

## Case Report

### A Case of Severe Hepatitis due to Varicella Zoster

Azarkar Z<sup>1\*</sup>

1. Assistant Professor of Infectious Disease, Birjand University of Medical Sciences, Birjand, Iran.

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Varicella is a common self limiting and usually benign disease. However, some serious complications are known. Age and immunologic impairment are predisposing factors for severe varicella (1). Varicella zoster Virus (VZV) is frequently associated with mild hepatitis and acute liver failure- with rare fatality. The pathogenic mechanisms underlying these complications are largely unknown. Recently mutations of VZV DNA have been described in immune depressed patients. However, they were not associated with severe clinical manifestations (2).

We describe a case of acute, severe hepatitis in healthy 16- year old boy with VZV infection.

#### Case presentation

A 16 year – old male without significant medical history was admitted to the infectious disease ward for evaluation of vomiting, abdominal pain, anorexia, low fever, and jaundice. Five days earlier he had developed fever, followed by common cold symptom. He had history of close contact with a chickenpox patient two weeks before. On admission physical examination showed jaundice and fever.

Initial laboratory findings were:

WBC count 7700/ $\mu$  L, platelet count 150000/ $\mu$

L, AST 3091 U/L, ALT 3289 U/L, LDH 841 U/L, ALP 550 U/L, bilirubin total 5.5 mg/dl, Direct bilirubin 4.4 mg/dl, PT=19 seconds.

HBS Antigen, anti HBC-IgM, anti HCV, anti cytomegalovirus (CMV) IgM, anti Epstein Barr virus (EBV)-IgM and anti –HAV-IgM were all negative.

CXR was normal. Ultrasonography revealed hepatosplenomegaly.

On the second day after admission coetaneous vesicles suggestive varicella appeared on the face and trunk.

Serum VZV IgM antibody and serum VZV PCR assay were positive.

Treatment with intravenous acyclovir 10mg/kg three times /day was started on the second day.

This kind of systemic manifestations and occasional visceral dissemination can occur. Patients with cellular immunodeficiency, regardless of age, are at risk for severe or disseminated cutaneous disease and visceral involvement (3).

Disseminated VZV have been well described in the adult population. This viral infection occur primarily in patients who are immunosuppressed, with renal transplantation, steroid use, and pregnancy reported as the most frequent causes of impaired immunity (4).

In adults with varicella infection hepatitis is asymptomatic with mild elevation of transaminases. Severe hepatitis with V2V in an immune competent host is uncommon (1).

In most previous reports patients have been described as immune compromised for one or several reasons such as splenectomy, renal transplantation, bone marrow transplantation, use of corticosteroid and AIDS. The most

\*Corresponding author: Zohre Azarkar, Assistant Professor of Infectious Disease, Birjand University of Medical Sciences, Birjand, Iran.  
Tel: (+98) 561 44 45 402  
Email: Drz.azarkar@yahoo.com

frequent symptoms described were cutaneous varicella lesions, acute abdominal or back pain, and fever (5-10).

Beby-Defaux reported a case of 28 years immunocompetent man. Who despite acyclovir therapy developed hepatitis, severe pneumonia, rhabdomyolysis and disseminated intravascular coagulation, followed by encephalopathy and multiorgan failure therapy (11).

Rogers et al and Rosset all described patients with severe varicella hepatitis without any skin lesions (12).

In this case the patient was a healthy boy with no history of VZV in his childhood. He got the infection from his brother two week, before admission.

The standard treatment for organ VZV infection is acyclovir 10mg / kg three times /day and this should be started immediately upon suspicion of diagnosis (10).

The patient received acyclovir for 10 days and treated successfully.

The mortality rate associated with VZV induced hepatic failure is very high. A few patients who survived had received acyclovir and /or underwent liver transplantation (13).

The clinical picture, combined with positive VZV serology and a positive skin biopsy specimen could be highly suggestive .A diagnosis of VZV infection can be established rapidly with VZV DNA detection in the serum by PCR (14). Histopathology examination of the liver contributes to the diagnosis .In typical cases there are areas of limited hemorrhagic necrosis multinucleate, ground glass nuclear staining and eosinophilic cawdry type A intra nuclear inclusion.

Electron Microscopy shows characteristic intracellular virions of herpesviridae. Herpes simplex and VZV can be differentiated by fluorescent staining, immune histochemistry, insitu hybridization, or PCR (10).

In the current case the diagnosis of VZV induced hepatitis was based on the history of contact with a chickenpox patient, suggestive cutaneous vesicles and positive serology and PCR for VZV.

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