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CASE REPORT

Splenic Infarction in a Case of Acute Promyelocytic Anemia

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ABSTRACT

Splenic infarction occurs due to occlusion of splenic vessels that leads to splenic tissue ischemia and necrosis. There are several reports regarding splenic infarction in patients with acute myelogenous leukemia (AML). Herein, we report a case of acute promyelocytic anemia (AML-M3) who presented with abdominal pain and splenic infarction.

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Introduction

Splenic infarction occurs due to occlusion of splenic vessels that leads to splenic tissue ischemia and necrosis.[1-4] There are several reports regarding splenic infarction in patients with acute myelogenous leukemia (AML). Here in, we report a case of acute promyelocytic anemia (AML-M3) who presented with abdominal pain and splenic infarction.

Case Presentation

The patient was a previously healthy 20-year-old woman who presented with blurred vision, abdominal pain and dizziness following hemorrhoid surgery. Physical examination revealed pallor, wide peripapillary retinal hemorrhage and splenomegaly. Blood examination on admission revealed: WBC; 54×10^9/µl, Hb;7 gr/dl and Platelet count;16×10^9/µl, LDH; 3300 IU/L (normal range up to 480), Prothrombin time (PT); 19.3 sec (12-15), INR; 1.67(1-1.2), Partial thromboplastin time (PTT); 28 sec (24-45), Fibrinogen level; 287 mg/dl (200-400), Fibrin degradation product (FDP)>20 mg/L (reference range less than 10 mg/L) and D dimer >2000 ng/ml (normal <255). The Patient underwent bone marrow aspiration and biopsy which revealed hypercellular marrow with more than 95% blasts and in spite of mostly abnormal promyelocytes compatible with AML-M3. Flowcytometry revealed blasts positive for CD33, CD117 and negative for HLA-DR, myeloperoxidase staining of the blasts were strongly positive. Molecular genetic study revealed PML-RAR-α positivity by RT-PCR. Abdominal ultrasound showed splenomegaly (spleen size: 165×90 mm) with heterogeneous echo and large peripheral hypoechoic geographic lesions suggestive for infarction. Abdominal spiral CT scan showed splenic enlargement with extensive peripheral hypodensity and lack of contrast enhancement in favor of infarction (figure 1). Chemotherapy with daunorubicin, All-trans retinoic Acid (ATRA) and arsenic trioxide was initiated for the patient. He also received FFP and platelet transfusions considering the patients' conditions and diagnosis of the patient. Initially, the

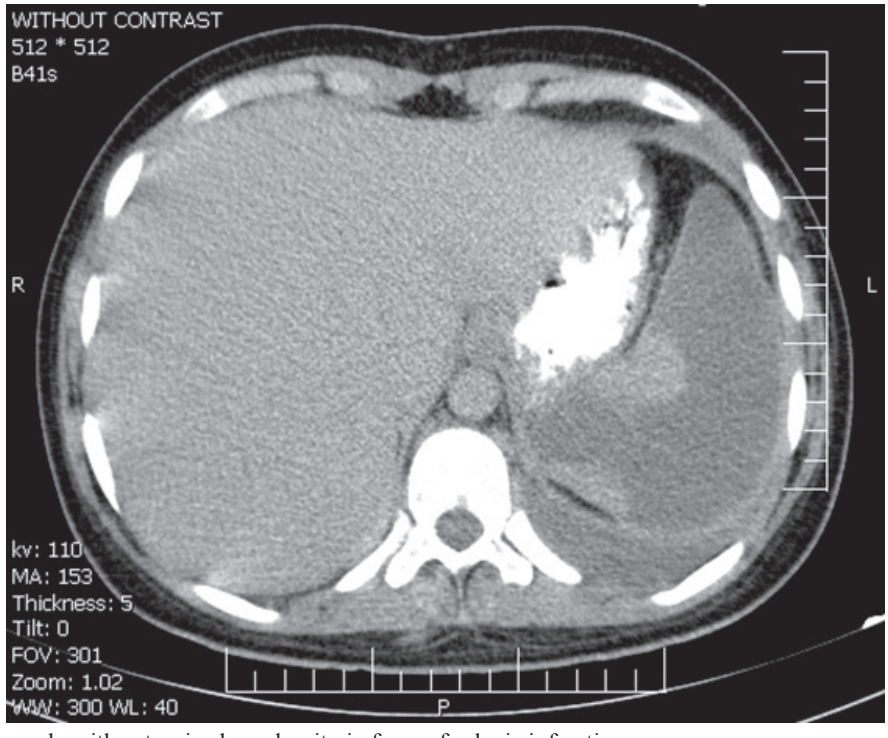

Figure 1: splenomegaly with extensive hypodensity in favor of splenic infarction.

patient underwent supportive care and close observation for management of the splenic infarction. Finally, surgical consultation was conducted on day 28 after induction of remission which splenectomy was recommended due to persistence of large splenic lesions on serial abdominal imaging but did not perform due to thrombocytopenia.

Discussion

In our patient with AML-M3, disseminated intravascular coagulation (DIC) in the spleen led to splenic infarction. Splenic infarction is defined as occlusion of the splenic vessels that leads to splenic ischemia and necrosis which may be total or segmental. A heterogenous group of diseases cause splenic infarction, mostly attributable to hematological malignancies and myeloproliferative disorders. However, benign hematological disorders such as autoimmune hemolytic anemia, hypercoagulable states, vascular disorders, trauma and iatrogenic etiologies such as pancreatectomy and liver transplant can also be influential.[1-8] Acquired protein C deficiency has been reported in AML.[9,10] Splenic infarction alone is not an indication for surgery and requires close follow-up. Surgery is indicated for persistent symptoms or subsequent complications such as hemorrhage, rupture, and abscess formation. Our explanation for splenic infarction in this case of AML-M3 was thrombotic complication due to AML and its inherent thrombotic tendency as disseminated intravascular coagulation.

Conflict of Interest: None declared.

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