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## Unusual Presentation of Ruptured Sinus of Valsalva Aneurysm

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### ABSTRACT

*Sinus of Valsalva Aneurysm (SVA) is caused by dilation, usually of a single sinus of Valsalva, from a separation between the aortic media and the annulus fibrosus. Indeed, a deficiency of normal elastic tissues and abnormal development of the bulbus cordis have been associated with the development of SVA. We report a 31-year-old man with unusual presentation of ruptured sinus of valsalva aneurysm. (Tanaffos2010; 9(3): 65-68)*

**Key words:** Aorta, Sinus of valsalva, Aneurysm

### INTRODUCTION

Sinus of Valsalva Aneurysm (SVA) is caused by dilation, usually of a single sinus of Valsalva, from a separation between the aortic media and the annulus fibrosus. Indeed, a deficiency of normal elastic tissues and abnormal development of the bulbus cordis have been associated with the development of SVA. We report a new presentation of ruptured sinus of valsalva aneurysm which has not been reported yet in our knowledge.

### CASE SUMMARIES

A 31-year-old man was admitted for work up of palpitations and atypical chest pain.

Cardiac examination was normal and revealed normal blood pressure. An ECG was performed (Figure 1). It showed normal sinus rhythm (NSR) with frequent APDs (Atrial Premature

Depolarization). No other abnormal signs or symptoms were seen. Serial cardiac enzyme tests were within normal limits and without any change. Considering the above-mentioned findings a 24-hour holter monitoring was performed. The result was NSR with frequent bigeminal APDs and even couplets (Figure 2).

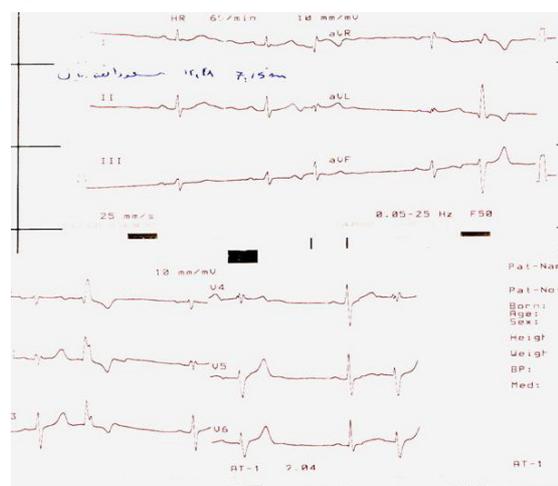


Figure 1. The first ECG of patient.

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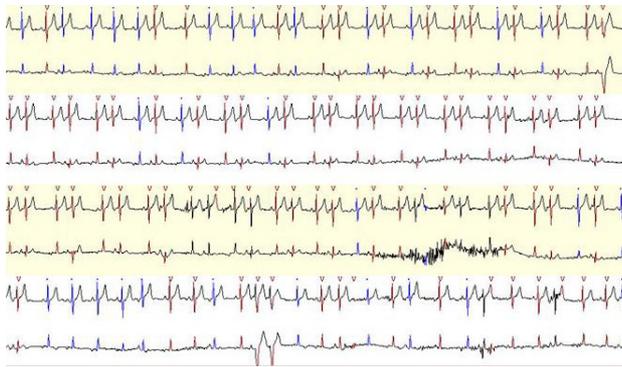
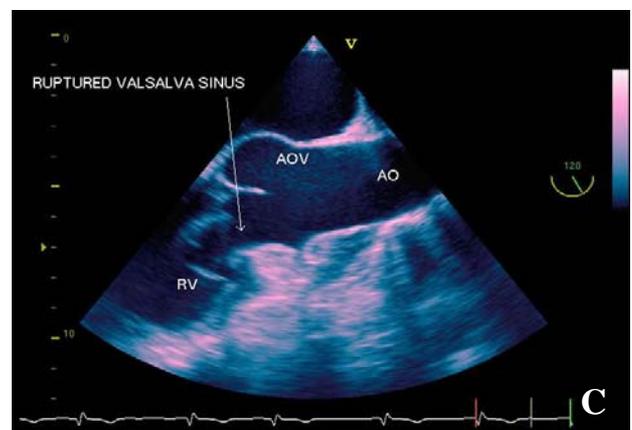
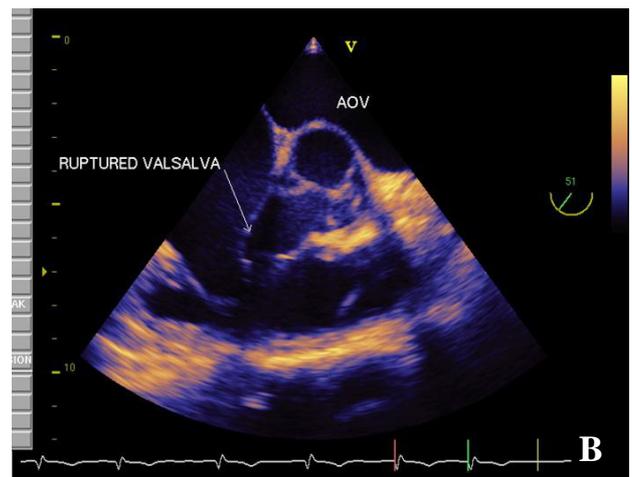
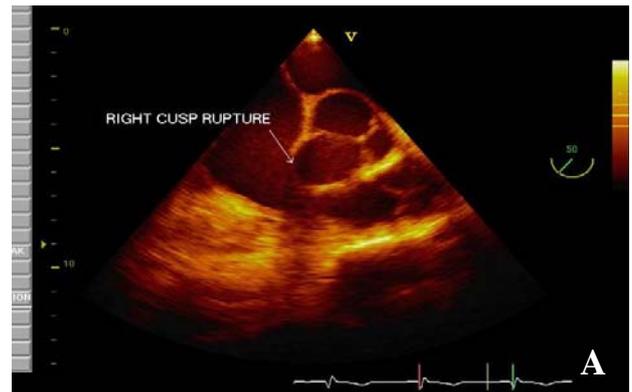


