Non obstructive Cholangitis with EBV (Epstein Barr Virus) Hepatitis

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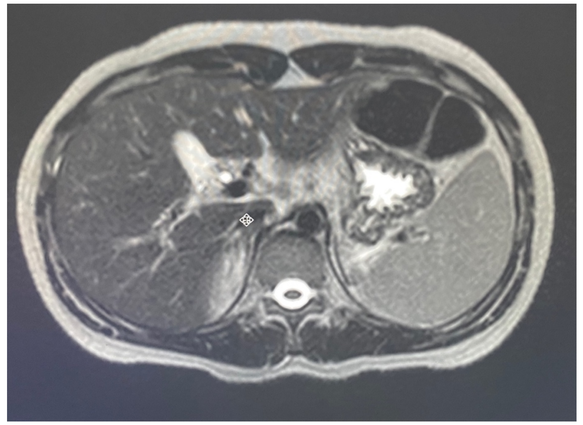
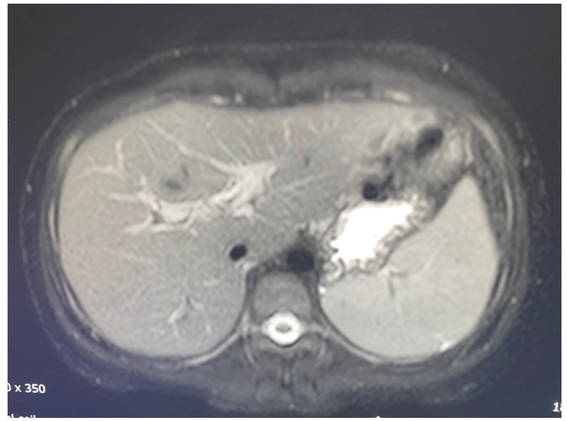
Introduction

Epstein-Barr virus (EBV) is a herpes virus known to cause infectious mononucleosis (1). It is a worldwide prevalent virus that has a seroprevalence of around 90-95% The incidence of EBV causing scleral icterus and cholestasis is less than 5%. To our knowledge the presence of EBV cholangitis in the absence of any underlying cause is absent from the literature. Herein, we report a case of EBV associated with nonobstructive acute cholangitis.

Case Presentation

A 21-year-old Hispanic female with past medical history of endometriosis, spina bifida occulta presented with right upper quadrant and epigastric abdominal pain for the past 4 days. It was non-radiating, constant, colicky in nature with nausea and vomiting. She also had fevers and chills with yellow discoloration of her skin, eyes, and urine. Her vitals were stable. The physical exam was significant for jaundice of the skin and scleral icterus with the right upper quadrant tenderness and negative Murphy's sign. On admission, lab results showed hemoglobin of 14.1 g/dl and white blood cell count of 8.5 K cells/ml, total bilirubin was elevated at 5.8. Liver enzymes including aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) were elevated at 630, 823 and 355 respectively. Lipase was normal. Abdominal ultrasound showed a severely contracted gallbladder with a thickened wall, and a small amount of pericholecystic fluid. MRI showed no gall stones but inflammation in periportal/peribiliary and in the porta hepatis with Gallbladder hydrops and CBD was 5 mm and no intra or extrahepatic biliary dilation (Figure 1 & 2).
Hepatitis Panel, HIV and other work up was negative but positive for mononucleosis screen and EBV DNA quantitative PCR which showed 2800 copies/mL. No surgical intervention was performed. The patient was treated supportively with analgesics and anti-emetics.





Figures 1 and 2: MRI of the abdomen showing periportal and peribiliary inflammation without biliary dilation of the intra and extra hepatic ducts

Discussion

We report the first case of primary EBV infection complicated by acute cholangitis. The pathogenesis of EBV associated cholangitis remains unclear. Our patient imaging showed absence of any stricturing or dilatation in the CBD. In 2020, Colbran & Ng reported a case of EBV hepatitis presenting as ascending cholangitis in an immunocompetent patient. Similar to our case, their patient presented with Charcot's triad however, our patient had peribiliary inflammatory changes which was not seen in their patient. EBV can decrease the production of systemic and intrahepatic pro-inflammatory cytokines which can interfere with the activity of the bile transport systems. Another proposed mechanism for the cholestasis that is seen in EBV infection could be attributed to abdominal lymphadenopathy. Thus, when considering diagnostic protocol for a young patient with acute cholangitis, in the absence of a clear etiology for jaundice, physicians need to be vigilant in ordering EBV workup in order to avoid the overuse of antibiotics, decrease the rate of antibiotic resistance and avoid unnecessary invasive procedures.

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