

Non-ruptured symptomatic splenic artery aneurysm (RCD code: I-1D.1)

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Abstract

Splenic artery aneurysm occurs in 1% of the population. Most splenic artery aneurysms are asymptomatic and are diagnosed incidentally. Symptomatic splenic artery aneurysm is usually detected due to rupture, while non-ruptured splenic artery aneurysm is rare. We present the case of a 69-year-old female who presented with signs of left abdominal pain and vomiting, and was diagnosed with splenic artery aneurysm. Diagnosis was made by CT scan and revealed a non-ruptured splenic artery aneurysm. Open abdominal surgery, endovascular treatment and laparoscopic surgery are treatment options for splenic artery aneurysms. Immediate treatment after diagnosis of symptomatic splenic artery aneurysm is recommended. JRC D 2018; 3 (8): 275–277

Key words: Splenic Artery, Aneurysm, Intra-abdominal, Iran

Splenic artery aneurysm (SAA) was first described by Beaussier in 1770 [1]. The incidence of SAA is between 0.1 – 0.2% in autopsies and can increase to 10% in people older than 60 years of age [2]. The incidence rate of splenic artery aneurysm in women is 4 times more likely than men [3]. It is estimated that 80% of splenic artery aneurysms are asymptomatic and diagnosed incidentally. Almost 20% of SAA cases are symptomatic and present with left abdominal pain [4]. Rupture occurs in 4% of cases [5]. The major aetiological factor of SAAs is arteriosclerosis. Other factors include congenital defects and trauma. Non-ruptured symptomatic SAAs occur with left-upper quadrant (LUQ) pain and vomiting [6]. Symptomatic SAA should be treated as an emergency [4]. We present the case of a 69-year-old female referred to Imam Khomeini Hospital, Sari, with complaints of left abdominal pain.

Case presentation

A 69-year-old Iranian female was referred to Imam Khomeini Hospital, Sari. The patient complained of continuous abdominal

pain in the LUQ, radiating to the back, which started to appear 10 days prior to admission. She reported 2–3 episodes of nausea and vomiting in the evening of that day. The patient's bowel movement was normal. Her past medical history was positive for heart disease. The patient was taking 80 mg Acetylsalicylic acid (ASA) tablets daily and was not allergic to any kind of food or medicinal substances. She was a non-smoker and addiction tests were negative. Family medical history was unremarkable. On admission, the patient was in a lying position, awake and conscious, and was negative for signs of toxicosis or other illnesses.

Blood pressure was 120/80 mmHg and her body temperature was 37 °C. The patient's pulse rate and respiratory rate was 72 beats per minute and 18 breaths per minute respectively. Physical examination of the head and neck revealed that conjunctivae were not pale and sclerae were icteric. Movements of the thorax upon respiration were symmetrical and tachypnea was not observed. Abdomen was soft upon palpation, distention was not detected. Tenderness was reported in the LUQ but neither rebound tenderness nor muscle guarding were observed. Digital rectal examination was unremarkable. Sensation and movement in all four extremities were normal.

