Splenic Infarction after Epstein-Barr Virus Infection: Case Report

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Abstract

We describe a 24-year-old man who was admitted for evaluation of left side abdominal pain. He described asthenia associated with night sweats and an abdominal pain radiating to his left shoulder. He was not febrile. Cervical nodes were palpable symmetrically with exudative pharyngitis. Ultrasound examination and abdominal CT showed a splenomegaly with multiple infarcts, without artery aneurysm, and without artery or vein thrombosis. The presence of high levels of IgM and IgG antibodies directed against the Epstein Barr virus Viral Capsid Antigen (VCA) and a high level of viral charge led to acute infectious mononucleosis. Splenic infarction is a relatively uncommon diagnosis. The etiologies are represented by a thromboembolism origin, by a hemopathy, by a digestive cause, by an infectious cause, by a chronic autoimmune disorder and by a coagulation disorder. It is considered a rare presentation of acute infectious mononucleosis. Currently, physiopathology mechanisms are still unclear and likely multifactorial.

Keywords: Epstein-Barr virus; Infectious mononucleosis; Splenic infarction

Introduction

Epstein-Barr virus is a ubiquitous virus, affected 95% of the world population [1]. The primary infection at Epstein-Barr virus is commonly without symptoms. However, it can be presented with the classic triad of pharyngitis, fever, and lymphadenopathy [2]. Splenic infarction represents a very rare complication of infectious mononucleosis. We describe a case of splenic infarction during infectious mononucleosis in an otherwise healthy individual.

Case Presentation

A 24-year-old boy, immunocompetent, was admitted with a 7 days history of left side abdominal pain. He described asthenia associated with night sweats and an abdominal pain radiating to his left shoulder, and increasing upon deep inspiration. The only past medical history was oral aphthous in childhood. He took no treatment, and had no cardiovascular risk factors. He didn't travel recently but had a new girlfriend. On admission, he was not febrile. Cervical nodes were palpable symmetrically with exudative pharyngitis. There were no skins, joint or ophthalmic signs. The only past medical history was oral aphthous in childhood. He took no treatment, and had no cardiovascular risk factors. He didn't travel recently but had a new girlfriend. On admission, he was not febrile. Cervical nodes were palpable symmetrically with exudative pharyngitis. There were no skins, joint or ophthalmic signs.
the increases demand. Also, these mechanisms might contribute to infarction in splenic areas vulnerable to ischemia. Second, infection can cause thrombosis through various mechanisms [11]. In the presence of inflammatory conditions, increased cytokine production due to sepsis disrupts the coagulation system [19]. The action of pro-inflammatory mediators activates platelets and leads to platelet adhesion [20]. Moreover, these pro-inflammatory molecules reduce the levels of anticoagulant proteins, and compromise the functioning of the elements of anti-coagulation mechanism [21,22]. Third, the presence of a transient hypercoagulable state during IM has been proposed [23]. In fact, antiphospholipid antibodies (aPLs), lupus anticoagulant, and reduced protein C and protein S have been reported in several patients with IM associated splenic infarction [17,24]. Studies demonstrated the presence of transient aPLs during an acute EBV infection, as in our patient [25,26]. An association between infections and aPL has been reported in several epidemiologic and experimental studies [27]. However, there is no consensus in the literature regarding the management of aPL carriers during EBV. An individual based approach should be practiced after considering additional risk factors for thrombosis. Further studies are needed to clarify the pathogenesis of splenic infarction associated with IM due to EBV infection.

**Conclusion**

We report a case with splenic infarction as a rare complication of IM due to EBV infection. When splenic infarct is observed on imaging studies, infectious etiologies including EBV should be considered. Physiopathology mechanisms are still unclear and likely multifactorial including intra-splenic structural changes and coagulopathy.

**References**


