CASE REPORT

TJG
The Turkish Journal of Gastroenterology

Spontaneous splenic infarction in an elderly cirrhotic patient with multiple comorbidities

Elife ERARSLAN[1], Alper BOZKURT[2], İlhami YÜKSEL[1], Hanzade Duygu DEMİR[3]

Departments of [1]Gastroenterology, [2]Radiology and [3]Internal Medicine, Etlik İhtisas Educational and Research Hospital, Ankara

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

Multipl ko-morbiditeli yaşlı sirotik bir hastada gelişen spontan dalak infarktı

Spontan dalak infarktı siroz ve portal hipertansiyonda nadiren görülmektedir. Klinik görünümü akut karın ağrısını taklit edebilir. Bu durumun tanısı klinik bulgular ve dalağın görüntülenmesi ile konur. Son yıllarda, ultrasonografi ve bilgisayarlı tomografi dalak enfarktı tanısı için popülarite kazanmıştır. Çok sayıda olguda infarkt bölgeseldir ve tedavi çoğunlukla konservatiftir. Biz burada siroza bağlı portal hipertansiyon ve aynı zamanda komorbiditeleri bulunan yaşlı sirotik bir olguda nadir görülen spontan dalak infarktını bildiriyoruz. 72 yaşında sirozu olan kadın hasta, iki gündür devam eden karın sol üst kadran ağrısı nedeni ile başvurdu. Özgeçmişinde kriptojenik siroz, konjestif kalp yetmezliği, kronik obstrüktif akciğer hastalığı ve hipertansiyon öyküsü vardı. Fizik muayenesinde palpasyonla splenomegali vardı. Batın ultrasonografisinde splenomegali ve dalak orta bölümde subkapsüler alandan hilusa doğru uzanan 62x35 mm çapında lobule konturlu hipoekoik alan izlendi. Batın tomografisinde dalakta 64x58 mm çapında subkapsüler hipodens lezyon görüldü. Doppler ultrasonografide dalak hilusundan lateral zona doğru uzanan kama şeklinde heterojen hipoekoik avasküler alan saptandı. Olgumuzda bilgisayarlı tomografi ve Doppler ultrason ile dalak infarktı tanısı kondu. Hastamızda altta yatan hastalıkların konservatif tedavisi yapıldı. Spontan dalak enfarktı sol üst kadran ağrısı ile başvuran ve komorbiditeleri bulunan sirozlu hastalarda göz önünde bulundurulmalıdır.

Anahtar kelimeler: Dalak infarktı, siroz, 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

Address for correspondence: Elife ERARSLAN
Etlik İhtisas Educational and Research Hospital,
Department of Gastroenterology, Ankara, Turkey
Phone: + 90 312 567 20 00
E-mail: elifeerarslan@gmail.com

Manuscript received: 27.07.2011 **Accepted:** 03.10.2011

Turk J Gastroenterol 2012; 23 (5): 596-598
doi: 10.4318/tjg.2012.0429

disease and hematologic malignancies (3). Diagnosis of splenic infarction is based on the clinical presentation and confirmed by splenic imaging (2). The clinical presentation can mimic other causes of acute abdominal pain (1). The most common symptom is left upper quadrant abdominal pain, often radiating to the left shoulder (2).

We report an elderly patient with cirrhosis and portal hypertension presenting with left upper quadrant abdominal pain who developed splenic infarction.

CASE REPORT

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/uL (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 U/L (9-36 U/L), total bilirubin 1.8 mg/dl (0.1-1.2 mg/dl), 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.

