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Research Paper

The case of left loin pain and aneurysm of the splenic artery

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Abstract

True splenic artery aneurysms (SAA) are rare, but potentially fatal, lesions. They account for 60% to 70% of patients diagnosed with visceral artery aneurysms. They should be differentiated from pseudo ones since the etiology is different. They risk of rupture is high if diameter is > 2 cm and in child-bearing years. In this case report; we describe an encounter of SAA in a patient presenting with severe left loin pain and shock due to acute pyelonephritis. The aneurysm was 1.2 cm and was partially thrombosed and calcified indicating its stability and chronicity. The etiology, types and management of SAAs is discussed.

Keywords: abdominal pain, aneurysm, pseudoaneurysm, rupture, splenic artery.

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INTRODUCTION

True splenic artery aneurysms (SAA) are rare, but potentially fatal, lesions. They are the third most common type of abdominal aneurysm, after aneurysms of the aorta and of the iliac artery, and account for 60% to 70% of patients diagnosed with visceral artery aneurysms (VAAs) [1]. Hepatic artery aneurysms account for 20% of VAAs and the celiac/mesenteric ones represent 10% only [2]. Since most SAAs remain asymptomatic; the true prevalence in general population is uncertain. Previous studies; have reported 0.098% in a large series to 0.78% in a study of 3600 arteriogram to 10.4% in autopsy series on patients > 60 years with special attention given to the splenic artery [3-5]. SAAs are 4 times more common in women but approximately 3 more likely to rupture in men [6]. A recent retrospective study found that 78% of SAAs occur in women, generally related to increased incidental or symptomatic findings that coincide with use of ultrasonography in pregnancy. The mortality after rupture of SAA in non-pregnant patients ranges from 25% to 40%. However, among pregnant patients, the maternal mortality due to SAA rupture was 75%, and fetal mortality was 95% [7]. In this case report, we present a woman with left loin pain associated with such SAA and discus its management.

THE CASE:

A 45-year-old woman presented with left flank pain for 2 weeks. The pain was dull aching in character and was associated with low grade fever, nausea and vomiting and frequency as well as burning micturition. She denied past medical illnesses and was a mother of 5 children, the last of whom was 8 years old. On her initial physical examination; she was conscious, oriented X3 and in distress of pain. She was temperature of 38 C, tachycardia and low blood pressure at 90/60 mm Hg. She did not have lymphadenopathy, goiter, jugular venous distension or oedema. Systemic examination did not show abnormality except for left loin and suprapubic tenderness. Laboratory investigations showed normal peripheral leucocytic and platelets counts. Hemoglobin was 10 g/L with normal MCV. ESR and CRP were high. Serum sugar, urea, creatinine, electrolytes and liver functions were normal. Urine routine and microscopy showed 1(+) protein with excess WBCs/HPF and 25 RBCs/HPF. Chest x-ray and ECG were normal. Abdominal and pelvic ultrasound did not show kidney abnormality yet disclosed a calcified hypoechoic cystic lesion medial to the spleen. CT angiogram of the abdominal aorta, using 128 slice MDCT scanner; confirmed a 1.2 cm aneurysm of the distal splenic artery with wall calcification and partial thrombosis (Figure 1, 2). The patient was treated with intravenous saline to maintain her blood pressure and Meropenum 1 g IV every 12 hours after urine and blood cultures. The latter disclosed urosepsis with significant growth of E. coli which was sensitive to Meropenum. Her general condition had improved and her left flank pain had disappearance of after 2 weeks of IV antibiotic therapy. Since the final diagnosis was an acute pyelonephritis; she

received 2 more weeks of oral Cefuroxime 500 mg twice daily as an outpatient. She was reassured that the calcification and partial thrombosis in the splenic aneurysm indicated its chronicity and that it was not contributing to its current illness and is unlikely to rupture with a size < 2 cm. Hence, no intervention was done and it appeared unchanged 1 year later.

Figure 1: Axial view of CT angiogram of the abdominal aorta; shows (A) non-ruptured, saccular 1.2 cm aneurysm in the distal splenic artery

