Case report

Spleenic infarcts as a rare manifestation of parvovirus B19 infection

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**Abstract**

**Introduction:** Human parvovirus B19 is a DNA virus most known for causing erythema infectiosum in children, and polyarthropathy or transient aplastic crisis in adults. However, various unusual clinical manifestations have also been reported in association with it. We describe a young patient who presented with splenic infarcts as a rare complication of B19 infection.

**Case report:** A 33-year old previously healthy man was admitted to our hospital because of a 5-day history of fever and headache. Imaging studies revealed two splenic infarcts. Endocarditis was ruled out, whereas serologic testing for B19 was indicative of acute infection.

**Discussion:** To our knowledge, three cases of thromboembolism in the setting of B19 infection have been reported up to now, including one occurrence of splenic infarction. These events were attributed to the development of a transient antiphospholipid antibody syndrome. In contrast, our patient did not have elevated titers of antiphospholipid antibodies.

**Conclusions:** Spleenic infarcts can be an atypical presentation of B19 infection. Parvovirus B19 may induce thromboembolic events, even in the absence of antiphospholipid antibodies.

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**Introduction**

Human parvovirus B19 is most known for causing erythema infectiosum in children, transient red cell aplasia which can be significant in those with hemolytic anemia or polyarthropathy in adults [1,2].

**Case report**

A 33-year old man was admitted to our hospital because of a 5-day history of continuous fever up to 39 °C, and diffuse headache. He was unmarried and lived with his parents in Thirasia Island near Santorini, working as a builder, was sexually active and consistently used condoms. He did not smoke, use illicit drugs, or misuse alcohol, had no pets or exposures to animals, and had never traveled abroad. The man reported no recent tick or mosquito bites, no ingestion of unpasteurized milk or milk products, and no sick contacts and had received all routine childhood vaccinations, had no known medical problems, and did not take any medications. His family history was negative for tuberculosis, cancer or autoimmune disease.

On physical examination, the patient appeared well and was in no acute distress; he was alert and communicative. His temperature was 39 °C, heart rate 73 beats per minute, blood pressure 120/80 mmHg, respiratory rate 16 breaths per minute, and oxygen saturation 99% while breathing ambient air. He had no synovitis, rash, photophobia, or nuchal rigidity. His abdomen was soft, with mild tenderness in the left upper quadrant on deep palpation. There was no hepatomegaly or splenomegaly, and no cervical, axillary, or femoral lymphadenopathy. Heart murmurs were not detected.

Laboratory analyses revealed normocytic, normochromic anemia (hemoglobin level, 12 g per deciliter), with a low reticulocyte count (0.3%), leukopenia (white-cell count, 3420 per cubic millimeter, with 68% neutrophils, 25% lymphocytes, 7% monocytes, and no eosinophils or band forms), thrombocytopenia (platelet count, 87,000 per cubic millimeter), and elevated levels of hepatic aminotransferases (aspartate transaminase 138 U per liter, alanine transaminase 111 U per liter), lactate dehydrogenase (1383 U per liter), creatine phosphokinase (1019 U per liter), and C-reactive protein (112 mg per liter). Clotting times (dilute prothrombin time and activated partial-thromboplastin time) were not prolonged. Urinalysis was normal. Multiple blood cultures were sterile.

An abdominal ultrasound examination revealed a mildly enlarged spleen, with two peripheral wedge-shaped hypoechoic lesions, which were characteristic of splenic infarcts. A computed
tomography (CT) scan of the abdomen (Fig. 1), obtained after the administration of intravenous and oral contrast material, confirmed the results of ultrasonography, demonstrating splenomegaly (with the spleen measuring 15.4 cm in the cranio-caudal dimension), and two wedge-shaped areas of hypoenhancement in the periphery of the spleen, features that were consistent with splenic infarcts. No other organ abnormality was observed. An electrocardiogram showed sinus rhythm. Transesophageal echocardiography showed no valvular vegetations.