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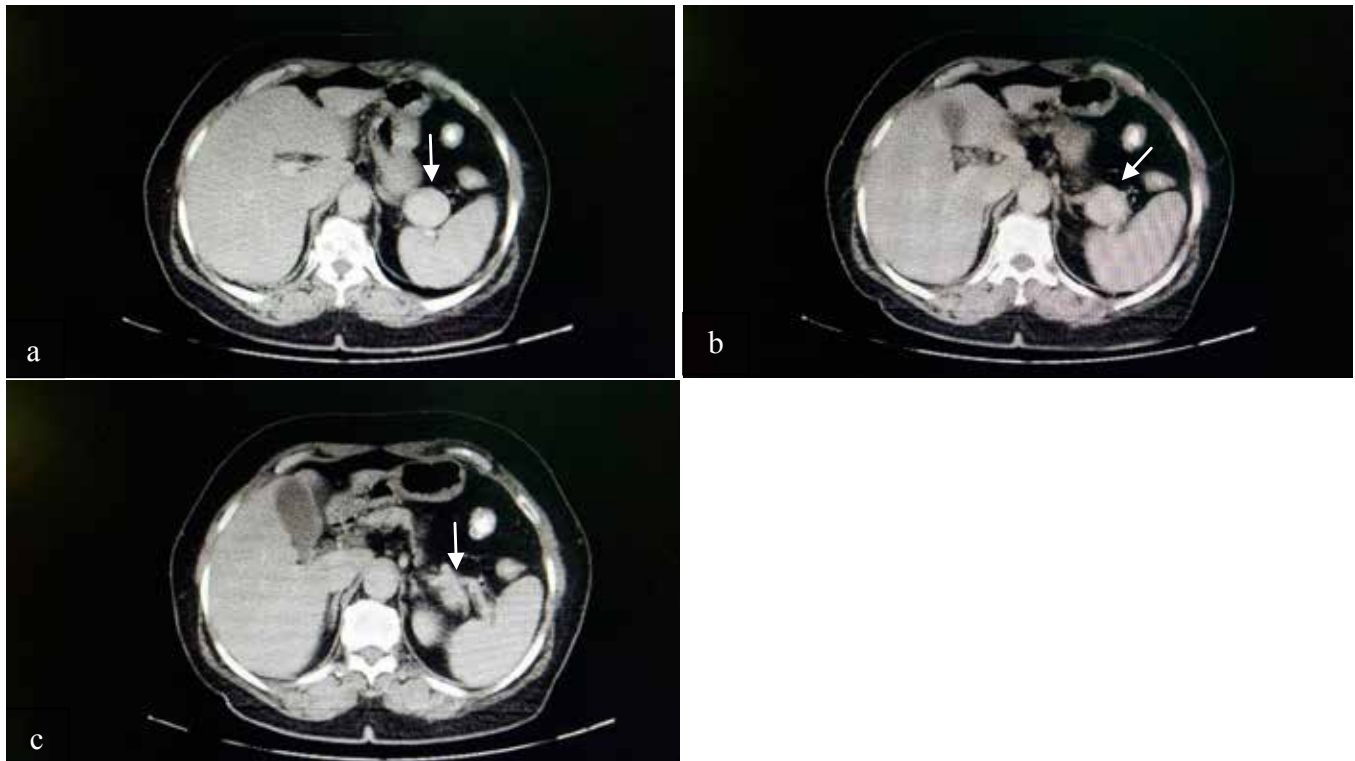


Figure 1. Aneurysm located in third distal part of splenic artery

Laboratory results included the following values: WBC: 13 000/ μ L [Normal range: 4 500 – 11 000/ μ L], PMN: 87% (Normal range: 45 – 75%), Hemoglobin: 11.5 g/dL (Normal range: 13.5 – 17.5 g/dL), MCV: 89 fL/cell (Normal range: 80–96 fL/cell), Plt: 235 000/ μ L (Normal range: 150 000 – 450 000/ μ L), Blood glucose level: 112 mg/dL (Normal <100 mg/dL, Prediabetic:100–125 mg/dL), Blood urea nitrogen: 32 mg/dL (Normal range: 7–20 mg/dL), Creatinine: 0.9 mg/dL (Normal range: 0.6 – 1.2 mg/dL), Na: 142 mEq/L (Normal range: 135–145 mEq/L), K: 5.2 mEq/L (Normal range: 3.5–5 mEq/L), Amylase: 52 U/L (Normal range: 30–110 U/L), Lipase: 37 U/L (Normal range: 30–110 U/L), PT: 13 s (Normal range: 11– 13.5 seconds), PTT: 25 s (Normal range: 25 – 35 s), INR: 1 (Normal range: \leq 1.1), AST: 37 U/L (Normal range: 10– 40 U/L), ALT: 45 U/L (Normal range: 7 – 56 U/L), ALP: 187 IU/L (Normal range: 44– 147 IU/L), Alb: 3.4 g/dL (Normal range: 3.5 – 5.5 g/dL), Total-protein: 5 g/dL (Normal range: 6 – 8.3 g/dL).

Ultrasonography revealed that liver size was but parenchymal echogenicity was increased (mild fatty liver). Gallbladder and bile ducts inside and outside of the liver and portal vein were normal. Pancreas size and parenchymal echogenicity was normal in the head and body region. Abdominal aorta was normal in the anterior-posterior (AP) diameter and no aneurysmal dilatation was observed. Ultrasonograms showed a cystic structure with a diameter of 33 mm which had veins and turbulent vessel flow. This was located between the superior bridge of the left kidney and superior bridge of the spleen. No significant lymphatic adenopathy was observed. A CT scan with contrast was performed for specific evaluation of vascular lesions and aneurysm size. CT scan revealed a hypo-dense, oval-shaped structure with a size of 27x40 mm, located in the distal one-third part of the splenic artery (Figure 1). Evidence

suggested aneurysmal dilatation of the splenic artery. No signs of flow or stranding were observed around lesion. Information on the patient's follow-up and outcome is not currently available.

