



Acute Right Heart Failure in Postpartum Period: An Unusual Case of Ruptured Sinus of Valsalva Aneurysm

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ABSTRACT

Sinus of valsalva aneurysms are uncommon and can be congenital or acquired. They may have variable clinical presentation ranging from asymptomatic cases to congestive heart failure and, in extreme situations, cardiac arrest. Both ruptured and nonruptured valsalva sinus aneurysms can cause deadly consequences, though the prognosis is excellent after treatment. As a result, timely and precise diagnosis is essential. Rupture of sinus of valsalva (RSOV) aneurysm in pregnancy during antepartum is a dreaded complication and can be life-threatening for both mother and fetus. However, it is infrequent with only few cases reported during pregnancy. We report a case of ruptured sinus of valsalva aneurysm in postpartum period with acute right heart failure following uneventful normal vaginal delivery and favorable outcome postpercutaneous intervention.

Keywords: Percutaneous intervention, Pregnancy, Ruptured sinus of valsalva aneurysm.

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INTRODUCTION

Sinus of valsalva aneurysm (SVA) is a rare congenital or acquired cardiac anomaly due to deficiency of fusion of the media of the aorta with the annulus fibrosis of the aortic valve resulting in abnormal dilatation or enlargement of the aortic sinuses. SVAs may rupture into any of cardiac chambers, usually the right-sided ones to form an aortico-cardiac fistula usually in second and third decade and account for 0.1–3.5% of all congenital cardiac defects.¹ Pregnancy may be a risk factor for the rupture of SVAs due to hemodynamic and hormonal factors although it is very rare. Rupture occurs commonly in antepartum period and patient may present with palpitations or acute heart failure. We present a case of rupture of SVA in postpartum period with uneventful normal vaginal delivery.

CASE DESCRIPTION

A 34-year-old woman, P3L3A1 post normal vaginal delivery day 24, presented to medicine emergency with complaints of epigastric pain, shortness of breath, and palpitation on exertion followed by pedal edema and progressive abdominal distension for 20 days. There was no history of orthopnea and paroxysmal nocturnal dyspnea. There was no past history of similar complaints and all her previous pregnancies were uneventful. There is no history of sudden cardiac death or similar complaints in family. On examination, she had tachycardia with pulse rate of 105 beats per minute which was waterhammer in character with no radio-radial or radio-femoral delay; blood pressure was 100/40 mm Hg in right brachial artery in sitting position and 114/50 mm Hg in right popliteal artery (Hill sign absent) with respiratory rate of 18/minute. Her Jugular vein pressure was raised to 7 cm H₂O above sternal angle and had bilateral pitting pedal edema extending up to mid-thigh. Her arm span-to-height ratio was less than 1 and no other peripheral signs of aortic regurgitation were present. Systemic examination revealed continuous machinery murmur best heard at left parasternal border, grade 4/6 with a palpable thrill across the precordium and loud P2 in the second left intercostal space. No murmur was heard on back of patient. Bilateral normal

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vesicular breath sounds were heard. Her abdomen was uniformly distended with no distended veins; shifting dullness was present with tender hepatomegaly, palpable up to 3 cm below right coastal margin. Blood investigations showed moderate anemia with hemoglobin—9.7 gm/dL, TLC—5700 mm³, platelets—2.89 lac/μL, transaminitis with ALT-104, AST-90, ALP-190, T.Bil-1.3 with normal cardiac biomarkers (CK-T-26) and D-dimer (234 ng/dL). ECG showed RVH with sinus tachycardia. Cardiomegaly was present on chest X-ray and ultrasound was suggestive of gross ascites with hepatomegaly. Ascitic fluid analysis revealed high SAAG ascites (SAAG-1.3). She was planned for 2D-Echo for acute onset right heart failure in postpartum period and meanwhile was managed conservatively with diuretics.

TTE was suggestive of ruptured sinus of valsalva aneurysm with left to right shunt from right coronary sinus to right ventricle with severe TR and dilated RA and RV (Fig. 1). Color Doppler echo showed the flow directed toward right ventricle from aorta during diastole. CT heart and great vessels showed presence of contrast-filled saccular outpouching arising from right coronary cusp and protruding across the interventricular septum into RV cavity measuring 1.3–1.5 cm in size with focal ill-defined inferior wall suggestive of rupture with no associated abnormality of vessels and heart (Fig. 2).

