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Case Report

A case of spontaneous splenic rupture causing hemorrhagic shock in a 74-year-old on Apixaban

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ABSTRACT

Spontaneous splenic rupture is a rare, life-threatening cause of atraumatic intra-abdominal hemorrhage and may occur in patients receiving anticoagulation. We present a 74-year-old woman with atrial fibrillation treated with apixaban who presented with one day of worsening abdominal pain and near-syncope without preceding trauma. She was hypotensive on arrival, with hemoglobin of 5.8 g/dl, elevated lactate, and acute kidney injury. Computed tomography (CT) of the abdomen and pelvis demonstrated a lower-pole splenic laceration with active contrast extravasation, subcapsular hematoma, and hemoperitoneum. She underwent immediate resuscitation with intravenous fluids, packed red blood cells, and prothrombin complex concentrate for anticoagulant reversal, followed by emergent exploratory laparotomy. Operative findings confirmed hemoperitoneum and an actively bleeding spleen with capsular avulsion; splenectomy was performed. Her postoperative course included intensive care monitoring, additional transfusion support, temporary pharmacologic thromboprophylaxis that was discontinued because of incisional bleeding, and post-splenectomy vaccination. She was discharged on postoperative day 7 without anticoagulation pending cardiology follow-up and remained clinically well at subsequent postoperative visits. This case highlights spontaneous splenic rupture as an important diagnostic consideration in anticoagulated patients presenting with unexplained abdominal pain, anemia, or hemodynamic instability despite no history of trauma. Prompt imaging, anticoagulant reversal, resuscitation, and definitive operative management are critical to survival, while post-hemorrhage anticoagulation decisions require individualized multidisciplinary assessment.

Keywords: Spontaneous splenic rupture, Traumatic splenic rupture, Apixaban, Direct oral anticoagulants, Hemorrhagic shock, Splenectomy

INTRODUCTION

Spontaneous splenic rupture (SSR) is a rare and life-threatening condition, most commonly associated with trauma but also observed in the setting of hematologic malignancies, infections, and anticoagulation therapy.[1] The use of direct oral anticoagulants (DOACs), such as apixaban, has increased significantly. Apixaban works by selectively inhibiting Factor Xa, a key enzyme in the coagulation cascade, thereby preventing clot formation. It is indicated for the treatment of deep vein thrombosis (DVT) and pulmonary embolism (PE), reduction in the risk of recurrent DVT and PE, prophylaxis of DVT following certain orthopedic surgeries, and prevention of stroke and systemic embolism in nonvalvular atrial fibrillation. However, these agents also increase the risk of major bleeding complications, including visceral organ hemorrhage.[2] SSR in the setting of apixaban presents

unique diagnostic and therapeutic challenges. Prompt recognition, resuscitation, and often surgical intervention are necessary to prevent mortality. Here, we present the case of a 74-year-old female who developed a spontaneous splenic rupture while on apixaban.

CASE REPORT

A 74-year-old Asian female presented to the ED with one day of worsening abdominal pain and near-syncope. She had a past surgical history of a radical nephrectomy for renal cell carcinoma (RCC) and a complex medical history, including atrial fibrillation, coronary artery disease, chronic kidney disease, diabetes mellitus, hemolytic anemia, hypertension, and hypothyroidism. She denied any trauma. A home blood pressure (BP) of 70/54 mmHg was reported. Her home meds included apixaban, amlodipine, atorvastatin, metoprolol, torsemide, hydralazine, losartan, synthroid and lantus. In the ED, her BP was 90/60 with a hemoglobin of 5.8 g/dl, BUN/Cr of 38/1.8 (baseline Cr 1.0), and lactate of 5.92 mmol/l. A computed tomography of the abdomen and pelvis (CT A/P) demonstrated a laceration of the lower pole of the spleen with active contrast extravasation, medial displacement of the spleen by a subcapsular hematoma, and peri-splenic hemoperitoneum extending into the abdomen and pelvis (Figure 1).

