PAD 44. Thereafter patient remains on follow-up on an outpatient basis weekly. Her LFT improved along with her general clinical condition improved significantly. CECT scan repeated on PAD 45 and 60 which revealed feature of multiple mild siderotic and regenerative nodules (Figure 2 D, E). The patient is followed up for 12 months now and she is free of any delayed complications such as gastrointestinal scarring.

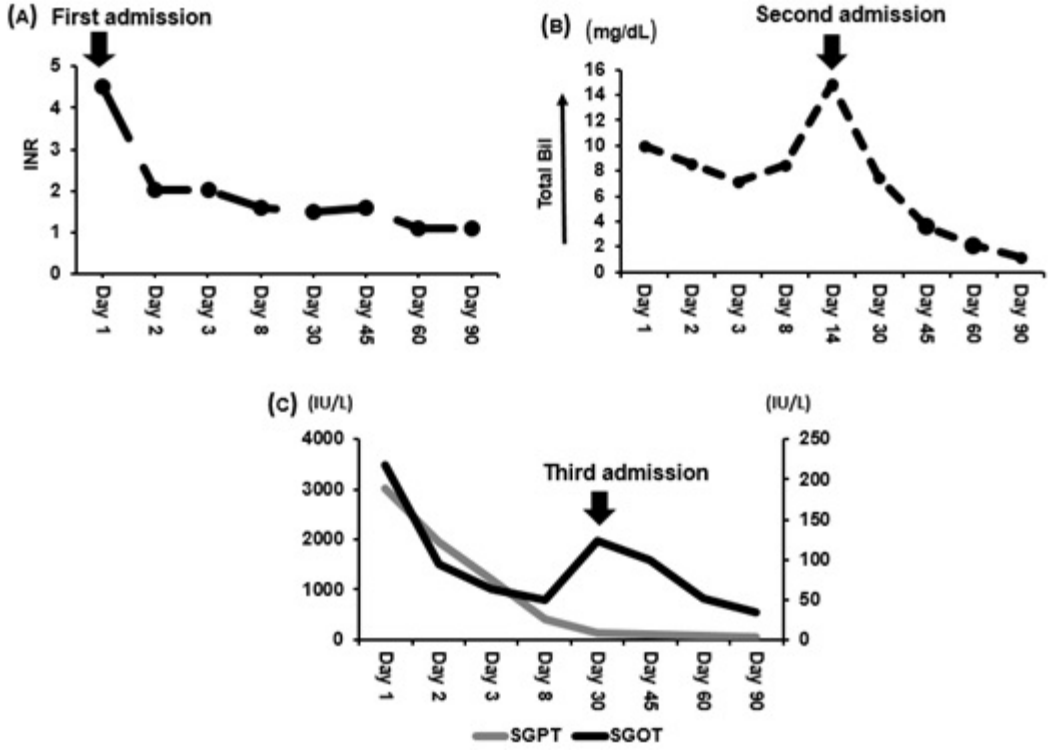

Figure 1: Change in INR (A), Total Bilirubin (B), SGPT/SGOT (C) profiles on post admission days (PAD).

