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

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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-

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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 × 30 × 20 cm, and ruptured through its posterior surface. We also found a ruptured hematoma, 10 × 15 cm in size, in the third segment of the left lobe of the liver. These 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 × 5 × 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.

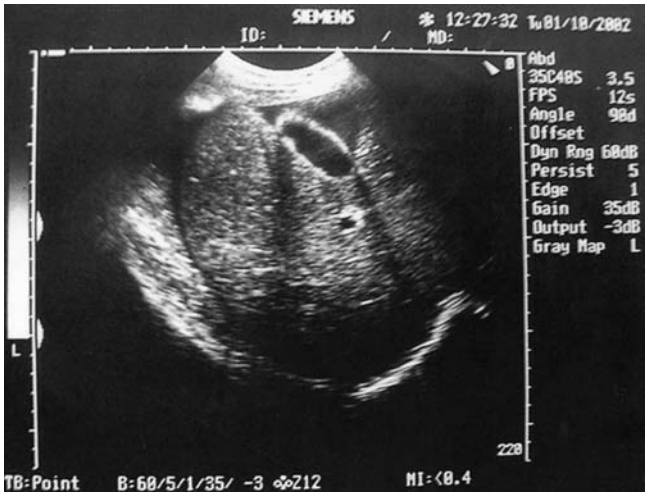

Fig. 1. Ultrasonographic appearance of the perihepatic fluid

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 × 15 cm, in the outer part of the spleen, caused by infarction. An unorganized hematoma, 21 × 13.5 × 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 × 6 × 5 cm in size, with a 2.5 × 4.0 × 7.0-cm hematoma beneath the surface.

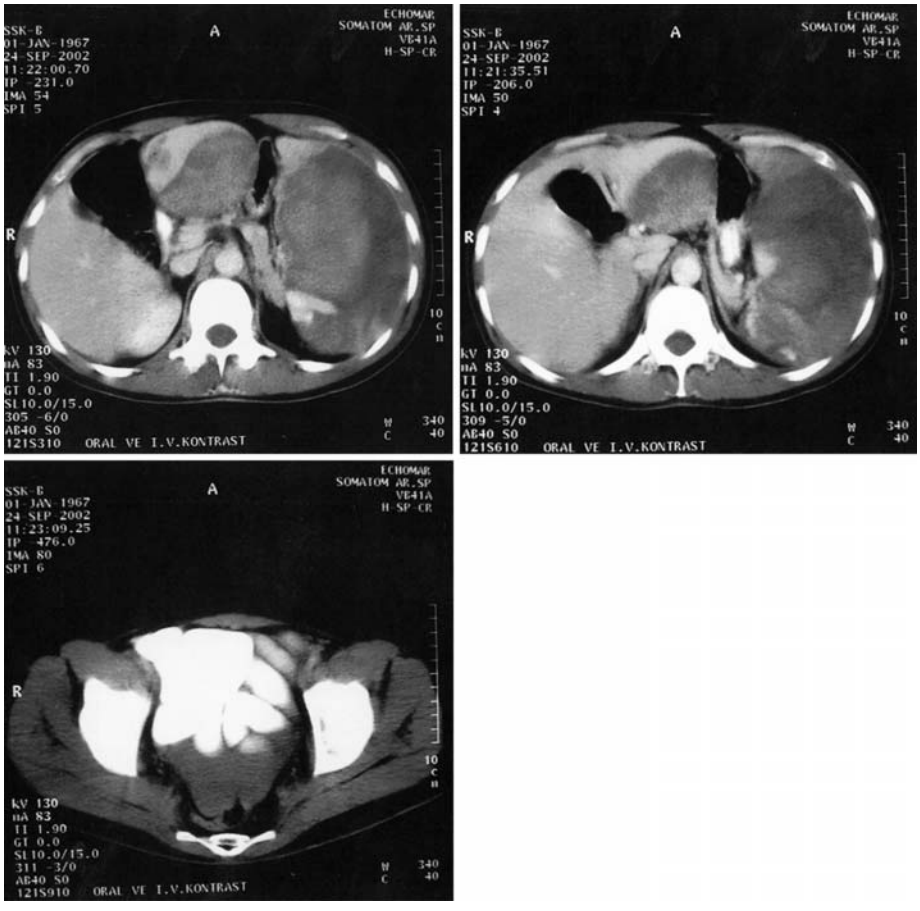

Fig. 2a–c. Computed tomography. **a** A splenic hematoma. **b** A hepatic and splenic hematoma. **c** Free fluid in the Douglas pouch

Fig. 3. Resected specimens of splenic and hepatic tissue

Organization of granulation tissue, with fibrin and erythrocyte exudation, was evident in the capsule of the liver.

