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SPLENIC ARTERY ANEURYSM IN A PREGNANT WOMAN – CASE PRESENTATION AND LITERATURE REVIEW

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ABSTRACT

Splenic artery aneurysms (SAA) are a rare condition, usually asymptomatic. They pose a risk of rupture, which can lead to massive hemorrhage and fatal consequences. A particularly vulnerable group includes pregnant women, whose physiology may contribute to the formation and growth of aneurysms. In this study, we present a case of the detection and treatment of a splenic artery aneurysm diagnosed in a pregnant woman. The article presents a review of the literature related to SAA, its genesis, symptoms, diagnosis, and possible ways of treatment.

KEYWORDS

Splenic Aneurysm, Pregnancy, Pregnancy Aneurysm

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Introduction

A splenic artery aneurysm (SAA) is an uncommon vascular anomaly characterized by a dilation of the splenic artery by 50% compared to the normal vessel diameter, which ranges from 0.43 cm to 0.49 cm, depending on the measurement standard[1]. The prevalence of SAA is reported to be less than 1% of the population [1], with some studies suggesting an incidence of 0.1–0.2%[2]. It is the most frequently occurring visceral artery aneurysm, accounting for approximately 60–70% of all visceral aneurysms and third most common of intra-abdominal aneurysms.[3,4]

Patients diagnosed with splenic artery aneurysms are predominantly women, which is a unique phenomenon among vascular malformations.[5] Women constitute up to 87% of all patients with detected SAA [6]. The majority of affected women are multiparous, indicating that previous pregnancies may serve as a risk factor[1]. The average age at diagnosis is approximately 48 years, although cases have been reported from adolescence to advanced age [7].

In most cases, splenic artery aneurysms remain asymptomatic and are incidentally discovered through angiographic examination or postmortem analysis. The first symptom of an SAA may be rupture, which carries a high mortality rate, particularly in pregnant women and their fetuses.

Case Presentation

A 24-year-old woman, at 17 weeks of pregnancy, presented to her attending obstetrician with complaints of loss of appetite and mild epigastric pain. The gynecologist performed an ultrasound egzamination and identified a pulsating mass in the left upper quadrant of the abdomen, measuring around 15 mm in diameter. Suspecting a splenic artery aneurysm (SAA), the gynecologist referred the patient to a vascular surgeon, whom she visited after nine days.

A Doppler ultrasound confirmed a 16 mm lesion located at the splenic hilum. A decision was made to monitor the patient at regular intervals of approximately 3–4 weeks. Due to the stable size of the aneurysm and the absence of significant symptoms, the aneurysm repair procedure was postponed until after delivery. Throughout this period, the patient remained under the care of both a vascular surgeon and a gynecologist.

At 34 weeks of pregnancy, a cesarean section was performed. A laparotomy was performed using the Cohen method, a transverse incision was made in the lower segment of the uterus, and the baby was delivered head first. The newborn girl was delivered alive, with an Apgar score of 6/10, but soon her condition improved. The walls of the uterus were curetted and its walls sewed. The surgical team was called into the operating room.

Given the location of the aneurysm, laparoscopic splenectomy was planned. However, due to significant arterial bleeding encountered during organ dissection, conversion to an open approach was necessary. The splenic vascular pedicle was transected using a vascular stapler, achieving hemostasis. The aneurysm, measuring 20 mm in diameter, was located in the distal third of the vessel. It was carefully dissected, and both the inflow and outflow vessels were ligated and further secured with a stapler. After achieving complete hemostasis, after placing of drains in the Douglas pouch, the abdominal wall was closed.

The patient underwent surgery successfully and had an uneventful postoperative course. Follow-up examinations showed proper wound healing. Three weeks after discharge, she received vaccinations against *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* type b. The girl developed normally.

Literature Review

Etiology

Atherosclerosis is the most common cause of SAA, accounting for 66–70% of cases [7,8]. Another predisposing factor is congenital arterial defects, specifically the absence of development of the internal elastic lamina, occurring in approximately 30% of cases[7,8]. Infectious aneurysms, though much rarer, have also been reported in the literature [9].

There is a significant association between pregnancy and SAA, but pregnancy itself is not currently considered a direct causative factor. In a study by Owens and Coffey, 24% of women in a cohort of 31 patients with SAA were pregnant at the time of diagnosis, and among women of reproductive age, 51% were pregnant[10].