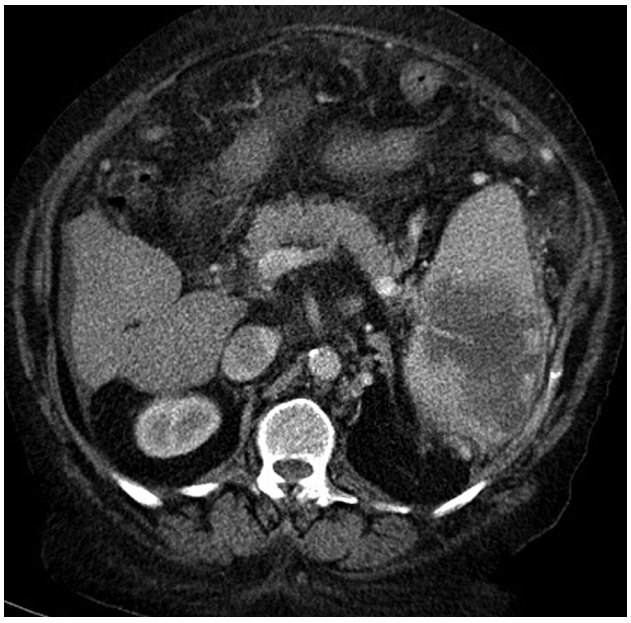

Figure 1. Abdominal computed tomography revealed a hypodense lesion (64x58 mm) extending from the central zone to the lateral zone of the spleen with a subcapsular extension.

DISCUSSION

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 intra-arterial 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

1. Antopolsky M, Hiller N, Salameh S, et al. Splenic infarction: 10 years of experience. Am J Emerg Med 2009; 27: 262-5.
2. Chin JK, McCormick PA, Hilson AJ, et al. Liver/spleen scintigraphy for diagnosis of splenic infarction in cirrhotic patients. Postgrad Med J 1993; 69: 715-7.
3. Lawrence YR, Pokroy R, Berlowitz D, et al. Splenic infarction: an update on William Osler's observations. Isr Med Assoc J 2010; 12: 362-5.
4. Matsui A, Shimada T, Sasaki N, et al. Splenic infarction in a child with portal hypertension secondary to biliary atresia. J Pediatr Surg 1997; 32: 648-9.
5. Ray S, Mridha AR, Ahammed M. Diffuse splenic infarction in a case of severe acute pancreatitis. Am J Surg 2011; 201: e23-5.
6. Jaroch MT, Broughan TA, Hermann RE. The natural history of splenic infarction. Surgery 1986; 100: 743-50.
7. Capron JP, Chivrac D, Dupas JL, et al. Massive splenic infarction in cirrhosis: report of a case with spontaneous disappearance of hypersplenism. Gastroenterology 1976; 71: 308-10.
8. Yuasa K, Yamada S, Uehara M, et al. An autopsy case of primary biliary cirrhosis with giant splenic infarction. Nippon Shokakibyo Gakkai Zasshi 1985; 82: 1591-5.
9. Jaroch MT, Broughan TA, Hermann RE. The natural history of splenic infarction. Surgery 1986; 100: 743-50.
10. Kurt M, Onal IK, Ibis M, et al. Splenic infarction: rare complication of N-butyl-2-cyanoacrylate injection for gastric varices. Dig Endosc 2010; 22: 74-5.
11. Kim J, Chun HJ, Hyun JJ, et al. Splenic infarction after cyanoacrylate injection for fundal varices. Endoscopy 2010; 42: E118.
12. Tan YM, Goh KL, Kamarulzaman A, et al. Multiple systemic embolisms with septicemia after gastric variceal obliteration with cyanoacrylate. Gastrointest Endosc 2002; 55: 276-8.
13. Cheng PN, Sheu BS, Chen CY, et al. Splenic infarction after histoacryl injection for bleeding gastric varices. Gastrointest Endosc 1998; 48: 426-7.
14. Yu LK, Hsu CW, Tseng JH, et al. Splenic infarction complicated by splenic artery occlusion after N-butyl-2-cyanoacrylate injection for gastric varices: case report. Gastrointest Endosc 2005; 61: 343-5.
15. Nordlinger BM, Fulenwider JT, Millikan WJ, et al. Splenic artery ligation in distal splenorenal shunts. Am J Surg 1978; 136: 561-8.
16. Dourakis SP, Alexopoulou AA, Hadziyannis SJ. Splenic infarct as a late complication of liver transplantation. Eur J Gastroenterol Hepatol 1998; 10: 805-8.