Repair of Sinus of Valsalva Aneurysm
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Citation

Abstract
We report a case of uneventful repair of Sinus of Valsalva aneurysm (SVA) in a young female without a PA catheter, which can be difficult to place because of the anatomy of the SVA and potentially hazardous by causing serious arrhythmia.

INTRODUCTION
Sinus of Valsalva aneurysm (SVA) was first described by John Thurman in 1940. SVA is a rare congenital anomaly, which usually is clinically silent but may vary from middle, asymptomatic dilation detected in routine 2-dimentional echocardiography to symptomatic presentation related to the compression of adjacent structures or intracardiac shunting caused by rupture of SVA into the right side of the heart. When SVA ruptures, few specific signs of left-to-right shunting may become apparent.

- A loud superficial “machine type” continuous murmur is accentuated in diastole
- A palpable thrill along the right or left lower parasternal border may be presented.

Most ruptured SVAs occur from puberty to the age of 30 years and often are diagnosed or presented clinically at this age. A ruptured SVA progresses in 3 stages as described by Blackshear and colleagues (1):

- Acute chest or right upper quadrant pain
- Subacute dyspnea on exertion or at rest
- Progressive cough, peripheral edema

Atypically SVA presents with infective endocarditis, which may originate at the ages of aneurysm.

Approximately 65-85% of SVAs originate from the right sinus of Valsalva, 5% - from left sinus (1).

Congenital SVA is more prevalent in Asia, caused by dilation, usually of a single sinus of Valsalva, from a separation between the aortic media and annulus fibrosis.

Rupture of the dilated sinus may lead to the intracardiac shunting with communication to the right atrium (10%) or directly into the right ventricle (60-90%). Cardiac tamponade may occur if the rupture involves the pericardial space. Associated structural defects in congenital SVAs included supracristal or perimembranous VSD (30-60%), bicuspid aortic valve (15-20%) or AR (44-50%).

CASE REPORT
A 28 year old female, originally from Philippines, presented with dyspnea on exertion, fever and chills. On PE she was found to have a murmur and work up revealed a fistula of sinus of Valsalva aneurysm, which has a clear left-to-right shunt passing from her aorta, specifically the right sinus of Valsalva, to her right ventricular outflow tract with evidence of a mobile fenestration and vegetation. The atrial valve was spared without evidence of endocarditis. She had a blood culture that showed Abiotrophia adiacea and she was on 6 weeks course of IV antibiotic prior to surgical repair.

According to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines, the patient had minor clinical predictor for increased perioperative cardiovascular risk. Preoperative electrocardiograms (ECG), left and right ventricular function were normal. We decided not to use a pulmonary artery catheter (PAC) for this case because of the location of SVA. Placement of PAC may be difficult, since the tip of the catheter may go to the aneurysm in right ventricle and cause arrhythmia (1) or perforation.

The patient received midazolam before line placement (PIV, arterial line, and CVP). After induction of anesthesia with etomidate, midazolam, fentanyl and vecuronium, orotracheal intubation was performed and the TEE probe was passed...
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with ease. Echo revealed normal size left ventricle, normal aortic valve, aneurysm of right sinus of Valsalva connected with right ventricle via fistula (Figures 1 and 2).

**Figure 1**

General anesthesia was maintained with isoflurane, vecuronium and fentanyl. During GA continuous ECG with online analysis of dysrhythmia and ST segment, invasive arterial blood pressure monitoring by radial artery cannulation, arterial saturation, CVP pressure by right IJ cannulation, ETCO2, temperature and urine output were monitored. After opening pericardium purse strings were placed in the ascending aorta for aortic cannulation and antegrade cardioplegia and in the SVC and IVC for venous cannulation; in the right atrium for the retrograde cardioplegia cannula and in the right superior pulmonary vein for LV vent insertion. Full dose heparin given and aprotinin infusion were started. CPB was instituted, aortic crossclamp was placed with the pump flow down. The aorta was transected just above the sinotubular junction. Right ventriculostomy was performed and the vegetation was excised. The fistula was closed via aortic and right ventricular approach using a pericardial patch. Postop TEE showed no residual fistula with good technical result. There was trace aortic insufficiency noted.

After aortic crossclamping was released the patient restored sinus rhythm and ventilation was resumed. She was rapidly weaned from CPB on no inotropic support. She was transferred to ICU intubated. She was extubated six hours later. On postoperative day one, the chest tubes were removed and she was transferred to Cardiac Surgery telemetry unit. She was discharged home on postoperative day three.

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