CYTOMEGALOVIRUS-ASSOCIATED SPONTANEOUS SPLENIC INFARCT IN AN IMMUNOCOMPETENT PATIENT

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INTRODUCTION

Cytomegalovirus (CMV) associated thrombosis has been reported in medical literature and mainly in immunocompromised population. However, cytomegalovirus associated splenic infarcts have rarely been reported.

CASE DESCRIPTION

A 36-year-old Caucasian woman with no significant past medical history who presented to the emergency department with fever, chills, night sweats, palpitation and mild abdominal pain was found to have acute CMV infection. The patient mentioned that she has used oral contraceptives prior to presentation. No family history of thrombophilia has been reported. CT scan of the abdomen and pelvis showed splenic infarct and an ultrasound of her spleen with doppler showed the same findings, i.e., splenic infarct with normal flow in the splenic artery. However, Echocardiography showed no vegetation, PFO or mural thrombus. Hypercoagulable workup was negative. Anticoagulation was deferred, oral contraceptives were stopped, and copper IUD was recommended, patient was managed conservatively due to involvement of small vessels only and lack of related symptoms. She continued to do well on follow up evaluations.

DISCUSSION

Acute CMV infection with various thrombotic manifestations have been reported in the medical literature. It is a transient risk factor for both arterial and venous thromboembolism and can occur in immunocompetent patients in the absence of other hypercoagulable factors. The patient in our case had 2 risk factors that might explain the reason behind splenic infarction: the first, being on oral contraceptives and the second is the acute CMV infection. A metanalysis on 97 patients in 2011 reported that the incidence of thrombosis among acute CMV infection in hospitalized patients was 6.4%, and the incidence of acute CMV infection among hospitalized patients with thrombosis was 1.9-9.1%.

IMAGING

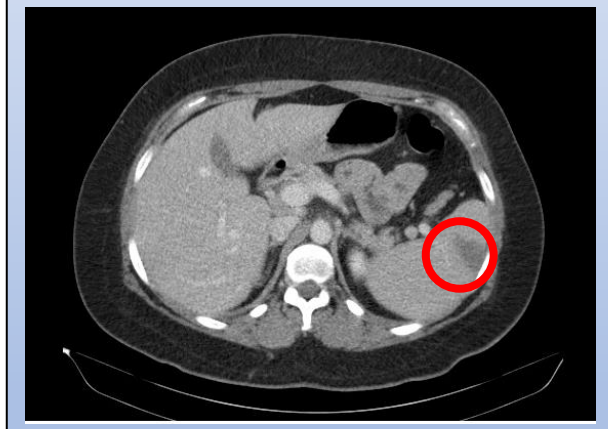

Figure (1)

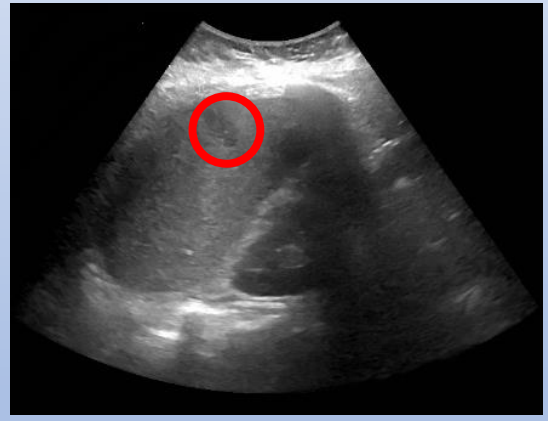

Figure (2)

Figure (1) : There is a wedge-shaped hypodense area at the lateral anterior spleen measuring 3.1 x 2.5 x 2 cm compatible with splenic infarct**. Figure (2)** : Splenomegaly with changes compatible with a focal infarction and normal flow seen in the splenic artery.

DISCUSSION Contd.

Several mechanisms have been described explaining the role of CMV in thrombosis. In vitro, CMV activates factor X and stimulates the production of factor VIII and vWF. It also binds to platelets via Toll-Like Receptor 2 and causes systemic endotheliitis at various sites in the body, leading to the expression of tissue factor. These mechanisms result in platelet and leukocyte aggregation, adhesion, and thrombin formation. In vivo, a transient increase in antiphospholipid antibodies and a decrease in protein C activation have been reported.

CONCLUSION

In conclusion, patients with unexplained arterial or venous thrombosis should be tested for CMV infection as a possible etiology.