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SPLENIC INFARCTIONS IN ACUTE EPSTEIN-BARR VIRUS INFECTION: A RARE BUT POSSIBLY UNDERDIAGNOSED COMPLICATION IN ADULTS

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ABSTRACT

Background: Although splenomegaly is a common finding in Epstein-Barr virus (EBV) infection, splenic infarction is rarely reported and may be under-recognised, especially in adults. Neurological complications such as aseptic meningitis are also uncommon but documented. The simultaneous occurrence of both complications in the context of primary EBV infection is exceptional.

Case description: We describe the case of a 56-year-old previously healthy man who presented with fever and abdominal pain. Abdominal computed tomography (CT) revealed splenomegaly and multiple wedge-shaped splenic infarcts. During admission, the patient developed transient disorientation, and cerebrospinal fluid analysis was consistent with aseptic meningitis. Serological and molecular testing confirmed acute EBV infection in blood, pharyngeal swab and cerebrospinal fluid. No underlying prothrombotic condition was identified. The patient recovered completely with supportive care, without the need for antiviral treatment or anticoagulation.

Conclusions: Splenic infarction is a rare but possibly underestimated complication of primary EBV infection, particularly in adults. Awareness of this association can help avoid unnecessary investigations or treatments. In selected patients, conservative management without anticoagulation may be appropriate.

KEYWORDS

Epstein-Barr virus, splenic infarction, aseptic meningitis, infectious mononucleosis

LEARNING POINTS

- Splenic infarctions are a rare but potentially severe complication of primary Epstein-Barr virus infection.
- EBV-related mononucleosis in adults may present with atypical features, including neurological involvement such as aseptic meningitis.
- Awareness of these complications may facilitate earlier diagnosis and appropriate supportive management.

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INTRODUCTION

Epstein-Barr virus (EBV) is a ubiquitous herpesvirus and the primary cause of infectious mononucleosis, classically characterised by fever, pharyngitis and lymphadenopathy. While the disease usually follows a benign course, especially in younger patients, adult presentations can be atypical and complicated by rare manifestations, including splenic infarction and neurological involvement such as aseptic meningitis.

CASE DESCRIPTION

A 56-year-old male with a medical history of hypertension and hypertriglyceridemia, with no known prior EBV infection, presented with a two-week history of malaise, persistent lower back and abdominal pain, and fever up to 39ºC. Physical examination was notable for fever but otherwise unremarkable, with no neurological deficits or lymphadenopathy.

Initial laboratory studies showed elevated liver enzymes (alanine aminotransferase 190 U/l, aspartate aminotransferase 194 U/l), hyperbilirubinaemia (total bilirubin 1.9 mg/dl), increased lactate dehydrogenase (805 U/l) and inflammatory markers (c-reactive protein 15 mg/dl, erythrocyte sedimentation rate up to 120 mm, procalcitonin 0.6 ng/ml). Peripheral blood smear revealed reactive lymphocytes. Coagulation was normal except for elevated D-dimer (1,081 ng/ml) and fibrinogen (500 mg/dl).

Chest radiograph was normal. An abdominal computed tomography (CT) scan revealed splenomegaly (15 cm) with four wedge-shaped hypodense areas consistent with splenic infarctions, as well as mediastinal and hepatic hilar lymphadenopathy *(Fig. 1)*. An MRI scan of the spine was normal.

During hospitalisation, the patient developed confusion and disorientation. Neurological examination showed a positive Babinski reflex; a brain CT was unremarkable. Lumbar puncture revealed lymphocytic meningitis (48 white blood cell count, 96% lymphocytes, glucose 44 mg/dl, proteins 124 mg/dl). EBV serology (immunoglobulins IgM and IgG) was positive, and a polymerase chain reaction blood test detected EBV DNA in the pharyngeal swab, blood and cerebrospinal fluid. All other infectious studies were negative.

The patient received supportive care without antiviral or anticoagulation therapy. He progressively improved, and all symptoms and laboratory abnormalities resolved after three weeks.

