Spontaneous splenic infarction in an elderly cirrhotic patient with multiple comorbidities

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Spontaneous splenic infarction has been seen rarely in cirrhosis and portal hypertension. The clinical presentation can mimic other causes of acute abdominal pain. The diagnosis of the condition is based on clinical findings and splenic imaging. In recent years, ultrasonography and computed tomographic scan have gained in popularity for the diagnosis of splenic infarction. Most reported cases are of focal infarction, and treatment is mostly conservative. Herein, we describe a rare case of spontaneous splenic infarction in an elderly cirrhotic patient with portal hypertension who also had comorbidities. A 72-year-old female previously diagnosed with cirrhosis was admitted for left upper quadrant abdominal pain for two days. Her medical history included cryptogenic cirrhosis, congestive heart failure, chronic obstructive pulmonary disease, and hypertension. Physical examination on admission revealed a palpable splenomegaly. Abdominal ultrasonography revealed splenomegaly and a hypoechoic area with lobulated contours measuring 62x35 mm extending from the subcapsular area to the hilus in the middle section of the spleen. Abdominal computed tomographic showed a subcapsular hypodense lesion of the spleen measuring 64x58 mm. Doppler ultrasound revealed a wedge-shaped heterogeneous hypoechoic avascular area extending from the central zone to the lateral zone of the spleen. In our case, diagnosis of splenic infarction was made by computed tomographic and Doppler ultrasonography. Our patient received conservative treatment for the underlying diseases. Spontaneous splenic infarction must be kept in mind in cirrhotic patients with underlying comorbidities presenting with left upper quadrant pain.

Key words: Splenic infarction, cirrhosis, portal hypertension

Spontan dalak enfarkt› siroz ve portal hipertansiyonda nadiren görülmektedir. Klinik görünümü akut karan a¤r›s›n taklit edebi-lir. Bu durumun tanısı klinik bulgular ve dala¤in görüntülenmesi ile konur. Son yıllarda, ultrasonografi ve bilgisayarl› tomogra-fi dalak enfarkt› tan›s› için popülarite kazanmıştır. Çok sayıda olguda enfarkt bölgesi doludur ve tedavi ço¤unlukla konservatiftir. Biz burada sira¤a ba¤l› portal hipertansiyon ve aynı zamanda komorbiditeleri bulunan yaşlı sirotik bir o¤lu nadir görülen spontan dalak enfarkt›n bildiriyoruz. 72 yaşında sira¤o olan kadın hasta, iki gündür devam eden karan ôngünü ile başvurdu. Özgeçmimizde kriptojenik sira¤, konjestif kalp yetmezliği, kronik obstruktif akci¤eler hastal›¤› ve hipertansiyon o¤kusu var-d›. Fizik muayenesinde palpsiyonun a¤r›s›n paralayıcılı oldu. Bat›n ultrasonografisinde splenomegali ve dalak orta bölümde sub kapsülar alanlardan hilusa doğru uzanan 62x35 mm çapında lobal konturlu hipoeohik alan izlendi. Bat›n tomografisinde dalakta 64x58 mm çapında sub kapsüler hipodens lezyon görüldü. Doppler ultrasonografide dalak hilusunda lateral zona do¤ru uzanan kama çektirilin heterojen hipoeohik avasküler alan saptandı. Olgumuzda bilgisayarl› tomografi ve Doppler ultrason ile dalak enfarkt› tan›n› s›kondu. Hastamızda alta yatan hastalıklar›n konservatif tedavisi yapıldı. Spontan dalak enfarkt› sol üst karan âgrıs ile ba¤-vuran ve komorbiditeleri bulunan sirotu hastalarda göz önünde bulundurulmalıdır.

Anahtar kelimeler: Dalak enfarkt, sira¤, portal hipertansiyon

INTRODUCTION

Splenic infarction is an uncommon ante-mortem diagnosis (1). Congestive splenomegaly is a frequent finding in patients with portal hypertension, but splenic infarction is uncommon in cirrhosis (2). The predominant causes of splenic infarction are thought to be bacterial endocarditis, sickle cell
A 72-year-old female previously diagnosed with cirrhosis and portal hypertension was admitted for left upper quadrant abdominal pain for two days. Her medical history included cryptogenic cirrhosis, congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD) and hypertension. She was on medication of spironolactone, acetyl salicylic acid, furosemide, propranolol, digoxin, perindopril, salbutamol, and tiotropium bromide. Physical examination on admission revealed palpable splenomegaly. Laboratory tests on admission included: hemoglobin 12.3 g/dl (12.2-18.1 g/dl), hematocrit 36.9% (37.7-53.7%), platelet count 158 K/μL (142-424), international normalized ratio (INR) 1.13, aspartate aminotransferase 35 IU/L (0-32 IU/L), alanine aminotransferase 20 IU/L (0-33 IU/L), alkaline phosphatase 178 IU/L (53-141 IU/L), gamma glutamyl transpeptidase 69 IU/L (9-36 U/L), direct bilirubin 0.5 mg/dl (0.0-0.3 mg/dl), total protein 7.6 g/dl (6.3-8.4 g/dl), and albumin 3.4 g/dl (3.8-5.1 g/dl). Abdominal ultrasonography (USG) revealed splenomegaly and a hypoechoic area with lobulated contours measuring 62x35 mm extending from the subcapsular area to the hilus in the middle section of the spleen. Therefore, abdominal computed tomography (CT) was performed, which described a hypodense lesion measuring 64x58 mm extending from the central zone to the lateral zone of the spleen with a subcapsular extension (Figure 1). Doppler ultrasound revealed a wedge-shaped heterogeneous hypoechoic avascular area extending from the central zone to the lateral zone of the spleen.

