Splenic infarction secondary to acute pancreatitis

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ABSTRACT

Background and objective: the close anatomic relationship of the pancreas with the splenic vessels and the spleen is responsible for splenic complications in the course of acute pancreatitis. Our objective was to report two cases of severe acute pancreatitis complicated by splenic infarction.

Patients: in a three-month period of time two patients were diagnosed with splenic infarction secondary to acute pancreatitis. In both cases splenic infarction diagnosis and follow-up were carried out using computed tomography.

Results: in the first case images clearly showed a narrowing of the splenic artery due to the inflammatory pancreatic condition. In the second case no involvement of the splenic vessels could be demonstrated, hence the only possible etiological explanation was a hypercoagulability state generated by pancreatitis.

Conclusions: it would be advisable that splenic complications were added to the list of relevant extrapancreatic manifestations. CT is very useful for the diagnosis and follow-up of splenic complications arising in acute pancreatitis.

Key words: Splenic infarction. Acute pancreatitis. Stenosis of the splenic artery.

INTRODUCTION

The pancreas is a retroperitoneal organ closely related to splenic vessels. This anatomical relationship is the reason why these vessels and the spleen are affected by inflammatory pancreas disease. One of the possible mechanisms of pancreatic necrosis may be microvascular obstruction by spastic vascular changes or increased intravascular coagulability (1-3). These vascular complications may also occur in other organs when the insult is severe enough. Cortical renal necrosis, splenic infarction, ischemic heart disease, cerebral ischemia, and retinal infarction have been reported. Splenic complications in

RESUMEN

Fundamento y objetivo: la estrecha relación anatómica del páncreas con los vasos esplénicos y el bazo es responsable de complicaciones esplénicas en el curso de la pancreatitis aguda. El objetivo es presentar dos casos clínicos de pancreatitis aguda grave que sufrieron infarto esplénico como complicación de la enfermedad pancreática.

Pacientes, participantes: en un periodo de tres meses, dos pacientes fueron diagnosticados de infarto esplénico secundario a pancreatitis aguda. En ambos casos el diagnóstico y seguimiento evolutivo del infarto esplénico se hizo a través de tomografía axial computarizada.

Resultados: en el primer paciente, las imágenes muestran de forma inequívoca la afectación de la arteria esplénica por el proceso inflamatorio pancreático. En el segundo, no se pudo demostrar afectación de los vasos esplénicos, por lo que la única posible explicación etiológica es un incremento de coagulabilidad intravascular.

Conclusiones: sería recomendable añadir las complicaciones esplénicas al conjunto de complicaciones graves extrapancreáticas de la pancreatitis aguda. La tomografía axial computarizada es de gran utilidad para la detección y seguimiento de las complicaciones esplénicas de la pancreatitis aguda.

Palabras clave: Infarto esplénico. Pancreatitis aguda. Estenosis de arteria esplénica.
acute pancreatitis (splenic vein thrombosis, pseudocyst, rupture, infarction, necrosis, hematoma, hemorrhage from erosion of splenic vessels), though infrequent, are life-threatening to patients. Presented herein are two clinical cases of serious pancreatitis complicated by splenic infarction in the course of pancreatic disease.

CASES REPORTED

Case 1

An 83-year-old man with a history of hypertension, chronic obstructive pulmonary disease, cerebrovascular ischemia with paresia of the upper right extremity, and treatment with dicumarin for atrial fibrillation was admitted to the emergency unit. He was suffering from continuous epigastric pain accompanied by nausea and vomits. Physical examination revealed arrhythmia, and a soft, non-tender abdomen. Laboratory data on admission: Red cell count, 3,830,000 per mm³; hemoglobin 121 g/L; white cell count 14,000 per mm³, with 90% polymorphonuclear cells; blood urea nitrogen 74 mg/dL; creatinine 1.8 mg/dL; blood amylase 733 U/L. On the ultrasound scan an enlargement of the pancreatic body and biliary lithiasis were observed. In the next seven days there was a progressive increase in blood amylase up to 1,476 U/L. A computed tomography (CT) showed an enlargement of the pancreatic tail with an ill-defined pseudonodular hypodense area of 4-5 cm in size in contact with the splenic hilum, suggesting acute pancreatitis. Three days later an ERCP was performed, which showed stenosis of the papilla and bile sludge. A sphincterotomy was carried out, and twenty four hours after this the patient reported pain in the upper left abdominal quadrant, irradiating to the shoulder. The pain persisted in spite of treatment with morphine derivatives. A new CT scan was performed, which showed an irregular, enlarged pancreas with peripancreatic exudate surrounding the splenic artery, which was narrowed. Shown too were a splenic infarction and left pleural effusion (Fig. 1), neither of which was modified in later tests. Forty days after admission the patient died from a mesenteric infarction.

