Hemoperitoneum secondary to a spontaneous rupture of the spleen mimicking a duodenal perforated ulcer: A case report

Key words: Spontaneous rupture of the spleen. Splenomegalia. Smoking habit. Acute abdomen.

Dear Editor,

Spontaneous rupture of the spleen without traumatic antecedent is a rare entity and it is usually related with the presence of pathological spleens, mainly associating with tumor, infectious or inflammatory diseases (1). We report a case of spontaneous rupture of a non-pathologic spleen in a patient with splenomegalia attributed to smoking habit.

Case report

A 40-years old male was admitted to the Emergency Department of our institution for acute pain located in epigastrium and left hypochondrium during the last 12 hours before admission. The patient complained of usual epigastric pain, which improved after taking omeprazole. The patient did not refer traumatic events in the last weeks. He smoked 2 packs a day and presented a previous clinical history of polyglobulia that has been already studied and attributed to smoking habit, having ruled out polycythemia vera after bone marrow study. The patient did not refer alcoholic habit. On physical examination, the patient presented a body mass index (BMI) of 23.8 kg/m², a heart rate of 115 bpm and the abdominal examination showed a pain located in the left upper quadrant with signs of peritonism. Laboratory data revealed a white blood cell count of 18,540 x 10³/µl, hemoglobin of 17.2 g/dl, C-reactive protein of 2.12 mg/dl and lactate 41 mg/dl, as only relevant findings. An abdominal CT scan showed hepatomegalia with steatosis and perihepatic and perisplenic free fluid (Fig. 1). Despite there was no evidence of pneumoperitoneum, the history of heartburn, abdominal examination with generalized peritonitis and free fluid observed at CT scan, perforation of duodenal ulcer was considered as the first diagnostic option. It was decided to perform an exploratory laparoscopy, aiming abundant hemoperitoneum and splenomegalia with spontaneous rupture of the inferior and medial pole of the spleen. A splenectomy was performed with stapling and section of the splenic vessels through Echelon Flex stapler® (Ethicon

Fig. 1. Abdominal CT scan. Hepatomegalia with signs suggestive of liver steatosis and perihepatic and perisplenic free fluid.
EndoSurgery®). The resected piece was extracted through a small left subcostal incision and a Blake drain was placed in the splenic bed. The patient recovered satisfactorily and was discharged on the 4th postoperative day. Pathologic examination showed a normal spleen.

Discussion

Spontaneous rupture of the spleen without a traumatic history is an infrequent entity, but it is usually related with the presence of pathologic spleens, mainly associated with neoplastic, infectious or inflammatory pathologies. The etiopathogenic mechanism of spontaneous splenic rupture has not been elucidated yet, but it has been postulated that many mechanisms are involved, being probably splenomegaly the most important of them, as happens in our patient. Other factors associated with spontaneous rupture of the spleen are splenic infarction, neoplastic or hematological splenic affection, cytoreductive chemotherapy... (1). It has been estimated in less than 7%, the rate of spontaneous ruptures of the spleen appearing in histologically normal visceras (2). Spontaneous rupture of a pathologic spleen is much more frequent and may occur in all the disorders that induce splenomegaly: hematological (policitemia vera, hemolytic anemia, coagulation disorders, hemophilia, Waldenström macroglobulinemia), infectious (infectious mononucleosis, cytomegalovirus, paludism, bacterial endocarditis, typhoid fever, Q fever, splenic tuberculosis, brucellosis, acute pancreatitis, Gaucher disease, Ehrles-Danlos disease, Niemann-Pick disease, portal hypertension, pregnancy, postendoscopia, dialysis, spleen alterations (cysts, hemangiomas, infarction, abscess, splenic artery aneurysms, cavernous hemangioma) (1).

Any cause that increases the pressure in the splenic venous territory and determines splenomegaly might favor the splenic rupture, considering this a risk factor for spontaneous rupture by itself (3). It is noted that any cause favoring splenic compression, such as the contraction of the diaphragm or the abdominal muscles, as in the mechanism of Valsalva or cough, might favor the spontaneous rupture of the enlarged spleen (1). In a smoker patient cough is a common phenomenon, simply because of irritation of the upper airway, and this could have been one of the causes that might trigger the spontaneous rupture of the spleen in the patient we present.

The association of polycythemia, splenomegaly and hepatomegaly, is characteristic of polycythemia vera, but in our patient this entity was ruled out by bone marrow study and finally all these signs were attributed to be secondary to the smoking habit. Polycythemia is defined as an increase in the red cell mass. It is characterized by an increase in the number of erythrocytes and/or the amount of hemoglobin. Poliglobulia is diagnosed when the hemoglobin level overcomes 18.5 g/dL in males and 16.5 g/dL in females. Tobacco causes tissue hypoxia secondary to carboxyhemoglobin and this triggers an increase in erythropoiesis and secondarily an increase in the red cell mass, which in turn stimulates the splenic hemocatheresis, determining the splenomegaly (4). Smoking has been also associated with non-alcoholic fatty liver disease, causing hepatomegaly and hepatic steatosis (5). Moreover, it is well known that smoking reduces the pressure of the lower esophageal sphincter, favoring the appearance of gastrointestinal reflux disease (6).

The occurrence of sudden epigastric abdominal pain with signs of peritonism, in a patient with clinical history suggestive of gastritis, should continue to guide drilling of duodenal ulcer as the first diagnostic option, even though free intraperitoneal fluid is observed at the imaging tests, but no pneumoperitoneum. However, spontaneous rupture of the spleen should be included in the differential diagnosis of this condition, especially in patients with previously known splenomegaly, but also in smokers.

Jaime Ruiz-Tovar, Gustavo Díaz, David Alias, Montiel Jiménez-Fuertes and Manuel Durán

Department of General Surgery and Digestive Diseases. Hospital Universitario Rey Juan Carlos. Móstoles, Madrid. Spain

References