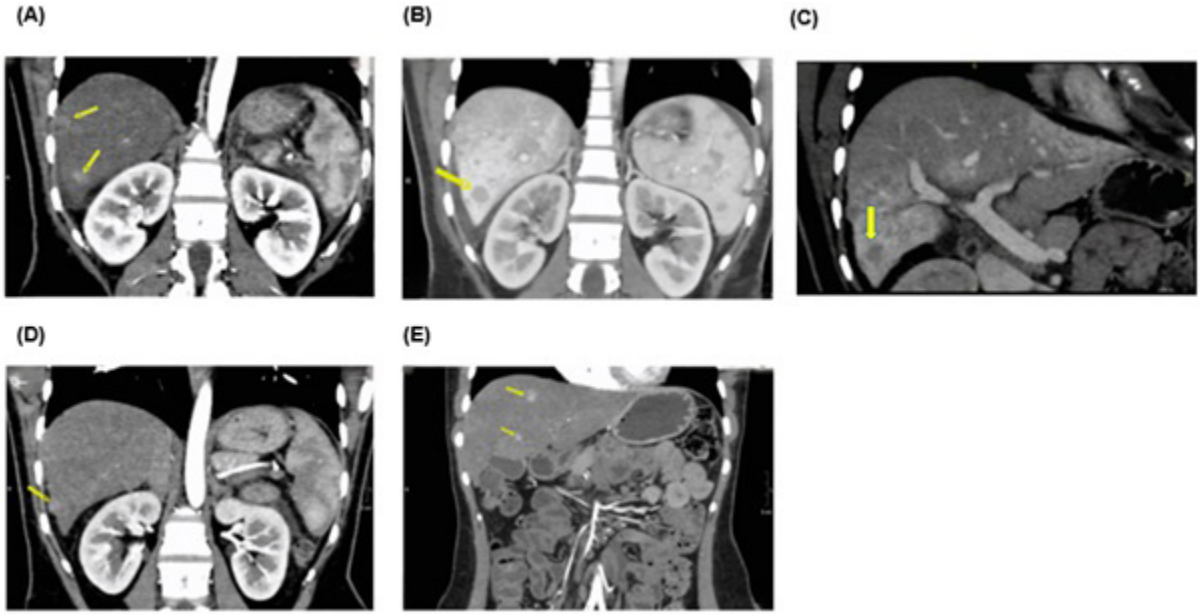

Figure 2: CT Triphasic done on PAD 0 (A) Multiple hyperdense nodules on both lobes, PAD 14 (B) Hyperdense siderotic nodules scattered in both lobes, PAD 30 (C) Mulitple hyperdense nodules and lesions representing regenerative nodules , PAD 45 (D) Liver shows volume redistribution with surface nodularity with dysplastic nodule in seg VIII , PAD 60 (E) Multiple siderotic regenerative nodules in segment V, VI and VIII.

4. Discussion

Hepatotoxicity is known consequences of iron poisoning and is associated with 50% of mortality which is found to be dose dependent. However, this present case of Acute Iron Hepatotoxicity is particularly unique because despite the Serum Iron (197 µg/dl) being significantly lower than the associated concentration considered to cause hepatoxicity (>1700 µg/dl) it caused severe Hepatoxicity. Our report is consistent with previous report that had 340 µg/dl serum iron which even though low caused hepatoxicity similar to our case [4]. The reason for such hepatotoxicity at such low level of iron is unclear till date. Liver biopsy might have been helpful but here it was clinically not indicated.

We also report longest clinical course in this case showing both biochemical and radiological trend. Our patient required three times admission for different reasons and all biochemical parameters settled down completely after 3 months of iron tablets overdose. Previous reports have also reported biochemical trend but for shorter duration. [4, 5] As reported previously we also managed this case with chelation therapy with parental as well as oral desferrioxamine along with other liver protective medicine. Liver transplantation is the rescue therapy for the cases of iron poisoning leading to acute liver failure. According the classification of effects of iron poisoning four stages have been proposed: Stage-1 (Stage of gastrointestinal toxicity), Stage-II (Stage of apparent stabilization or quiescent phase), Stage- III (Stage of mitochondrial toxicity), Stage-IV (Stage of gastric scarring) [6]. Our patient presented to us in stage III where liver transplant is indicated. Previous reports showed that despite of liver transplantation done, iron overload associated with acute liver failure carried a poor prognosis [7]. However we managed the case without liver transplantation and followed her up for longer period and did a closed monitoring. Management of acute iron hepatotoxicity even with lowest serum level should include gastric lavage, chelation therapy as deferoxamine and monitoring of biochemical and radiological changes with a closed follow up.

5. Conclusions

Acute Iron Hepatoxicity is a rare in adults and very few such cases have been reported so far. This case report is the first to mention the radiological and the biochemical changes for the longest period. Our case by far has lowest serum level (197 µg/dl) reported in literature. Therefore, it may provide more insight about management of iron overload as the literature is scant for such a case.

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