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A CASE OF ACUTE IRON POISONING IN AN ADULT PRESENTING AS FULMINANT HEPATIC FAILURE

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Abstract :

Fulminant hepatic failure (or acute liver failure) is defined as the rapid development of hepatocellular dysfunction, specifically coagulopathy and mental status changes (encephalopathy) in a patient without known prior liver disease 1 Acute liver failure is a medical emergency associated with a high mortality rate because of the development of cerebral edema, infectious complications, and multiorgan failure. Despite advances in medical management, mortality rates in patients with acute liver failure remain high in the absence of emergency liver transplantation. The predominant cause of acute liver failure differs markedly throughout the world. In the United States and other western countries, medications, including acetaminophen and idiosyncratic drug toxicity are the most commonly identified causes of acute liver failure. In France, Japan, and India, severe acute HBV infection is a leading cause of acute liver failure. 2 This clinical report summarizes the management of acute fulminant hepatic failure due to acute iron toxicity secondary to suicidal ingestion of ferrous sulphate tablets.

Keyword : Acute iron poisoning, acute iron poisoning in an adult, Deferoxamine, acute fulminant failure

INTRODUCTION:

Acute liver failure is a clinical syndrome that represents the final common pathway of severe liver injury resulting from numerous infectious, immunologic, metabolic, vascular, and infiltrative disorders. [2]

In general, patients with hyper acute liver failure are more likely to develop cerebral edema and to recover without liver transplantation. By contrast, patients with subacute or late-onset hepatic failure are more likely to present with evidence of portal hypertension such as ascites and to have a low rate of survival without transplantation.

The mechanism of liver injury in acute liver failure is most often severe hepatocellular necrosis, as occurs with acetaminophen toxicity or viral hepatitis. [2] Acute liver failure can also result from severe cellular or mitochondrial dysfunction, as occurs with some forms of drug toxicity (e.g., antiretroviral agents), Wilson disease, and acute fatty liver of pregnancy. [3] In the following case report, we describe the case of acute fulminant hepatic failure in a 28 year old female who ingested 20 iron tablets (amount about 50mg/kg of ferrous salt).

CASE REPORT:

A 28 year old female was admitted in our department on 30-06 -12 with history of treatment ina private Hospital from 27-6-2012 for alleged history of consumption of unknown tablets, about 20 in number. Gastric lavage aspirate done at the private was coffee brown in color. Patient had been referred to Government Royapettah Hospital on 30-06-2012 as she became drowsy and her liver enzymes were elevated (SGOT- 3750 U/L, SGPT- 2990 U/L). History taken after admission in our department revealed that patient had consumed about twenty ferrous sulphate tablets, all at once (which were given to her during Antenatal checkup containing 2000mg amount of elemental iron). Each ferro tablet contained 335 mg of dried ferrous sulphate equivalent to 100 mg of elemental iron.

On examination, patient was found to be icteric, tachypnoiec, afebrile with blood pressure of 130/80 mmHg in right upper limb in supine position and pulse rate 98 per minute and her oxygen saturation was 96%. On further examination, Patient was found to be drowsy, arousable to painful stimuli. Doll's eye movement was present. Pupils were normal in size, equally reacting to light. Bilateral plantar reflex was extensor. GCS SCORE was E 2 / V 3 / M 4 = 9/15. On examination of cardiovascular system, first and second heart sounds were heard and are of normal intensity. No murmurs were heard. On examination of respiratory system, lungs were clear. Abdomen was soft with no organomegaly. Bowel sounds were present. Patient was provisionally diagnosed as a case of "IRON TABLET POISONING / FULMINANT HEPATITIS" and treatment was started. Patient's blood
investigations were done and the results came as serum iron level 250 g/dl, prothrombin time of 3.73 INR, serum bilirubin 8.4 mg/dl, SGOT 510 U/L, SGPT 2360 U/L.

Patient was treated with intravenous deferoxamine and supportive management in the form of injection mannitol (0.5 to 1 g/kg), intravenous dextrose infusion, injection vitamin K, parenteral antibiotic coverage, L-Ornithine –L-Arginine supplement and bowel wash. During the course of treatment, Patient's condition improved and consciousness returned. Full blood count, renal function test, chest X-ray was normal. ECG was within normal limits. CT- Brain plain was normal. Serum iron level declined to105 g/dl on 02-07-2012.

Serum aminotransferase levels also declined to 110 U/L (SGOT) and 130 U/L (SGPT). Medical Gastroenterologist opinion was obtained as "***Fulminant Hepatic Failure due to Iron poisoning***". *HbsAg and AntiHCV tests were Negative.* PT/INR and liver function tests gradually became normal.

As patient's general condition improved, Patient was discharged on 21-07-2012. Patient diagnosed as a case of "ACUTE IRON TOXICITY / ACUTE FULMINANT HEPATIC FAILURE".

DISCUSSION:

Iron toxicity is one of the leading causes of death particularly in Children younger than 6 year. [4] Chronic Iron Poisoning is common especially in patients requiring multiple transfusions of red blood cells in sickle cell disease, Thalassemia, myelodysplasia syndromes. Accidental or suicidal ingestion of iron in adults is not that common.

