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Case Report

Spontaneous splenic rupture and multiple lung embolisms due to cytomegalovirus infection: a case report and review of the literature

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SUMMARY

A 53-year-old woman presented with a spontaneous splenic rupture. The splenic rupture was considered a complication of a primary cytomegalovirus (CMV) infection as were multiple pulmonary embolisms. CMV infections are common but are most often asymptomatic, and to our knowledge only 15 cases complicated with splenic rupture have been published.

1. Introduction

Cytomegalovirus (CMV) infections are common worldwide. Most primary infections are asymptomatic, but the virus can cause mononucleosis, a high prolonged fever, hepatitis, or pneumonitis. Splenomegaly has been reported in about a third of patients with CMV mononucleosis during a primary infection.[1] However, splenic rupture is a rare complication.[2] The case of a previously healthy woman with CMV infections and a

2. Case report

A 53-year-old woman presented to the emergency department with a 24-h history of severe pain in the upper abdomen and vomiting. No history of trauma was present, although she reported having fainted several times at home due to the pain. Her previous medical history included Graves' thyrotoxicosis and a thyroidectomy for a toxic goiter, and she was taking levothyroxine. On initial clinical examination she was found to be pale and in pain, with tenderness in her upper abdomen on palpation; no bowel sounds were noted on auscultation. A digital rectal examination was normal. Her vital signs were within normal limits, except for a heart rate of 110 beats/min. Laboratory tests showed the following: hemoglobin 77 g/l, white blood cell count 15.6×10^9/l, and C-reactive protein 33 mg/l.

A computed tomography (CT) scan of the thorax and abdomen showed free fluid in the abdomen, measuring as blood in Hounsfield units, and a splenic rupture (Figure 1). The patient

day 4 of her admission she suffered from tachypnea and tachycardia. A CT angiography of the thorax was performed, which showed multiple lung embolisms. This was confirmed with scintigraphy of the lungs. She was treated with low molecular weight heparin. The patient was discharged on day 11 post admission with anticoagulation therapy planned for 6 months.

The results of the serological testing strongly indicated a primary CMV infection. CMV was detected by PCR technique in two serum samples taken during the hospitalization (1000 copies/ml). Both CMV IgG and IgM were detected in the first serum sample, and rising CMV IgG and declining CMV IgM levels were seen 1 month later. The CMV IgG antibodies in the first sample were of low avidity, as typically seen during the first weeks after seroconversion. Furthermore, seroconversion for CMV IgG could be established to have taken place from 2010 to 2012. However, Epstein–Barr virus (EBV) viral capsid antigen IgM antibodies were also detected in the first serum sample. This reactivity was

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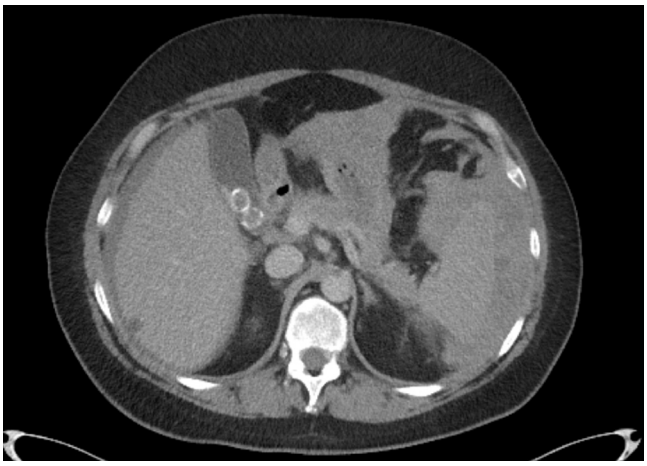

Figure 1. CT scan of the abdomen showing a splenic rupture and free intraperitoneal fluid.

considered as unspecific reactivity or cross-reactivity against CMV, as it was unaltered over time and EBV IgG was already present in 2010. EBV was not detected by PCR in any sample tested.

3. Discussion

A spontaneous splenic rupture is a serious and potentially life-threatening condition. It is a well-known complication of primary EBV infection, but splenic rupture is not mentioned in the textbooks of infectious diseases or virology in connection with CMV.

In a systematic review of 845 patients with an atraumatic splenic rupture, 137 or 14.8% were caused by a viral infection, most commonly infectious mononucleosis (EBV) and malaria. CMV infection was the cause in 13 cases.[2] For reasons that are not known, spontaneous splenic rupture is more often reported in men, with a male to female ratio of 2:1. Of note we did not find any reports regarding children or adolescents. All previously reported patients have been immunocompetent adults, as was the patient reported here. Two of the previously reported cases had a possible predisposing factor for splenic rupture during CMV infection. A 38-year-old male reported by Horwitz et al. had a history of malaria infection,[3] and a 29-year-old male reported by Maillard et al. suffered from a pyruvate kinase deficiency.[4] Both of these diseases can cause splenomegaly.

In addition to the splenic rupture, the disease in our patient was complicated with multiple lung embolisms. Venous thrombosis with or without associated pulmonary embolism has been reported in case reports and small case series of immunocompetent patients with acute CMV infections, often in the absence of known risk factors for hypercoagulability.[5] The underlying mechanism is unknown. We considered the pulmonary embolisms in our patient to have been caused by her primary CMV infection as she had no other risk factors.

CMV is a member of the herpes virus family and causes life-long infections. CMV infections are common worldwide. In general, the prevalence of infection increases with age, and in developing countries the prevalence is higher (up to 100%) and the infections are acquired earlier in life than in developed countries. In Scandinavia, more than half of all young adults have been infected with CMV. Most primary infections are asymptomatic, but the virus can cause mononucleosis, a high prolonged fever, hepatitis, or pneumonitis. Whether symptoms are more common in adults than in children is not known.

Splenomegaly has been reported in about a third of patients with CMV mononucleosis during a primary infection.[1] However, a splenic complication with splenic rupture or splenic hematoma associated with CMV is rare. We have only found 15 published cases, nine of these in English

Non-operative management, as in our case, should be the treatment of choice in hemodynamically stable patients. Splenectomy should be reserved for patients who are hemodynamically unstable on admission and who do not respond to resuscitation, and for those in whom conservative treatment fails. However, splenectomy is associated with potential adverse effects, such as an increased risk of severe bacterial infections, especially infections caused by encapsulated bacteria, including Streptococcus pneumoniae, Haemophilus influenzae, and Neisseria meningitidis. In the previously mentioned review by Renzulli et al., 114 out of 774 patients with atraumatic splenic rupture, irrespective of the etiology, were treated conservatively, including seven with a splenic arterial embolization. In 16 of those patients, conservative management failed and the patients underwent surgery.[2] The mortality for the published cases is 0%, but in the review by Renzulli et al., the mortality rate including splenic rupture related to all infectious causes was 8.7%.[2]

In conclusion, a primary CMV infection can cause a splenic rupture or a subcapsular hematoma without a history of trauma in immunocompetent adults. Conservative management is the preferred treatment for these patients.

Conflict of interest: None declared.

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