Uncommon Convergence: Splenic Infarct In A Patient With Sickle Cell Trait & CMV Infection

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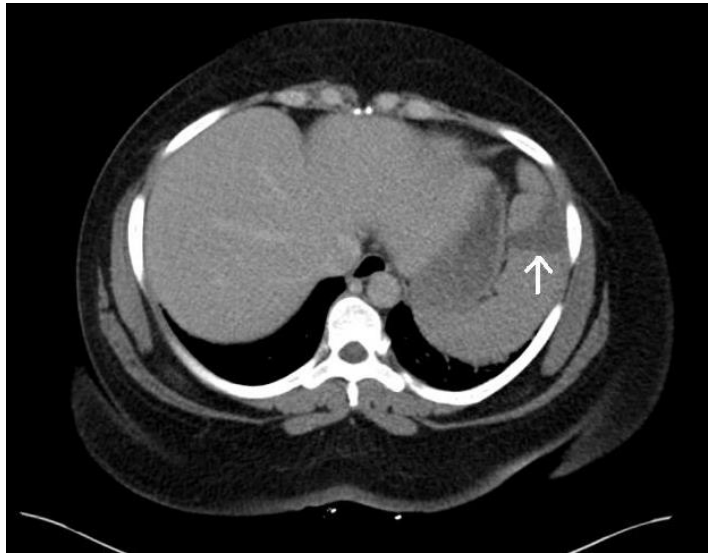
Introduction

- Sickle cell trait, characterized by genetic heterozygosity for sickle cell hemoglobin, is generally considered a benign condition with minimal clinical manifestations.
- While splenic infarcts are a common complication of sickle cell disease, they are rarely observed in individuals with sickle cell trait.
- This case report presents a unique and interesting scenario of a splenic infarct in a patient with sickle cell trait in the setting of a cytomegalovirus (CMV) infection.

Case Description:

- A 31-year-old African American female with history of sickle cell trait presented to the hospital with 2 days of left sided abdominal pain worsened with deep inspiration associated with nausea and poor oral intake.
- 1 week prior to the presentation she had extreme fatigue and myalgias but no fever or chills, which resolved in 2 days.
- Lab work, on presentation, was significant for leukocytosis 14K/uL with atypical lymphocytes, transaminitis (AST 107 U/L, ALT 100 U/L) and elevated alkaline phosphatase 170 u/L.
- CT scan of abdomen showed splenomegaly, spleen measuring 14.9 cm, with a peripheral wedge-shaped hypodensity along the superolateral aspect of the spleen suggestive of splenic infarct.
- The patient was admitted for further evaluation. While in the hospital, the patient was found to have CMV hepatitis with elevated CMV IgG and IgM. CMV PCR quantitative showed 4,120 copies/mL.
- Extensive work up for other infectious causes was negative. She received one dose of Ganciclovir while in the hospital but then anti-virals were discontinued as the patient was immunocompetent and could likely clear infection without intervention.
- Lab work for thrombophilia was sent and the patient was briefly started on Eliquis which was stopped due to heavy menstrual bleeding.



CT Abdomen showing wedge shaped splenic infarct (white arrow)

- Upon follow up after discharge, the patient had complete resolution of her symptoms and her liver function test normalized.
- Repeat quantitative CMV DNA analysis showed decreased viral load (114 copies/mL). The work up for thrombophilia was negative.

Discussion:

- Etiology of splenic infarcts in patients with sickle cell disease is usually multifactorial. Young patients with sickle cell anemia often have splenomegaly due to extensive extra-medullary hematopoiesis. However, over time deformed sickled red blood cells that cannot traverse the splenic vasculature cause infarctions and microinfarctions of the spleen. This results in a shrunken and dysfunctional organ.
- However, splenic infarcts are highly uncommon in patients with sickle cell trait. Triggers like severe prolonged exercise, high altitude exposure, and extreme dehydration ca rarely cause splenic infarct in such patients.
- Previous studies have theorized that CMV may induce a procoagulant response with infection although the mechanism is currently unknown.
- It is theorized CMV may contribute to tissue factor activation, platelet activation, and endothelial inflammation. (1)
- CMV may induce a tissue factor mediated response leading to the activation of inflammatory mediators.
- CMV may activate factor X and stimulate production of factor VIII and von Willebrand factor. (2) CMV may bind to platelets via toll like receptor 2 activating platelet adhesion via P selectin on the cell surface. (3)
- CMV can cause endothelial inflammation at various locations leading to increased expression of tissue factor ultimately activating clotting cascade.
- Overall, this case report provides valuable insights into the potential complications of CMV infection in patients with sickle cell trait.
- It emphasizes the need for a high index of suspicion for alternative causes of splenic infarcts, especially in the presence of viral infections.
- Further research is needed to better understand the mechanisms by which CMV infection can contribute to thrombosis and to develop effective strategies for prevention and treatment.

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