Hepatitis A immunoglobulin M (IgM) antibody, hepatitis B surface antigen and core IgM antibody, a polymerase chain reaction (PCR) assay for hepatitis C viral RNA, and serologic tests for cytomegalovirus (CMV), herpes simplex virus, coxsackie A virus, echorovirus, human immunodeficiency virus, Rickettsia species, Coxiella burnetii, Brucella melitensis, and Leishmania were negative, as was real-time reverse transcriptase PCR for H1N1 influenza A virus. Antibody tests for Epstein–Barr virus (EBV), coxsackie B virus, and Toxoplasma gondii showed evidence of past infection. Parvovirus B19 IgM antibodies were positive, while parvovirus B19 immunoglobulin G (IgG) antibodies were negative, indicating an acute infection. Titers of anticitrullinophilin and anti-β2-glycoprotein I antibodies were within normal limits. The patient received symptomatic treatment with antipyretic and analgesic agents. Over the following few days, his clinical and laboratory status gradually improved. On the fifth hospital day, his fever resolved. He was discharged home on the tenth hospital day. Whether he developed parvovirus B19-specific IgG antibodies during the convalescent phase remained unknown, because the relevant test could not be performed in his island.

Discussion

Splenic infarction occurs as a result of the occlusion of the splenic artery or its branches due to thrombosis or embolism [3]. The most common etiologies are hematologic diseases, including myeloid disorders, lymphomas, hemoglobinopathies, and hypercoagulable states; cardioembolic events; viral infections; and trauma [4,5]. The majority of patients present with pain in the left upper quadrant or left flank or both. However, as in this case, a surprising percentage of patients have no symptoms localized to the splenic area. The most frequent laboratory abnormality is elevated lactate dehydrogenase levels, as seen in our patient [6].

A major concern, when evaluating a patient with fever accompanied by splenomegalgy and splenic infarcts, is bacterial endocarditis. This diagnosis was ruled out by sterile blood cultures and normal transesophageal echocardiography as well as recovery without antimicrobials. Additionally, the acuity of our patient’s illness, and his rapid and complete recovery argued against hematologic malignancies, being most consistent with a viral infection. The viral infections that have been associated with splenic infarcts are infectious mononucleosis [7,8] and acute CMV infection [9]. Both conditions were excluded on the basis of negative serologic testing.

Our patient did not present with any of the classic syndromes associated with PVB19 infection. Nevertheless, he had a remarkably low reticulocyte count. Asymptomatic transient reticulocytopenia is a hallmark laboratory feature of PVB19 infection. PVB19 replicates primarily in erythroid progenitors. Virus-induced cytotoxicity results in cessation of red cell production. In individuals without hemolytic anemia, this transient arrest of red cell production will lead to only a minimal drop in hemoglobin levels but in patients with increased destruction of red cells, who depend on continual rapid production of erythrocytes (hemoglobinopathies or hemolytic anemias), this suppression of erythropoiesis can cause a transient aplastic crisis with severe anemia [1,10].

A wide range of unusual clinical manifestations have been reported in association with PVB19 infection, including vasculitis, myocarditis, glomerulonephritis, peripheral neuropathy, meningitis and encephalitis, immune thrombocytopenia, and hemolytic-uremic syndrome [11]. Three cases of thromboembolism have also been described, to our knowledge: a man with a splenic infarct [12], a woman with multiple pulmonary emboli [13], and a woman with right pulmonary arterial and superior mesenteric venous thromboses [14]. Similar to our case, all these occurrences involved young previously healthy patients who recovered fully. In regards to pathogenesis, they were attributed to the development of a transient antiphospholipid antibody syndrome. Our patient did not have elevated titers of antiphospholipid antibodies. Consequently, other unknown mechanisms also account for the thromboembolic phenomena observed in the setting of PVB19 infection.

Conclusions

Splenic infarcts may be an atypical presentation of PVB19 infection. PVB19 infection should be considered in the differential diagnosis of fever accompanied by splenic infarcts in immunocompetent young adults. Clinicians must be aware of PVB19 propensity to induce thromboembolic events, regardless of the presence of antiphospholipid antibodies.

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