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CASE REPORT Molly Lewandowski, MD & Samantha Nohava, Medical Student, UMKC

SPLENIC INFARCTION

Splenic infarction is a rarely encountered disorder, usually occurring as a complication of another disease process. Due to the distribution of the splenic vasculature, infarcted areas are limited to specific segments of the spleen and rarely extend to all of the parenchyma. Wedge-shaped, hypodense regions are the characteristic appearance of splenic infarction on imaging studies.

CASE:

A 57 year old African American male presented to Truman Medical Center with a five day history of severe left upper quadrant abdominal pain, radiating to his left shoulder and neck. He also complained of nausea, dyspnea and mild chest pain but denied fever; the patient had visited the ER two days prior with similar complaints and his symptoms were partly relieved with a GI cocktail and ranitidine. However, the above symptoms worsened and he returned for further evaluation. His past medical history was remarkable for hepatitis C, hypertension, IV drug use, chronic tobacco use, BPH and a history of medication noncompliance.

On presentation, his vital signs revealed a BP of 170/79, P 90, R 20 and a temperature of 98 F. Labs demonstrated a leukocytosis of 12.2 but were otherwise unremarkable. A CXR had findings suggestive of COPD and an EKG was normal. A CT of the abdomen with contrast was obtained; this showed a medial upper pole wedge-shaped area of hypodensity in the spleen, consistent with a splenic infarct (**image on the next page**). Given his history of IV drug use and his current leukocytosis, blood cultures were obtained and a trans-thoracic echocardiogram was ordered; the latter was essentially normal and did not demonstrate evidence of endocarditis. The blood culture grew only a non-Bacillus species that was determined to be a contaminant. Additional testing included a monospot and lupus anticoagulant, both of which were negative.

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During the hospitalization, further history was obtained which revealed the potential etiology of his infarction. One week prior to admission, while injecting a drug, he missed the vein and removed the needle, noticing a clot in the syringe; nevertheless, he proceeded to inject the drug into another vein and his presenting symptoms developed within 12 hours of that event. The patient remained afebrile throughout his hospital course; he was treated with IV saline, morphine and oxycodone-acetaminophen for pain control and lisinopril for his hypertension. His pain improved significantly over the next 72 hours and he was discharged on oral analgesics.



SPLENIC INFARCTION MARKED BY ARROW

DISCUSSION:

Splenic infarcts are usually limited to one segment or pole of the organ because the lobar arteries that supply the spleen do not anastomose with one another, thus giving rise to lobes known as segments. For this reason, conservative surgery of the spleen is possible, when indicated.

A literature review turned up multiple possible causes for splenic infarction including coagulation disorders such as antiphospholipid syndrome, autoimmune disorders such as Wegener's granulomatosis, and infectious causes such as HIV, CMV, aspergillosis, EBV, salmonella and malaria. Splenic infarction has also been described as a complication of pancreatitis, a microvascular complication of diabetes mellitus or a consequence of systemic emboli. Laxity of ligaments supporting the spleen can lead to a "wandering spleen" which is another cause of infarction, resulting from torsion.

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The classic presentation of splenic infarction includes left upper abdominal pain, nausea, vomiting and early satiety. CT angiography with contrast is the modality of choice for diagnosing splenic infarction, revealing a hypodense, wedge-shaped region as illustrated above. Leukocytosis and anemia are commonly found. Standard management of splenic infarction includes hydration, oxygenation and pain control. Depending on the size of the infarct, symptoms typically resolve within 7-14 days.

Complications of splenic infarction include abscess formation, pseudocyst development, hemorrhage, subcapsular hematoma or splenic rupture; however, all of these complications are uncommon. In the majority of cases, the ischemic tissue undergoes fibrosis and heals completely, thus precluding the need for splenectomy. Preserving the spleen is especially important due to its role in preventing infections; following splenectomy, patients are at significant risk for overwhelming infections, including sepsis, from encapsulated bacteria such as *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Neisseria* species; patients who have massive splenic infarcts and/or must undergo splenectomy should thus be vaccinated against these organisms.

CONCLUSION: This case highlights a rare complication of IV drug abuse and demonstrates the value of CT angiography in the diagnosis of splenic infarction. As discussed above, splenectomy is not often necessary and should be avoided to prevent medical complications associated with hyposplenism.

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