

## Management of Splenic Artery Aneurysms during Pregnancy

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### ABSTRACT

Splenic artery aneurysms are considered rare; however, they are the most common visceral artery aneurysm in reality. The incidence of splenic artery aneurysm increases in pregnancy and may result in significant maternal and fetal mortality if that would develop. This review summarizes etiologic, clinical, and diagnostic features and management of splenic artery aneurysms during pregnancy in the light of recent literature.

Keywords: Splenic artery, aneurysm, pregnancy

## INTRODUCTION

Splenic artery aneurysm (SAA) is defined as an abnormal dilatation of the splenic artery with a diameter of more than one centimeter. Although SAA is a rarely seen clinical condition, it could be life-treating in case of rupture. SAA is the most common visceral artery aneurysm and the third most common intra-abdominal aneurysm, followed by abdominal aorta aneurysm and iliac artery aneurysm [1,2]. Beussier first described the SAA in 1776, Macleod reported the successfully treated ruptured SAA in 1940; but only the mother survived. The first case in which the mother and baby survived after treatment for ruptured SAA was reported in 1967 [3].

### Epidemiology

Since most patients are asymptomatic, the actual prevalence of SAA is unknown. However, the

prevalence of SAA varies between 0.01% and 10.4% in autopsy series performed to patients aged 60 and older [4,5]. The incidence of incidentally detected SAA during angiography was reported as 0.78% in angiography performed 3600 patients [6].

The SAA is four times more common in females than in males, and the lesion is usually noticed during pregnancy [7]. Fifty-eight percent of patients are women of childbearing ages, and 95% of cases are detected during pregnancy [8]. More than 400 cases of SAA rupture have been reported, 30% of these being pregnancy-related [9]. The risk of rupture of an existing SAA during pregnancy has been estimated to be 25%.

### Etiology

The precise etiology of the SAA is not known. However, various factors such as angiodysplasia,

portal hypertension, pancreatitis, liver transplantation, pregnancy, grand multiparity (five and more delivery), diabetes mellitus, polyarteritis nodosa, atherosclerosis, deficiency of alfa-1 antitrypsin, intracranial aneurysm, and infection could lead to the formation of an aneurysm. Among the above, there is a robust correlation between SAA and pregnancy [10-13].

The weakness of the arterial wall and high blood pressure are the leading causes of the formation of an aneurysm in an artery. Hormones released during pregnancy, such as estrogen, progesterone, and relaxin, have significant effects on the arterial wall. Estrogen and progesterone can lead to subintimal thickening, medial fibrodysplasia, and glycosaminoglycan accumulation in subintimal and medial layers of the splenic artery. In addition to estrogen and progesterone, relaxin improves the effects of these hormones, and it increases wall elasticity of the splenic artery. Furthermore, microcystic degeneration is detected in patients with portal hypertension [7,9,10,14,15].

Pressure on the splenic arterial wall rises due to the physiologic elevation in cardiac output and blood volume, which causes portal congestion during pregnancy. In the third trimester, the proximal blood flows velocity of the splenic artery increases because of the pressure of the uterus to iliac vessels [16]. The risk of aneurysm and rupture of the splenic artery increases during pregnancy, especially for multiparous pregnant women as a result of those total changes.

In the case of ruptured SAA, the mortality risk is 25% of the general population. However, maternal mortality risk is 70%, and fetal mortality risk is 90% during pregnancy [10,17].

### **Clinical Features**

Eighty to ninety percent of SAA patients are asymptomatic. SAA can be detected by angiography or during laparotomy incidentally, or it may be found in postmortem examination in the case of the rupture [13,18]. Nevertheless, clinical features could be a divergent pattern.

### **Symptoms and Findings**

Abdominal pain is the most frequently observed

symptom of SAA, and sudden catastrophic cardiovascular collapse with rupture is a common presentation. The pain may involve the epigastrium or the right and left upper quadrants [7]. Non-specific symptoms such as lack of appetite, nausea, and vomiting can be observed. Intraperitoneal hemorrhage may result in sharp and severe pain spreading to the back and left shoulder due to irritation of the diaphragm (Kehr's sign). Again, because of intraperitoneal hemorrhage, tenderness may develop in the entire abdomen, and this sensitivity is more severe on the uterus fundus in pregnant women [3]. Symptoms of syncope or convulsion-like contractions and short-term unconsciousness afterward have been reported in ruptured SAA patients in pregnancy [19-21].

