Case Report

Acute liver failure due to organophosphorous

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ABSTRACT

A case of acute liver failure who lost five of his family members due to consumption of some food stuff made at home. Autopsy of viscera of the deceased revealed organophosphorous. Only one member who presented as ALF survived. A 33yr old male presented with reeling of head and vomiting after 8-10 hrs of consuming some home made food. He was treated outside for 1 day and shifted to Apollo Hospital Bhubaneswar due to deterioration of condition. On examination he was conscious but disoriented and icteric, hemodynamics and urine output was normal. Investigations revealed serum bilirubin 6.3/2.4mg/dl, ALT - 10,225u/l, AST - 12,865u/l, PT - 13.1/66.1sec, INR - 8.89, TLC - 5,000/cm³, Creatinine:1.1mg, Serum Ammonia 138mmol/l. Other infective markers for viral hepatitis, dengue, malaria and leptospira were negative. Patient went to encephalopathy within an hour. In view of Jaundice, Coagulopathy and encephalopathy diagnosis of ALF was made and Patient was managed with ICU care, head end elevation, mannitol, Inj N-acetyl cystine, Inj vitamine K, IV fluids, IV antibiotics and other supportive treatment. Possibility of need of liver transplantation was discussed but family expressed inability to afford. The patient was not bleeding hence FFP not given. However with conservative management he started improving after 72 hrs and fully recovered within 10days.

Investigations: LFT, Infective markers for viral hepatitis, dengue, malaria and leptospira. Diagnosis: Acute Liver Failure

Key words: Acute liver failure, Organophosphorous, Encephalopathy.

INTRODUCTION

Acute liver failure is a rare but life-threatening critical illness that occurs most often in patients who donot have preexisting liver disease. Sudden loss of synthetic and detoxification function of liver results in jaundice, encephalopathy and coagulopathy which leads to multiorgan failure.¹²

Acute liver failure is the culmination of severe liver cell injury from a variety of causes including viral hepatitis, toxins, metabolic disorders and vascular insult.³ The etiology of ALF varies demographically. In India, Acute viral hepatitis is the most common cause of ALF.⁴,⁵ Drugs (Acetaminophen) are the most common cause of ALF in the west.⁶ While Amanita phalloides mushroom toxin is associated with dose-related toxicity. It is much more common in Europe and California than in the remainder of the United States.⁷ Drug-induced liver injury may be dose-dependent and predictable as in Acetaminophen toxicity.ALF due to acetaminophen can occur if a large dose (150 mg/kg) is consumed as in deliberate self-poisioning. It can also occur with substantial drug ingestion over hours to days as occur in unintentional poisoning.⁸ Other form of drug-induced liver injury is idiosyncratic drug reaction .It is often unpredictable and independent of dose.⁹ Wilson's disease accounts for 6-12% of cases of ALF. It should be suspected when patient has very high serum bilirubin with hemolysis only modest elevation of transaminase and low alkaline phosphatase at presentation.¹⁰

Organophosphorous compounds are organic compounds containing phosphorus.¹¹ Organophosphate poisoning results from exposure to organophosphates (OPs), which cause the inhibition of acetylcholinesterase (AChE), leading to the accumulation of acetylcholine (ACh) in the body. Organophosphate poisoning most commonly results from exposure to insecticides or nerve agents. OPs are one of the most common cause of poisoning world wide.¹²,¹³ OP pesticide exposure occurs through inhalation, ingestion and dermal contact.
Although viral hepatitis is the most common cause of ALF in India, a Case was found out Organophosphorous as etiological agent of isolated acute liver failure.

CASE SUMMARY

A 33yr old male presented with reeling of head and vomiting after consuming some home made food. He and five of his other family members took a home made food made up of Rice, Bengal gram, oil where Rice is fermented overnight. After 8-10 hrs of consumption they got sick, were admitted to different hospitals with reeling of head, vomiting, abdominal discomfort and respiratory distress. But one after the another succumbed to death in different hospitals. The last person got panicked and shifted to Apollo Hospital, BBSR. On examination he was conscious, disoriented and icteric, hemodynamics and urine output was normal. Investigations revealed serum bilirubin-6.3/2.4 mg/dl, ALT-10, 225u/LAST-12, 865u/l, PT-13.1/66.1sec, INR-8.89, TLC-5,000/cmm, Creatinine-1.1mg, Serum Ammonia-138 mmol/l. Other infective marker for viral hepatitis, dengue, malaria and leptospiira were negative.

Patient went to encephalopathy within an hour and was managed in line of acute liver failure. Family members expressed inability for liver transplant. Patient improved within 72hrs and fully recovered within 10days.

DISCUSSION

Acute liver failure is a broad term which is used interchangeably with fulminant hepatic failure. Fulminant hepatic failure is generally used to describe the development of encephalopathy within 8 weeks of the onset of symptoms (Jaundice) in a patients with a previously healthy liver. Acute liver failure can be subclassified into "hyperacute" as onset within 7 days, "acute" as onset between 7 and 28days, and "sub-acute" as onset between 28days and 24 weeks. The diagnosis of ALF is based on the triad of Jaundice, altered mental status and coagulopathy.

The initial manifestation of ALF is nonspecific with anorexia, fatigue, abdominal pain, and fever. With advancing liver injury signs of ALF emerges. Patient develops jaundice, encephalopathy, coagulopathy, hemodynamic instability, acute renal failure, ascites, lung injury, sepsis metabolic abnormalities.

In tropical countries like India, the differential diagnosis of ALF should include severe infection with Plasmodium Malaria, Dengue fever, Leptospirosis, Rickettsial infections, Enteric hepatitis, Hepatic tuberculosis, Amoebic liver abscess.

But ALF due to organophosphorous is not reported in literature.

Here a case was found in India who developed the features of ALF like jaundice, encephalopathy, coagulopathy but the infective markers for viral hepatitis, dengue, malaria and leptospiira negative. He developed the features of ALF after consuming some home-made food. Five family members died only one person survived who later on developed ALF. The autopsy of all deceased family members revealed presence of Organophosphorous in the food.

TREATMENT

The most important aspect of treatment of acute liver failure is to provide good intensive care support, including protection of the airway. Specific therapy is also dependent on the cause of the patients liver failure and presence of any complications. Careful attention should be made to patients fluid management and hemodynamics. It is crucial to monitor their metabolic parameters, assess for infection, maintain nutrition, and promptly recognize GI bleeding.

Liver transplantation is the definitive treatment in liver failure. In selected cases in whom no allograft is immediately available, consider support with a bioartificial liver. This is a short-term measure that only leads to survival if the liver spontaneously recovers or is replaced.

In the above case patient was managed with ICU care, head elevation, mannitol, N-acetylcysteine, vitamine K, IV fluid, IV antibiotics and supportive treatment. Patient started improving after 72 hrs. He was fully recovered within 10days without liver transplant.

CONCLUSION

So it is prudent to anticipate that as survived patient had taken same food along with the deceased and as his initial presentation was similar to all other in whom autopsy study revealed organophosphorous compound. Same must be the cause of ALF in survived patient. The patient survived without liver transplant.

Organophosphorous can cause acute liver failure in isolation and patient of acute liver failure may survive without liver transplant.

REFERENCES