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RUPTURE OF A SPLENIC ARTERY ANEURYSM IN THE FIRST TRIMESTER OF PREGNANCY

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□ **Abstract—Background: Rupture of a splenic artery aneurysm during pregnancy is a rare and serious event, occurring mainly during the third trimester. The risk of rupture for an existing splenic artery aneurysm is very high during pregnancy (from 20% to 50%). When it does rupture, the maternal mortality rate is around 75% and the fetal mortality rate 95%. Of 110 cases reported in the literature, only one ruptured during the first trimester. Objectives: The aim of this case report is to make emergency physicians aware of this diagnosis, because only if it is considered can it be managed rapidly and appropriately. Case Report: We report the case of a 6-weeks pregnant patient referred to our institution in hemorrhagic shock who died of a ruptured splenic artery aneurysm shortly after surgery. The initial diagnosis considered was a ruptured ectopic pregnancy. Conclusion: We report this case to increase awareness of splenic artery rupture during pregnancy, even during the first trimester. © 2011 Elsevier Inc.**

□ **Keywords—pregnancy; hemoperitoneum; splenic artery aneurysm; hemorrhagic shock; ectopic pregnancy**

INTRODUCTION

Spontaneous hemoperitoneum during the first trimester is an uncommon major complication of pregnancy, related most often to ruptured ectopic pregnancy. We report the case of a patient with a spontaneous hemoperitoneum resulting from a ruptured splenic artery aneurysm (SAA). The literature is presented, differential diagnoses are discussed, and potential causes for the increased risk of ruptured aneurysm during pregnancy are considered.

CASE REPORT

This 36-year-old woman, gravida 2 para 1, had an unremarkable history, except for a cesarean section during her first pregnancy. She was 6 weeks pregnant when she was referred to the emergency department (ED) for a suspected ruptured ectopic pregnancy.

The patient developed severe abdominal pain, predominantly on the left side. Several hours later, the pain persisted and she contacted her physician who advised that she go to the ED. She remained at home until the abdominal pain increased and she lost consciousness. The emergency mobile medical service was called.

On arrival at the ED she was in extremis, with severe hypovolemic shock, no peripheral pulse, no obtainable blood pressure, a heart rate of 150 beats/min, cutaneous mottling, bilateral mydriasis, and no response to nociceptive stimuli. As the patient was resuscitated, abdominal ultrasound was performed and showed a massive hemo-

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peritoneum. The clinical picture and term of pregnancy suggested a ruptured ectopic pregnancy. Intensive resuscitation in the ED included orotracheal intubation, subclavian central venous catheter, multiple transfusions (3 units of packed red blood cells, 3 of fresh frozen plasma, fibrinogen 3 g), norepinephrine, and atropine infusion.

The initial laboratory results revealed a hemoglobin level of 3.2 g/dL, a platelet count of 72,000/mL, potassium of 7.4 mmol/L, and bicarbonate of 6 mmol/L. Arterial pH was 6.04, consistent with severe metabolic acidosis.

After 60 min of resuscitation, her hemodynamic status improved sufficiently for transfer to the operating room for surgical exploration (suspected ruptured ectopic pregnancy). The laparotomy with Pfannenstiel incision from her previous cesarean scar revealed a profuse hemoperitoneum estimated at more than 3 L. Inspection of the pelvis did not show an ectopic pregnancy. A xiphopubic laparotomy was performed and vascular surgeons were called in. The patient went into cardiac arrest; external cardiac massage revived her in less than a minute.

Exploration of the abdominal cavity found no gynecologic lesions, but rather bleeding from above the mesocolon resulting from a splenic artery aneurysm that was ruptured for 2 cm of its distal third. Due to the patient's hemodynamic instability and signs of gastrointestinal compromise, the aneurysm was excised and the artery clamped, without splenectomy.

Overall, the patient received 16 units of packed red blood cells, 15 of fresh frozen plasma, and 2 of concentrated platelets, as well as 6 g of fibrinogen. At the end of the surgery, her hemodynamic condition was stable; bilateral mydriasis was present, and slight diffuse bleeding persisted, related to disseminated intravascular coagulation. Postoperative laboratory results revealed: hemoglobin of 6.4 g/dL, a platelet count of 65,000/mL, potassium of 4.7 mmol/L, bicarbonate of 19 mmol/L, and an arterial pH of 6.75.

She was transferred to Intensive Care, but died on arrival after a second cardiac arrest, despite further attempts at resuscitation.

DISCUSSION

Our review of the literature found 110 cases of a ruptured SAA during pregnancy, but only one during the first trimester, and only 18 cases in which both mother and child survived. The overall prevalence of SAA is $< 1\%$ in the general population: 0.16% in a series of autopsies and 0.78% in a series of angiographies (1). Its prevalence during pregnancy is not known with precision. Despite this rarity, SAA is the third most common visceral aneurysm after those of the aorta and iliac arteries (2). In 80% of cases it is located on the distal third of the splenic artery, as in our case (3). It is four times more frequent in women than men (2,4). Outside of pregnancy, the risk of rupture can be estimated at 3–5.3%, with a mortality rate of 25% (1). During pregnancy, the risk of rupture increases substantially to 20–50%, with a mortality rate of 75% in women and 95% for their fetuses (2,5–7).

The risk factors for SAA are: female gender, portal hypertension, congenital vessel anomalies, degenerative arterial diseases, inflammatory processes, vascular trauma, and pregnancy (4). There is an increased risk associated with multiparity and in the third trimester of pregnancy (5,7–9). The rupture rate during pregnancy seems to be around 12% in the first two trimesters, 69% in the third, 13% during labor, and 6% in the postpartum period (10). The risk of rupture is proportional to the diameter of the aneurysm (especially when it exceeds 2 cm).

The etiological hypotheses for the increased prevalence of these aneurysms during pregnancy include a hemodynamic and a hormonal hypothesis. The hemodynamic hypothesis is associated with an increase in blood volume and cardiac output, with an augmentation in the splenic shunt, which may increase the mechanical constraints on vessel walls and cause local impairment (1,4,7). This effect may be cumulative from pregnancy to pregnancy (4). At the same time, hormones may explain the histologic remodeling found in this type of aneurysm, where atherosclerosis is very rare. Estrogens, progesterone, and relaxin may influence arterial wall remodeling and induce fibromuscular dysplasia of the tunica media and fragmentation of the elastic fibers, thereby promoting development of the aneurysm (1,4,9). Pathological examinations of aneurysms often find such dysplasia, but other abnormalities are also observed, including intramural hemorrhages and chronic inflammatory or infectious phenomena responsible for the arterial damage (9). As in most reported cases, the postmortem pathology examination failed to clarify the mechanism of occurrence of the aneurysm.

The development of a SAA during pregnancy is generally asymptomatic, and 95% remain asymptomatic until they rupture (1,4). Prodromic signs of rupture, documented in 5% of cases, are non-specific and include intermittent left side pain, epigastric pain, or chest pain irradiating into the left arm. Nausea and vomiting may occur. Signs may also mimic cholecystitis (1). Rupture of a SAA causes intense abdominal pain focused in the epigastric or left upper quadrant, sometimes with radiation in the left arm associated with shock that may occur within several minutes. In 25% of cases, rupture occurs in two stages (double-rupture phenomenon, as in our case) and may allow an effective treatment (1,5). In the first stage, the hemorrhage is contained in the