GAVIN PUBLISHERS

Annals of Case Reports

Hussain F, et al. Ann Case Rep: ACRT-155.

Case Report

DOI: 10.29011/2574-7754/100055

Cerebral Edema Treated Successfully After Liver Transplant in a Patient with Acute Iron Toxicity

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Citation: Hussai F, Rashid Khan T, Bhattarai P, Raman L (2018) Cerebral Edema Treated Successfully After Liver Transplant in a Patient with Acute Iron Toxicity. Ann Case Rep: ACRT-155. DOI: 10.29011/2574-7754/100055

Received Date: 15 December, 2017; **Accepted Date:** 29 December, 2017; **Published Date:** 09 January, 2018

Summary

We report a case of a 13-year-old female transferred to us in hepatic coma due to intentional ingestion of reported 27mg/kg of elemental iron that yielded in an iron level of 246 mcg/dl, 5 days after suspected ingestion. The patient presented in shock, liver failure and subsequently developed cerebral edema with impending herniation. During her course, she was treated with Continuous Renal Replacement Therapy (CRRT), Plasma Exchange (PLEX) and eventually received liver transplant with complete resolution of her neurological symptoms.

Keywords: Cerebral Edema; Iron; Liver Transplant; Toxicity; Resolution

Background

Most cases of iron poisoning, whether accidental or intentional, present acutely after the ingestion. Many patients will be asymptomatic with ingestion of elemental iron less than 10mg/kg. Most clinical guidelines recommend transferring patients with ingestion of 40mg/kg of elemental iron to hospital facilities [1] with severe toxicity levels seen at 60mg/kg [2]. Patients who present late after ingestion are less likely to ultimately survive [3].

Case Report

A 13-year-old previously healthy female presented with altered mental status, metabolic acidosis and fulminant hepatic failure. The patient was in her usual state of health 5 days prior to presentation when she had an argument with her parents. The following morning after her argument, she reported having emesis, fatigue and anorexia to her mother. She however felt better for a few days until the morning of presentation when she was difficult to arouse.

Upon arrival to an outside ER patient had a Glasgow Coma Scale of 7 (E2, V1, M4). She had delayed capillary refill, no uterus and an unremarkable abdominal exam. Her vital signs were notable for tachycardia (135 bpm), hypotension (75/45 mmHg) and tachypnea (34/min).

Initial blood gas showed severe metabolic acidosis (pH <6.97, CO2 22, bicarb 4.8) with elevated lactate (>11 mol/L). Complete metabolic panel revealed acute liver failure (AST>7500 units/L, ALT 10000 units/L, total bilirubin 4.3 mg/dL) with coagulopathy (INR 19.5, fibrinogen 85 mg/dL, PT 138 sec, PTT 56 sec, D-dimer 3.99 mcg/mL), hyperammonemia (275 mmol/L) and hypoglycemia (7 mg/dL). Complete blood count, hepatitis panel and urine toxicology were unremarkable. Her acetaminophen was <10 mcg/mL. Salicylate and ethyl alcohol levels were negligible. X-ray of her abdomen and head CT both were normal.

She was resuscitated and transferred to our Pediatric Intensive Care Unit (PICU) for transplant evaluation. Immediately upon arrival, due to unclear etiology of her liver failure, she was empirically started on N-acetyl cysteine. Admission labs revealed elevated iron (246 mcg/dL) and ferritin levels (8117 ng/mL). Her repeat acetaminophen, salicylate and ethyl alcohol level were negligible. Repeat x-ray of her abdomen and pelvis however showed radiopaque substances in her rectum concerning for ingestion of pills (Figure1) here. Upon questioning, parents revealed there was a bottle of iron with 90 capsules of which only 14 remained. Each capsule contained 18 mgs of elemental iron. Thus, she could have ingested up to 1368 mg (27 mg/kg) of iron. There was also history of constipation for a few days, which could have led to the non-passage of the tablets. Parents also reported that she was normal prior to the day of presentation and the only medications that were missing were the iron tablets.

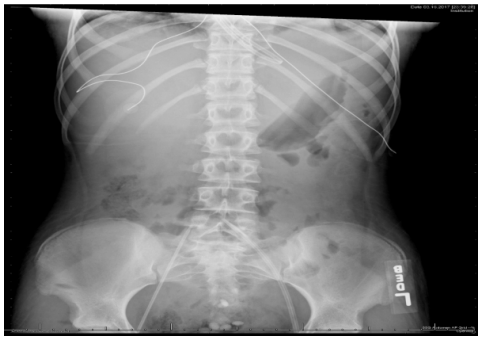

Figure 1: Abdomen and pelvis radiograph 5 days after ingestion showing the radio opaque substances.