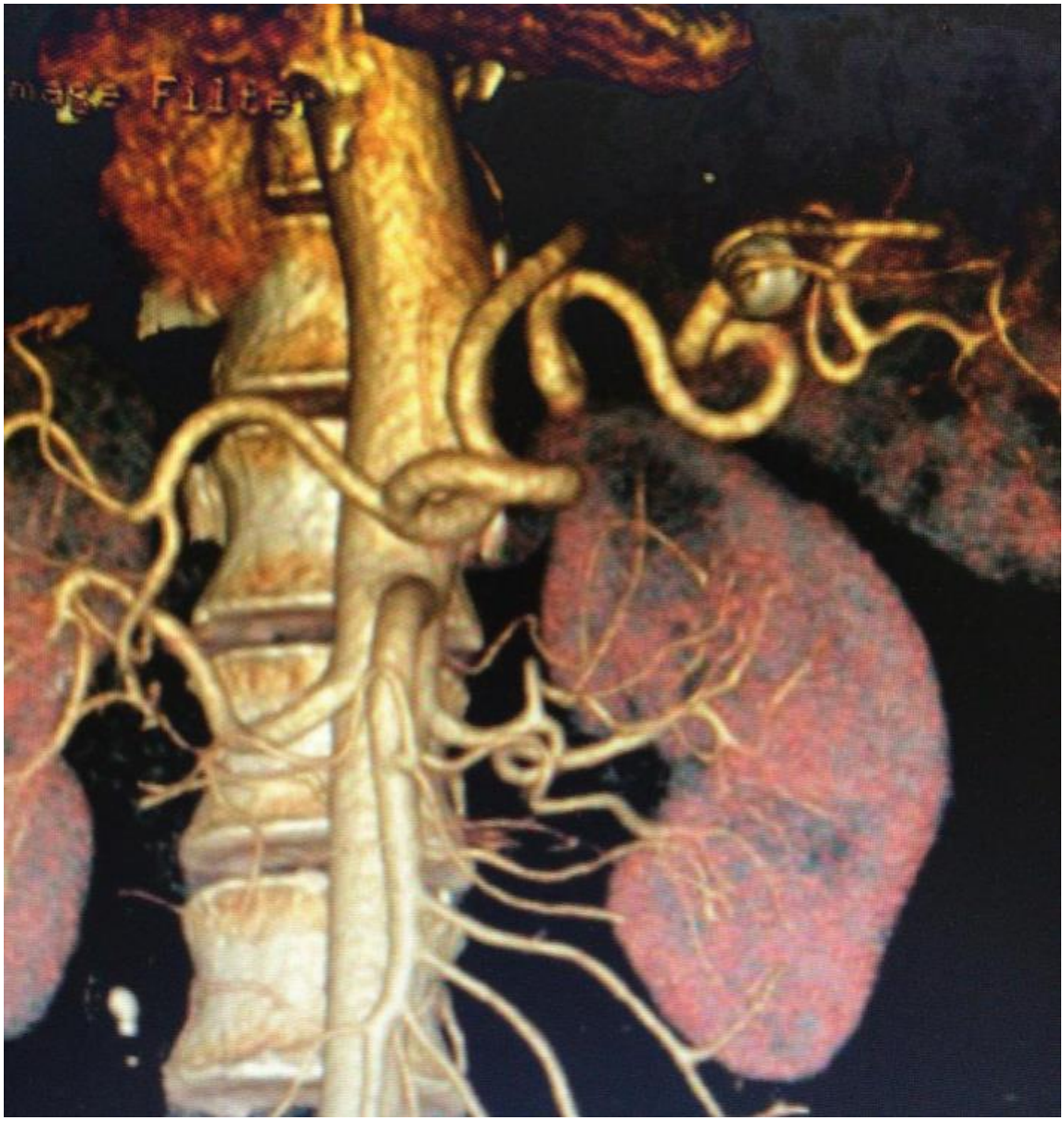

Figure 2: 3-D reconstruction, in coronal plane, of contrast-enhanced arterial phase MDCT; shows 1.2 cm aneurysm arising from distal portion

DISCUSSION

The normal diameter of the splenic artery is 0.45 cm. Hence, SAA is considered when its diameter exceeds 1 cm [8]. SAAs develop as true or pseudo ones and each has different etiology and prognosis. True SAAs are rarely associated with fibromascular dysplasia yet are common associate of systemic hypertension and portal one with cirrhosis as well as in liver transplantation and pregnancy [6]. Contrary to abdominal aneurysms; their etiology is not due to atherosclerosis, though in histology, 80-99% of which show medial degeneration and mural thrombi which simply reflect secondary events rather than primary ones [1]. SAAs are usually asymptomatic and rarely require more than periodic CT follow up. However, the risk of rupture is high in; (a) those with diameter > 2 cm, (b) women in the childbearing years due to vascular dilatation and increase portal pressure during pregnancy [9], (c) cirrhotic patients and especially those planned to undergo orthotopic liver transplant or Porto venous shunting procedures [6]. Spontaneous rupture is typically associated with 2 phases. The first bleed manifest as left upper quadrant pain with hemodynamic instability and GI bleed. The initial bleed may be tamponade in the lesser sac to be followed shortly with massive intraperitoneal flooding [10]. Treatment of SAAs include; (a) Endovascular approach with Fusiform ones are better treated with a stent graft (covered stent), while tortuous, saccular aneurysms are treated with aneurysmal coiling techniques, (b) laparoscopic ligation of the mid-splenic artery via stapling or clipping to splenectomy alone for distal SAAs near the hilum. Splenectomy with distal pancreatectomy may be necessary when the aneurysmal wall is severely inflamed and adherent to the tail of the pancreas. The laparoscopic approach carries the advantage of the rapid recovery, shorter hospital stay, and less postoperative pain compared with the open approach. The laparoscopic approach is also suitable and safe in pregnant SAA patients. Finally; open surgical approach with/without splenectomy may be indicated in large SAAs, those with impending rupture and hilar ones [11]. Pseudoaneurysms of splenic artery are different. They develop subsequent to necrotizing pancreatitis, perforated peptic ulcer or postoperative injury [12]. Because of the high risk of rupture and the high mortality rate if splenic artery pseudoaneurysm ruptures, the earliest possible intervention is deemed necessary. Transarterial embolization of pseudoaneurysm using coils, thrombin injections, or gelfoam if the patient is hemodynamically stable and diagnosis certain [13]. Ligation of pseudoaneurysm

with or without pancreatectomy (distal, partial, or total) and splenectomy is the standard treatment when the patient is hemodynamically unstable or embolization has failed [14]. Good surgical judgment and early consultation with a vascular surgeon should be undertaken because surgical intervention has been reported to carry mortality and morbidity risks of 1.3% and 9%, respectively [15]. In conclusion; SAAs once detected, should be differentiated from pseudo ones since the etiology and management is different. In the case of SAAs; the size, exact location in the splenic artery, and associated co-morbid states especially with pregnancy should be defined to assess risks of its rupture and hence its subsequent management.

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