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Herpes Simplex Virus Induced Acute Fulminant Hepatitis in Immunocompetent Patient

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Abstract
HSV Induced acute fulminant hepatitis is a rare and a fatal disease. It is always overlooked as a cause of fulminant hepatitis which delays the diagnosis and treatment leading to fatal outcome. Occurrence of HSV hepatitis is rare in immunocompetent patients and these patients doesn't have mucocutaneous lesions making it difficult to diagnose. HSV Hepatitis has very good response to acyclovir and has proven to be beneficial in treating these patients. But due nonspecific presentation and delay in diagnosis, it often proves fatal.

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I. Introduction

Acute fulminant hepatitis is a life threating condition which involves acute injury to the liver parenchyma leading to impairment of its function in patients with previously normal functioning liver [1]. It is characterized by high grade fever, abdominal pain and jaundice. Presentation of HSV related acute fulminant hepatitis is similar to the other causes of acute fulminant hepatitis. HSV related acute fulminant hepatitis often presents with triad of high grade fever, markedly reduced leucocyte count and elevated aminotransferases levels [2]. Other features include coagulopathy, encephalopathy and thrombocytopenia. Rash or mucocutaneous lesions are rarely seen in immunocompetent patients [3]. HSV infection can also cause hemolytic anemia which can co-exist in these patients [4]. This condition can be diagnosed by doing DNA PCR and presence of antibodies against HSV [5]. Acyclovir remains one of the lifesaving drug in patients of HSV related fulminant hepatitis. Timely initiation of acyclovir in these patients can improve the outcome in these patients [2, 3].

Case Presentation

18 years male presented with high grade fever, pain in abdomen and dark colored stools since 5 days. He never had similar complaints in past. There was no history of any drug intake or exposure to any toxins. On clinical examination patient was conscious, alert, irritable, and febrile. He had fever of 104 Fahrenheit, tachycardia of 120 beats per minute with normal blood pressure and oxygen saturation. General examination revealed severe pallor and icterus. He had tender abdomen with palpable liver and spleen on per abdominal examination. His rest of systemic examination was normal. His fundus examination revealed vitreous hemorrhage with retinal detachment.

He had normal chest x-ray and electrocardiogram. He had low levels of hemoglobin, WBC, platelets and hemoglobin concentration. His mean corpuscular volume was high. His total bilirubin levels were raised with indirect bilirubin levels more than direct bilirubin levels. There was more than tenfold rise in aminotransferases levels. His international normalizing ration was high. His alkaline phosphatase levels were in normal range. His serum electrolytes and renal functions were in normal range. His Vitamin b12 was in normal range. His serum iron, total iron binding capacity and transferrin saturation was on the lower side. His total protein was in normal range with marked elevated LDH and CRP levels. His protein and hemoglobin electrophoresis were normal. His ANA western blot was negative for autoantibodies. He was negative for hepatitis A, B, C, D and E. His G6PD levels were in normal range. There was no evidence of PNH clone in gated neutrophils and monocytes on immunophenotyping. He was negative for cytomegalovirus and epstein-barr virus. His serological markers were strongly positive for HSV 1 and 2. Peripheral smear examination revealed macrocytosis, microcytosis, anisopoikilocytosis, pencil cells, tear drop red blood cells with 74% neutrophils, 20% lymphocytes and 6% atypical virocytes platelets count was reduced. Bone marrow aspiration studies revealed hypercellular bone marrow with normal myeloid/erythroid ratio with no abnormal cells. USG of abdomen revealed moderate hepatosplenomegaly. CT of Abdomen revealed moderate splenomegaly. Table 1 shows all the lab parameters

