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Ruptured Slpenic Artery Aneurysm-Unusual and Interesting Imaging Findings

Case Report

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Abstract

Splenic artery aneurysms (SAA) are rare and can be fatal if they rupture. They present with abdominal pain, melena, distension and tenderness of abdomen. We are reporting this case, since rupture of SAA is rare occurrence and there are a few unusual imaging findings observed. MDCT played a vital role in diagnosing the rupture, facilitating the treatment. Presence of gas within the infarct is not always due to infection, it may be non-suppurative as in our case.

Introduction

Splenic artery aneurysms (SAA) are rare and can be fatal if they rupture. Precise etiology of true aneurysm of splenic artery is unknown. It is associated with hypertension, portal hypertension, cirrhosis, liver transplant, pregnancy, arteritis, collagen vascular disease, Alfa anti-trypsin deficiency or inflammatory/infectious diseases, pancreatitis. Splenic artery pseudo-aneurysm (SPA) is less prevalent than true aneurysm and occurs in less than 1% in general population [1]. In true aneurysm there is dilation of splenic artery with all layers intact. Pseudo aneurysm occurs following disruption of one or more layers of vessel wall. Underlying causes of splenic artery aneurysm may be trauma, infection or weakness of vessel wall from exposer to pancreatic enzymes. Due to widespread use of cross-sectional imaging, there is an increased detection rate of splenic artery aneurysms. The true aneurysm often shows atheromatous plaque/calcification. Pseudo-aneurysm is usually multilocular contour on imaging. However, on many occasions, it is difficult to differentiate true from pseudoaneurysm on clinical and imaging grounds. Hemorrhage and abdominal pain are the most common presenting symptoms [2]. Secondary hemorrhage may involve pancreatic duct, peritoneum, retroperitoneum, adjacent organs or into pseudocyst if present. The chance for rupture of SPA is higher than SAA due to lack of all the three layers of the vessel wall. We are reporting this case, since rupture of SAA is rare occurrence and there are a few unusual imaging findings observed in our case.

Case Details

40 years male who was a chronic alcoholic presented with abdominal pain and shortness of breath for 2 days. Patient had one episode of melena 2 days back. No history of constipation, vomiting or hematemesis. No other comorbidities were present. On examination

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there was abdominal distension and diffuse tenderness all over the abdomen. There was no guarding or rigidity. Patient was dehydrated. Pallor was present. SPO2 was 100% on room air. Respiratory rate: 28 breaths/min. Pulse rate: 126 bpm. BP: 100/60 mmHg. GRBS mg/dl. GCS: E4V5M6. On blood investigations, hemoglobin was low (8.3gm/dl), PCV-25%, TLC-22,700/ul, urea- 45mg/dl, creatinine-1.2mg/dl, Na-132, K- 4.1 and Cl-92 mmol/l. The 2DEcho showed structurally normal heart with good LV function.

Ultrasonography of abdomen revealed hepato-splenomegaly. There was peri-splenic heterogeneously hypoechoic collection with evidence of internal echoes. Splenic artery showed out-pouching ofsize-1.2x1.1 cms close to the hilum. Ying-yang sign was observed on color Doppler examination. On CT scan of abdomen there was a large hypodense lesion in spleen with areas of hyperdensity suggestive of bleed and large infarct (Figure 1). A few air pockets were observed in the infarcted parenchyma. A rounded area of central hypo and peripheral hyperdensity was noted within splenic parenchyma close to the hilum. In arterial phase there is contrast pooling in the central part of hypodense lesion close to splenic hilum which was arising from splenic artery (Figure 2). On portal phase there was increased opacification of central enhancing area with active contrast leak into surrounding splenic parenchyma within the areas of bleed suggestive of active bleed from ruptured pseudo-aneurysm (Figure 3). There was moderate hemoperitoneum. Pancreas was atrophic and showed parenchymal calcification suggestive of chronic pancreatitis. (Figure 4) clearly demonstrated the SAA and contrast media extravasation (CME) was noted in volume rendering image.