There is strong evidence that the development of SAA may be associated with portal hypertension. This hypothesis explains the correlation between SAA and pregnancy, during which physiological increases in portal venous pressure occur due to hormonal changes and increased intra-abdominal pressure caused by the growing uterus[11]. Other reports indicate an association between SAA and liver cirrhosis, particularly in patients

listed for liver transplantation, suggesting that portal hypertension plays a role in aneurysm formation[12] Although pregnancy remains the most significant risk factor, systemic hypertension also contributes by increasing the dilatory forces acting on the vessel walls. Atherosclerosis, due to its weakening effect on arterial walls, is also considered a factor that may increase the risk of SAA[13.]

Pathology

The splenic artery is one of the three branches of the celiac trunk, giving off branches to the stomach and pancreas before reaching the spleen[14] (Gray's Anatomy/Bochenek). It runs along the superior border of the pancreas, surrounded by the omental bursa. SAA usually occurs as solitary lesions, most commonly located in its distal one-third.[6] A study conducted by Maher on 217 patients diagnosed with unruptured SAA found that the mean diameter of SAA was 2.1 cm in females and 2.6 cm in males. In contrast, among patients with ruptured SAA, the mean diameter was 2.3 cm for females and 3.2 cm for males. SAAs with a diameter exceeding 5 cm are referred to as giant SAAs, and literature reports cases with diameters reaching up to 15 cm.[13,15]

Symptomatology

SAAs are usually asymptomatic. However, they may present with nonspecific symptoms, the most common being epigastric pain unrelated to food intake. The pain is localized in the epigastrium or left upper quadrant.[7] Additionally, SAA may cause nausea, vomiting, a feeling of fullness, loss of appetite, and pain in the left flank and shoulder.[13,16] In rare cases, SAA may lead to massive gastrointestinal bleeding, mimicking gastritis or malignancies. [17]

Rarely, it may also compress the splenoportal confluence and proximal portal vein, leading to splenomegaly and portal hypertension with all its consequences and symptoms. [18]There are also reports of massive SAAs causing chronic pancreatitis, likely due to compression of the pancreatic duct. This resulted in extensive fibrosis of the pancreatic parenchyma, loss of exocrine tissue, and disturbances in enzyme outflow, ultimately leading to disease progression.[19,20]

Diagnostics

Most cases of SAA are diagnosed incidentally during imaging studies conducted for other reasons. Due to the nonspecific symptoms, a large proportion of cases are detected via abdominal CT or ultrasound. A special group to consider when discussing SAA are pregnant women. In the majority of cases, SAA in this group is identified during routine ultrasound examinations conducted in each trimester of pregnancy, as was the case in the study presented in this article.

DSA (Digital Subtraction Angiography) remains the gold standard in the diagnosis of SAA, allowing precise visualization of the lesion and facilitating surgical planning.[21] Some authors propose screening for SAA. A study conducted by McMahon et al. suggests that routine radiological screening for every pregnant woman is not justified due to the low risk of rupture, estimated at 0.1%. However, given the dramatic survival statistics associated with this event, it is recommended to pay special attention to the possibility of SAA in high-risk women during routine obstetric ultrasound examinations.

Treatment

The decision regarding the management of splenic artery aneurysm (SAA) is based on its diameter, presenting symptoms, patient condition, and location. Due to the high risk of rupture, intervention is recommended for aneurysms larger than 2 cm in diameter and in symptomatic patients. Some sources also suggest treatment for women of reproductive age, pregnant patients, those with cirrhosis eligible for liver transplantation, as well as for all pseudoaneurysms and patients with SAA >25 mm who are suitable for surgery. [1,22] The choice of treatment depends on the capabilities of the medical center and the operator’s expertise.

According to a study by Lim, which included 576 patients between 2008 and 2018, endovascular procedures were the most frequently used treatment modality. These procedures were also the safest for patients, with a mortality rate of only 0.5%, compared to a 4.9% mortality rate associated with open surgery. This technique is advantageous for both patients and operators, as it allows the procedure to be performed under local anesthesia while minimizing tissue trauma. This makes it particularly suitable for elderly patients, for whom it may be the only viable treatment option. Additionally, endovascular treatment enables spleen preservation, which is another significant advantage.

In most reported cases, coil embolization was the most commonly used technique. Some procedures also involved glue embolization, vascular plug embolization**,** or stent-graft implantation for SAA. Endovascular procedures were associated with postoperative complications in 25% of cases, and 5.5% of patients required reintervention[22.] Importantly, this technique is suitable even for giant aneurysms. Trochimczuk described the successful endovascular treatment of a 26 cm aneurysm[23]. Despite promising outcomes, endovascular treatment has its limitations. It appears to be the preferred method for proximal and mid-segment SAAs, but its use may be unfeasible or require reintervention for distal or hilar aneurysms.[24] According to Lim, 10% of patients underwent laparoscopic treatment. In reported cases, no intraoperative deaths were recorded, and 5.6% of patients experienced postoperative complications, though none required reintervention. Despite being performed under general anesthesia, laparoscopic surgery carries a lower risk than open surgery and remains an option for pregnant patients.

