SPONTANEOUS SPLENIC RUPTURE SECONDARY TO RIVAROXABAN: RARE BUT RAISING

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ABSTRACT

A 68-year-old male presented to our hospital with sudden onset of left-sided chest and abdominal pain. The patient was previously treated for left lower lobe pneumonia and reported no history of the chest or abdominal trauma. His medical history included atrial fibrillation that was managed by rivaroxaban 20 mg once daily. Computed tomography of the abdomen demonstrated a splenic haematoma. The patient rapidly deteriorated and developed hypovolemic shock. Emergency laparotomy revealed haemoperitoneum of 3500 mL, and the ruptured spleen warranted splenectomy. The postoperative course was complicated by a pancreatic fistula formation that eventually resolved.

KEYWORDS spontaneous splenic rupture; rivaroxaban;

Introduction

Spontaneous splenic rupture (SSR), due to the use of novel oral anticoagulants (NOAC), has been recently reported in the literature and became a new phenomenon [1, 2, 3]. It occurs in a non-compromised spleen and meets Orloff and Peskin criteria (no history of trauma before the operation, no perisplenic adhesions or scarring, no evidence of disease that can affect spleen adversely and normal microscopic and macroscopic appearance of the spleen) [4].

Rivaroxaban is a selective inhibitor of factor Xa blocking thrombin production indicated for the prevention of thromboembolism in non-valvular atrial fibrillation [5]. It serves as an alternative to warfarin. Here we present a case of SSR in a 68-year-old gentleman who was on rivaroxaban along with low-dose aspirin.

Case Report

A 68-year-old man presented to the emergency department (ED) of our hospital with a productive cough, left-sided chest pain and shortness of breath. His medical background included ischaemic heart disease, atrial fibrillation and benign prostate hyperplasia. His medications included low-dose aspirin, atorvastatin, metoprolol, ramipril, and digoxin. Also, he was also taking rivaroxaban 20 mg daily for anticoagulation (added one year ago after the diagnosis of non-valvular atrial fibrillation was made). There was no history of bleeding or clotting disorders.

The diagnosis of left lower lobe pneumonia was established by the chest radiograph (Fig.1). The treatment with ceftriaxone and clindamycin was initiated in the community. After 48 hours, he represented with sudden onset of left-sided chest pain and left flank abdominal pain. He denied any history of abdominal or chest injury. The pain was of sudden onset, sharp stabbing in the left flank and radiated to the left side of the chest. On clinical examination, his abdomen was soft but mildly tender in the left hypochondrium.

The patient’s condition rapidly deteriorated with a drop of systolic blood pressure (sBP) from 120 to 80 mm Hg. On haematological tests, his serum haemoglobin reduced from 129 to 90 g/L. Prothrombin time (PT) was 19 seconds (normal range, 9 – 13 seconds), activated partial thromboplastin time (APTT) was 35 seconds (normal range, 24 – 39 seconds), international normalized ratio (INR) was 1.6 (normal range, 0.9 – 1.2) and platelet count was 210 x 10^9/L (normal range, 140 – 400 x 10^9/L).
He suddenly became clammy and pale. Differential diagnosis at this stage included rupture of the abdominal aortic aneurysm or dissection. The chest radiograph (Fig. 2) and computed tomography (CT) scan (Fig. 3, 4) showed massive left-sided pleural effusion and a splenic haematoma of 13.8 x 8.5 x 7.8 cm in size with blood in the abdominal cavity.

Rapid transfusion of three packed red blood cells, two fresh frozen plasma and infusion of prothrombin 5000 IU were initiated after consulting with the haematologist. The patient was transferred to the operation theatre for emergency surgical intervention. During laparotomy, 3500 mL of blood and clots were evacuated. The spleen appeared to have a capsular rupture at the upper pole with a subcapsular haematoma. Splenectomy was performed, and the left intercostal drain was inserted at the same time for effusion. The patient was transferred to the intensive care unit and subsequently discharged from the hospital on the 8th postoperative day.

On the 10th postoperative day, he developed an abdominal collection of the fluid with a high content of lipase with no pancreatic duct injury on magnetic resonance cholangiopancreatography (MRCP). It was drained with a pigtail catheter and eventually reduced after treatment with octreotide.

Discussion

Due to ongoing and more widespread use of NOAC, the frequency of bleeding complications tends to grow accordingly. The literature sources show that spontaneous splenic ruptures caused by anticoagulants and antiplatelet agents are not infrequent, composing up to 9.1% of all spontaneous splenic ruptures [6, 7]. The causes of pathologic splenic rupture, as compared to spontaneous one, are identifiable and range from abdominal injury and infections (infectious mononucleosis, babesiosis, bacterial endocarditis [8, 9, 10]) to pregnancy, haematological and autoimmune conditions such as systemic lupus erythematosus [11, 12, 13].

Some authors suggest that the diagnosis of splenic rupture should be kept in mind when dealing with the patients on any anticoagulation therapy and presenting with left-sided abdominal or lower chest pain, or even chest infection [14]. However, there is still a bias if the combination of Rivaroxaban with aspirin and/or lipid-lowering agents increases bleeding potential. It was observed that the vitamin K antagonists (warfarin) or low molecular weight heparins (LMWH) caused a spontaneous splenic rupture in the otherwise healthy patients [15, 16]. Ri-

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varoxaban, as a selective inhibitor of factor Xa, currently does not have antidotes that can stop the bleeding at the early stages. Along with the recent reports, our case provides another evidence for uncontrolled bleeding potential in a patient on rivaroxaban and low-dose aspirin. The clinician should be particularly cautious when starting the patient on the combined anticoagulation and antiplatelet therapy.

Another consideration may be paid to possible bridging therapy for the patients on such combined regimen, e.g. withholding rivaroxaban temporarily or switching to a more controlled anticoagulant during the admission to hospital or intensive treatment. The indications to such bridging for rivaroxaban, however, should be elaborated further. As rivaroxaban and other xabans do not have certain tests to check the risk of bleeding, safe use of these medications is recommended.

Conclusion
In the patients on NOAC, even in the absence of abdominal trauma, one should have a high index of suspicion of solid organ’s rupture like the spleen. Spontaneous splenic rupture may present late with severe haemodynamic compromise and can pose a diagnostic dilemma.

Early surgical intervention is potentially a lifesaving measure in this regard. Further search for and application of reversal of xabans in the clinical scenario alike is needed.

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Competing Interests
The authors declare no conflict of interest.

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