International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Patel N et al. Int J Reprod Contracept Obstet Gynecol. 2021 May;10(5):2119-2121
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20211555

Case Report

Splenic artery aneurysm rupture in pregnancy: a learning experience

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Received: 16 March 2021
Revised: 12 April 2021
Accepted: 13 April 2021

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ABSTRACT

Splenic artery aneurysm (SAA) is a rare clinical entity that carries a risk of rupture and life threatening hemorrhage. The objective of this clinical case report is to highlight this rare occurrence during pregnancy to avoid misdiagnosis. A 25 year old primigravida in her 3rd trimester of pregnancy was brought to trauma and emergency care with severe epigastric pain with rapid onset of unexplained shock. The shock was out of proportion to external or visible blood loss or any other injury. She was taken for emergency laparotomy. Intra-operatively, hemoperitoneum was encountered in peritoneal cavity due to ruptured splenic artery aneurysm. Massive transfusion protocol was activated and aneurysmectomy with splenectomy was performed. Post operatively, the patient was transferred to intensive care unit. Unfortunately, the patient succumbed due to complications of irreversible shock. Pregnant women with SAA can present with non-specific symptoms and hypovolemic shock. In such cases of hemorrhage, obstetricians should always keep in mind the other non-obstetric causes of intra-abdominal bleeding.

Keywords: Pregnancy, Intra-abdominal hemorrhage, Splenic artery aneurysm rupture, Maternal mortality

INTRODUCTION

Splenic artery aneurysm (SAA) is a rare clinical entity that carries the risk of rupture and fatal hemorrhage. Rupture of SAA during pregnancy can be disastrous for both mother and fetus.[1] Patients with SAA are usually undiagnosed and remain asymptomatic until it ruptures. During pregnancy, the development of shock is very rapid.[2] Hence, ultrasonographic diagnosis, clinical judgment, and rapid shifting of patient to operation theatre remain the mainstay of treatment. Hereby, we are reporting a case of SAA rupture during 3rd trimester of pregnancy.

CASE REPORT

A 25 year old primigravida female, in her 36 weeks of pregnancy, was brought to trauma and emergency care with chief complaints of severe epigastric pain and giddiness. The symptoms were abrupt in onset, approximately three-four hours before arrival. The patient had a history of vomiting two days back and there was no history of fever. There were no other symptoms suggestive of pre-eclampsia. Physical examination showed a lethargic and confused female. She was severely pale with a rapid thready pulse of 180/min hypotensive with blood pressure of 90/60 mmHg, respiratory rate of 34/min and oxygen saturation (SpO_2) of 98% on room air. Abdominal examination revealed a distended abdomen with generalised tenderness and fundal height of 34 cm. On auscultation, fetal heart sounds were absent. On P/V examination, the cervix was 3 cm dilated, 30% effaced, head fixed and the station was -3. There was no bleeding P/V.

Ultrasonography of the abdomen revealed free fluid in the abdomen and absence of fetal cardiac activity. Her laboratory results were as follows: hemoglobin (HGB)=8.7, total count (TC) =22.7, platelet count

(PC)=143, international normalized ratio (INR)=2.27, serum creatinine (Cr)=1.61, serum total protein=3.7, albumin=1.8, lactate dehydrogenase (LDH)=2435, serum glutamic-pyruvic transaminase (SGPT)=949 and serum glutamic oxaloacetic transaminase (SGOT)=1999.

In view of unexplained shock with the possibility of intra-abdominal hemorrhage, the patient was planned for emergency laparotomy.

Pre-operatively, the pulse and blood pressure went non-recordable. Hence code blue was activated and the patient was resuscitated. Massive transfusion protocol was activated. Following which, the emergency laparotomy was done under general anaesthesia and was performed by professor in MS, Obstetrics and Gynaecology (OBGY) with her team of junior doctors. A midline infra-umbilical incision was used to access the abdomen. Upon entering the peritoneal cavity, around 1.5 litres of hemoperitoneum was encountered. There was no evidence of uterine rupture. A trans-curvilinear incision was given on the lower uterine segment and a dead fetus of around 2.5 kg was delivered and there was no evidence of retro-placental collection. Following which, the uterus was exteriorized and the adnexa was evaluated completely. The uterine incision was closed in layers. A search for other causes of intraperitoneal hemorrhage was done and a large hematoma was visualized in the lesser sac that was rapidly growing in size (Figure 1). The rest of the bowel and stomach appeared to be normal. An Intra-operative consultation was obtained from general surgeon and vascular surgeon. On arrival, the lesser sac was opened. Following which, there was a sudden gush of blood from a ruptured artery. A proximal clamp was applied and control was taken. The ruptured artery was then identified to be a splenic artery aneurysm. Splenectomy with aneurysmectomy was done. The splenic artery was ligated (Figure 2) and the spleen was sent for histopathological examination. After confirming hemostasis, her abdomen was closed while keeping 2 drains. A total of 6 packed cell volume, 4 fresh frozen plasma and 3 platelet concentrates were transfused over the duration of surgery.