Investigations	Patients values	Reference range
Hemoglobin	2.1 gm/dl	14-17.5 gm/dl
MCV	104 femtoliter	80-100 femtoliter
Platelet	20000 / microliter	150000 to 450000 / microliter
WBC	1600 / microliter	4500 to 11000 / microliter
Sodium	135 mEq/L	135-145 mEq/L
Potassium	3.5 mEq/L	3.5-5.5 mEq/L
Creatinine	0.4 mg/dl	0.7-1.3 mg/dl
Urea	40 mg/dl	6-40 mg/dl
Total bilirubin	10.5 mg/dl	0.3-1.2 mg/dl
Direct bilirubin	2.5 mg/dl	0.1-0.3 mg/dl
Indirect bilirubin	8 mg/dl	0.2-0.8 mg/dl
Total protein	6.2 g/dl	6-8.3 g/dl
Albumin	3.6 g/dl	3.4-5.4 g/dl
globulin	2.6 g/dl	2-3.9 gm/dl
SGOT	2711 unit/liter	8-45 units/liter
SGPT	2241 unit/liter	7-56 units/liter
Alkaline phosphatase	86 U/L	70-369 U/L
PT-INR	2.4	< 1.1
Serum iron	31.14 microgram/dl	70-180 microgram/dl
Total iron binding capacity	200.91 microgram/dl	250-450 microgram/dl
Transferrin saturation	15.50 %	20-40 %
Vitamin B12	911 pg/ml	120-914 pg/ml
G6PD	17.8 U/gm of hemoglobin	6.4-20.0 U/gm of hemoglobin
C reactive protein	34 mg/dl	Less than 6 mg/dl
LDH	2633 mIU/ml	230-460 mIU/m
HSV 1+2 IgG	15.4	Negative – less than 0.9, Positive more than 1.1

Patient was started with acyclovir therapy with other supportive management including adequate hydration, antibiotic therapy, antioxidants, and micronutrient supplementations. Patient improved gradually over 10 days of therapy.

II. Discussion

Acute fulminant hepatitis is a life threating condition which involves acute injury to the liver parenchyma leading to impairment of its function in patients with previously normal functioning liver. Paracetamol poisoning remains one of the major cause of fulminant hepatitis in entire world. Hepatitis A, B, C, D and E infection also remains one of the major cause of acute hepatitis. Acute fulminant hepatitis can also be seen in autoimmune, ischemic and toxin related conditions [1]. Rarely non hepatitis virus like CMV, EBV and HSV can cause acute fulminant hepatitis [1, 2]. HSV Hepatitis is highly fatal but a treatable condition provided there is an early diagnosis and treatment. Diagnosis of HSV hepatitis is challenging as it is clinically similar to other cause of hepatitis. Mucocutaneous lesions are rarely seen in immunocompetent patients [3]. Herpes infection can cause hemolysis in these patients leading to picture of nonimmune hemolytic anemia [4].

HSV can cause acute fulminant hepatitis by various mechanism like dissemination of virus into the liver and other viscera due to impaired macrophages and cytotoxic T lymphocyte function or reactivation of latent HSV infection [1]. Diagnosis of HSV infection can be done by isolation of virus from lesion or detection of antibody against antibodies in blood. Various techniques includes microscopy, biochemical assay and nucleic acid amplification technique. ELISA remains a simple and rapid diagnostic tool as compared with western blot assay [5].

Acyclovir remains one of the cornerstone therapy in treatment of HSV hepatitis. Acyclovir is converted to its active form by thymidine kinase. It is a nucleoside analogue that prevents virus multiplication. In patients of acyclovir resistant hepatitis foscarnet or cidofovir can be used. Various studies have suggested that early use of acyclovir can give a better clinical outcome in patients of HSV induced acute fulminant hepatitis [1].

III. Conclusion

HSV hepatitis is a highly fatal and a treatable condition if it is diagnosed early and treated on time with acyclovir therapy. HSV infection should be suspected in patients with triad of high grade fever, decreased leucocyte and raised liver enzymes. Mucocutaneous lesion are often absent in immunocompetent patients and

can primarily presents as HSV hepatitis rather than mucocutaneous lesion, hence absence of mucocutaneous lesions does not rule out HSV infection in immunocompetent patients. Use of acyclovir as an empirical therapy in patients of acute fulminant hepatitis should be a routine practice until the exact etiology of fulminant hepatitis is known.

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