Splenic infarction is an uncommon form of splenic pathology (2,4). Embolic events, either of cardiovascular etiology or as a result of a hypercoagulable state and hematologic disorders, are associated with splenic infarction in about two-thirds of the cases (1,4). The most frequent causes of splenic infarction include myelofibrosis, bacterial endocarditis, sickle cell disease, and hematologic malignancies (3,5). Other unique causes of splenic infarction include splenic vascular disease, Gaucher disease, infiltrative diseases (sarcoidosis and amyloidosis), pancreatitis, collagen-vascular diseases (systemic lupus erythematosus, polyarteritis nodosa), and nonhematologic malignancy (5,6). Splenic infarction must be suspected in patients with known hematologic or thromboembolic conditions who develop left upper quadrant pain and signs of localized or systemic inflammation.

Spontaneous splenic infarction secondary to cirrhosis and portal hypertension is believed to be uncommon (4,7,8). In a review of a large series of patients with splenic infarction, only 3 of 152 cases were as a result of portal hypertension (4). Iatrogenic infarction may occur during selective intraarterial infusion of vasopressin for gastrointestinal bleed, resulting in angiographically demonstrable splenic artery spasm and subsequent splenic infarction in cirrhotic patients (9). There are also case reports of splenic infarction after cyanoacrylate injection to gastric fundal varices (10,11), histoacryl embolization (12-14), splenic artery ligation (15), and liver transplantation (16).
Splenic infarction can be the presenting symptom of other underlying illnesses, so a high index of suspicion for this condition is appropriate in the presence of predisposing conditions for thrombosis, left flank pain, and splenomegaly. Antopolsky et al. (1) reported that predisposing factors to splenic infarction were present in 71% of the patients. The most common predisposing factors were atrial fibrillation, occurring in 23% of the patients, while 8% of the patients had a history of previous splenic infarction. Essential hypertension, diabetes mellitus, COPD, and CHF were present in 31%, 23%, 8%, and 8% of the patients, respectively (1).

Splenic infarction is the result of an ischemic event in the spleen. However, the mechanism of the splenic infarct in cirrhosis and portal hypertension is unclear (2,7). In a case report of a cirrhotic patient, splenectomy was performed after massive spontaneous splenic infarction. Multiple thromboses of the small arterial and venous vessels were shown in the histological examination; however, the etiology of this infarct remained unclear (7). Various mechanisms have been described for splenic infarction in the course of cirrhosis. It could be, in part, similar to those postulated in splenic infarction secondary to hematological malignancy (2). These include increased splenic mass (congestive splenomegaly) with increased oxygen requirement, or decreased oxygen-carrying capacity due to anemia (due to hypersplenism or gastrointestinal bleeding) (2,4). The probable mechanism of the splenic infarction in our elderly patient with cryptogenic cirrhosis and portal hypertension is anoxia, which may have developed due to the underlying CHF and COPD.

Diagnosis of splenic infarction is based on the clinical presentation and confirmed by splenic imaging (2). Several diagnostic modalities may be used to definitively diagnose splenic infarction. The most commonly obtained radiographic modalities used for diagnosis of splenic infarction include CT, nuclear imaging and USG (5). Contrast CT scan is currently the best noninvasive test available to diagnose splenic infarctions (1). The diagnosis of splenic infarction in our case was made by CT and Doppler USG.

Specific treatments aimed at correcting the identified underlying cause should be undertaken. The indications for splenectomy are expanding subcapsular hematoma, splenic pseudocyst, abscess, and splenic rupture (9). However, splenectomy is rarely needed. The prognosis varies depending on the process responsible for the splenic infarction (5). Our patient had conservative treatment for the underlying diseases and did not require splenectomy.

Spontaneous splenic infarction is seen rarely in cirrhotic patients. It must be kept in mind in cirrhotic patients with underlying comorbidities presenting with left upper quadrant pain.

REFERENCES