SPONTANEOUS RUPTURE OF THE SPLEEN – A RARE CASE

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The study presented two cases of spontaneous rupture of the spleen. Both patients were treated at the Department of General, Oncological, and Endocrinological Surgery. The first patient underwent splenectomy. The postoperative course was uneventful. The second patient was subject to pharmacological treatment because of non-acceptance to surgical intervention. The patient died during the following hospitalization. Both patients received oral anticoagulants, due to atrial fibrillation and an implanted heart valve. History of previous trauma was absent. The study presented a detailed description of the clinical course of splenic rupture.

Key words: spontaneous splenic rupture, oral anticoagulants

Rupture of the spleen is a severe, life-threatening condition. The most common cause of splenic rupture is attributed to thoracic and abdominal injuries. Spontaneous splenic rupture is less often observed in cases of infectious diseases (malaria, Q fever, AIDS), neoplastic diseases (lymphomas, solid tumors) or anticoagulation therapy (1). In such cases the clinical course is similar to that observed during traumatic rupture. However, diagnosis in such cases is often a surprise to most physicians. Dominating symptoms include abdominal pain and hypovolemic shock. Patients often require emergency surgical intervention.

The study presented two cases of spontaneous splenic rupture treated at the Department of General, Oncological, and Endocrinological Surgery in Kielce.

CASE REPORTS

1. A fifty-year old male, non-smoking farmer (history number: 22348/2009 and 24565/2009) was admitted to the hospital, due to abdominal pain lasting several hours. Symptoms developed spontaneously and were not connected with meals. The following were not present: history of abdominal injury, nausea, vomiting and defecation disturbances. The patient was on oral anticoagulation for a period of one year, due to persistent atrial fibrillation. Additionally, he received metoprolol and propafenon. The physical examination was as follows: severe general condition, irregular heart rate, and low blood pressure (60/40 mm Hg), peripheral pulse was absent, the abdomen tense, without pathological resistance. Digestive tract and spontaneous intra-articular bleeding were not observed. Laboratory parameters were as follows: INR – 3.9, prothrombin index 29.6%, Hb – 8.77 g/dl, Ht – 25.3%, PLT – 529 K/µl, troponin-normal. The chest X-ray was normal, the plain abdominal film showed no abnormalities. The ultrasound examination showed an enlarged spleen (190 mm), with a non-homogeneous, echostructure and echogenicity, and presence of subcapsular fluid compartments. Near the lower pole of the spleen one observed a 20 mm layer of fluid. The patient was subject to contrast computer tomography which demonstrated an enlarged spleen with the possibility of its rupture, and
a 25 mm thick subcapsular hematoma. Peritoneal cavity fluid was also observed.

The patient received colloids and crystalloids, which lead towards blood pressure normalization. Oral anticoagulants were discontinued and 3 units of freshly frozen plasma (FFP) were transfused. On the second day of hospitalization the patient complained of severe abdominal pain with blood pressure reduction (90/60 mm Hg). The patient was qualified for emergency surgery. During the operation we observed a bloody content in the peritoneal cavity, a subcapsular hematoma of the spleen, and a ruptured splenic capsule. Splenectomy was performed. The histopathological examination result was as follows: fresh hemorrhagic lesions, with unchanged parenchyma. Due to hemoglobin reduction (7.5 g/dl) the patient received 3 units of FFP during the postoperative period. The INR value one day after surgery was 1.05. Due to the presence of atrial fibrillation the patient received a prophylactic dose of low molecular weight heparin (LMWH). The postoperative course was complicated by posterior tibial vein thrombosis of the left lower leg which required the administration of therapeutic doses of LMWH (6-th postoperative day). No other complications were noted. The patient was discharged from the hospital eight days after the operation with continuation of therapeutic (7 days) and prophylactic LMWH. The patient denied any thoracic or abdominal cavity injuries, prior to hospitalization.

One month after the operation the patient was once again admitted to the hospital, due to abdominal pain. The patient’s condition was stable; the physical examination showed no abnormalities; the postoperative wound was properly healed. The patient discontinued anticoagulation therapy without good reason. Laboratory parameters were as follows: Hb-11.9 g/dl, Ht – 35%, PLT – 1403 K/µl, prothrombin time – 13.7 s., prothrombin index – 94%, INR-1.2, APTT-33.9 s, D-dimers 508 µg/l (normal values ranging between 63-246 µg/l). Abdominal ultrasound showed complete recanalization of the posterior tibial left vein. LMWH therapy was once again initiated for a period of 14 days. The patient’s general condition improved and pain symptoms regressed. Control ultrasound showed lack of portal vein recanalization. The patient was discharged from the hospital with the recommendation to continue LMWH therapy, and eventual oral anticoagulation. However, the patient did not come in for follow-up and his fate is unknown.

