Splenic Infarct: A Rare Presentation in a Pediatric Patient

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ABSTRACT

A previously healthy 16-year-old male presented with a two day history of persistent epigastric pain. His physical examination was significant for tenderness in the left hypochondriac region with a palpable spleen 2cm below the left sub-costal margin. A CT scan of the abdomen showed a splenic infarct. Heterophile and EBV VCA IgM antibody test were positive. This is a rare case of infectious mononucleosis presenting with splenic infarct in an adolescent male without co-morbidities.

Keywords: *infectious mononucleosis; splenic infarct.*

INTRODUCTION

Several mechanisms have been proposed to explain the underlying pathophysiology of splenic infarction associated with infectious mononucleosis. These include both hypoxemia and increased thrombotic tendency due to underlying conditions. Acute EBV infection also induces excess B cell proliferation and consequently, increased production of various antibodies. It has been shown that EBV also leads to transient increase in thrombotic markers. Beyond these hypotheses, specific pathophysiologic mechanisms leading to splenic infarct need further investigation.

The clinical presentation of infarction may include signs and symptoms characteristic of hemorrhagic shock or septic thromboembolism which often results in splenic abscesses. Moreover, classic signs of severe sepsis with left upper abdominal pain are typical.[1] CT scan is the current diagnostic modality of choice because there are no known specific laboratory tests to make an accurate diagnosis. CT scan with gadolinium contrast and three dimensional reconstruction can be used for better image enhancement.[2-4] Subcapsular hematoma evident on the CT scan may be a sign of splenic rupture.

We present a case of a 16-year-old male with EBV positive infectious mononucleosis associated with splenic infarction as a sequela. The patient was managed conservatively with subsequent resolution of clinical symptoms.

CASE REPORT

A previously healthy 16-year-old male presented with a two day history of persistent epigastric pain worsening on inspiration and improving at rest. He also reported one episode of non-bilious, non-bloody emesis. The patient denied fever, headache, sore throat, myalgia, weight loss and arthralgia. He was visiting the United States from the Dominican Republic. He also denied any history of trauma, recent URI or previous episodes of similar symptoms.

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There was no family history of any known illnesses. On physical examination, there was no erythema or exudates of the pharyngeal mucosa and there was no cervical lymphadenopathy. There were no bruises or petechiae. Abdominal exam was significant for tenderness in the epigastrium with increased tenderness in the left hypochondriac region. The splenic tip was palpable 2 cm below the left sub-costal margin in the splenic axis. Cardiovascular, respiratory, musculoskeletal and neurological exams were unremarkable.

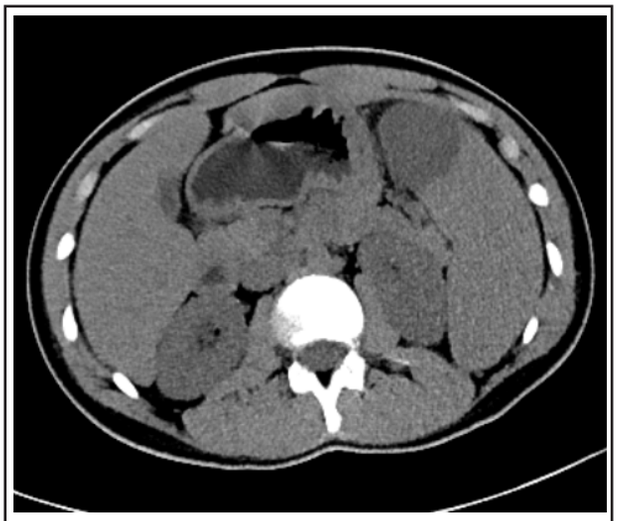

Figure 1.CT scan showing mild hepato-splenomegaly with a 4.3 cm sub-capsular low-attenuation area without capsule destruction consistent with infarction.

INVESTIGATIONS

The CBC on admission showed Hb of 12.3 g/dl and Hct of 36.2%, MCHC of 35.2 g/dl, WBC of 6.76 with 11% Atypical Lymphocytes. Other labs included ALT/AST: 154/105 U/L, PT: 16.7 Sec, INR: 1.53, PTT: 35 Sec, Total bilirubin: 1.5 mg/dl, LDH 552 U/L and Uric Acid 4.8 mg/dl. The peripheral smear was normal. CT scan of the abdomen showed mild hepato-splenomegaly with a 4.3 cm sub-capsular low-attenuation area without capsule destruction consistent with infarction. Heterophile antibody test and EBV VCA IgM were positive thus confirming acute infectious mononucleosis with splenic infarct.

OUTCOME AND FOLLOW UP

The patient symptomatically improved over the following two days with conservative management. The laboratory tests were repeated and showed normalization of PT/INR (13.9/1.3) as well as a downward trend in AST: 74, ALT: 124, Total bilirubin: 1.3, LDH 486. He was discharged with instructions to avoid contact sports for three weeks and to follow up with his primary care physician in the Dominican Republic.

DISCUSSION

Splenic infarct in children is a rare finding without pre-existing hematological abnormalities. During history taking or clinical examination, hematological or other etiologies are mostly identified. In this case, the other causes were excluded based on history, physical examination and peripheral blood smear. In some patients when specific risk factors are not identified, an uncommon underlying etiology is uncovered. Splenic infarction with EBV positive mononucleosis without comorbidities is exceedingly rare. The associated comorbidities include hereditary spherocytosis, protein C deficiency,[5] sickle cell disease[6] and pyruvate kinase deficiency.[7] EBV has been seen to cause transient elevated antiphospholipid antibodies and in one case resulted in infarction.[8] When comorbid disorders are excluded, splenic infarctions with infectious mononucleosis have only been reported three times.[9,10] Since the diagnosis of infectious mononucleosis is usually made clinically, CT scans are not routinely performed and the incidence of splenic infarction due to infectious mononucleosis cannot be determined.[11] In children surgery is indicated only in the presence of complications with hypersplenism or focal splenic tumors.[12]

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