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Case Report

Non-suicidal intentional ingestion of iron tablets and severe intoxication: the result of adolescent boy's impulsive risky behaviour in the school

Krutika Rahul Tandon*, Rahul Tandon, Amola Khandwala, Tanvi Mehta

Department of Pediatrics, Pramukhswami Medical College, Bhaikaka University, Karamsad, Gujarat, India

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***Correspondence:**
Dr. Krutika Rahul Tandon,
E-mail: tandonkrutika72@gmail.com

ABSTRACT

Acute iron poisoning is a potentially fatal intoxication in children. As the Iron preparations are commonly administered to pregnant women, lactating mothers, toddlers, it is easily available at home. So younger children are prone to consume it accidently. Although iron is a therapeutic drug in recommended dosages, excessive iron in the free state can produce toxicity by affecting multiple cellular processes by catalysing redox reactions with lipid peroxidation and free radical formation. The severity of intoxication depends on the amount of elemental iron ingested. Serious toxicity is usually associated with a dose of >40 mg/kg of elemental iron. Levels more than 100 mg/kg are almost always fatal. We report a case where a 12-year male child intentionally taken 60 tablets of iron (ferrous fumarate) at his school as a part of competition or bet to other schoolmate and presented with acute iron poisoning with hepatic encephalopathy to us. Important initial laboratory parameters were AST-4,879 U/L, Prothrombin time-60 sec and Iron level-213 microgram/dl. With timely specific management i.e., deferoxamine infusion along with all required intensive care supportive management in PICU the patient was discharged successfully. We chose to report this case to highlight the risky behaviour of adolescence who usually grows physically and emotionally earlier but their prefrontal lobes are yet immature to take proper and correct decision. Thus, impulse activity may prove fatal for them.

Keywords: Lipid peroxidation, Deferoxamine, Iron intoxication, Hepatotoxicity

INTRODUCTION

Acute poisoning including iron poisoning generally occurs by accident in younger children whereas it is usually intentional in adolescents and adults. The incidence of serious iron poisoning has drastically decreased over the last 30 years.[1] When there is an overdose, the serum iron level exceeds the body's binding capacity causing free iron to circulate which in turn produces an increase in reactive oxygen species (ROS) or so-called oxygen, free radicals. This exerts toxic effect on mitochondria by shunting electrons away from the electron transport chain and thereby uncoupling the oxidative phosphorylation leading to anaerobic metabolism and so metabolic acidosis. Iron affects almost every organ in the body being a systemic intra-cellular poison.[2] It can cause acute periportal hepatic necrosis and occasionally pulmonary damage, renal damage, and pancreatic necrosis.[2]

In the market there are multiple formulations of iron are available to correct anemia in the form of injections, tablets, and syrups. Even it is free government supply at various community and national programs. So, it is not difficult to have it or get it. Iron poisoning is mostly seen in children. Clinical outcome is variable depending on the quantity of iron ingested, other drugs ingested, and the time to receive treatment. Hepatotoxicity associated with acute iron exposure is unusual and largely confined to the pediatric age group, with only sporadic reports of adult

cases. Hepatotoxicity has been associated only with serum iron levels greater than 1,700 microgram/dL.[3] Unlike younger children or adults, the Adolescents age group has entirely different emotional and psychological status. They are more responsive and easily influence by their peers, and thus more likely to change their decisions and alter their behavior in response to social pressure. This can result in a greater likelihood of engagement in risky behaviors, alone and/or in groups, to elevate social status.[4] In our case, the bet was made with other schoolmates that who can consume higher number of iron tablets?

CASE REPORT

A 12-year-old boy was brought to our emergency department (ED) with the altered state of consciousness by the school teacher. The school teacher gave history of consumption of iron tablets (~60) by the patient 3 days prior as a part of bet which he came to know on that day only from another student. The patient had vomiting and diarrhea for which treatment from a local doctor was taken on OPD basis and then remain asymptomatic for 2 days before altered sensorium. In the emergency department, the patient was unconscious with yellowish discoloration of the eyes. On admission the child had normal body temperature, 102/min heart rate (normal rhythm), well palpable pulses, respiratory rate 30/min, oxygen saturation 100% on air, blood pressure 112/88 mmHg and icterus. Glass glow coma scale of less than 8 warranted elective intubation. No other significant positive findings. The initial laboratory reports were-Hb-12.5 gm/dl, TLC-11800 cumm, platelet-1,08000 cumm, arterial blood gas-pH-7.48, $PaCO_2$-26, PaO_2-296, HCO_3-19. Electrolytes and RFT was normal, SGPT/SGOT-4879/1384u/l, PT/INR-60.2/5.27, APTT-49.9. His serum iron was 213 microgram/dl and ferritin-1011.9 microgram/l and TIBC-244 microgram/dl.