On gross examination, resected the spleen was 14x12x5 cm and weight was 330gm. The capsule was congested. Microscopic section revealed a large area of infarct necrosis. A few areas of normal architecture were seen. At hilum there were dilated vessels. A localized part of splenic artery was dilated with prominent intimal thickening, fibrin deposition and luminal thrombus. The final diagnosis was splenic artery true aneurysm with thrombosis (Figures 5, 6). In the peritoneum, a large blood clot was noted close to the splenic hilum. There was adhesion of omentum with the pancreatic tail.

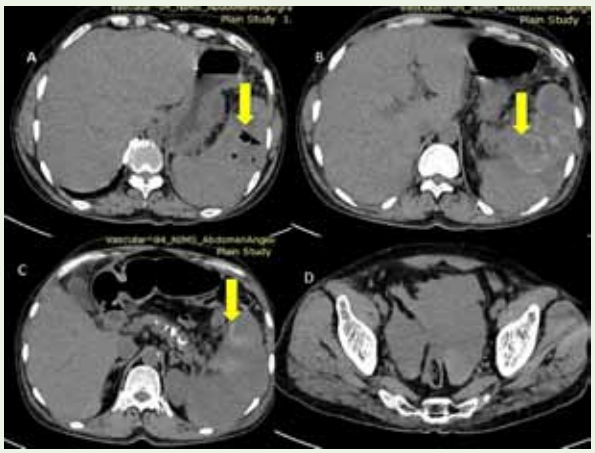

Figure 1: NECT demonstrating large hypodense lesion in spleen suggestive of infarct with areas of air pockets indicated by arrow in (A), rounded mixed density at splenic hilum indicative of SAA indicated by arrows in (B). hyperdensity suggestive of bleed within infarct (indicated by arrow in C) and extensive calcification in pancreas (C) and hemoperitoneum(D)

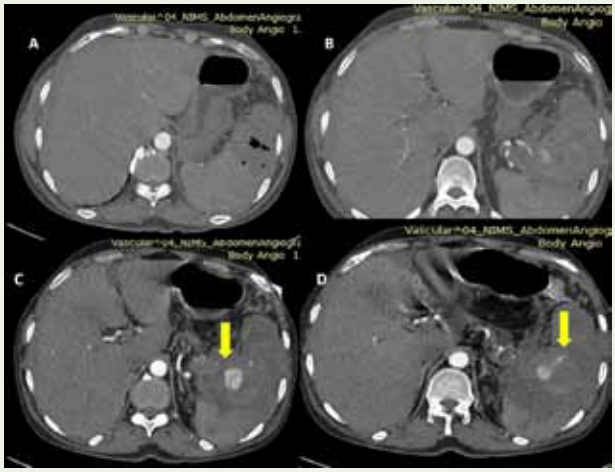

Figure 2: Arterial phase image reveals large infarct with air pockets (A), faint visualisation of SAA in B. Well opacified aneurysm with surrounding thrombus as indicated by arrow in C. There is active leak as indicated by arrow in D.

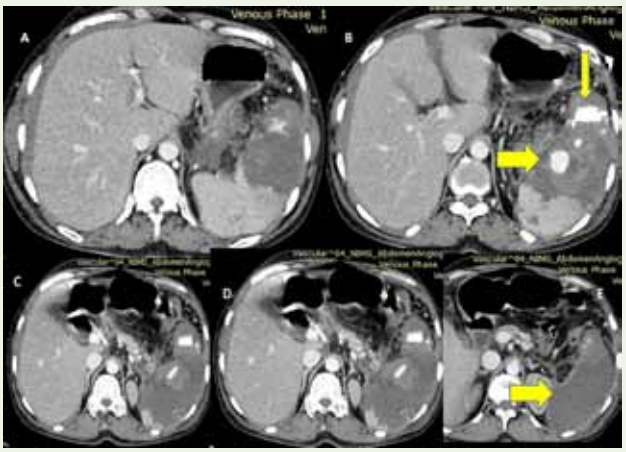

Figure 3: Portal phase images shows infarct in A, the SAA indicated by thick arrow in B having the size similar in both arterial and portal phase image and increasing size of Contrast media extravasation in portal phase as compared to arterial phase is indicated by thin arrows in same image B. Figures C and D show the aneurysm and contrast leak. The large infarct of spleen is also well depicted (arrow in E).