Thus, with the diagnosis of iron toxicity, she received 18 gms of deferoxamine over 24 hours, which reduced her ferritin levels. She was not on any sedative medications and was difficult to arouse and thus received 4 rounds of PLEX for her concerning neurological exam. Her renal failure was treated with CRRT. On day 3, her neurologic exam worsened with unequal and nonreactive pupils. Her head CT at that time showed extensive cerebral edema with impending herniation (Figure 2).

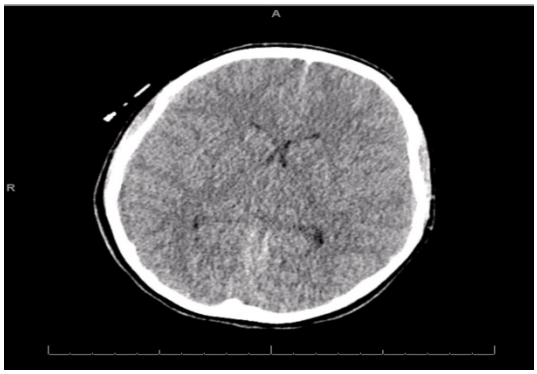

Figure 2: Head CT showing cerebral edema on day 3 of hospitalization.

She underwent a liver transplant on day 6 of admission with improvement in her liver function and neurological status. Biopsy of the native liver showed moderate perivenular parenchymal collapse, moderate hepatocanalicular cholestasis and moderate steatosis. She was successfully extubated on day 18 and CRRT was stopped on day 19. She was ultimately discharged home on Hemo Dialysis.

Discussion

Iron poisoning is generally characterized by four stages. Within the first phase (0-6 hours), iron causes direct mucosal injury, manifesting as vomiting, diarrhea and GI bleeding. During the latent phase, from 6-24 hours, iron causes ongoing cellular toxicity, but local toxicity can subside, and the patient can appear improved. The third stage, lasting 12 hours to 5 days after ingestion, often starts abruptly with shock, metabolic acidosis and can progress to liver failure. Finally, 2-6 weeks later, patients may end up developing strictures, leading to gastric outlet or small bowel obstruction [2].

Iron circulates bound to transferrin and is stored as ferritin. Small amounts of iron are lost via the urine, feces and skin. With increased iron levels, free radicals damage the GI mucosa, allowing iron to passively enter the body down its concentration gradient. The liver is particularly vulnerable in an overdose because of its unlimited ability to uptake iron [2]. Serum iron concentrations are only useful in predicting severity of intoxication if they are obtained within 4-6 hours after the ingestion because it is rapidly cleared from the serum [4].

As iron causes significant local toxicity, whole bowel irrigation is recommended if the patient is alert and can tolerate this. Our patient was intubated and sedated when she arrived, and the tablets had already passed to the rectum so we were hopeful that irrigation with lactulose (given per rectum and via NGT) would remove these pill fragments and concurrently treat her hyperammonemia. The mainstay of severe iron toxicity treatment includes deferoxamine, which binds to ferric iron and is excreted by the kidneys. The recommended starting dose is 15 mg/kg/hr with a maximum of dose of 80 mg/kg/day in children or 6-8g/24 hours in adults [2]. Deferoxamine can cause shock, hypotension, pulmonary edema and ARDS [2-5]. For our patient, this did reduce iron and ferritin levels significantly; however, she developed pulmonary edema during her treatment.

As the patient presented in shock and developed renal failure, we initiated CRRT, which has been successfully used in other cases [6]. Charcoal Hemoperfusion (CH) has been commonly used in the past to bind protein-bound intoxicants but with the diminished availability of CH cartridges as well as decreasing expertise among health professionals with using these membranes, CH treatment has become less common and was not used in our case [7].

Successful use of plasma exchange has been reported in other cases of severe iron intoxication, combined with deferoxamine and exchange transfusion as well [8]. In our case PLEX was mainly used for her concerning neurological status as her ferritin levels was not helpful in guiding therapy.

There is one other reported case of liver transplant in treating iron overdose [9]. To our knowledge this is the first reported case of such a delayed presentation, stage three of iron ingestion with significant complications including cerebral edema with impending herniation with resolution of symptoms after liver transplant.

Declaration of Interest

The authors have no conflicts of interest or financial involvement to report.

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