Case 2

An 83-year-old woman with a history of hysterectomy, hepatitis B, and repeated episodes of biliary pain was admitted as an emergency case with diffuse abdominal pain, nausea, and vomiting during the previous 12 days. On physical examination superficial and deep pain were observed in the upper abdomen, especially in the upper right quadrant. Laboratory data on admission: White cell count 17,000 per mm³, with 86% polymorphonuclears; blood amylase 5,203 U/L; total bilirubin 1.55 mg/dL; GOT 111 U/L; GPT 152 U/L; GGT 203 U/L; pH 7.222, pCO₂ 28.8 mmHg; pO₂ 80 mmHg; bicarbonate 13.7 mmol/l; 48 hours after admission serious coagulation changes developed: Prothrombine time 50% and cefalin time 48.2 seconds. CT scan displayed acute lithiasic cholecystitis, choledocolithiasis, diffuse enlargement of the pancreas, and ascitis, with multiple areas of infarction in the spleen (Fig. 2). Within 3 days of admission an endoscopic sphincterotomy was performed. The patient deteriorated progressively, and died on the seventeenth day from a hemorrhagic shock of digestive origin.

DISCUSSION

Splenic infarction is the result of arterial and venous compromise in the splenic vessels, either intraluminal or extraluminal; 50-72% of cases have been reported as associated with chronic myeloid leukemia and myelofibrosis (4-6). It is also associated with hemolytic anemia, hypercoagulability states, embolic disorders (atrial fibrillation and endocarditis), vascular disease, trauma, surgical complications (pancreatectomy and liver transplant), and other infrequent causes such as splenic vein thrombosis, amyloidosis, sarcoidosis, and adult respiratory distress syndrome (4-11).
Splenic infarction secondary to acute pancreatitis is believed to be uncommon, but the number of reported cases seems to indicate a greater incidence than previously thought (12-15). Retrospective radiological studies have found rates of 7%, thus demonstrating a relationship between this complication and the severity of pancreatitis (14).

The clinical spectrum varies from asymptomatic infarction to hemorrhagic shock secondary to massive subcapsular hemorrhage with free rupture to the abdominal cavity. One third of cases are clinically silent. The most common symptom is pain in the upper left abdominal quadrant. Additional symptoms are fever, chills, nausea and vomiting, pleuritic pain, and pain in the left shoulder (Kher’s sign) (4,5). The aim of medical treatment is pain relief with narcotics or non-steroidal anti-inflammatory drugs. Surgery is only indicated when complications exist. In general, most splenic infarctions do not require surgical treatment. Indications for surgical treatment include sepsis, abscess, hemorrhage, and pseudocyst formation (5). Prognosis varies depending on the process responsible for the splenic infarction.

Various mechanisms have been described as responsible for splenic infarction in the course of acute pancreatitis. The most frequently cited is splenic vein thrombosis, be it by direct extension of the local inflammatory process, by the hypercoagulability state induced by pancreatitis, or both (1). Direct compression of the splenic vessels, including the splenic parenchyma by a pseudocyst of the pancreatic tail, has been considered a cause responsible for infarction of the spleen (2). Compression of the splenic artery, inflammation of its wall, or permanent arterial spasm may be other mechanisms in the development of splenic infarction. Angiographic studies of patients with acute pancreatitis have detected ischemic changes such as diffuse spasm and the obstruction of large splenic vessels, including the splenic artery in 41% of cases, this incidence being closely related to Ranson’s score (3).

In the first case described, the stenosis of the splenic artery demonstrated by CT appears to be the mechanism responsible for the development of splenic infarction. This stenosis may be secondary to inflammatory changes in the wall of the artery, or even to direct compression of the inflamed pancreatic tissue. With the possibility that pancreatitis and splenic infarction were secondary to embolism from atrial fibrillation in this patient, the hypothesis already described in the literature (9,10) seems improbable. This improbability is based on the anticoagulation status of this patient, an analysis of the tomographic images, and the existence of gallstones as responsible for the etiology of pancreatitis. In the second case it a hypothesis is difficult to establish. Nonetheless, in view of the lack of evidence regarding the involvement of splenic vessels, an increase in the intravascular coagulability of splenic vessels must be considered as the factor responsible for the multiple areas of splenic infarction. The delay in hospital admission allowed no follow-up study, with the diagnosis of splenic infarction being made on the day of admission.

Based on current knowledge, it seems advisable to add splenic complications to the serious extrapancreatic complications of acute pancreatitis, and to pay greater attention to potential spleen changes in this context. A CT scan is highly useful in the detection and follow-up of splenic complications in acute pancreatitis.

REFERENCES