A case of Sinus of Valsalva Aneurysm with Junctional Tachycardia and later complete heart block

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Introduction:
Aneurysms of the sinuses of valsalva are thin-walled outpouchings most commonly involving the right or non-coronary sinuses. Because they are asymptomatic, they are rarely discovered before they rupture and form aorto-cardiac fistula. We present a rare case of unruptured aneurysms of noncoronary and left sinuses of valsalva burrowing into the interventricular septum producing an unusual junctional tachycardia and later complete heart block.

Case presentation:
34 year old lady presented with history of recurrent episodes of paroxysmal palpitations of one and a half year duration. Each episode was associated with significant discomfort and hemodynamic compromise requiring DC cardioversion to terminate the tachycardia. The tachycardia was not responsive to i.v adenosine or verapamil. The ECG taken during the episode of tachycardia shows tachycardia with a rate of 240/min, RBBB morphology, QRS width 100msec, axis 150 and features of AV dissociation (figure 1). Basal ECG showed RBBB with similar QRS axis(figure 2). Her echocardiogram and CT angiogram (figure 3 and 4) revealed two sinuses of valsalva aneurysms, one from non coronary sinus and the other from left coronary sinus. She underwent electrophysiological studies from two institutions. In one study baseline intervals were within normal limits. No VA conduction or dual AV node physiology was observed. No accessory pathway noted. No AVNRT or atrial flutters were inducible. In another study done during tachycardia AV dissociation was seen during tachycardia. His potential was seen preceding each QRS and HV interval was similar to HV interval in sinus rhythm. EP study was consistent with a diagnosis of junctional ectopic tachycardia. She was started on amiodarone and there was no recurrence of tachycardia after and she was asymptomatic for one year on 100 mg/day of amiodarone.

One day she presented to the emergency department with symptoms of extreme fatigue and near syncope. Her ECG showed infrahisian complete heart block with very low ventricular escape rate (figure 5). She was subjected to a temporary pacemaker insertion. There were no features of rupture of sinus of valsalva aneurysm. The complete heart block was thought to be due to pressure effect by the enlarging aneurysm at the HIS bundle region. She underwent an aortogram (figure 6 and 7) which showed two aneurysms but larger size than prior CT. Mild aortic regurgitation was present and there was no ventricular septal defect. Her coronaries were normal.

She underwent excision and patch repair of the two aneurysms and with a provisional plan for dual chamber permanent pacemaker insertion if the heart block persists. After successful surgery, her rhythm got back to sinus after 3 days post op and she was discharged in stable condition in sinus rhythm. Surgeon could also avoid a potential aortic valve replacement and she was left with mild Aortic regurgitation. For the first three months of surgery she was put on warfarin and aspirin. After surgery on a six month follow up, she has no recurrence of tachycardia off amiodarone and she in NYHA class 1 without any medications.

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Discussion:

Sinus of valsalva aneurysms was first reported by J Hope in 1835\(^1\). Most aneurysms are congenital in origin but may be seen acquired after bacterial endocarditis, atherosclerosis, dissection or chest trauma\(^2\). Congenital aneurysms of the aortic sinuses of valsalva are thought to result from weakness in the aortic media at its junction with the annulus fibrosus\(^3\). Aneurysms appear as small diverticuli or finger-like protrusions that extend most commonly from the right or non-coronary sinus. The right ventricle and right atrium are common termination sites for aneurysms of the right coronary sinus. Aneurysms of the non coronary sinus usually enter the right atrium\(^4\). Rarely, aneurysms present because of compression to other cardiac structures producing symptoms\(^5\). Sinus of valsalva aneurysms are usually asymptomatic and are rarely discovered before they rupture and form aorto-cardiac fistula. They come to clinical attention most typically in adolescence and young adulthood when the protruding structure ruptures into the receiving chamber. Acute rupture of a large aneurysm causes retrosternal or epigastric pain and severe dyspnea from congestive heart failure. By contrast, perforation of a small aneurysm may go unnoticed until congestive heart failure develops from the long standing volume overload\(^6\). Coronary artery compression by a sinus of valsalva aneurysm is an unusual mode of presentation\(^7\). Rarely, sinus of valsalva aneurysms burrow into the interventricular septum, causing AV conduction defects\(^8\). Mild aortic regurgitation is expected from distortion of aortic cusp and root enlargement as a result of long-standing volume overload. Severe aortic regurgitation should cause the suspicion of aneurysm rupture into the left ventricular outflow tract or secondary to endocarditis affecting the aortic valve leaflets\(^9\).

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References:

**Clinical Cases**

**Basal ECG**

**Complete AV block**