DISCUSSION

Splenic infarction is an uncommon complication of infectious mononucleosis, but one that seems to be increasingly recognised due to wider use of abdominal imaging. Most published cases involve young, otherwise healthy individuals, and are typically discovered when left upper quadrant pain prompts further investigation. However, the true incidence may be underestimated. In a recent review, only a small number of EBV-associated splenic infarcts had been reported until 2010, but the number has increased significantly in the last decade[1].

In this case, the diagnosis of EBV infection was made in a 56-year-old man, an age at which primary infection is rare. Serologic and molecular findings were consistent with acute primary infection, suggesting late exposure despite an otherwise normal immunocompetent background. While uncommon, delayed EBV seroconversion has been reported, particularly in individuals with limited early-life exposure to the virus or in certain geographic or socioeconomic contexts[2]. The absence of known comorbidities or immunosuppression in our patient supports the idea that age alone should not rule out primary EBV infection.

The pathogenesis of splenic infarction in this context remains poorly understood, but several mechanisms have been proposed. These include increased splenic metabolic demand due to lymphoid hyperplasia, transient hypercoagulability (sometimes associated with antiphospholipid antibodies or reduced protein C/S levels), or local vascular compression due to rapid splenic enlargement[3-5]. Some cases have also reported anatomical anomalies that could predispose to vascular compromise[6]. In our patient, no underlying thrombophilia or embolic source was found. Coagulation tests were largely normal, and the clinical course was favourable without specific therapy.

Whether anticoagulation is indicated in these cases is controversial. Most published reports describe conservative management unless there is evidence of ongoing thrombosis, embolic risk, or coexisting prothrombotic conditions. Some authors have used prophylactic anticoagulation empirically[5],

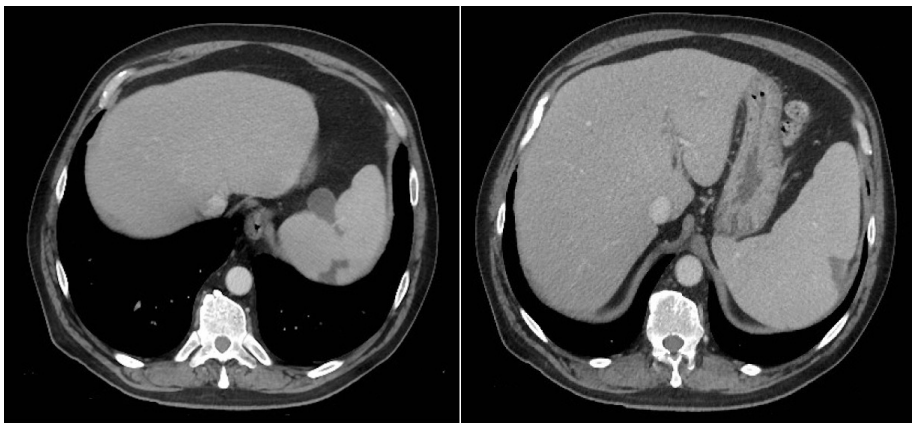


Figure 1. Abdominal CT scan showing splenomegaly and multiple wedge-shaped hypodense areas consistent with splenic infarctions.

while others have managed patients with observation alone. In our case, given the absence of thrombophilia and the patient's clinical improvement, we opted for supportive care without anticoagulation, with close follow-up. This approach aligns with other reports where spontaneous resolution occurred without complications such as splenic rupture or haemorrhage[4,7].

Interestingly, this case also included neurological involvement in the form of transient encephalopathy and cerebrospinal fluid findings consistent with aseptic meningitis, with EBV DNA detected by a polymerase chain reaction blood test. While rare, central nervous system involvement has been described in 1–5% of EBV infections, particularly in adults[8]. However, in this patient, the splenic infarctions were the initial and most clinically relevant finding. The meningitis likely represents an additional, albeit less prominent, complication.

Overall, this case underscores the importance of considering EBV infection in the differential diagnosis of splenic infarction in adults, even in the absence of classical mononucleosis features. It also supports the idea that these events may be more common than previously thought and often resolve with conservative management. Increased clinical awareness may help avoid unnecessary interventions and facilitate appropriate supportive care.

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