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## A case of acute acalculous cholecystitis during the course of reactive Epstein-Barr virus infection

Reaktif Epstein-Barr virus enfeksiyonunda gelişen akut taşsız kolesistit olgusu

## To the Editor,

A previously healthy 8-year-old girl was admitted to our hospital with a seven-day history of fever, abdominal pain, and vomiting. On physical examination, exudative tonsillopharyngitis and cervical adenopathy were noticed. Abdominal examination revealed tenderness in the right upper quadrant. The liver and spleen were both enlarged. Laboratory investigations revealed a white blood cell count of 10.240/mm³ (74% lymphocytes); alanine aminotransferase (ALT) 496 IU/L (N: 0-39 IU/L), aspartate aminotransferase (AST) 569 IU/L (N: 0-36 IU/L), alkaline phosphatase (ALP) 434 IU/L (N: 118-360 IU/L), gamma-glutamyltransferase (GGT) 103 IU/L (N: 0-23 IU/L), total serum bilirubin 4,6

mg/dL with a direct fraction of 3,2 mg/dL. Throat, blood and urine cultures showed no growth. Ultrasonographic (US) examination of the abdomen demonstrated a markedly thickened, edematous gallbladder wall with pericholecystic fluid and no evidence of gallstones or dilatation of the biliary tract. IgG antibodies for Epstein–Barr viral capsid antigen (VCA) were positive at a level of 62.21 (reference: <9), IgG antibodies for Epstein-Barr nuclear antigen (EBNA) were positive at a level of 87 (reference: <5), EBV DNA was detected by real-time polymerase chain reaction (PCR) and quantified in plasma with a viral load of 74.000 copies/mL confirming the diagnosis of reactive Epstein-Barr

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virus (EBV) infection. Feeding was stopped, intravenous rehydration therapy was started. The patient's fever regressed on the fifth hospital day and US findings resolved on the seventh hospital day. Biochemical results and hepatosplenomegaly resolved within two months.

The present case had both cholestatic hepatitis and acute acalculous cholecystitis (AAC) during the course of infectious mononucleosis due to EBV reactivation. AAC is an inflammatory process of the gallbladder in the absence of gallstones that rarely occurs in childhood during the course of infectious disease (1, 2). US criteria for AAC consisted of distention of the gallbladder, thickened gallbladder wall over 3.5 mm, gallbladder wall edema, pericholecystic fluid, nonshadowing echogenic materials or sludge, localized tenderness (sonograp-

hic Murphy's sign), and striated gallbladder wall (3). Only 13 cases of AAC associated with EBV infection have been reported all from 2007 and all had primary infection (4). Twelve of them were female similar to our case. Although direct invasion of EBV and/or irritation of the gallbladder from the bile stasis are considered to be possible causes of AAC during the course of acute EBV infection, the exact mechanisms have not been elucidated yet (5). Our patient had both EBV viremia and cholestatic hepatitis, so we thought that both of these pathogenetic mechanisms might be valid. Treatment of EBV is usually supportive and most cases of cholecystitis resolve spontaneously.

In conclusion, AAC may complicate the course of EBV infection and reactivation of EBV may be associated with AAC.

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