Discussion

Splenic artery aneurysm is the third most common type of intra-abdominal aneurysm [5, 7]. Mean age for SAA was 60.1 years for females as evaluated in a study by Abbas et al [5]. An estimated 80% of cases are asymptomatic and diagnosed accidentally, however, 20% are symptomatic and indicated by left abdominal pain and nausea or vomiting [4]. Symptoms include left abdominal pain radiating to the back and pain in the left hypochondriac region [8]. About 1% of cases of SAA are non-ruptured symptomatic SAAs [5], similar to our case. A patient with symptomatic non-ruptured SAA presents with signs including left upper quadrant abdominal pain, chest pain, and flank pain. Pain characteristics could be diffuse or constant and can precede rupture from minutes to years [5, 6].

In previous studies, rupture of SAA was seen in about 4% of cases [5] and manifested by left shoulder pain, sudden abdominal pain in the epigastrium, and rapidly appearing hypovolemic shock [4, 6, 9]. In the case of our patient, the diameter of SAA was 3.3cm, while the mean diameter of non-ruptured SAA measures approximately 2 cm [5]. There are several methods to diagnose SAA including plain abdominal X-ray, ultrasonography (US), computed tomography scan (CT), magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and endoscopic US.

US is non-invasive and radiation-free, however, conditions such as obesity could affect its sensitivity [4]. Real-time ultrasonography can reveal masses located in the abdomen, but it does not provide any information on the vascular nature of those masses [10]. CT provides three-dimensional cross-section imaging, however, exposure to radiation limits the use of CT during pregnancy [4]. Diagnosis is usually made by contrast-enhanced CT scan [11], similar to our case. Digital subtraction angiography or transcatheter angiography via femoral artery is the gold standard for diagnosis of SAA [11].

Patients with SAA have a longer and more tortuous splenic artery than the normal population. Females with increased tortuous splenic artery are at higher risk for SAA [12]. All symptomatic SAAs should be treated as soon as possible as an emergency [4]. Not all SAAs need intervention, as most SAAs remain small and asymptomatic [5].

Treatment options for SAA include open abdominal surgery, endovascular treatment (coil embolisation or stent) and laparoscopic surgery [13, 14]. Open abdominal surgery is still the gold standard for treating SAA [13]. The location of an aneurysm determines the type of procedure in open surgery [14]. Patients age, operative risk, and clinical status are additional factors which determine the type of intervention performed [5]. Suitable intervention for proximally located, tortuous aneurysms is aneurysmectomy with end-to-end reconstruction, however, an aneurysm at the middle or distal end of a splenic artery is treated by excision of the lesion along with splenectomy [11, 13, 14]. Mortality and morbidity for open abdominal surgical intervention is 1.3% and 9% respectively [13]. Endovascular treatments are becoming more popular due to their high success rate and low morbidity [11, 13, 14]. Current endovascular techniques include transcatheter embolisation, percutaneous injection, endovascular stent grafts [13], and detachable balloon occlusion [11]. The first-line treatment option for asymptomatic SAA diagnosed incidentally is embolisation. Laparoscopic treatment is a less invasive approach to SAA in comparison to open abdominal surgery. Despite its relative safety, this treatment is contraindicated in haemodynamically unstable patients, or those with risk of rupture and large aneurysms [13]. Laparoscopic techniques include splenic artery ligation, resection of an aneurysm, and obliteration of an aneurysm with the vascular stapling device [5].

Conclusion

Splenic artery aneurysms are rare, diagnosed incidentally, and may become symptomatic in patients. Non-ruptured symptomatic aneurysm is extremely rare. Symptomatic splenic artery aneurysm carries a risk of rupture, although if diagnosis is early, the risk of rupture will decrease. Patients with signs of splenic artery aneurysm (left abdominal pain, nausea and vomiting) should be immediately referred for further diagnostics with the aim of decreasing rupture risk and its consequences.

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