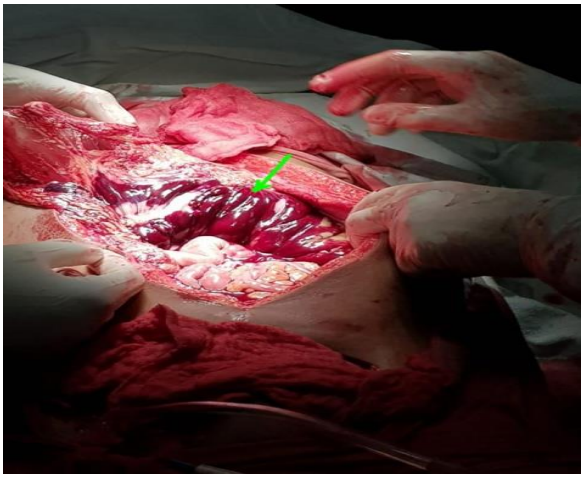

Figure 1: A large hematoma in lesser sac.

Postoperatively the patient was transferred to the intensive care unit where her pulse showed tachycardia and blood pressure was not recordable. She was transfused with 4 fresh frozen plasmas and 10 cryoprecipitated antihemophiliac factors. The patient collapsed in ICU and code blue was activated. Unfortunately, the patient succumbed to complications due to irreversible shock.

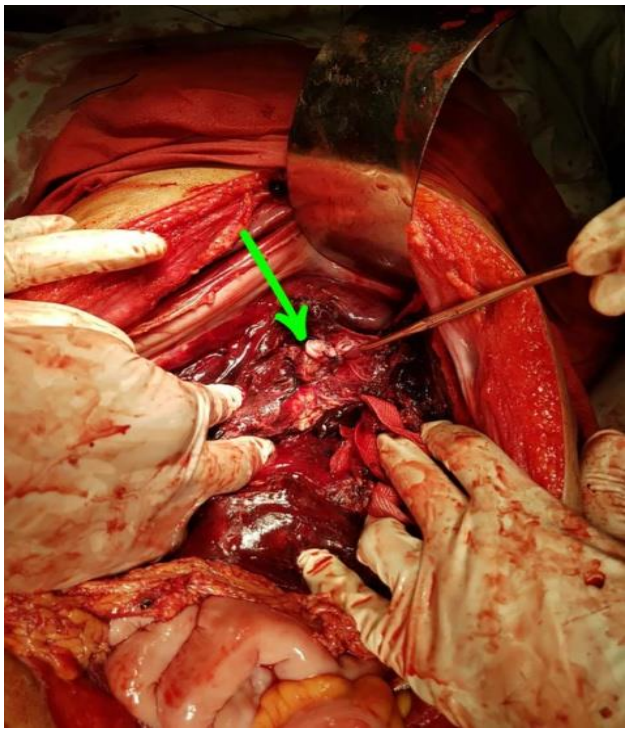

Figure 2: Splenic artery aneurysm stump.

Grossly, the spleen was enlarged and the external surface was smooth and unremarkable. The capsule was intact.

On histopathological examination, the section showed changes of chronic venous insufficiency. The splenic sinusoids were dilated and congested with atrophy of white pulp and fibrosis. The arteries at hilum showed medial calcification.

DISCUSSION

Splenic artery aneurysm is the most common visceral artery aneurysm and the second most common site of intra-abdominal aneurysms secondary to the aorta. Splenic artery aneurysms may be more prevalent in women, usually as a consequence of atherosclerosis.[3] Pregnancy has a strong association with the formation of splenic artery aneurysm.[2] However, the incidence of SAA in pregnancy is unknown. The underlying pathologic processes in the development of most SAAs are related to atherosclerosis, arterial fibrodysplasia, and arteritis. Associated risk factors include female gender, multiparity, and portal hypertension. Several reports have established an association between multiparity and SAA development, but the precise interactions of hormonal influence,

fibromuscular dysplasia, and wall stress are not yet fully understood. Significant maternal and fetal mortality has been documented to be close to 75% for pregnant women and about 95% for the fetus. Rupture occurs almost exclusively in the 3rd trimester.[1] The changes associated with pregnancy increase the risk of formation and rupture of the aneurysm. Symptoms are nonspecific and the diagnosis is often made during laparotomy.[4] Hence, in a majority of the cases, the clinical signs of a ruptured SAA in pregnancy are masked. 70% of cases of SAA rupture during pregnancy are misdiagnosed as uterine rupture. Concealed abruptio placenta is another one of the most common differential diagnosis in cases of SAA.[5] Routine screening of the splenic artery by ultrasound and Doppler should be considered in pregnant women with unexplained upper abdominal pain.[6]

Once ruptured, it can cause severe hypovolemic shock with end organ damage as with our case. The initial laboratory findings were suggestive of acute kidney injury, secondary myocardial infarction and acute liver injury.

CONCLUSION

Splenic artery aneurysm is a rare clinical entity that remains asymptomatic in the majority of cases. But, the presence of SAA in a pregnant female and its rupture during pregnancy is disastrous to both mother and fetus. For pregnant women presenting with unexplained shock and hemodynamic instability, wherein no apparent cause is found, the obstetrician should have a high index of suspicion for non-obstetric causes of intraperitoneal hemorrhage. A multidisciplinary approach is a key to success.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Patel N, Barot KS, Patel J, Patel S, Sapre S. Splenic artery aneurysm rupture in pregnancy: a learning experience. Int J Reprod Contracept Obstet Gynecol 2021;10:2119-21.