Management and outcome

The patient was managed in ED and then in PICU with ventilatory support for 5 days. As per history, physical examination and laboratory reports iron poisoning with hepatoxicity. Due to unavailability of deferoxamine at our hospital we were able to start its infusion only after 12 hours of admission meanwhile he received infusion of N-acetylcysteine and other supportive management. As INR was high with bleeding from RT, ET and rectum, 6 units of fresh frozen plasma were also given. Next day lab report was repeated showed serum iron 144 microgram/dl, PT/INR-20/1.75, SGPT/SGOT-1348/250 U/L. On the 5th day of admission, the patient was extubated, put on a low flow oxygen device, and RT feeding started with a high carbohydrate diet. Ultrasound abdomen on 6th day done showed heterogeneous texture of liver suggests acute insult. On 10th day of admission patient vital and systemic examination was normal with lab report showed serum iron 135 microgram/dl, S. ferritin 432 microgram/l, SGPT and SGOT-473,103 U/L, and PT/INR-22.7/1.96. So shifted to the general ward. On the 11th day patient was again having excessive sleepiness complaint with vitals normal and maintaining normoglycemia, so shifted to PICU for close observation but he was not deteriorated further. Patient was discharged on day 15 with counselling session to clinical psychologist.

DISCUSSION

Iron is toxic in the free state and therefore, in the body, it binds to proteins such as ferritin and transferrin to avoid tissue damage. In Overdose, the binding capacity of these proteins is overwhelmed, iron becomes free to exert its action on various organs and organ systems with hazardous consequences. A dose >40 mg/kg of elemental iron can be potentially toxic. The case presented here had ingested an almost lethal dose of >49.5 mg/kg and presented with evidence of hepatocellular injury in form of encephalopathy. Iron poisoning has four clinical stages.[5] Features of acute gastrointestinal irritation dominate the period up to 6 hours after ingestion, and most patients do not develop other features or progress beyond this stage. Hypotension may arise at this stage either due to hypovolemia due to continuous vomiting or due to blood loss due to gastric erosion. In the second stage, which starts about 6-12 hours after ingestion, the clinical features start showing remission. Few patients may progress to stage 3, which is 12-48 hours after ingestion, wherein there is a recurrence of symptoms in the form of shock and metabolic acidosis. Acute kidney injury and hepatocellular failure may develop in a few patients during this stage. The last (fourth) phase, which usually develops in young children 2-6 weeks after ingestion, is characterized by the recurrence of vomiting due to gastric or duodenal stenosis caused by the healing of gastric erosions. Our index case was in stage 3 in form of hepatic encephalopathy stage IV. Hepatotoxicity is believed to be a dose-related phenomenon.[9] Serum iron concentrations of 1000 microgram/dL are associated with clinically important hepatotoxicity. But serum concentrations alone cannot be relied upon to determine prognosis because peak concentration varies with the amount and formulation of iron ingested, and the time at which a serum iron level can be drawn is dependent on the timing of presentation. Liver failure was reported in an adult patient with a peak serum iron level of 340 µg/dL.[6] This was seen in our case. The serum iron levels can be unreliable in the late presentation of the patient since the iron is re-distributed intra-cellularly. Intravenous deferoxamine is the definite antidote for iron intoxication.[7,8] The recommended maximal dosage of deferoxamine is 80 mg/kg over 24 hours. DFO binds with ferric iron (Fe3+) in the blood to form water-soluble ferrioxamine, which is renally excreted. Other supportive care is the mainstay of iron poisoning. Few cases of severe iron intoxication have been managed with exchange transfusion in the past, and this remains a therapeutic option in children with severe toxicity. We had given to our patient 50 mg/kg injection deferoxamine

over 8 hours initially by 15/mg/hr infusion rate. Compared to children and adults, adolescents tend to be good at recognizing social and emotional information, seeking out new experiences, meeting new people, and confronting various challenges.[10] However, as a part of normative development, adolescents are also characterized by sensitivity to reward, emotionality, risk-taking, and impulsivity, with a tendency to act in the spur of the moment and to make rash choices regardless of the consequences as we have seen in this.

Although peak serum iron level (213 micrograms/dL) was significantly lower than that reported to cause hepatotoxicity (1,700 micrograms/dL), significant elevations in aminotransferases (4,879 U/L), total bilirubin (4 mg/ dL), and prothrombin time (60 seconds) was there in our case.

CONCLUSION

Early recognition is necessary to ensure appropriate therapy and the prevention of fatalities. Intentional non-suicidal poisoning may occur in adolescent age groups and their mental fragility may prove fatal for them. This case reminds us that emotional and psychological fragility of adolescence can take hasty and impulsive decisions which could prove fatal for them. Hepatotoxicity can prove fatal also but timely recognition, appropriate specific and supportive therapy can save life also.

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