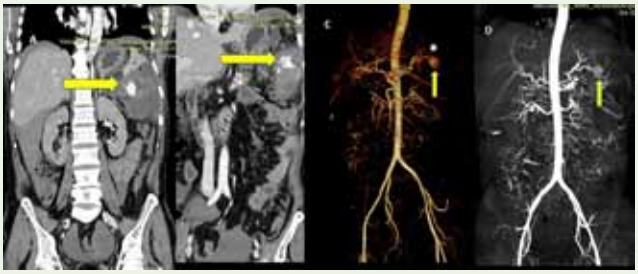

Figure 4: The coronal reconstruction and volume rendering CT angiogram clearly depicts the aneurysm from splenic artery having a peripheral thrombus (arrow in A) with active contrast extravasation (arrow in B). In image C and D, the aneurysm is well shown arising from splenic artery indicated by arrow.

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Figure 5: Gross specimen showing splenic artery aneurysm and congested splenic capsule.

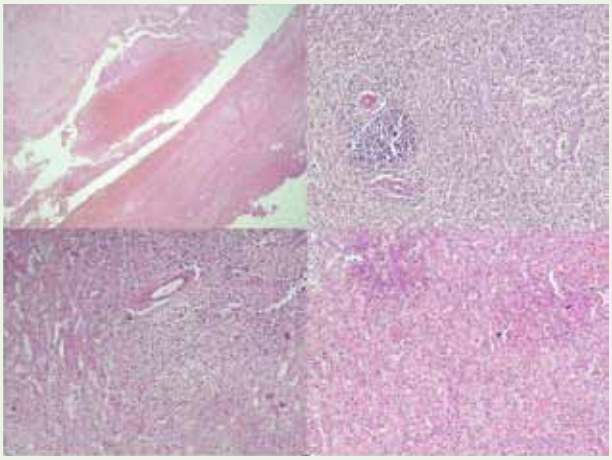

Figure 6: Multiple sections studied show splenic parenchyma having large area of infarct necrosis. A few areas of preserved normal architecture with red pulp composed of venous sinusoids and white pulp composed of lymphoid follicles. Sections from hilum shows dilated vessels, part of splenic artery is identified which is dilated and shows prominent intimal thickening and fibrin deposit in and luminal thrombus. Upper left luminal thrombus, upper right some preserved lymphoid follicle, lower left infarct like material, lower right red pulp and venous sinusoid.

Discussion

Splenic artery aneurysms are infrequently encountered and critical to recognize. Pathologically, the aneurysms are subdivided into true aneurysms (77%) and pseudo-aneurysms (13%) [3]. However, the term splenic artery aneurysm (SAA) is used interchangeably. The true aneurysm (SAA) is nothing but localized widening of splenic artery. Splenic pseudo-aneurysm (SPA) is different from true aneurysm histologically in that a collection of blood forms between the tunica media and tunica adventitia rather than circumferential dilatation of the vessel. Wall weakening in SAA occurs through traumatic, inflammatory, infective or iatrogenic causes [1]. Aneurysm is common in splenic artery due to proximity of artery to pancreas. Other arteries that can be involved are gastroduodenal, pancreatico-duodenal, hepatic and left gastric [4]. In case of pancreatitis, pancreatic enzymes cause a necrotizing arteritis with destruction of vessel wall structures and fragmentation of elastic tissue leading to aneurysm/pseudoaneurysm [1]. Though repeated acute exacebations of inflammation in the background of chronic pancreatitis is a possible mechanism for SAA formation and rupture, it is difficult to say for sure that our patient had such repeated acute episodes of pancreatitis in absence of suggestive history.