As she had no associated underlying structural heart lesions and was in the postpartum period, percutaneous closure was planned. Following percutaneous transcatheter device closure with cocoon

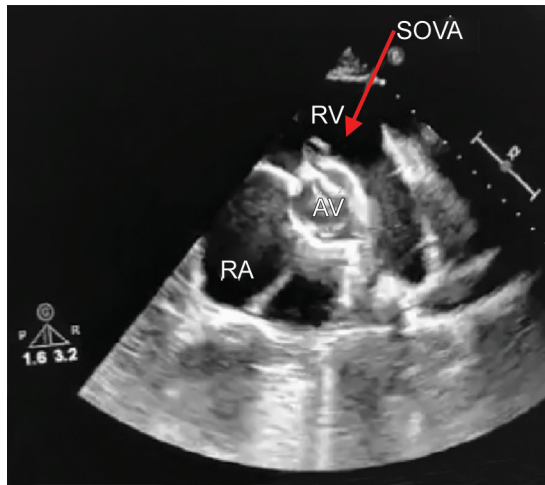


Fig. 1: TTE in PSAX view (AV level) showing tricuspid aortic valve with sinus of valsalva aneurysm of right coronary cusp opening into right ventricle cavity. SOVA, sinus of valsalva aneurysm; RA, right atrium; RV, right ventricle; AV, aortic valve

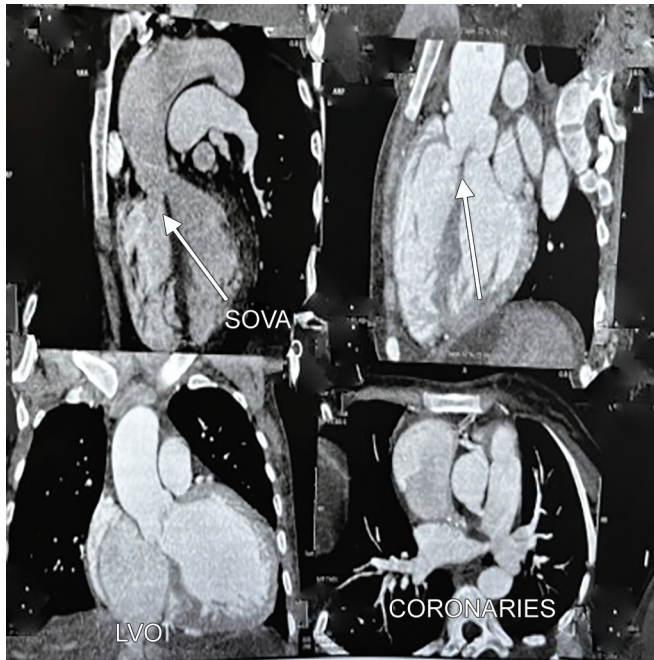


Fig. 2: CT heart and vessels showing SOVA (sinus of valsalva aneurysm) (white arrow) originating from aorta and opening into right ventricle in upper right and left images. Lower left image depicts normal left ventricle outflow tract (LVOT) and lower right image shows normal coronary artery anatomy

occlude, her symptoms and signs of right heart failure abated within a week. Her post-op TTE showed closure of defect with minimal residual shunt. She is doing well at follow-ups and is taking aspirin for 6 months to prevent coronary ostium thrombosis.

DISCUSSION

SVA ruptures most commonly into the right ventricle but may also rupture into the right atrium and result in acute onset right

heart failure with or without hemodynamic instability, associated with acute onset chest pain. The patient characteristically has a continuous murmur heard over the left parasternal area or apex. RSOV can be diagnosed with a transthoracic echocardiogram (TTE) or a trans-esophageal echocardiogram (TEE). CT and MRI can be used preoperatively to define the anatomy, delineate the size of the defect, and evaluate associated cardiac lesions more precisely.² The treatment options for repair of a RSOV include open surgical repair or percutaneous closure (PC).

Rupture of sinus of valsalva (RSOV) has been reported in pregnancy with few published case reports; though it is unknown how pregnancy precipitates the rupture. The hyperdynamic circulation in pregnancy results in increased flow across the aortic valve and subsequently increased stress on the dilated sinuses. In the immediate postpartum period, emptying of uterus relieves the obstruction of the inferior vena cava and results in marked increase in preload to the heart. These mechanical stresses render the aneurysm increasingly prone to rupture.³ Pregnancy-related hormones also cause changes in mechanical properties of connective tissues and could theoretically lead to rupture of the sinus of valsalva.

Most of the case reports such as those by Latzman³ and Agrawal⁴ have included patients presenting during the antepartum period. Our patient had an uneventful pregnancy and started developing symptoms of right heart failure only four days after a normal vaginal delivery. Hence, she likely developed the rupture either during labor or in the immediate postpartum period. Her presentation was unique as she had no symptoms during her pregnancy or in her previous pregnancies. Considering no underlying structural abnormalities, the patient underwent percutaneous device closure and has subsequently improved symptomatically. This has underlined the success of percutaneous closure as a minimally invasive approach with good post-op outcomes.

CONCLUSION

We have shown a case with postnatal presentation of ruptured sinus of valsalva. It is an important and potentially treatable cause of acute onset right heart failure in antenatal and postnatal patients. Hence, it requires careful clinical evaluation and prompt intervention considering good response rates to both open surgery and percutaneous closure.

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