Sudden onset of epigastric or left upper quadrant pain with common abdominal tenderness and hemodynamic instability should be considered as SAA rupture. The epidural anesthesia used during delivery may mask the clinical picture of the rupture [22]. The non-stress test (NST) could be regular at the beginning of the last trimester. However, the shunt between splanchnic vessels and the placental circulation is attempted to compensate for maternal hemodynamic changes. That condition can lead to acute fetal hypoxia and abnormal NST waves [23].

In 20-25% of patients with SAA rupture, rupture-induced bleeding is initially confined to the omentum or coagulum occlusion of the Foramen Winslow may confine hematoma within the lesser sac, this may last up to 48 hours, but when the pressure within the lesser sac increases, the bleeding enters the peritoneum so that rupture becomes "free." This condition is called a two-stage rupture or double rupture phenomenon and clinically presents as sudden onset abdominal pain and shock following a hypotension episode that resolved with fluid replacement [10,15,18,24].

A case of acute portal hypertension in the early post-pregnancy period due to splenic artery rupture into the splenic vein has also been reported [25]. Hemorrhage could be aggravated due to the effect of coagulopathy and thrombocytopenia in women with cirrhotic portal hypertension

and hypersplenism.

As a result, clinical suspicion is crucial and life-saving for the diagnosis of SAA and SAA rupture.

### Diagnosis

Clinical suspicion is crucial, as radiologic studies like x-ray, abdominal computed tomography (CT), and angiography are limited during pregnancy because of teratogenicity. Most of the cases cannot be diagnosed until the surgery is performed during rupture [8,26].

An abdominal x-ray before pregnancy can incidentally display ring like calcifications which occur due to an atherosclerotic aneurysm [27].

CT (Figure 1) and magnetic resonance imaging (MRI) can provide a three-dimensional evaluation of the splenic artery (Figure 2). CT is not permitted, and MRI is questionable during the first trimester of pregnancy. Angiography is the golden standard for diagnosis of SAA (Figure 3), and during angiography, treatment options like stent placement or embolization can be performed, but contrast material injection is also not suggested to prevent the fetus from harmful effects; another disadvantage of angiography is its invasive nature. Post-interventional infectious complications, arterial puncture, and related complications may develop during the procedure [2,10,28,29]. According to the risks of angiography, this method should be kept in the last resort in the third trimester, whether for diagnostic or therapeutic purposes.

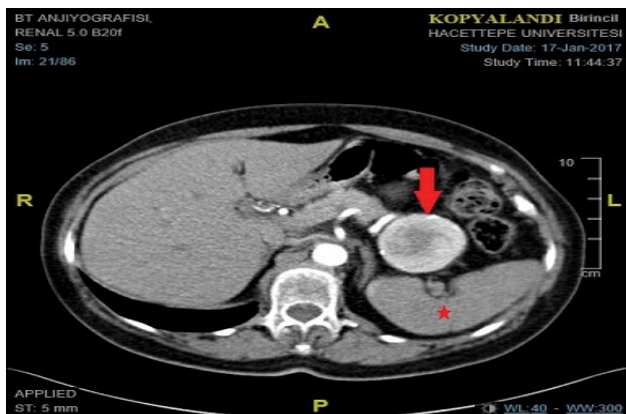


Figure 1. A CT image of 35 years old woman's SAA. A saccular aneurysm is shown with a red arrow, and the spleen is shown with an asterisk.

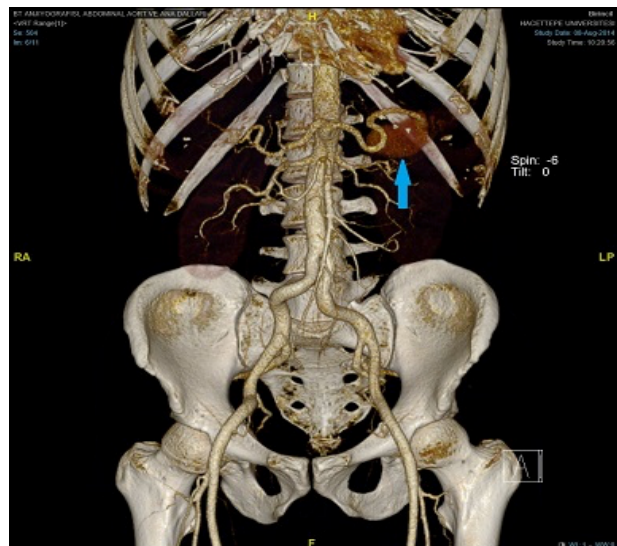


Figure 2. Three dimensions rendered CT image of SAA. The aneurysm is shown with a blue arrow.