Figure 2. 24h-holter monitoring

A transthoracic echocardiography (TTE) was performed. Mitral valve prolapse (MVP) was the pathologic finding. In his Exercise Tolerance Test (ETT) according to Bruce protocol, he reached stage 3 (METs = 10.1) without any abnormal changes except numerous PACs which decreased in numbers during peak exercise. Standard  $\beta$ -blocker therapy was started reducing the frequency of APDs although not terminating them. The patient was discharged because of the partial response to medical treatment and pain free status. Two months later, he came back with intermittent chest pain, palpitations and dyspnea. A new ECG was taken which revealed no changes relative to previous recordings. On the other hand, a new TTE study revealed a high velocity flow throughout the right ventricle (RV) in parasternal and 4-chamber views. Due to these findings a Trans Esophageal Echocardiography (TEE) was performed showing rupture of the right sinus of Valsalva aneurysm into RV (Figure 3 A-D). The patient underwent cardiac catheterization. An apparent flow from aorta to the RV was seen in aortic root injection. Prolapse of the right Judkins catheter was demonstrated from the aorta into the RV. In addition, biventricles opacified during left ventricular injection (Figure 4 A,B).

Because of these findings, the patient was referred to a cardiac surgeon. In the follow-up visit, 3 weeks

after the operation, a TTE study showed no abnormal flow and closed connection between aorta and RV. The most important and interesting finding was the complete disappearance of the APDs in the last ECG (Figure 5). The patient has been symptom free since then.



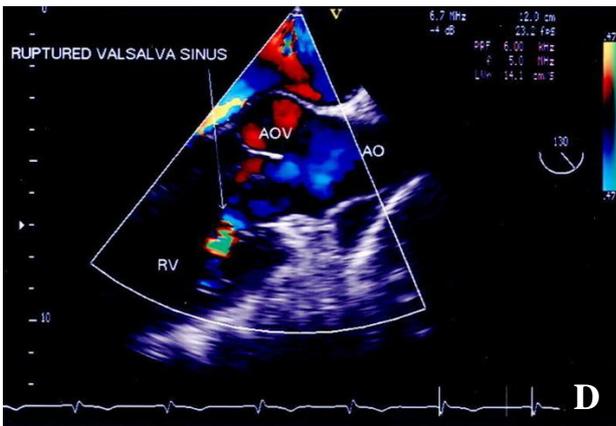


Figure 3 (A-D). Preoperative transesophageal echocardiography of patient.

