



Superior Mesenteric Vein Thrombosis with Pulmonary Embolism: A Rare Presentation of Acute Cytomegalovirus Infection

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Acute cytomegalovirus infection usually presents as mononucleosis syndrome. We describe a 28 year old immunocompetent patient who presented with superior mesenteric vein thrombosis and subsequent pulmonary embolism, a rare manifestation of an acute cytomegalovirus infection.

Patient Description

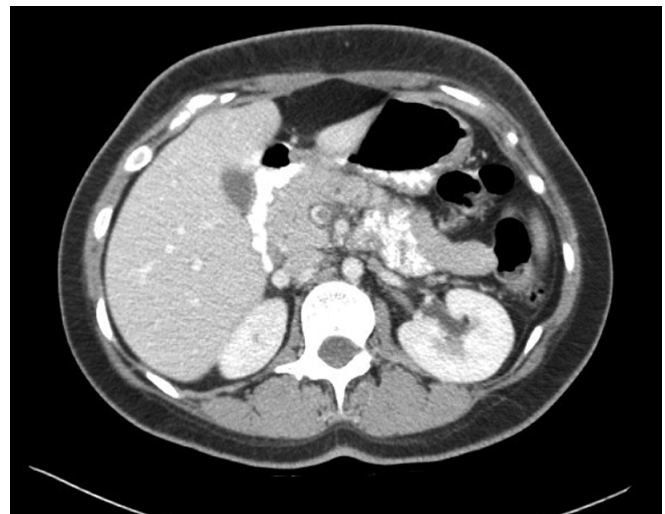
A 28 year old woman was admitted with fever and flank pain. Two weeks previously she developed a high fever and pain localized to her right flank and back accompanied by vomiting. Her primary physician prescribed empiric antibiotic treatment but there was no improvement. An abdominal ultrasound performed 14 days after the onset of fever demonstrated a small right perirenal fluid collection. She was then admitted to the hospital for further workup.

She was in general good health and had two normal pregnancies, both of which had terminated in cesarean sections. She was not obese and did not smoke. Mild combined dyslipidemia was controlled by diet alone. Her only regular medications were oral contraceptive pills. Her family history revealed that her mother took warfarin after she had had a stroke at a young age. Thrombophilia workup was negative. A grandmother suffered from pulmonary embolism while receiving treatment for cancer, and one uncle had a postoperative deep vein thrombosis.

Physical examination was normal except for mild tenderness to deep palpation

in her right flank. Urinalysis was normal. The leukocyte count was 8000/ μ l. Mild lymphocytosis (4320/ μ l, 49%) was present. Biochemistry screen, including liver enzymes, was normal. Erythrocyte sedimentation rate after 1 hour was 72 mm and C-reactive protein was elevated (143 mg/L, normal 0–8).

A contrast-enhanced abdominal computed tomography showed a small amount of free peritoneal fluid around the liver, in the right paracolic gutter and in the pelvis. Enlarged lymph nodes were seen in the portocaval space. Infiltration of abdominal fat was noted. An irregular soft tissue mass measuring 7.5x2.5x2 cm was encircling the superior mesenteric vein. An 8 mm filling defect was seen in the proximal part of this vein near its junction with the splenic vein [Figure]. While the patient was awaiting a needle biopsy of the mass, she suddenly developed severe acute dyspnea. CT angiography of the chest demonstrated multiple bilateral segmental filling defects compatible with pulmonary embolism.



Axial section from a contrast-enhanced CT of the abdomen demonstrating a filling defect in the superior mesenteric vein with a surrounding soft tissue mass

She was transferred to the intensive care unit and prescribed enoxaparin, which led to rapid clinical improvement. Tests for anticardiolipin antibodies and beta-2-glycoprotein antibodies were negative. Protein C activity, free protein S antigen level, and anti-thrombin III levels were normal. Polymerase chain reaction for Factor V R506Q and prothrombin G20210A were also negative. She was heterozygous for the MTHFR C677T mutation. Partial thromboplastin time was slightly prolonged (38–49 seconds, normal values 20–36 sec). Lupus anticoagulant screening test was positive at 82 sec (normal 33–44 sec). A confirmation test performed by the dilute Russell viper venom time

(LAC-RVVT) was 1.56 (normal ratio 0–1.3). Cytomegalovirus-immunoglobulin M antibodies were found and further tests showed low avidity of antibodies, compatible with an acute infection. PCR for cytomegalovirus was weakly positive. Subsequent tests showed a rise in IgG levels. A repeat CT study of the abdomen 3 weeks later showed partial recanalization of the superior mesenteric vein and complete resolution of the mass.

Comment

We describe a 28 year old immunocompetent patient who presented with superior mesenteric vein thrombosis and subsequent pulmonary embolism as a manifestation of an acute cytomegalovirus infection. CMV can cause vasculopathy and thrombosis in immunocompromised patients [1] and, rarely, also in immunocompetent individuals [2]. Venous thrombosis during an acute CMV infection has a predilection for involvement of the portal vein and its tributaries. Squizzato et al. [3] recently reviewed all previously reported cases of portal vein thrombosis

associated with acute CMV infection. Ten of the 11 patients were young adults (29–40 years old). Abdominal pain was reported in 7 of the 11 patients, and mild elevation of liver enzymes in each case. All presented with incomplete thrombosis of the portal system (partial thrombosis of the portal trunk or branch thrombosis) without intestinal ischemia. Predisposing factors (oral contraceptives or prothrombin G20210A mutation) were detected in 7 of the 11 patients. None of their patients developed symptomatic pulmonary embolism. Regression of thrombosis was rapid and usually complete after the disease subsided.

The pathogenesis of venous thrombosis during CMV infection is unclear. It is suggested that the procoagulant effect of CMV occurs via infection of endothelial cells [4]. It is assumed that the combination of viral hepatitis and local vessel inflammation with a systemic prothrombotic state may initiate local thrombosis especially in genetic predisposed individuals. In our patient, there was no biochemical evidence of hepatitis. It can be speculated that lupus anticoagulants induced by CMV infection together with the local abdominal inflammation and oral contraceptive use resulted in venous thrombosis. Inflammation in and around

the superior mesenteric vein wall [Figure] was a prominent feature in our patient and was initially interpreted as a soft tissue mass requiring biopsy. The mass completely regressed with anticoagulant therapy and was therefore part of the local inflammation.

References

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LAC = lupus anticoagulant
PCR = polymerase chain reaction
Ig = immunoglobulin
CMV = cytomegalovirus