Figure 3. An angiography image of 21 years old woman's multiple saccular splenic artery aneurysms (Shown with a red arrow)

Ultrasonography (USG) is the first method to use as it is noninvasive, mostly available, and cost-effective. However, it has some limitations such as user dependency, low sensitivity for smaller lesions, and technical difficulties due to obesity or bowel gas. USG can be used in case of clinical suspicion to support the diagnosis, by showing intraabdominal fluid presence [2,7,8,10].

Obstetric problems like ectopic pregnancy rupture, placental abruption, amniotic fluid embolus, and uterine rupture must be kept in mind with non-obstetric reasons like acute gastritis, cholecystitis,

visceral perforations, pulmonary embolus, and other arterial aneurysm ruptures in differential diagnosis [10,30].

Regular surveillance is not recommended because of the disease's rarity. However, pregnant women with risk factors mentioned in the etiology section, such as multiparity, portal hypertension, and atherosclerosis, could be included in surveillance programs [2,31,32].

Although there is no consensus about surveillance method and frequency, the best surveillance method could be USG according to convenience, accessibility, and cost-efficiency. Patients with at least one risk factor should be screening for SAA during pregnancy [31,32]. The most logical surveillance frequency in patients with risk factors could be evaluated in terms of SAA during regular prenatal USG screening instead of a strict screening protocol according to the trimester [32]

#### Treatment

There is no consensus for the treatment of asymptomatic SAA patients, and the appropriate treatment of SAA is unclear, but the treatment of >2 cm and symptomatic aneurysms are suggested. The risk of rupture under 2.5 cm diameter aneurysms is quite low. Incidentally diagnosed SAA during pregnancy with < 2 cm diameter can be followed by USG every six months but ruptured SAA under 0.5 cm cases also reported [2,8].

Effective treatment of the SAA depends on the localization of an aneurysm, patient's age, surgical risks, and clinical condition. Embolization or stent placement can be the proper approach for cases diagnosed before pregnancy [33]. A case of angiographic embolization during the third trimester of pregnancy is also reported [29].

Ligation without reconstruction can be appropriate if an aneurysm is located at the proximal segment because the blood accumulation from short gastric arteries can prevent splenic infarct. If an aneurysm is in the middle, 1/3 of splenic artery resection of an aneurysm and reconstruction can be performed. For distal 1/3 or hilus located aneurysms, splenectomy with resection of an aneurysm is the best option. Mortality of elective surgery varies between 0.5 – 1.3 %, and morbidity is 9 %. The appropriate time

for elective surgery for pregnant women is the beginning of the second trimester as embryogenesis is completed, and the uterus is not advanced enough to prevent exposure [8,27,34].

Minimally-invasive techniques including transcatheter embolization, laparoscopic ligation, or percutaneous angiographic embolization may be performed in pregnancy if the location and size of the aneurysm are appropriate. Surgical intervention is recommended in the second trimester, although laparoscopic aneurysm resection and splenectomy have been performed successfully in the third trimester [35,36].

Treatment of ruptured SAA is an unequivocally urgent surgery after adequate fluid resuscitation. Tranexamic acid or massive transfusion protocols can be applied during the time of surgery. When there is a suspicion of intraperitoneal bleeding, the vertical incision must be preferred to Pfannenstiel incision. Cross-clamping of supra celiac aorta, depending on fetal status, can be life-saving before definitive surgery. Aneurysm resection after splenectomy must be the treatment [20,37]. If delivery during emergent surgery is planned, fetal removal after splenic artery ligation is suggested, because any secondary bleeding from the uterus could be mortal [2,7,8,10].

According to a study, attendance of a general surgeon to the operation in case of SAA, rupture during delivery with gynecologist increase the survival significantly [8].

Polysaccharide pneumococcal and influenza vaccines are necessary for the prevention of post-splenectomy sepsis [38].

In conclusion, SAA during pregnancy is a rare condition, but it can be mortal. Clinical suspicion is crucial, as radiologic diagnostic tools are insufficient in this clinical condition. Surveillance for and managing SAA in any women of childbearing potential at high risk (e.g., portal hypertension) is essential. Emergent surgery can be life-saving both for the mother and for the fetus in case of rupture.

**CONFLICT of INTEREST:** The authors declare that there is no conflict of interest.



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