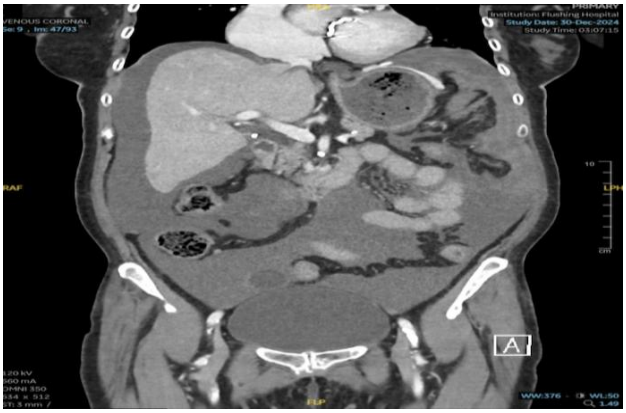

Figure 1: CTA shows a lower pole splenic laceration with active contrast extravasation and moderate hemoperitoneum.

She received 2 units of packed red blood cells (PRBCs), intravenous fluids, prothrombin complex concentrate (PCC), and was taken emergently to the OR. An exploratory laparotomy confirmed hemoperitoneum with an actively bleeding spleen with capsular avulsion. A splenectomy was performed. A Jackson-Pratt drain was placed, and the patient received 2 more units of PRBCs and 1 unit of platelets intraoperatively. She was transferred to the surgical intensive care, received additional blood products, antibiotics, and was extubated the following day. Her 24-hour post-operative labs showed lactate 1.29 mmol/l), hemoglobin up to 11.4 g/dl, her kidney function also improved with a Cr of 1.5. She was downgraded to the floor on postoperative day 4, started on Lovenox, but it was discontinued after bleeding from the midline incision was noted. She was discharged on postoperative day 7, on no anticoagulants, pending outpatient cardiology follow-up. Appropriate vaccines were administered. The patient presented for a follow-up on day 10 and day 25 doing well, but still on no anticoagulants as she had experienced again bleeding at the wound site when started on ASA two weeks after discharge.

DISCUSSION

The spleen is an intraperitoneal organ located in the left upper quadrant of the abdomen, protected posteriorly by the 9th to 11th ribs. It plays a major role in both hematologic and immunologic function, including the filtration of aged or damaged red blood cells, hematopoiesis in certain disease states, and the production of antibodies through its white pulp. The spleen is highly vascularized, primarily supplied by the splenic artery, a branch of the celiac trunk, which enters the hilum and branches extensively within the organ. Venous drainage occurs via the splenic vein, which joins the superior mesenteric vein to form the portal vein. Due to this rich vascular network and its friable parenchyma, the spleen is particularly susceptible to hemorrhage, in cases of trauma or spontaneous rupture, the latter of which is rare and often associated with underlying pathology or anticoagulant use.

Anticoagulants, i.e. DOACs, warfarin (Coumadin), aspirin (ASA), and low molecular weight heparins like enoxaparin (Lovenox) are estimated to be widely prescribed to over 8 million Americans.[3] DOACs offer advantages over warfarin, including fewer dietary restrictions and less monitoring, but bleeding risks persist-especially when used concurrently with CYP3A4 or P-glycoprotein inhibitors, or with antiplatelet agents.[4] Warfarin's narrow therapeutic window and interaction with numerous medications and foods necessitate close INR monitoring.[5] ASA, even at low doses, can significantly increase bleeding risk when combined with other anticoagulants.[6] Lovenox, while more predictable than unfractionated heparin, may accumulate in renal impairment, raising the risk of hemorrhagic events. Rarely, these bleeding complications can manifest as spontaneous splenic rupture, a potentially fatal and underrecognized consequence in anticoagulated patients.[7]

SSR is defined as atraumatic splenic hemorrhage that occurs without an external force or injury. It is most commonly associated with underlying pathologies such as hematologic malignancies (e.g., lymphomas, and leukemias), infectious diseases (e.g., Epstein-Barr virus, malaria), inflammatory disorders, and anticoagulant use.[1,8] In a systematic review by Renzulli et al approximately 30% of SSR cases were idiopathic, while over 70% had identifiable risk factors, with hematologic disorders being the most common.[1] SSR is rare compared to splenic trauma associated with blunt abdominal injury, which accounts for 40%–50% of intra-abdominal injuries following blunt trauma.[9] Splenic injuries have been

classified as grade I (minor capsular tears or small subcapsular hematomas) to grade V (completely shattered spleen or hilar vascular injury).[2] Management in the setting of SSR, as with traumatic cases, must be tailored to the patient's clinical stability, imaging findings, and comorbid conditions. High-grade with ongoing hemorrhage require typically splenectomy and non-operative management for the hemodynamically stable consisting of close observation, fluid resuscitation, transfusions and adjunctive angioembolization achieving success rates exceeding 90% in select cases.[10]