Pathophysiology of Iron toxicity is of two types, Corrosive toxicity and Cellular toxicity**.** [5] [6] The severity of acute ingestion changes in relation to factors such as quantity, toxicity and pharmacokinetics, the time elapsed from exposure, age and clinical condition of the patient. Clinical manifestations may vary, even if the dose ingested is the same, from mild gastrointestinal disorders toto hypovolemic shock, metabolic acidosis, liver failure or death. Iron is extremely corrosive to gastrointestinal tract. [7]Patients may become hypovolemic because of significant fluid and blood loss.

Also Absorption of excessive quantities of ingested iron impairs oxidative phosphorylation and mitochondrial dysfunction. A direct negative inotropic effect of iron on the myocardium also is demonstrated in animal models. Early coagulopathy unrelated to hepatotoxicity can also occur due to free iron inhibition of thrombin formation. Liver is one of the organs most affected .Other organs such as the heart, kidneys, lungs, and the hematologic systems also may be impaired. End result is significant metabolic acidosis in acute iron poisoning, while chronic iron overload may cause death due to *myocardial* siderosis. [7] Ingestion of elemental iron exceeding ***20 mg/kg*** *c*auses gastrointestinal toxicity; elemental iron exceeding ***40 mg/kg*** causes moderate intoxication; elemental iron exceeding ***60 mg/kg*** causes severe toxicity and may be lethal. [7] In our case, the patient has ingested 50 mg/Kg of elemental iron.

Gastrointestinal symptoms start as soon as 6 hours of ingestion. It takes up to 24 hours to develop metabolic acidosis. Patient may also develop stupor and coma as early as 6 to 8 hours and can last up to 2 days. Abnormalities include hypotension, coagulopathy and metabolic acidosis. Patients are at high risk of infections and may develop multi organ dysfunction and shock during this stage. Patient may progress hepatic failure. [3] [7] [8] Most patients die during this stage. Patient who survives may develop scarring of gastrointestinal tract as a delayed complication weeks after severe poisoning. [9]

Serum iron levels (at least 4 hours post-ingestion) 350-500 g/dl may lead to mild to moderate toxicity, levels higher than 500 g/dl may lead to hepatotoxicity and levels higher than 1000 g/dl may lead to severe toxicity. [10] [11] although elevated serum iron concentrations may be an additional indicator of potentially serious toxicity, lower concentrations cannot be used to exclude the possibility of serious toxicity. A single serum iron concentration may not represent a peak concentration or may be falsely lowered by the presence of deferoxamine, unless an atomic absorption technique is used for measurement.[12] [13] In our case, the patient's serum iron level was 250 g/dl and presented with acute Fulminant liver failure. Please note that patient's serum iron level was measured four days after the ingestion of iron

Imaging studies like x-ray KUB film can determine if radiopacities are present, as iron tablets are radiopaque for a few hours post ingestion.[14] Absence of radiopacities does not rule out a significant or lethal ingestion.

Other investigations needed are liver function testsm coagulation studies, glucose levels because hepatic dysfunction is common In all cases, treatment should be given as soon as possible and it is advisable to contact the nearest Poison control center. Management of acute iron poisoning is by stabilization of vital signs, limiting absorption of iron by decontamination (Gastric lavage, activated charcoal), enhancing elimination of iron (chelation by Deferoxamine).

Deferoxamine has been available since the 1960s as a specific chelator for patients with acute iron overdose or chronic iron overload. It has high affinity and specificity for iron. [7]

Deferoxamine chelates free iron and iron transported between transferrin and ferritin, but not the iron present in transferrin, hemoglobin, hemosiderin and ferritin. 100 mg of Deferoxamine mesylate binds 8.5mg of iron. It is to be noted that Deferoxamine is not a substitute for standard measures generally used in iron toxicity (e.g., induced emesis, gastric lavage).

Intravenous administration of deferoxamine should be considered in acute iron poisoned patients with any of the following findings: metabolic acidosis, repetitive vomiting, toxic appearance, lethargy, hypotension, or signs of shock. Deferoxamine administration also should be considered for any patient with an iron concentration >500 µg/dL. Deferoxamine should be initiated as an intravenous infusion, starting slowly and gradually increasing to a dose of 15 mg/kg/h. Patients who have concentrations <500 µg/dL or who do not appear toxic should be treated supportively without administration of parenteral deferoxamine [7]

Contraindications for Deferoxamine are hypersensitivity and severe renal disease or anuria. Rapid intravenous infusion can cause flushing, urticaria, hypotension. Rare fatal cases of mucormycosis have been reported. [15] Patient has to be monitored for changes in renal function. End points of treatment are clinical improvement, resolution of anion gap and acidosis, return of urine color to baseline, serum iron level 100 mcg/dl.[16]

CONCLUSION:

This case report highlights the potential hazards of the most commonly prescribed, over the counter drugs, even though of rare occurrence. A thorough knowledge regarding toxicity of these commonly prescribed drugs is imperative in the management of life threatening complications caused by accidental or suicidal ingestion of these drugs. This case report also highlights the potential reversibility of life threatening complications with timely and effective supportive management.

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