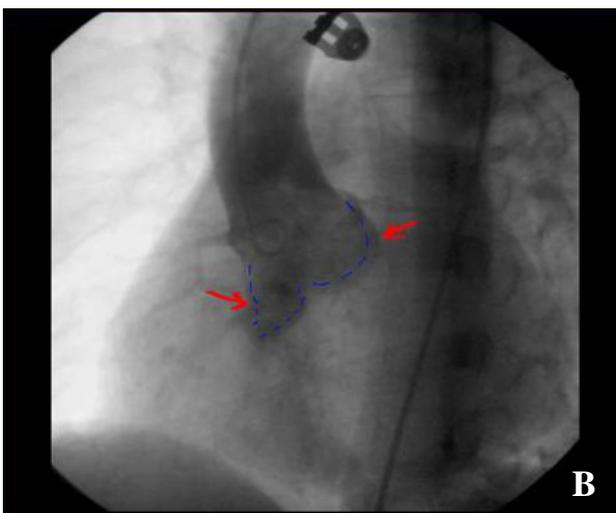
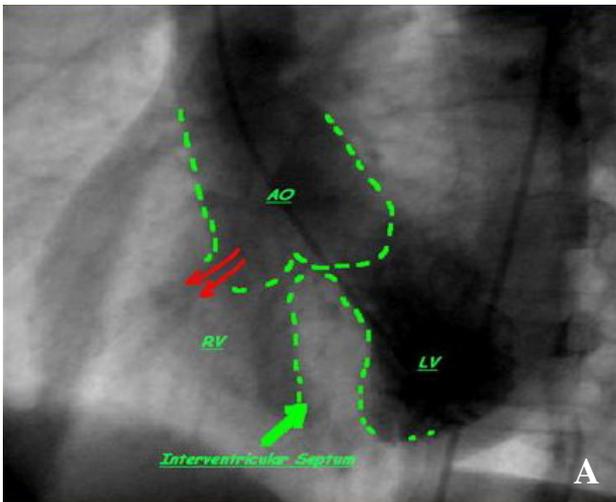


Figure 4 (A,B). Angiocardiographic views of the patient.

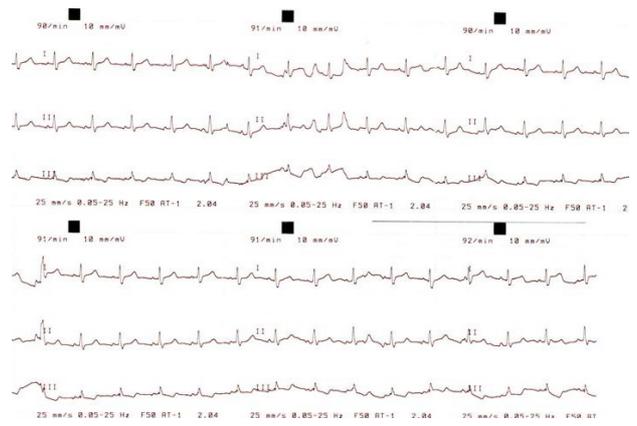


Figure 5. Post-op ECG.

## DISCUSSION

John Thurman first described SVA in 1840, and Hope further described it in 1939. Approximately 65-85% of SVAs originate from the right sinus of Valsalva, 10-30% of them originate from non-coronary sinus while aneurysms originating from the left sinus are exceedingly rare (<5%)(1).

Male to female ratio is 4:1. Rupture of the dilated sinus will lead to intracardiac shunting when a communication is established with the right atrium (Gerbode defect [10%]) or directly into the right ventricle (60-90%) (2,3). In the USA, SVA was present in 0.09% of large autopsy series and its incidence ranged between 0.10-0.23 percent in the Western surgical studies (4). It is of great importance that SVA is more prevalent in Asian surgical series (0.46-3.5%) (1) and correlates with more supracristal ventricular septal defects (VSDs) (5, 6). Most ruptured SVA cases occur from puberty to age 30 years and are often diagnosed or presented clinically at this age. Surgery is the definitive therapy and urgent surgical repair is recommended in all patients with ruptured SVA, especially with intracardiac shunting (7). Recently interventional treatments have been implemented such as occlusion of the defect with ASD closure devices, with most studies using Amplatzer devices (8, 9).

Complications include myocardial infarction due to coronary artery compression (10), complete heart block due to AV node compression, right ventricular outflow tract obstruction (11), sudden cardiac death, infective endocarditis (12), tamponade, and rarely a potential source of cerebrovascular emboli (13).

There are reports indicating that palpitation or syncope may present secondary to obstruction of the left or right ventricular out flow tract (11), but there have been no reports of frequent APDs in the literature. Although APDs could be due to sympathetic over-activity, its consistency without signs or symptoms of heart failure may indicate a pressure effect of aneurysm on cardiac atrial tissues and coronary vessels supplying SA node. This case introduces a new presentation of an old disease which should be considered by the treating physician (14).

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