Transient Splenic Infarct in a Patient with Epstein Barr Virus (EBV) infection

Brian Sowka, DO; Padmavathi Mali, MD; Gastroenterology of Gastroenterology; La Crosse, WI

GUNDERSEN MEDICAL FOUNDATION

INTRODUCTION

- There have been few case reports of splenic infarcts as a complication of EBV infection in the literature.
- This case is unique as it presented with a transient splenic infarct which resolved in few days.

CASE DESCRIPTION

- A 60-year-old male with hypertension presented to GI clinic for transaminitis.
- Hospitalized a week prior with three weeks of chills, myalgias, arthralgias, and abdominal pain.
- Laboratory evaluation during admission showed elevated transaminases AST 118 (0-40 U/L), ALT 111 (0-40U/L), and Alkaline Phosphatase 223 U/L (40-129 U/L), total bilirubin was 1.0, LDH was elevated at 512, and mild pancytopenia.
- CT scan of the abdomen showed faint wedge-shaped splenic hypodensities concerning for infarctions. He was started on enoxaparin which was later transitioned to aspirin. Transthoracic echocardiogram showed no evidence of endocarditis and blood cultures were negative.
- Pancytopenia improved but transaminases increased to AST 463, ALT 482, ALP 482, and Bilirubin rose to 1.4. A CT of the chest was performed to exclude pulmonary embolism which showed resolution of the splenic infarcts.



Figure 1 – Axial CT demonstrating multiple visible hypodensities in the spleen



Figure 2 – Axial CT for pulmonary embolism with resolution of splenic infarcts

CASE DESCRIPTION

 He was discharged home and a follow-up workup for elevated liver tests showed a negative viral hepatitis panel, negative autoimmune work-up, and EBV antibody to Viral Capsid IgM and IgG returned elevated at 121 and 53 respectively. EBV antibody to Early Antigen was also elevated at 127. Over the next few weeks, his clinical symptoms and transaminases improved.

DISCUSSION

- There are few prior reports of splenic infarction as an uncommon complication of infectious mononucleosis related to EBV infection.
- This patient had a splenic infarct which resolved in a few days with no other etiology which was identified.
- The mechanism related to thrombosis and infarct formation is thought secondary to the proinflammatory state causing platelet adhesion in EBV infection.
- In our patient, this effect was transient likely secondary to perfusion abnormalities and he recovered well. This case reinforces the importance of ruling out EBV infection in patients with elevated liver tests and splenic infarct.

REFERENCES

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