In most cases, the aneurysm is excluded by clipping or ligation of the different and afferent vessels. Due to the presence of extensive collateral circulation between the splenic artery and pancreatic and gastric vessels, spleen preservation is generally possible when the aneurysm is in a typical location. If collateral circulation is insufficient to maintain organ perfusion, end-to-end anastomosis may be performed. An alternative approach is laparoscopic splenectomy, which is indicated when the aneurysm is located in the splenic hilum. [25]

However, laparoscopy also has its limitations. Retrogastric and intrapancreatic aneurysm location may effectively hinder access to the lesion, making the ligation impossible, necessitating alternative treatment approaches.Before the advent of endovascular interventions and laparoscopy, open surgery was the gold standard for SAA treatment. Despite its high invasiveness and associated risks, it still plays a role in SAA management, although its use is now limited.Traditionally, laparotomy with bypass grafting has been performed for proximally located SAAs, while splenectomy has been the preferred approach for hilar aneurysms.

Rupture

The primary and most life-threatening complication of a SAA is its rupture. Although it is a rare- SAA rupture occurs in 2-10% of symptomatic patients, its consequences can be critical for the patient.[16]The risk of rupture increases with an aneurysm diameter of 2 cm or more. Additionally, pregnancy is a significant risk factor for SAA rupture.[26]Some authors also suggest the presence of genetic defects that may cause rupture in an otherwise healthy artery.[27]The literature reports a 25% mortality rate in cases of rupture, which increases to 75% for pregnant women and 95% for the fetus.[28] Symptoms of SAA rupture may initially include nonspecific complaints such as nausea and vomiting. Due to blood extravasation and irritation of the phrenic nerve, Kehr's sign may occur, presenting as acute pain radiating to the left shoulder or left side of the back.[29]SAA rupture is associated with the *double-rupture phenomenon*, which is due to the anatomical location of the splenic artery. When the aneurysm initially ruptures, blood often collects within the lesser sac, temporarily containing the hemorrhage and leading to a transient clinical stabilization. This results in acute severe pain at the moment of rupture but without classic signs of hypovolemic shock. Typically, within 6-92 hours, a secondary rupture into the peritoneal cavity occurs, causing sudden and massive hemorrhage with rapid clinical deterioration manifesting with peritoneal signs, acute abdominal pain, and a drop in blood pressure.[16]If any of the above symptoms occur, urgent imaging is required. However, diagnostic procedures should not delay fluid resuscitation and surgical intervention to control bleeding due to its life-threatening character. A plain abdominal X-ray (RTH) is often performed in cases of acute abdomen but may not directly reveal the cause. However, calcification of the vessel in the left upper quadrant can suggest the diagnosis of SAA.For pregnant patients, ultrasound is the preferred diagnostic method due to its minimal teratogenic effects. The presence of a fluid collection in the lesser sac or free fluid in the peritoneal cavity on ultrasound indicates an urgent need for surgical intervention.Angio-CT is also a possible diagnostic option, though its use is limited due to accessibility issues and potential teratogenic effects.[2]

In cases of SAA rupture, the treatment of choice is laparotomy.[30] This approach allows for rapid access to the bleeding site regardless of the aneurysm's location.Some authors suggest that endovascular management may be an option if the patient is stable and not pregnant. Manian reported a case in which a vascular plug was successfully used to occlude blood flow to the aneurysm as a definitive treatment.[31] Others propose coil embolization as a method to stabilize the patient and delay laparotomy.[32]However, these techniques remain uncommon, with open surgery still being the primary treatment modality.

Conclusions

SAA is an interdisciplinary condition concerning general and vascular surgeons, gynecologists, as well as family medicine physicians, who are often the first to encounter affected patients. Vigilance in recognizing this condition is crucial, as even large SAAs often present with nonspecific symptoms, and disease progression increases the risk of life-threatening rupture.[22]A particularly vulnerable group includes pregnant women, in whom physiological changes due to fetal growth and hormonal alterations may mask SAA symptoms.Diagnostic and therapeutic options are broad and depend on the specific case and the capabilities of the medical center.The prognosis of SAA is influenced by multiple factors, the most significant being its symptomatic presentation and diameter. SAA rupture is a life-threatening emergency requiring immediate intervention. While diagnostics can aid in management, they should not delay the initiation of intensive treatment.

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