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Case Report

Infectious mononucleosis hepatitis: A case-report

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Abstract

46-year-old Mexican-born who lived in Mexico City was admitted to the hospital for evaluation with a two-week history of fever, jaundice, and malaise. Physical examination he had cardiac murmurs. The liver was palpable 2 cm below the costal margins. Liver-function tests showed hypertransaminasemia. The patient had a high titer of anti-EBV IgM antibodies, but tests for all other antiviral antibodies were negative. The liver biopsy shows EBV latent membrane protein.

Key words: Epstein-Barr virus, liver, hepatitis.

Introduction

The Epstein-Barr virus (EBV) was discovered 36 years ago by electron microscopy of cells cultured from Burkitt's lymphoma tissue by Epstein, Achong, and Barr.[1] Four years later, in 1968, EBV was shown to be the etiologic agent of heterophile-positive infectious mononucleosis.[2] EBV DNA was detected in tissues from patients with nasopharyngeal carcinoma in 1970.[3] In the 1980s, EBV was found to be associated with non-Hodgkin's lymphoma and oral hairy leukoplakia in patients with the acquired immunodeficiency syndrome.[4,5] Since then, EBV DNA has been found in tissues from other cancers, including T-cell lymphomas and Hodgkin's disease.[6]

EBV is a member of the herpesvirus family. The viral genome is encased within a nucleocapsid, which is, in turn, surrounded by the viral envelope. Before the virus enters the B cell, the major envelope glycoprotein, gp350, binds to the viral receptor, the CD21 molecule (the C3d complement receptor),[7] on the surface of the B cell.

On the other hand, patients with infectious mononucleosis commonly have hepatic involvement but isolated symptomatic hepatitis is unusual. Although rare cases of liver failure have been reported, there is no evidence that Epstein Barr virus causes chronic liver disease.

Case-Report

46-year-old Mexican-born who lived in Mexico City was admitted to the hospital for evaluation with a two-week history of fever, jaundice, and malaise. His family history was unremarkable, and he had no history of liver disease. He had not drunk unpasteurized milk or consumed alcohol. He smokes for the last 10 years. He did not abuse drugs, and he was heterosexual. He had taken no medications recently.

The patient's temperature was 37.8 °C. He had cardiac murmurs. The liver was palpable 2 cm below the costal margins. No other abnormalities were noted on examination.

The laboratory results included an abnormal erythrocyte sedimentation rate (35 mm/hr); hemoglobin, 13.7 g per deciliter; mean corpuscular volume, 91.5 microm3; white-cell count, 11,600 per cubic millimeter, with 78.3 percent neutrophils and 10 percent atypical lymphocytes; and platelet count, 196,000 per cubic millimeter. The results of the liver-function tests were as follows: serum aspartate aminotransferase level, 121 U per liter (normal, 13 to 34); serum alanine aminotransferase level, 295 U per liter (normal, 8 to 44); alkaline phosphatase level, 123 U per liter (normal, 37 to 114); total bilirubin level, 1.01 mg per deciliter (normal, 0.5 to 1.3); serum albumin level, 2.88 g per deciliter; and a normal prothrombin time. The patient had a high titer of anti-EBV IgM antibodies, but tests for all other antiviral antibodies were negative. The cultures of blood, urine and stools were sterile. The liver biopsy shows EBV latent membrane protein *(Figure 1)*. A diagnosis of infectious mononucleosis with hepatitis was made. The patient's jaundice and malaise improved with supportive therapy, and he was discharged from the hospital. Three weeks later, however, fever reappeared, accompanied by chills. His temperature was 40 °C, and he was rehospitalized. The second bout of fever, this time accompanied by chills. The chest film showed small bilateral pleural effusions. Abdominal, and abdominal computed tomography revealed cardiomegaly. Echocardiography is urgently indicated. The hemodynamic decompensation calls for urgent treatment, we indicated broad-spectrum antibiotics.

The patient was transferred to the intensive cardiac care unit, and treatment was begun.

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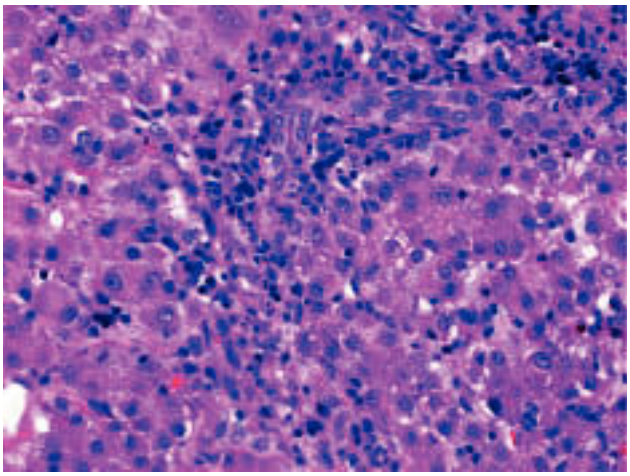

Figure 1. The liver biopsy shows EBV latent membrane protein.

Discussion

Epstein-Barr virus is a ubiquitous virus associated with a variety of different diseases and disorders. The manifestations of Epstein-Barr virus-associated diseases or disorders within the liver, which involve a broad spectrum of histologic and clinical features, ranging from hepatitis through lymphoproliferative disorders to lymphoma. An important aspect of Epstein-Barr virus expression and infection is the biology of the Epstein-Barr virus. Documentation of infection can be performed using serology to detect the interaction of Epstein-Barr virus with the immune system, and the detection of EBV proteins and use of molecular biologic techniques to identify the presence of EBV RNA, and DNA sequences. Of particular utility are in situ hybridization, Southern blot analysis, and polymerase chain reaction as diagnostic methods to identify specific RNA or DNA sequences. The histopathologic findings present in liver associated with Epstein-Barr virus have reviewed extensively.[8]

The immunopathogenetic mechanisms, which are responsible for liver damage in EBV infection, are not exactly known. EBV receptors have been found on Blymphocytes, but not on hepatocytes,[9] although EBV infection of hepatocytes has recently been demonstrated;[10] as a result, EBV does not seem to have a direct cytopathic effect. Infected B-cells start to proliferate[11] and the explosive lymphoproliferation evoked by EBV is normally down-regulated by the immune defence response by endogenous interferon, suppressor T-cells, natural killer cells, EBV-specific antibodies and cytotoxic T-cells.[12] Congenital or acquired disorders of cellular immune response seem to be responsible for the lesions in liver and bone marrow in fatal EBV infections.[13,14] T-cells and natural killer cells found in liver parenchyma in such cases would be implicated in liver cell necrosis.[13] Although EBV infection is a benign disease, may occasionally be fatal, particularly in children. EBV infection is rare in elderly subjects and appears to have a self-limited course.[15]

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