Annals of Clinical Case Reports

Case Report
Published: 25 Nov, 2021

Splenic Infarction after Epstein-Barr Virus Infection: Case Report

*Ulysse Turrian**

Department of Vascular Medicine, Toulouse University Hospital, France

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 lest 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 of inflammation. Left side pain was reproduced by palpation. Routine laboratory results showed an inflammatory syndrome with CRP at 82 mg/l (N<5 mg/l) associated with leucocytosis 26.7 G/L (with a normal range of 4 G/L to 10 G/L), with 49% lymphocytes, including hyperbasophilic lymphocytes. There were elevated levels of aspartate aminotransferase, alanine aminotransferase (AST 190 IU/L and ALT 578 IU/L, with a normal range of 0 to 50), Lactate Dehydrogenase (LDH 493 IU/L, with a normal range of 135 IU/L to 225 IU/L), alkaline phosphatase (404 IU/L, with a normal range of 40 to 129), and gamma- Glutamyltransferase (gGT 452 IU/L, with a normal range of 0 to 60). Ultrasound examination and Abdominal CT showed a splenomegaly with multiple infarcts, without artery aneurysm, and without artery or vein thrombosis (Figure 1, 2). Serologic viral screening for Hepatitis B and C Viruses (HBV, HCV), Human Immunodeficiency Virus (HIV), Cytomegalovirus (CMV) and EBV, were negative. 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. Other causes of splenic infarction were excluded. Electrocardiogram, cardiac echography, blood cultures, rheumatoid factor, thyroid function test, urinalysis were all normal. Thrombophilic screening showed a positive lupus anticoagulant. Anticardiolipin, anti-B2-glycoproteine-1 antibodies were negative, and protein C, protein S, antithrombin were normal. The patient was treated symptomatically. Anticoagulation treatment was stopped at 7 days, after the complete cardiovascular etiology screening. Abdominal pain decreased and aminotransferase and LDH levels gradually normalized. Lupus anticoagulant was controlled negative. No abdominal

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Correspondence:
Ulysse Turrian, Department of Vascular Medicine, Toulouse University Hospital, Toulouse, France,
E-mail: turrian.u@chu-toulouse.fr

Received Date: *29 Sep 2021*
Accepted Date: *10 Nov 2021*
Published Date: *25 Nov 2021*

Citation:
Turrian U. Splenic Infarction after Epstein-Barr Virus Infection: Case Report. Ann Clin Case Rep. 2021; 6: 2045.

ISSN: 2474-1655

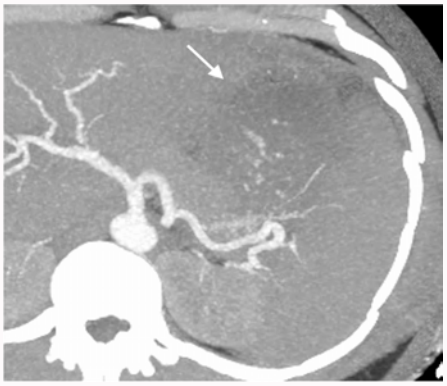

Figure 1: Contrast enhanced CT demonstrating a splenic infarct.

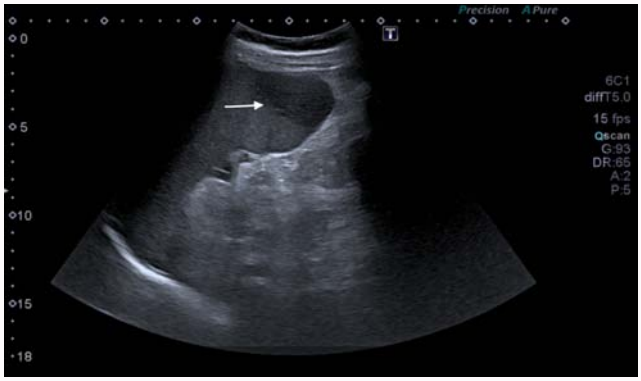

Figure 2: Ultrasound examination showing splenomegaly with a splenic infarct.

imaging was performed routinely.

Discussion

Splenic infarcts are infrequent [3]. The etiologies are represented by a thromboembolism origin (Cardiac, aorta), by a hemopathy (Sickle cell trait, lymphoma, leukemia), by a digestive cause (liver cirrhosis, pancreatitis, cancer), by an infectious cause (endocarditis, CMV, EBV), by a chronic autoimmune disorder (systemic lupus erythematosus) and by a coagulation disorder [4,5]. There are sometimes cocaine use and no etiology. Thromboembolism origin and hematologic disease must be searched in priority. Spontaneous splenic infarction is a rare complication of infectious mononucleosis. Its exact incidence is not known, and only 32 cases have been so far reported in medical literature between 1961 and 2021 [6-12]. In our case, causes of splenic infarction were excluded, except an acute infectious mononucleosis. The link between Epstein-Bar virus and splenic infarction is few reported in literature. That why, physiopathology mechanisms are still unclear and likely multifactorial. Many were proposed. First, splenomegaly is associated with histological modifications and leads to an alteration of the splenic microcirculation [13-15]. In the histopathologic findings of splenic rupture associated with IM, the splenic cord showed excessive hypercellularity, which compacts the sinus structure and interrupts blood flow [16]. Hypercellularity in splenic pulp will increase oxygen consumption. In addition, an increased level of circulating immune complexes due to B cell proliferation, promoting leukocyte aggregation and adhesiveness, has been associated with splenic infarction with EBV [17]. An alteration of splenic microcirculation creates hypoxemia [18]. Arterial blood supply may be insufficient for 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 EBD 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 aPLs 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.

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