Acute Infectious Mononucleosis Complicated by Splenic Infarcts

Fadi Al Akhrass*, Nicole Rex, Asim Kichloo, Larry Zhao and Muhannad Antoun

1Infectious Diseases and Internal Medicine Departments, Pikeville Medical Center, USA
2Kentucky College of Osteopathic Medicine, University of Pikeville, USA

Case Blog

A 45-year-old white female presented with a four-week history of worsening abdominal pain, sore throat, fever, and night sweats. She described a week-long history of nausea, and left sided pleurisy. Laboratory workup performed two-week previously showed leucopenia, transaminitis, and reactive monospot test. Symptomatic treatment was instituted without improvement. She had a past medical history of diabetes mellitus type 2, morbid obesity, hypertension, and dyslipidemia. She had no significant social history. On admission, she was slightly tachycardic with pulse of 94/ min, blood pressure of 113/81 mmHg, respiratory rate of 20/minute, and temperature of 98.7 °F. She had no cervical lymphadenopathies or muco-cutaneous lesions. Cardiac and lung examinations were normal. The patient’s abdomen was obese, with left upper quadrant tenderness, but no peritoneal irritation signs. Oganomegaly was not appreciated. Peripheral edema and calf tenderness were absent. Routine laboratory results showed a white blood cell count of 10.1×10³/μL with 77% lymphocytes, and elevated levels of aspartate aminotransferase, alanine aminotransferase, and lactate dehydrogenase (293, 483, and 475 IU/L, respectively). A contrasted-enhanced-computed tomography of the abdomen showed hepatosplenomegaly and numerous focal hypodense regions in the spleen consistent with splenic infarcts (Figure 1). Multiple blood cultures showed no growth, and echocardiography excluded endocarditis. Epstein Barr (EBV) viral capsid antigen immunoglobulin M (VCA-IgM) antibodies were at high levels and detected EBV PCR. Other causes of splenic infarction were excluded. Splenectomy was performed due to persistent symptoms.
and concern of hematologic etiology. Immunohistochemical stain was positive for EBV in few splenic cell nuclei (Figure 2). Her post-operative stay was uneventful and she received appropriate post-splenectomy vaccinations.

We assume the cause of splenic infarction was insufficient blood flow to oxygenate the entire spleen during its acute enlargement. This case illustrates the need for EBV to be thought about early in the differential diagnosis for splenic infarction.