2. A 55-year old patient (history number 7000/2010 and 16691/2010) on continuous dialysis, due to end-stage renal failure was admitted to the hospital because of abdominal pain, fever, and poor general condition. Symptoms were present for one week and were not connected with food consumption, or history of abdominal injury. The patient underwent previous surgery (no documentation): aortic valve implantation. The patient was on oral anticoagulation – acenokumarol. Laboratory parameters were as follows: INR - 2.9, urea – 109 mg/dl, creatinine – 7.96 mg/dl. Abdominal ultrasound showed an enlarged spleen 153x70 mm, with echostructure disturbances and solid-fluid areas. Therefore, computer tomography was performed demonstrating an enlarged spleen 160x150x90 mm with an area of infarction. The splenic parenchyma was location to an irregular hypodense mass, 90x65x60 mm in size, possibly corresponding to a hematoma. Fluid was present in the left pleural cavity and surrounding the spleen. The patient was qualified for surgery to which he did not agree. Oral anticoagulation was discontinued and the patient received LMWH prophylaxis. During hospitalization control ultrasound examinations showed an evolving splenic hematoma with fluid presence. The patient was discharged from the hospital at his own request after 16 days of hospitalization without consent for surgical intervention. The patient was readmitted to the hospital in August, 2010 due to abdominal pain and ultrasonographic presence of a splenic hematoma. The ultrasound examination showed an enlarged spleen (160 mm) with a hypoechochogenic mass, 29x27 mm in size (splenic infarction) and fluid with hyperechochogenic lesions-evolving hematoma. Laboratory parameters showed features of end-stage renal failure requiring systematic hemodialysis every other day. On the seventh day of hospitalization, before planned surgery the patient experienced sudden cardiac arrest. Resuscitation proved ineffective and the patient died.

DISCUSSION

Spontaneous rupture of the spleen is a rare disease entity. It is often observed as a compli-
cation of Q fever (2). Literature data described two cases connected with rickettsia infections. Symptoms are most often unspecific including headaches, fatigue, fever, dry cough and chest pain. Abdominal symptoms include nausea, vomiting, pain and diarrhea. The physical examination often reveals an enlarged liver. Considering the two described cases rickettsia infection symptoms were absent, and serological tests were not performed.

Polycystic disease is another cause of spontaneous splenic rupture. The parenchymal organ contains multiple blood-filled compartments (2). The disease is asymptomatic, often accidentally diagnosed. Causative factors include drugs and toxins, such as alcohol, corticosteroids, oral contraception, tamoxifen, azathioprin, and androgens. There is also a connection between immunological system disturbances (in patients with AIDS). The histopathological examination result showed no signs of cystic disease.

Spontaneous splenic rupture was also described in case of a patient with histiocytosis (1). The rupture may be caused by the accumulation of histiocytes, which damage the architecture of the organ. Literature data described one case of spontaneous splenic rupture during anticoagulation therapy. Bodaoui et al. (4) presented a patient who was on oral anticoagulation, after artificial heart valve implantation. The patient underwent splenectomy. However, the description of the mechanism of splenic rupture during anticoagulation therapy was not found. Chun et al. (5) described another patient on oral anticoagulation with diagnosed splenic rupture.

Spontaneous splenic rupture due to splenic metastases is another problem (6). In 2003, Georg et al. (7) presented a group of 670 patients with splenic lesions from which 41 were diagnosed with atraumatic splenic rupture (7). Most patients were diagnosed with malignant splenic cancer. Both our patients were clinically free of neoplastic disease. Considering Polish literature Barałkiewicz et al. (8) presented a case of spontaneous splenic rupture as a complication of infectious mononucleosis. Jankowski et al. (9), described a patient with spontaneous splenic rupture after myocardial infarction treated by means of streptokinase and heparin. In 2009, the British Journal of Surgery presented a meta-analysis comprising 632 publications describing 845 patients with spontaneous splenic rupture. Two large patient groups were identified: one with pathological splenic lesions responsible for atraumatic rupture (93%), and one with idiopathic splenic rupture (7%) (10). In case of both our patients imaging examinations showed no signs of cystic disease.

REFERENCES


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