differentiated carcinoid and for the other three was poorly-differentiated (atypical) carcinoid (4-6).

Levendoglu et al. (5) reported two cases of cecal adenoendocrine carcinomas with poorly- differentiated endocrine component. A case with atypical carcinoid as the endocrine component was reported from Japan and that patient died with local recurrence nine months after surgery (4).

In conclusion, as more such rare tumors with a spectrum of morphological combinations are recognized and reported, their clinical behavior and prognosis will become better understood. However, strict criteria for diagnosis should be fulfilled and supported by immunohistochemistry to avoid an overlap with the poorly-differentiated adenocarcinoma.

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Epstein-Barr virus-induced severe hepatitis in an immunocompetent infant

İmmun yetmezliği olmayan bir süt çocuğunda "Epstein-Barr" virüsüne bağlı ağır hepatit

To the Editor,

Epstein-Barr virus (EBV) infection causes infectious mononucleosis (IM) in older children and adults, while it is often subclinical in young children. Mild or moderate elevations of transaminases are seen in almost all cases but they rarely exceed 1000 IU/L (1,2). Bilirubin is mildly increased in 45% of cases, whereas jaundice is seen in only about 5% of patients (3). Severe hepatitis and liver failure associated with EBV infection have been rarely reported and are mostly related to congenital or acquired immunodeficiency (4). Severe liver injury is the main cause of death among fatal cases of EBV infection (5). We report a case of EBV-induced severe hepatitis in an immunocompetent 10-month-old male infant.

A previously healthy 10-month-old boy presented with a one-week history of fever, vomiting and jaundice. On admission, his liver and spleen were

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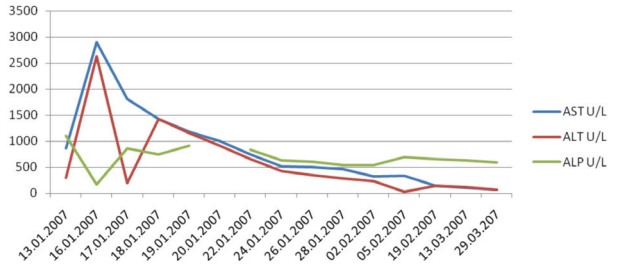

Figure 1. Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) levels during the course of the disease.

palpable 3.0 cm below the costal margin. Laboratory data revealed a normal leukocyte, hemoglobin and platelet count, but 20% atypical lymphocytes in peripheral blood smear. Other initial investigations were as follows: aspartate aminotransferase (AST) 861 IU/L, alanine aminotransferase (ALT) 293 IU/L, alkaline phosphatase (ALP) 1102 IU/L, gamma-glutamyl transpeptidase (GGT) 126 IU/L, total bilirubin 6.8 mg/dl (direct 5.4 mg/dl), and a prolonged prothrombin time (PT) of 29.7 sec. The levels of transaminases during the follow-up are shown in Figure 1. Abdominal ultrasonography revealed minimal hepatosplenomegaly and minimal ascites. Serum total immunoglobulins and lymphocyte sub-group analysis were in normal limits. EBV viral capsid IgM and IgG were both positive and serum EBV DNA level was 2551 copies/ml. Other serological and metabolic tests showed no abnormality. On the third day, transaminases and bilirubins had risen to peak level (AST 2898 IU/L, total bilirubin 15.2 mg/dl, direct bilirubin 8.8 mg/dl) and platelets were decreased to 51000/mm^3. The patient was managed supportively (i.e. infusion of fresh frozen plasma, glucose and HepatAmine). He was discharged on the 23rd day and followed at two-week intervals. The serum EBV DNA level became negative 2.5 months after admission to the hospital.

In our case, although it was not possible to perform histologic evaluation of the liver tissue, the positive serologic tests and high level of serum EBV DNA polymerase chain reaction (PCR) were compatible with primary EBV infection. Although it is rare, the possibility of EBV infection should be considered in children with acute liver failure even in the absence of an immunodeficient state. Serum EBV DNA load is a reliable indicator that can be used in the diagnosis and clinical follow-up.

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