After spending 5 days in our intensive care unit for respiratory problems, the patient was transferred to a ward. He was discharged on the 9th postoperative day after an uneventful recovery and is now well.

Discussion

Spontaneous rupture of the spleen or liver as separate entities has often been described; however, the simultaneous rupture of these organs is rare.[4,5] The primary risk factors for spontaneous splenic rupture (SSR) are splenic infiltration by hematological disease, splenic infarct, coagulation disorders, male sex, adulthood, and severe splenomegaly. Hematological cancers such as acute lymphocytic leukemia, acute myelogenous leukemia, chronic myelogenous leukemia, Hodgkin's lymphoma, viral infections with Epstein-Barr, hepatitis, human immunodeficiency virus, and bacterial infections such as pneumonia, meningococcemia, endocarditis, tuberculosis, dengue fever, Legionnaire's disease, Q fever, fungal infections, pancreatitis, rheumatologic diseases (such as systemic lupus erythematosus), immune thrombocytopenic purpura, rheumatoid arthritis, polyarteritis nodosum, Wegener's granulomatosis, and pregnancy are included in the wide spectrum of conditions predisposing to SSR.[1,5–8] Worldwide, the most common infectious agent associated with SSR is malaria.[1,9] There are also reports describing SSR in patients with amyloidosis.[10,11] Some procedures, such as electroconvulsive therapy, implantation of automatic defibrillators, shock wave lithotripsy, colonoscopy, and transesophageal echocardiography, are also associated with SSR.[1] In most of these conditions or diseases, the spleen is directly affected and the predisposing factors for rupture are increased size, inflammation, and changes in structural integrity.[12]

Rupture of the liver without trauma is also very unusual and most cases are associated with pregnancy and hepatic tumors.[5] Subcapsular liver hematomas and ruptures are rare fatal complications of hemolysis, elevated liver enzymes, and low platelets syndrome (HELLP).[2] Despite the wide spectrum of conditions predisposing to rupture of the spleen or liver individually, simultaneous rupture of both these organs is almost always caused by anticoagulation therapy.[4,13] By performing all of the known blood and serological tests, we excluded any pathological condition as the cause of rupture of these two organs. Bleeding is the leading complication of anticoagulant therapy, with an incidence of 20%, and may occur in unusual locations, resulting in complex and life-threatening sequelae.[3,13] The most common site of anticoagulant-related bleeding resulting in acute abdomen is the wall of the small intestine,[3] which occasionally causes rupture of the abdominal viscera. The exact cause of SSR during thrombolytic or anticoagulant therapy is unknown, but the exacerbated anticoagulation in patients on anticoagulant therapy would suggest that this phenomenon is most likely related to excessive derangement of the hemostatic mechanism.[14] The toxic effects of coumarin-derivative anticoagulants on the liver have been extensively studied. Autopsies of humans who died from generalized hemorrhage have revealed changes in the liver ranging from dilatation of the sinusoids and structural disorganization to frank parenchymal hemorrhage and subcapsular ecchymoses.[3] Atraumatic hepatic bleeding is usually preceded by subcapsular hematoma with subsequent rupture of the capsule.[14]

In conclusion, it is well known that warfarin sodium can cause severe bleeding or rupture of the abdominal viscera even when administered in the therapeutic range. Therefore, the possibility of splenic or liver rupture should be considered if a patient on thrombolytic or anticoagulant therapy presents with acute severe abdominal pain and shock, regardless of a history of trauma or known risk factors for spontaneous rupture.

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