

VARICELLA ZOSTER INDUCED FULMINANT HEPATIC FAILURE: A CASE REPORT

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Abstract- Varicella Zoster Virus (VZV) infection progresses to Fulminant Hepatic Failure (FHF) which is very rare and fatal. Mostly diagnosis of VZV infection becomes difficult due to initial normal liver function tests and absence of early typical vesicular rash. Hence ideal management could not be provided. Low dose steroid therapy carries a risk for VZV to progress to FHF, as the patient becomes immunocompromised. This is a case of VZV induced FHF of a 36 years old female patient who was on low dose steroid therapy for bronchial asthma management. She developed vesicular rashes on second day of admission and had elevated liver enzymes. Treatment as done with IV Acyclovir. On fourth day, there was persistent rashes and rapidly rising liver enzymes and resulted in multi organ failure and death.

Keywords: fulminant hepatic failure, varicella zoster, hepatitis, vesicular rash

INTRODUCTION

Varicella Zoster Virus (VZV) presents clinically as primary (chicken pox) or secondary (herpes zoster) infection. Primary infection usually produces generalized vesicular rash. The virus then presents latency in the dorsal root ganglia and may later reactivate as secondary infection in form of “shingles” or herpes zoster.^[1] Fulminant Hepatic Failure(FHP) due to VZV is extremely rare and deadly. Antiviral treatment with Intravenous (IV) acyclovir can be effective if initiated in a timely manner, but comorbidities and complications frequently result in high mortality.^[2]

CASE REPORT

A 36-year old lady presented to emergency department with a one-day history of abdominal pain. She had a pain score of 5-6 in Wong-Baker Faces pain rating scale. There was no history of vomiting or burning micturition. She had a medical history of dyslipidemia and was on Atorvastatin. She was recently diagnosed with bronchial asthma and was on low dose steroid therapy. No recent travel history or interaction with sick contacts was mentioned at the initial encounter at the emergency department.

The patient had normal vital signs. Abdominal examination showed tenderness in right iliac fossa, epigastrium and suprapubic. Initial laboratory parameters including Complete Blood Count (CBC), urine routine, Renal Function Test (RFT) and Liver Function Test (LFT) were within the normal range except C-Reactive Protein(CRP 20.2 mg/L), urine epithelial cells (30-35) and Aspartate aminotransferase (AST 127 U/L). Hence the patient was admitted for further evaluation.

On first day, the patient was given pain medications and IV antibiotics with IV hydration and repeat set of labs was ordered.

On second day morning, there was persisting abdominal pain along with vesicular rashes on face, chest and extensive erosion on oral mucosa. The diagnosis of VZV hepatitis was immediately considered and medical team was involved. Tzanck smear showed multinucleated cells indicating VZV infection. Hence acyclovir was started by medical team.

On third day, patient had persisting abdominal pain, fever and vesicular rash all over the body. The vesicles appeared crusted. The other work ups for hepatitis was non reactive and continued IV Acyclovir along with supportive medicines.

On fourth day, the patient had rapidly rising liver enzymes, thrombocytopenia and she developed myocarditis and sepsis. The patient was shifted to Multi-Disciplinary Intensive Care Unit (MDICU) and died on same day due to cardiac arrest.

DISCUSSION

VZV can affect both immunocompromised and immunocompetent adults. Dissemination of VZV infection and fulminant hepatitis occurs in immunocompromised patients. Medication history of steroid therapy as a trigger for temporary immunodepressed state should be considered. Steroid was reported as a possible risk for disseminated VZV infection only in one case.^[3]

In terms of symptoms, our patient was initially presented with right upper quadrant pain and tenderness, without the typical vesicular rash of primary varicella infection that appeared on second day. This was related to a case series (4 patients) wherein 3 patients, the initial abdominal pain appeared 2-4 days before the typical herpetic vesicular skin lesions, while the fourth case had not developed any lesions at all.^[4] Absence of skin lesions delay early diagnosis as abdominal imaging findings indicating visceral VZV has been rarely reported.^[5]

With regard to diagnosis, there was one day delay due to absence of lesions and the initial normal laboratory reports. This was in supportive of other cases which showed such delay in diagnosis due to absence of rash.^[6] Our patient seemed to show FHF on second day. The differential diagnosis of FHF includes ischemia, toxins, medications, autoimmune, hepatitis, metabolic and infectious causes with VZV being the rarest. In this case, the patient had no history of ingestion of any toxins related to cause hepatic failure.^[7]

Concerning management, treatment was started with intravenous acyclovir from the second day.^[7] Early IV acyclovir is the key to treatment of VZV infection. Other considered therapies include VZV immune globulin, liver transplant and supportive care. Since VZV induced FHF has high mortality rates and as early treatment is critical to survival, VZV hepatitis should be considered in differential diagnosis of all patients with liver failure who presents with a rash.^[8-12]

CONCLUSION

Acute VZV infection may present as FHF and the diagnosis is mostly challenging based on initial presentation as the vesicular rashes appear lately. Hence detailed contact history should be noted and medication history to be reviewed thoroughly to identify any risk factors that may exist. Low dose corticosteroid carries a risk for VZV infection to progress to FHF. Early diagnosis and management of VZV infection is the key to survival.

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