Simultaneous Rupture of the Liver and Spleen in a Patient on Warfarin Therapy: Report of a Case

METIN KAPAN1, SELIN KAPAN1, ILHAN KARABICAK1, and ISIL BAVUNOGLU2

Departments of 1Surgery and 2Emergency, Cerrahpasa Medical Faculty, Istanbul University, Istanbul, Turkey

Abstract
Although there are many reports describing spontaneous rupture of either the spleen or the liver, the simultaneous rupture of both organs is a rare event, especially during anticoagulant therapy. We report a case of spontaneous rupture of the spleen and liver in a patient on warfarin therapy for deep venous thrombosis.

Key words Spleen · Liver · Rupture · Warfarin therapy

Introduction
The spontaneous rupture of an abdominal organ without any underlying pathology is rare. Spontaneous splenic rupture is associated with many conditions, including hematological diseases, splenic infarcts, viral or bacterial infections, pancreatitis, rheumatologic diseases, pregnancy, and occasionally, anticoagulation therapy.1 Most cases of spontaneous liver rupture are associated with pregnancy, although there are some reports of liver rupture occurring during anticoagulation therapy.2 Therapeutic doses of oral anticoagulants have been associated with spontaneous rupture of apparently normal abdominal viscera, but simultaneous rupture of the spleen and liver is a rare event.3

Case Report
A 32-year-old man was transferred to our emergency unit from a district hospital, for investigation and treatment of acute abdomen. On admission he had a high fever and complained of severe abdominal pain. He had been taking warfarin sodium for 2 weeks to treat a deep venous thrombosis, but there was no apparent history of abdominal trauma. He had been given blood and fresh frozen plasma transfusions with broad-spectrum antibiotics for 3 days in the intensive care unit of the district hospital, but because his general condition continued to deteriorate, he was transferred to our emergency unit. Physical examination revealed generalized rigidity over the abdomen with rebound tenderness in all quadrants. His blood pressure was 120/80 mmHg; pulse rate, 96/min; and respiratory rate, 20/min. Laboratory data were as follows: hematocrit, 23%; leukocytes, 20 800/ml; prothrombin time (PT), 19%; PT-INR, 1.65; and activated partial thromboplastin time, 41%. Ultrasonography showed a 16-cm mass in the left lower lobe of the liver, suggesting an abscess or hematoma, with free fluid in the abdomen (Fig. 1). There was also a subcapsular intraparenchymal collection of fluid and heterogeneity, suggesting an infarct or hematoma in the spleen. Computed tomography of the abdomen showed a splenic hematoma(Fig. 2a), a fluid collection, 6 cm in diameter, in the lower segment of he left hepatic lobe (segment 3), and accumulation of free fluid in the abdominal cavity, suggesting splenic and hepatic rupture (Fig. 2b,c). To exclude any underlying concomitant disease, various blood tests and serological tests were done. The results of serological tests for hepatitis A, B, and C, Brucella abortus, cytomegalovirus, Epstein-Barr virus, and Venereal Disease Research Laboratory-Rapid Plasma Reagin were negative. Only the C-reactive protein level was elevated (39.3). The levels of immunoglobulin (Ig) A, IgC, nuclear antibodies, rheumatoid factor, Complement 3, Complement 4, antithrombin III, protein C, and Protein S were all within the normal range. No Plasmodium was detected in the peripheral smear of blood. Thus, we concluded that the rupture of the spleen and liver was not caused by any condition other than the anticoagulation therapy. After rapid intrave-
nous fluid and blood resuscitation, we performed an exploratory laparotomy, which revealed a large volume of hemorrhagic fluid within the abdomen. The spleen was enlarged, to $20 \times 30 \times 20$ cm, and ruptured through its posterior surface. We also found a ruptured hematoma, $10 \times 15$ cm in size, in the third segment of the left lobe of the liver. There was no congestion in the splenic or hepatic veins or in the inferior vena cava and there was no sign of collateral formation suggesting portal hypertension. After the aspiration of about 1800 ml hemorrhagic fluid, we performed splenectomy (Fig. 3) and an atypical resection of the third segment of the left hepatic lobe with placement of an absorbable gelatine sponge, $7 \times 5 \times 1$ cm (Spongostan standard, Johnson & Johnson, Skipton, UK) in the hepatic bed for hemostasis. After irrigating the abdominal cavity, we inserted a silicon drain, attached to closed suction, into the splenic region and closed the incision.

Histopathological examination of the resected specimens revealed a spleen enclosed in a thickened capsule with underlying coagulation necrosis. Red pulp in the peripheral splenic tissue was wide with thick-walled dilated sinusoids. There was a pale area, $16 \times 15$ cm, in the outer part of the spleen, caused by infarction. An organized hematoma, $21 \times 13.5 \times 6.5$ cm, made up of diffuse erythrocytes and fibrin, was also seen. Macroscopic examination of the resected hepatic tissue revealed a hepatic segment, $9.5 \times 6 \times 5$ cm in size, with a $2.5 \times 4.0 \times 7.0$-cm hematoma beneath the surface.

**Fig. 1.** Ultrasonographic appearance of the perihepatic fluid

**Fig. 2a–c.** Computed tomography. **a** A splenic hematoma. **b** A hepatic and splenic hematoma. **c** Free fluid in the Douglas pouch.