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Case report

# Epstein-Barr virus hepatitis can cause transient hepatopulmonary syndrome

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ABSTRACT

*Introduction:* Hepatopulmonary syndrome is commonly seen in the patients with chronic liver disease. Acute liver diseases are rarely associated with HPS. We have reported here a case of Transient HPS caused by Epstein-Barr virus hepatitis.

*Case report:* The patient was a 31 years old man that came to hospital due to RUQ pain and yellowish skin. In examination the patient was tachypnic and O2 saturation was 71% with prominent JVP. ver enzyme and bilirubin were high. All viral hepatitis was negative except anti viral capsid antigen-antibody of EBV. In Blood gas PaO2 was 54 mmHg, O2 saturation 73% and alveolar-arterial gradient was 18 mmHg. Stress Echocardiography with saline injection reported pulmonary arterial pressure 32 cmHg with delayed opacification of left atrium.

*Conclusion:* transient HPS can be manifestation in the acute hepatitis caused by EBV infection.

## 1. Introduction

Epstein-Barr virus (EBV) is a widely diffused herpesvirus that is spread by close contact between susceptible persons and asymptomatic EBV shedders [1]. EBV can affect virtually all body systems and has been associated with such different disease manifestations as acute cholestasis or hepatitis [2,3] Infectious mononucleosis is an acute viral disease in children and Young with a high incidence. It is commonly a benign, self-limited, multisystem lymphoproliferative disease generated by EBV [4]. Acute hepatitis with cholestatic pattern is a rare manifestation in primary Epstein-Barr virus infection [5]. Hepatopulmonary syndrome (HPS) is most commonly seen in patients with chronic liver disease, particularly those with portal hypertension [6–8]. For diagnosis of HPS, first, intrapulmonary artery dilatations must be documented by either contrast echocardiography, lung scanning, or pulmonary angiography. Second, any of the above findings occurring in the setting of liver disease and hypoxemia (PaO2 < 70 mm Hg) should be considered enough clinical information to diagnose of syndrome [9]. Here we report a Hepatopulmonary syndrome in a patient who presented with diagnosed acute hepatitis caused by Epstein-Barr virus.

## 2. Case report

The patient was a 31-year-old man without previous medical disorder that came to hospital due to epigastric & RUQ pain and yellowish skin. Other complaints of patient were anorexia, constipation, sore throat and nausea/vomiting. The primary examination vital sign was stable, mild tonsillar erythema, abdominal tenderness in right upper quadrant and lymphadenopathy in inguinal right side. After 2 day of hospitalization patient due to dyspnea (platypnea) and decrease O2 Saturation admitted in the ICU. In the examination the patient was tachypnic and O2 saturation was 71% with prominent Jugular vein. In the laboratory tests were high liver enzyme and bilirubin (hepatocellular pattern). In blood gas PaO2 on room air was 54 mmHg, O2 saturation was 73% and alveolar-arterial gradient was 18 mmHg. All viral hepatitis (HCV, HBV, HAV) are negative except anti-viral capsid antigen (VCA IgM) antibody of EBV. ceroluplasmin for evaluation of WILSON Disease was normal. Rheumatologic marker (ANA, C3, C4, Anti dsDNA) was normal range.

Table 1 demonstrated laboratory tests During hospitalization of patient. Chest radiography was normal without parenchymal lung disease and lymphadenopathy. The color Doppler sonography and abdominopelvic sonography had only prominent Spleen size measuring 14.7 and have not thrombosis and liver disorder.

Stress Echocardiography with saline injection reported normal Ejection Fraction (60%) with pulmonary arterial pressure 32 cmHg and delayed opacification of left atrium. No evidence of Pulmonary thromboembolism in CT Angiography. The patient improved after

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**Table 1**
Laboratory test.

| | | | |
|---|---|---|---|
| WBC cells/mcl | 16.1 | 13.64 | 11.2 |
| HB gm/dl | 12.4 | 11.1 | 11.5 |
| PLT cell/mcl | 166 | 103 | 292 |
| AST u/l | 423 | 186 | 88 |
| ALT u/l | 642 | 211 | 163 |
| ALK.p u/l | 376 | 570 | 347 |
| B.Total mg/dl | 11.99 | 5.9 | 2.3 |
| B.Direct mg/dl | 7.66 | 2.6 | 1 |
| LDH u/l | 1007 | 907 | 654 |

Abbreviation: WBC: white Blood Cell, HB: Hemoglobin, PLT: Platelet ALT: Alanine transaminase, AST: aspartate transaminase, ALK.P: Alkaline Phosphatase, B: Bilirubin, LDH: Lactate Dehydrogenase.

treatment by steroid and subsided all signs and symptoms. On follow up the signs and symptoms not returned and laboratory tests became normal.

## 3. Discussion

The EBV is a common human herpes virus that produces a long-lasting infection in B cells [10]. Acute hepatitis with the cholestatic pattern is a rare manifestation of primary Epstein-Barr virus infection [5]. Hepatic involvement is usually characterized by mild elevations of liver enzymes seen in 90% of the patients and self-limited; severe cholestasis with jaundice are uncommon [11]. EBV can cause of hilar lymphadenopathy and bilateral alveolar and interstitial infiltration, followed by pulmonary symptoms [12,13]. But in these cases was not orthodeoxia. In our case, EBV arrives with severe hepatitis that is the rare manifestation of EBV. However, HPS is most commonly seen in the patients with the chronic liver disease, particularly those with portal hypertension [7–9]. Acute liver diseases are rarely associated with HPS [14–16]. Temporary HPS has been reported in a patient with acute Hepatitis A Virus [17]. In this case by clinical manifestation (Hypoxemia and Platypnea) and high pulmonary arterial pressure in echocardiography that was a clue for the diagnosis of HPS and laboratory test that show elevation of Liver enzymes, HPS cause by EBV was diagnosed. Other differential diagnosis excluded by physical examination, laboratory test, and Imaging. Our patient after receiving steroid, and other supportive care improved and on follow up, the signs and symptoms not returned and laboratory tests became normal.

## 4. Conclusion

This case shows transient hepatopulmonary syndrome can be a manifestation in the acute hepatitis caused by EBV infection, and show always HPS not occur in the chronic liver disease.

## Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.rmcr.2018.03.016.

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