LETTERS

Splenic emboli owing to left ventricle mural thrombus: an unusual cause of acute abdominal pain

Dear Editor,

Splenic infarction is a rare cause of acute abdominal pain that can occur in a variety of settings including both malignant (e.g. myelofibrosis, lymphoma and leukemia) and benign hematological disorders (e.g. sickle cell hemoglobinopathies, polycythemia vera and hypercoagulable states). Embolic disease (e.g. atrial fibrillation, infective endocarditis, valvular disease, valve replacement or aortic atheromas) can also cause splenic infarction. Thromboembolic complications, although rare, have been seen in approximately 2% of old patients surviving an acute myocardial infarction.

We report a patient who presented with acute abdominal pain located in the upper-left-quadrant, and was diagnosed with multiple splenic infarcts originating from a large mobile mural thrombus located in the left ventricle, which resulted from a recent acute myocardial infarction.

A 67 year-old man was admitted to our hospital complaining of acute abdominal pain at the upper-left-quadrant accompanied with diffuse chest pain that was aggravated on deep breaths. Both symptoms were worsening over the preceding 48 hours and associated with fever, chills and dyspnea. On admission, the patient’s temperature was 37.4°C; heart rate was 105 beats/min; arterial blood pressure was 130/74 mmHg; his respiratory rate was 20 breaths/min and oxygen saturation measured by pulse oximetry was 93%. On physical examination decreased breath sounds, fine crackles within the left lower lobe and significant tenderness within the upper-left-quadrant of his abdomen, were found.

His medical history was remarkable for an anterolateral myocardial infarction, occurred one month before his presentation, for which, a subsequent two-vessel coronary artery bypass grafting procedure had been performed. Chronic obstructive pulmonary disease and a previous myocardial infarction were also accounted in his past medical history.

Laboratory investigation revealed the following: white-blood-cell count: 15,700 /mm$^3$ (neutrophils: 91%); hematocrit: 43%; hemoglobin: 14.1 g/dL; platelet count: 245,000/mm$^3$; C-reactive-protein: 9.63 mg/dL (normal: 0-0.8 mg/dL); D-dimers: 1544 ng/mL (normal<500ng/mL); lactate-dehydrogenase: 1130 U/L (normal: 240-480 U/L) and SGOT: 61 U/L (normal: 0-38U/L).

Chest x-ray showed a small left pleural effusion whereas abdominal ultrasound was normal, not indicative of a splenic infarction. An abdominal CT scan revealed multiple wedged-shaped areas of decreased density within the spleen, suggesting multiple infarcts. A subsequent transesophageal echocardiogram demonstrated a large mobile mural thrombus originating from the left ventricle, together with wall-motion abnormalities.

Low-molecular-weight-heparin treatment was initiated (enoxaparin, 1mg/kg twice daily, sc) that was converted to oral anticoagulation on the third day. The patient was discharged 7 days after his admission with complete resolution of the abdominal pain. He remained under treatment with warfarin for 3 months. At his reevaluation, no abnormal symptoms or sings were observed. An abdominal ultrasound depicted the remaining splenic infarcts without any additional findings, while a transesophageal echocardiogram showed a normal left ventricle function.

In conclusion, splenic infarction as a complication of cardiac embolic disease should be included in the differential diagnosis of an acute abdominal pain.

References

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