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A CASE OF FULMINANT HEPATIC FAILURE

KEYWORDS

Dr. Kavi chakravarthi M.G
Post graduate, Sri Ramachandra university, Chennai.

Dr Mohini Singh
Associate professor, Sri Ramachandra university, Chennai.

Dr S. R. Ramakrishnan
Professor, Sri Ramachandra university, Chennai.

INTRODUCTION

Acute liver failure (ALF) occurs due to a sudden severe insult to a previously normal liver or atleast well compensated liver disease, which leads to the development of progressive hepatic encephalopathy (HE), coagulopathy and jaundice. It is a broad term that encompasses both fulminant hepatitis and sub fulminant hepatic failure (or late-onset hepatic failure). Fulminant hepatic failure is characterized by the development of severe liver injury with impaired synthetic capacity and encephalopathy (within 8 weeks) whereas, sub fulminant hepatic failure is reserved for patients with liver disease for up to 26 weeks before the development of hepatic encephalopathy. ALF is a rare disease but has the potential to rapidly progress to multi-organ failure leading to death.

The rarity and severity of this disease results in a uniquely challenging illness, particularly in relation to its study and the development of effective treatments.[1] In this report, we discuss a 26 year old woman with hepatitis A infection which progressed on to fulminant hepatic failure managed with plasmapheresis.

CASE REPORT

A 26yr old female presented with jaundice, low intermittent fever and vomiting for 7 days, history of altered sleep pattern There was no history of liver disease, blood transfusion, tattoos, drug intake or alcohol abuse. There was no family history of liver diseases. On examination her blood pressure of 120/80, pulse rate of 76 respiratory rate 16, she was noted to be icteric. Abdominal examination awaited tender hepatomegaly on central nervous system examination flaps was present Cardio-pulmonary examinations was normal.

Initial investigations revealed Hb of 12.7g/dl, total count of 1800 cells/mm^3 and platelet count of 1.86 lakh cells/mm^3. Evaluation of liver function showed a total bilirubin of 11.01 mg/dl with highly elevated liver enzymes (SGOT 1140 IU/L and SGPT 8022 IU/L) and an albumin of 3.7g/dl. INR value was 2.3 Serum ammonia level was raised. Renal function and electrolytes were normal. See. Table 1

Viral hepatitis B, C & E serologies were negative. Anti-HAV IgM antibodies was reactive. Anti-human immunodeficiency virus antibody was negative. Anti-nuclear antibody, anti-smooth muscle antibody and anti-mitochondrial M2 antibody were also negative. Tests for malaria, dengue, leptospirosis were negative. Initial blood and urine culture were negative. Ultrasound abdomen showed tender hepatomegaly and mild ascites. Anti-HAV IgM antibody positivity with raised liver enzymes, raised ammonia levels and prolongation of prothrombin time (PT) indicated a diagnosis of ALF with hepatic encephalopathy due to hepatitis A.

Patient's sensorium worsened she was started on antiencephalopathy measures with syrup lactulose, high bowel wash, probiotic and tablet rifaximin was given, anti coagulopathy measures with injection of vitamin K was given and FFP was transfused. Despite these measures, patients sensorium and liver parameters further worsened hence was put on ventilatory supportShe underwent two cycles of plasmapheresis which improved her Glasgo Coma Scale. Her condition gradually improved and she was extubated. Her liver parameters dropped within normal limits and she was discharged and advised regular follow up.

INVESTIGATIONS

Table 1 showing Lab Investigations done Day One

Investigations	Values
Hb %	12.7
TC	1,800
Platelet Count	1..86
PT	34.5
PTT	36,2
INR	2.3
Sodium	138
Potassium	4.1
Bicarbonate	21
Chloride	93
BUN	52
Creatinine	1.3
TB	11.1
DB	6.1
SGOT	1100
SGPT	822
Ammonia	86

Table 2 showing Lab Investigations done after Plasmapheresis

Investigations	Values
TB	14.2
DB	9.1
SGOT	163
SGPT	63
PT	67.5
PTT	41.6
INR	4.5

DISCUSSION

In 1970, Trey and Davidson defined "fulminant hepatic failure" as a severe liver injury potentially reversible in nature and with the onset of hepatic encephalopathy within 8 weeks of first symptoms in the absence of preexisting liver disease.[2] In 1993, O'Grady et al. based on data from King's College subdivided ALF into hyperacute, acute, and subacute presentation depending on the interval from onset of disease to onset of encephalopathy.[3]

Jaundice and encephalopathy can be caused by two groups of

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conditions, diseases that lead to acute liver failure and diseases that may either mimic acute liver failure. Commonest among all causes of jaundice and encepahalopathy are hepatotropic viruses such as hepatitis A, B & E.[4]

Of the hepatotropic virus ,Hepatitis A virus (HAV) infection is a major cause of acute hepatitis worldwide.[5] ,but Acute hepatitis A rarely develops into acute liver failure (ALF), which sometimes requires liver transplantation or cause death.[6] HAV is an enterically transmitted virus, belonging to the Picornaviridae family, and replicates in the liver. HAV is generally a non-cytopathic virus and is believed to cause hepatocyte injury by host immune mechanisms.[7]

Sainokami et al. reported that patients with severe hepatitis A are more than 41 years old.[8] Although there are certain patients that are aged less than 40 years old with severe hepatitis A. Elderly patients tend to be associated with severe HAV genotype 1a infection.[9, 10] With the coexistence of chronic hepatitis B, the fatality rate of hepatitis A is higher than that without HBV infection (0.009 vs. 0.05%).[11]

The most feared complication of viral hepatitis is fulminant hepatic failure featured as encephalopathy, coagulopathy, cerebral edema. It is primarily seen in hepatitis B and D. Incidence of hepatitis A is 10%.

Management consists of intensive care support, treatment of specific etiology if present and early detection of candidates for liver transplantation.[13, 14] Liver assist devices and hepatocyte transplant remain experimental and further advances are required.[15]

A study done by Demetriou et al [16] in which 171 patients with fulminant/subfulminant hepatic failure showed that survival rates were higher in patients who were on plasmapheresis. Jens Otto et al [17] showed plasmapheresis helps in decreasing the arterial ammonia in patients with acute liver failure.This could be explained by increased hepatic urea synthesis and possibly by increased glutamine synthesis in muscle tissue.Freeman et al reported that survival following plasmapheresis appeared substantially better than in a non randomized group of similar patients not plasmapheresed. Plasmapheresis significantly decreased serum bilirubin, aspartate aminotransferase and plasma ammonia concentrations.[18]

Fulminant hepatitis is usually treated by liver transplant. In this case, the patient fulfils king's hospital criteria (↑prothrombin time>100, serum bilirubin greater than 17.5 mg/dl) for liver transplant. But alternative approach with plasmapheresis proved to be effective. Thus plasmapheresis prevents the associated complications arising with liver transplant. Identification of underlying cause for fulminant hepatitis plays an essential role in proceeding with the treatment methods.

Conclusion

Plasmapheresis can be thought as a treatment for Acute Liver Failure patients in those with in whom liver transplantation is not an option. Further research is warranted in the usefulness of plasmapheresis in acute liver failure

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