

Epstein-Barr Virüs Associated Acute Acalculous Cholecystitis

Ferit Celik, Fatih Tekin, Tansu Yamazhan, Fulya Gunsar

Ferit Celik, Fatih Tekin, Fulya Gunsar, Ege University Medical School, Department of Gastroenterology, Izmir, 35100, Turkey

Tansu Yamazhan, Ege University Medical School, Department of Clinical Microbiology, Izmir, Turkey

Correspondence to: Fatih Tekin, Ege University Medical School, Department of Gastroenterology, Izmir, 35100, Turkey

Email: drtekinfatih@gmail.com

Telephone: +90-232-3904357

Fax: +90-232-3427764

Received: May 2, 2014

Revised: May 21, 2014

Accepted: May 28, 2014

Published online: July 21, 2014

ABSTRACT

We hereby present the case of a 48-year-female who presented with signs and symptoms of acute cholecystitis. Radiological investigations revealed no stone or sludge in the gall bladder or bile ducts. There were also lymphocytosis, monocytosis, high fever and cervical lymphadenopathies. Serology tests revealed the presence of acute Epstein-Barr virüs (EBV) infection. The final diagnosis was EBV related acute acalculous cholecystitis (AAC). The patient was discharged with complete clinical and biochemical recovery by conservative therapy. The correct diagnosis avoided unnecessary surgical therapy and overuse of antibiotics.

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Key words: Acalculous cholecystitis; Epstein-Barr virüs

Celik F, Tekin F, Yamazhan T, Gunsar F. Epstein-Barr Virüs Associated Acute Acalculous Cholecystitis. *Journal of Gastroenterology and Hepatology Research* 2014; 3(7): 1179-1180 Available from: URL: <http://www.ghrnet.org/index.php/joghr/article/view/786>

INTRODUCTION

Acute acalculous cholecystitis (AAC) is the inflammation of the gallbladder with the absence of calculi. There exist many causes of AAC, however, Epstein-Barr virüs (EBV) related AAC is an

extremely rare condition. Primary EBV infection has clinical signs such as fever, pharyngitis, lymphadenopathy, hepatosplenomegaly and hepatocellular dysfunction. We hereby present the case of a 48-year-female who was finally diagnosed to be EBV related ACC.

CASE REPORT

A 48-year-old female was admitted to our hospital with fever, malaise, vomiting, jaundice and right upper abdominal pain. She had no medical history. Physical examination revealed a high body temperature of 39.1°C and a positive Murphy's sign suggesting acute cholecystitis. Also there were bilateral palpable cervical lymphadenopathies. Hemoglobin was 11.2 gr/dL, WBC: 5580/mm³, neutrophil: 32.8% (Normal range: 45-68%), lymphocyte: 45.5% (Normal range: 22-31%), monocyte: 19.7% (Normal range: 0-7%), platelet count: 105.000/mm³ (Normal range: 150,000-450,000), SGOT: 221 IU/L (Normal range: 5-35), SGPT: 165 IU/L (Normal range: 5-40), ALP: 516 IU/L (Normal range: 90-260), GGT: 224 IU/L (Normal range: 7-32), total bilirubin: 14,43 mg/dL (Normal range: 0,1-1), direct bilirubin: 12.9 mg/dL (Normal range: <0.25). Other laboratory investigations were found to be normal.

Abdominal ultrasonography (USG) showed pronounced thickening of the gallbladder wall, and a positive sonographic Murphy's sign. There was no stone or sludge in the gall bladder or bile ducts suggesting AAC. Magnetic resonance imaging confirmed the ultrasonographic findings (Figure 1). Oral feeding was stopped, intravenous hydration and antibiotic was started. Neck USG revealed many bilateral cervical lymphadenopathies with thick cortexes of which the largest one was 20×12 mm in diameter.

A possible concomitant viral infection was suspected due to the presence of high fever, lymphocytosis, monocytosis, and cervical lymphadenopathies. Hepatitis A, B, C, cytomegalovirus, human immunodeficiency virus were all excluded, however EBV-VCA (viral capsid antigen) IgM was found to be positive twice. EBV-DNA was also positive. Antibody to EBV nuclear antigen (EBNA) was found to be negative. Intravenous antibiotic was stopped since our final diagnosis was EBV related AAC. On the 14th day of hospitalization, patient was discharged with complete clinical and biochemical recovery.



Figure 1 Magnetic resonance imaging with accompanying cholangiopancreatography showing the marked thickening of the gallbladder wall (arrows). There was no stone or sludge within the gallbladder or biliary ducts.

DISCUSSION

Although cholestatic hepatitis and thickening of the gallbladder wall due to acute EBV infection are well-known features, EBV related AAC is an extremely rare condition. Possible mechanisms of AAC during EBV infection are the presence of bile stasis^[1] and direct viral invasion of the gallbladder^[2], however, the exact pathogenesis remains uncertain. A recent paper by Beltrame *et al*^[3] reviewed 7 cases with EBV related AAC which all of them were female. There has been no clear explanation for why EBV related AAC occurred in females. Six of the patients successfully treated with conservative therapy, and only one patient underwent cholecystectomy. Unfortunately, the pathological findings of the resected specimen was not documented in that patient^[4]. Some of these patients had tonsillar pharyngitis and cervical lymphadenopathy. In the present case, presence of high fever,

lymphocytosis, monocytosis and cervical lymphadenopathy brought us to a suspect of a viral infection, and EBV infection was diagnosed serologically. AAC was diagnosed by well documented clinical and radiological features. No other possible causes of AAC^[5,6] were present. Patient was discharged in well clinical condition and complete improvement in biochemical results. We suggest that development of AAC must be kept in mind in patients with cholestatic hepatitis due to acute EBV infection to avoid unnecessary surgical therapy and overuse of antibiotics.

CONFLICT OF INTERESTS

There are no conflicts of interest with regard to the present study.

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Peer reviewer: Yoshiaki Kawaguchi, Associate Professor, Department of Gastroenterology, Tokai University School of Medicine, 143 Shomokasuya, Isehara, 259-1193, Japan.