Although rare, SSR is a medical emergency and early detection of diagnosis is crucial, as complications include massive intra-abdominal hemorrhage, disseminated intravascular coagulation, rapid onset of hemorrhagic shock and multi-organ failure. Mortality rates are significant, ranging from 10–15% and higher if diagnosis and surgical management are delayed.[1]

In the elderly patients, SSR is a particularly life-threatening condition, as its presentation can be subtle and easily overlooked. Older adults are especially vulnerable due to age-related changes in splenic tissue, declines in renal and hepatic function, comorbidities, and frequent concomittant use of antithrombotic medications. The high mortality rate of SSR increases significantly in older adults due to delayed recognition and impaired physiologic reserve.[1,5] Notably, classic signs such as Kehr's sign or left upper quadrant pain may be absent in elderly patients, and presentations can mimic other abdominal or cardiovascular conditions, leading to misdiagnosis. Prompt imaging, typically with contrast-enhanced CT and early surgical consultation are critical for improving outcomes in this high-risk population. Polypharmacy, defined as the use of five or more medications, affects nearly 40% of adults over 65 and increases the likelihood of drug-drug interactions, altered drug metabolism, potentiating the effects of anticoagulants and serious bleeding events.[6,11,12] Our patient was on amlodipine, atorvastatin, metoprolol, torsemide, hydralazine, losartan, synthroid and lantus, in addition to apixaban for anticoagulation.

The literature supports that SSR is a rare, but recognized complication of anticoagulant use. A 2009 systematic review acknowledged increasing incidence with the widespread direct anticoagulants (DOAC) use.[1] Although DOACs are generally associated with a lower risk of major bleeding compared to warfarin, they have been implicated particularly in elderly patients or those with underlying splenic pathology. Reports by Kocael and Kwon of SSR in elderly patients on rivaroxaban, apixaban emphasize the importance of considering this diagnosis in anticoagulated patients presenting with unexplained abdominal pain or hemodynamic instability, even in the absence of trauma.[13] Nguyen et al highlighted the difficulty in early detection of SSR in non-traumatic setting, a point echoed by Hyun et al, as patients often present with shock and vague symptoms, making the diagnosis elusive.[14,15]

In addition to splenic rupture, it must be stressed that anticoagulation has been linked to spontaneous bleeding in various sites, including the retroperitoneum, gastrointestinal tract, and muscle compartments. Retroperitoneal and rectus sheath hematomas, though rare, can present without trauma and carry high morbidity, particularly in elderly patients.[16] Intracranial hemorrhage remains the most serious complication, especially in those with hypertension or cerebral amyloid angiopathy.[17] These risks highlight the need for careful monitoring and individualized assessment in anticoagulated patients.

This case demonstrates the challenges in balancing the need for anticoagulation with the risk of hemorrhage. The central controversy remains in how to manage anticoagulation following such bleeding episodes. While guidelines from the Anticoagulation Forum recommend resuming anticoagulation within 7–14 days in high-risk thrombotic patients, this recommendation is based on limited data and excludes patients with visceral hemorrhage.[18] Witt et al conducted a meta-analysis suggesting that resumption is generally safe, but cautioned that individualized assessment is critical.[19]

CONCLUSION

This case illustrates that SSR may be unexpected in patients on DOACs, but can be a real threat, especially in the elderly or those with impaired renal function. SSR should be in the differential diagnosis of those presenting with abdominal pain and hypotension, even in the absence of trauma. Early imaging, rapid resuscitation, and surgical intervention are essential for survival. Multidisciplinary collaboration is critical in managing the balance between bleeding and thrombotic risks, particularly when anticoagulation is necessary for conditions like atrial fibrillation. The case challenges clinicians to weigh thrombotic and bleeding risks in nuanced clinical contexts. Further research should focus on anticoagulation alternatives or procedural strategies like catheter ablation to reduce reliance on long-term anticoagulation, and development of specific risk-stratification tools for SSR prevention and safe anticoagulation resumption algorithms tailored to decrease visceral bleeding events.

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