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Fatal Acute Liver Failure by Herpes Simplex Virus- A Case Report

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ABSTRACT

Although exceedingly rare, fulminant hepatic failure in patients can develop with primary or reactivation of infection due to herpes simplex virus (HSV). The diagnosis is frequently suggested by the presence of mucocutaneous involvement. Markedly elevated transaminases with leucopenia and a relatively low bilirubin level may provide clues to the diagnosis. Here we present the case of a 83-year-old male who developed herpes simplex virus hepatitis leading to fulminant liver failure and death after being on steroids.

Keywords: Fulminant hepatic failure, herpes simplex virus, transaminases

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INTRODUCTION

HSV hepatitis is rare but prognosis of HSV associated fulminant hepatitis is poor. It carries a high mortality risk, even though an effective antiviral treatment is available. This may be explained by the fact that the diagnosis is usually delayed, so that initiation of treatment is too late. The delay in diagnosis is essentially due to the lack of specific symptoms. In fact, acyclovir given in the early stages of fulminant hepatic failure may prevent mortality.

Case Report:

A 83 year old male, known case of Chronic Obstructive Pulmonary Disease (COPD), Diabetes mellitus type II and hypertension on regular treatment was admitted at our hospital with history of drowsiness since night. He had been discharged 1 day ago after being treated for 1 week for acute exacerbation of COPD for which he had received steroids. Examination revealed that he was afebrile, anicteric and mildly confused. His vitals were stable. He had no oral or genital lesions and skin was clear. His abdomen was soft and nontender with no organomegaly. On admission, he was in hypoglycemia with blood sugar level 55 mg%. 25% Dextrose was given intravenously and after that his sensorium normalized.

Table 1: Laboratory Values at Previous admission, Readmission and at Death.

	Previous admission	Readmission		At Death
Date	01-01-2012	08-02-2012	10-02-2012	14/2/2012
Hb (g/dl)	11.3	10.4	10.6	4.9
TLC (/mm^3)	8380	11960	6330	1930
NEUTROPHILS (/mm^3)	93	89	85	81
LYMPHOCYTES (/mm^3)	4	6	5	9
PLATELET (lakhs/mm^3)	2.08	1.69	0.96	1.09
PCV				15.3
BIL IRUBIN TOTAL (mg/dl)	0.3		0.2	1.6
BILIRUBIN DIRECT (mg/dl)	0		0	1.1
BILIRUBIN INDIRECT (mg/dl)	0.3		0.2	0.5
SGOT (U/L)	21		1004	9300
SGPT (U/L)	29		585	2855
TOTAL PROTEIN (g/dl)	5		5	5.4
ALB (g/dl)	2.7		2.6	2.9
GLB (g/dl)	2.3		2.3	2.5
ALP (U/L)	83		70	251
PT (sec)	11			21.7
aPTT (sec)	25.4			52.3
INR	0.9			1.79
CREATININE (mg/dl)	1.5	1.1	1.4	3.4
UREA (mg/dl)	66	48	43	64

His initial investigations on present admission revealed Haemoglobin level of 10.4g/dl, total leucocyte count (TLC) of 11960 cells/mm^3 with 89% neutrophils, platelets 169000/mm^3,

urea 48 mg% and creatinine was 1.1 mg%. His liver function tests were not done on the day of admission and were normal one week ago (Table 1).

On day two of admission, he had fever and increasing drowsiness. His TLC dropped to 2280 cells/mm^3 and platelet count to 54000/mm^3. LFT revealed Total bilirubin 0.2 mg%, SGOT and SGPT increased to 1004 IU and 585 IU respectively. ALP was normal.

His urine output started falling and was becoming drowsy and was shifted to ICU. HbsAg ,IgM Anti-HCV, IgM Anti-HAV, IgM Anti-HEV, IgM Anti-EBV, IgM Anti-CMV, IgM Anti-HSV, IgM Anti-Dengue were sent. On fifth day, SGOT and SGPT went into thousands international units. PT, aPTT, INR, urea, creatinine increased further and TLC, platelet count, urine output decreased. He was intubated and mechanically ventilated .He started bleeding profusely. Multiple units were transfused for correcting coagulopathy. Hypotension was managed with ionotropes. Hemodialysis was done in view of metabolic acidosis and fluid overload. He had upper GI bleed and died on day seven in hospital. IgM HSV titres came positive (Ratio-1.5) after he had expired (> 1.2 positive). All other viral serologies were negative. His cause of death was determined to be herpes simplex viral hepatitis causing fulminant liver failure with dissemination to kidneys and possibly other organs.

DISCUSSION

Herpes simplex virus (HSV) is extremely common throughout the world. It is estimated that up to 80% of adults contract HSV throughout their lifetime and that most infections are asymptomatic or produce only mild nonspecific viral symptoms.[1] HSV hepatitis is rare and accounts for only 1% of all acute liver failure cases and only 2% of all viral causes of acute liver failure (ALF)[1,2]. ALF due to suspected viruses with negative serology for A-E should be tested for other viruses i.e. infectious mononucleosis, cytomegalovirus, herpes simplex, dengue to detect the cause.

It occurs most commonly in organ transplant patients, in the third trimester of pregnancy or in patients who are otherwise immunocompromised, but up to 25% of patients who develop HSV hepatitis are immunocompetent.[3]

HSV hepatitis presents with nonspecific flu-like symptoms including fever, myalgias, and abdominal pain. Only 30–50% show characteristic herpetic skin lesions. Laboratory investigations often show leucopenia, thrombocytopenia, and coagulopathy [3,4]. Renal failure is not uncommon in these patients and it has been shown to occur in up to 65% of patients with HSV related ALF.[3,5] Disseminated intravascular coagulopathy is frequently reported and encephalopathy is a late sign of the disease.[5,6] Ninety percent of patients with HSV hepatitis have a characteristic liver profile, known as “anicteric hepatitis”.[4,6] Anicteric hepatitis refers to a liver profile showing a significant increase in transaminases (100–1000 fold) with a relatively normal or low bilirubin [1,3,4,6]. There may be a marked elevation of SGOT greater than SGPT.[4]

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Antemortem diagnosis of HSV hepatitis is difficult. Investigations to aid in the diagnosis for HSV hepatitis are limited. The simplest and widely available for us was IgM HSV serology. Other tests are not easily accessible.

Antiviral treatment with acyclovir has been used successfully.[3,4,5] The extent of disease at the initiation of acyclovir plays a large role in its effectiveness, but outcomes probably improve with earlier initiation of therapy.[3]

CONCLUSION

Physicians should consider HSV hepatitis in patients with fulminant liver failure of unknown cause. A thorough examination, including examination of the oropharynx and a complete pelvic exam, may provide clues to the diagnosis. If possible, liver biopsy is the gold standard for diagnosis. In any patient presenting with flu-like illness, especially with skin lesions and anicteric hepatitis, HSV should be suspected and early treatment with acyclovir should be strongly considered.

Abbreviations:

IgM Anti-EBV - Immunoglobulin M Anti Epstein Barr virus, IgM Anti-CMV - Immunoglobulin M Anti Cytomegalovirus, IgM Anti-HSV - Immunoglobulin M Anti Herpes simplex virus, IgM Anti-Dengue - Immunoglobulin M Anti Dengue virus, PT- Prothrombin time, a PTT - Activated partial thromboplastin time, INR - International normalized ratio, FFP - Fresh frozen plasma.

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