Fistulous

Communication to adjacent organs in background of pancreatitis is also a dreaded complication. GIT bleed and hemoperitoneum should prompt the suspicion of rupture as in our patient. SAA has potential risk of rupture in 10% with mortality rate of 10-25% in non- pregnant patients and up to 70% during pregnancy [5]. When aneurysm is > 2 cm, the chance of rupture is higher [6]. Portal hypertension, liver transplantation and pregnancy increase the chances of rupture in SAA [7]. Mortality is high (90% to 100%) with bleeding into peritoneal cavity or retroperitoneal cavity. With aggressive treatment the mortality is 18-29% [8].

Prompt reorganization of hemorrhagic shock, aggressive hemodynamic stabilization, an focused diagnostic approach and specific treatment are the key to the management of ruptured SAA. Our patient was a chronic alcoholic with chronic pancreatitis presented with sudden rupture of SAA. MDCT played a vital role in early and accurate diagnosis and immediate surgery saved our patient. The images of our patient were analysed and following issues were discussed. CT is useful for demonstration of CME. Since it is not a dynamic scan it cannot demonstrate the degree, rate and quantity of active bleed. Arterial CME appear as high attenuation similar to adjacent artery. CT can differentiate contained aneurysm from ruptured aneurysm. Intra-parenchymal pseudo-aneurysms have similar appearance to active hemorrhage on initial scan and do not increase in size on delayed phases and follow the blood pools on all phases. In contrast, active hemorrhage appears as high density due to contrast leak which increases in size. This point is well demonstrated in figures 2 and 3, and CME is arterial. Venous CME demonstrates extravasation only in late equilibrium phase and is less defined than arterial contrast extravasation. CT cannot detect intermittent extravasation and is not useful in clinically stable patient. Radionuclide scans have advantage of detecting slower bleeds and in stable patients.

Infarcts are wedge shaped, hypodense situated peripherally Splenic infarct may show liquefactive necrosis and intra-parenchymal gas formation. Air-pocket may suggest either abscess formation or they may be due to liberation of oxygen from Oxyhemoglobin in splenic infarct [9]. Rankin believes that amount of gas in infarct depends on amount of infarct and vascularity of tissue [10]. Splenic infarct with gas formation is also described by Levy JM et al in their case report [11]. Multiple bubbles of gas present in infarct throughout the parenchyma with central distribution. These air pockets are usually nonsuppurative. Splenic abscess is fatal outcome of splenic infarct. Differentiation between the infarct with pockets of gas from splenic abscess is important as splenic abscess needs intervention and infarcts are managed conservatively. Abscess appears as round /oval hypodense lesion containing gas and has enhancing capsule. Nonsuppurative air pockets are noted in our case and microscopic examination of operated specimen revealed infarct necrosis in spleen and there was no evidence of abscess.

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Another explanation of air pockets in our case may be due to SAA eroding adjacent colon Causing melena and air pockets in splenic parenchyma. On laparotomy there was adhesion of pancreatic tail and omentum. However, there was no communication of colon with splenic parenchyma. Hence the possibility of fistulous communication is less likely. When the infarct matures it undergo three processes-complete resolution or contraction/scarring or liquefaction. Portal phase is best for detecting infarct. SAA with low risk of rupture may be followed up 6monthly. When there is suspicion of increasing size more than 2cms in diameter or patients having symptoms or if patient is pregnant, SAA should be treated aggressively. All false aneurysms of splenic artery should be treated as soon as diagnosed, irrespective of size, symptoms or rupture [12]. Endovascular treatment with either embolization or stent graft applications are treatment of choice for SAA. However surgical intervention is considered in case of rupture as done in our patient.

Conclusion

SAA /SPA though rare, can rupture and present with abdominal pain, melena, distension and tenderness of abdomen. Prompt reorganization of hemorrhagic shock, aggressive hemodynamic stabilization, focused diagnostic approach and specific treatment are the key to save the patient. MDCT played a vital role in diagnosing the rupture, facilitating the treatment in our patient. Though we thought the aneurysm to be pseudo-aneurysm on imaging findings, pathological examination confirmed it as a true aneurysm. Presence of gas within the infarct is not always due to infection, it may be non-suppurative as in our case.

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