Murmur After Myocardial Infarction: Is Always a Post Myocardial Infarction Ventricular Septal Defect: Case Report

Miyokard İnfarktüsü Sonrasında Gelişen Kardiyak Üfürüm: Her Zaman Post-Miyokard İnfarktüsü Ventriküler Septal Defekt midir?

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ABSTRACT Symptoms of acute left heart failure dominate in both ventricular septal defects occurring after transmural myocardial infarctions (post MI VSD) and rupture of a sinus Valsalva aneurysm (SVA), which may pose a difficulty in differential diagnosis. Ruptured SVA mandate prompt and urgent surgical correction. Also patients with post myocardial infarction VSD should undergo emergent surgery. The success of the surgery in both situations depends on the prompt medical stabilization of the patient and the prevention of cardiogenic shock. Implantation of an intraaortic balloon pump would have been life-saving in case of a post-MI VSD, but would have increased the aorto-right atrial shunt and aggravate the left heart failure in a ruptured SVA, thus may have even led to the loss of the patient. Simultaneous coronary artery disease and ruptured SVA is extraordinarily rare. We present here, a case of ruptured SVA with concomitant coronary artery disease, who had an MI two weeks before.

Key Words: Sinus of valsalva; heart septal defects, ventricular; myocardial infarction

ÖZET Transmural miyokard infarktüsünden sonra gelişen ventriküler septal defekt (post MI VSD) ve rüptüre olmuş sinüs Valsalva anevrizması (SVA) olgularının her ikisi de öncelikli olarak akut sol kalp yetersizliğinin klinik semptomları ile seyrederler. Benzer klinik tablolar ayırıcı tanıda zorluk oluşturabilir. Hem post MI VSD hem de rüptüre olmuş sinüs Valsalva anevrizması acil cerrahi girişimi gerektirir. Her iki durumda da cerrahi tedavinin başarısı hastanın stabil hale getirilmesi ve kardiyojenik şokun önlenmesine bağlıdır. Post MI VSD olgusunda intraaortik balon pompası yerleştirilmesi hayat kurtarıcı rol oynayabilirken, rüptüre olmuş bir SVA olgusunda ise aorto-sağ atrial şantı arttırarak sol kalp yetersizliğini daha da ileri duruma taşıyabilir ve hatta hastanın kaybedilmesine sebep olabilir. Koroner arter hastalığı ile birlikte aynı anda rüptüre SVA görülmesi oldukça nadir rastlanan bir klinik tablodur. Bu makalede, iki hafta önce miyokard infarktüsü hikayesi bulunan bir olguda koroner arter hastalığı ile birlikte eşzamanlı rüptüre sinüs Valsalva anevrizmasının tanı ve tedavi süreci anlatılmaktadır.

Anahtar Kelimeler: Valsalva sinüsü; kalp septum kusurları, ventriküler; miyokard infarktüsü

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A 64 years old female patient was transferred to our clinic for cardiac catheterization with a diagnosis of post myocardial infarction ventricular septal defect (post-MI VSD). She had a history of anterior MI and pulmonary edema two weeks before for which she had been intubated. Bilateral coarse rales and a grade 3/6 systolic murmur on the mesocardiac spot had been reported. Her echocardiography report revealed moderate left ventricular systolic dysfunction, mild-moderate mitral, moderate tricuspid insufficiency and a post-MI VSD. The final diagnosis had been acute anterior MI and a consecutive post-MI VSD.

Catheterization revealed 80% stenosis of the left anterior descending artery (LAD) ostium, and 70% stenosis of the first Obtuse Marginal (OM1) artery. There was also a radio-opaque material flow out of the aortic sinus (Figure 1). Subsequent echocardiography revealed this shunt was actually from the aortic root to the right atrium. Thus this was a ruptured aneurysm of the sinus of Valsalva rather than a post-MI VSD.

In the operation, it was actually a ruptured aneurysm of the non-coronary sinus opening into the right atrium (Figure 2), as was also shown in perioperative transesophageal echocardiography (Figure 3). The aorta was fixed with a pericardial patch, two bypass grafts were done, a small incidentally found patent foramen ovale was closed, and the tricuspid valve was fixed with a De-Vega annuloplasty. She was discharged in good condition after 7 days uneventfully.

DISCUSSION

Sinus of Valsalva aneurysm (SVA) is rare consisting, 1.2% of all congenital heart diseases and 0.2-1.5% of all cardiac surgical interventions. SVA is caused by a congenital deficiency of fusion between the aortic media and the fibrotic tissue of the aortic sinuses.[1] Complicated (symptomatic and/or ruptured) SVA is diagnosed early with their associated arryhtmic, embolic and coronary complications.[2] The most common complication is rupture usually in the right half of the noncoronary aortic sinus.[1,3]

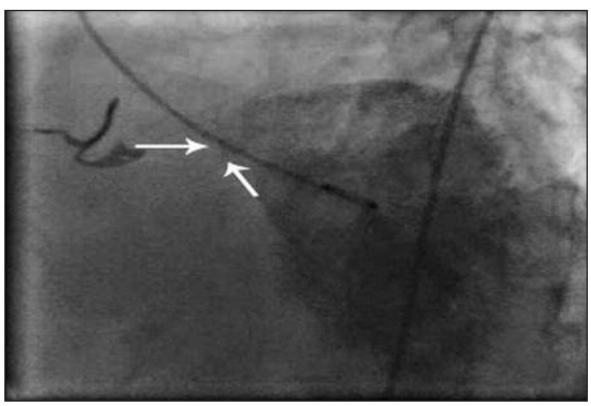

FIGURE 1: Flow of the contrast material out of the aortic sinus into the right atrium during left ventricular catheterization. Arrows mark the aortic tear.

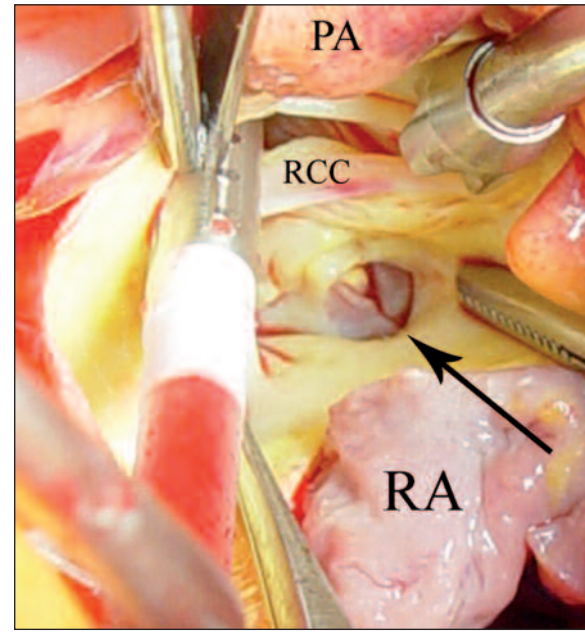

FIGURE 2: Intraoperative surgeon's view of the ruptured noncoronary sinus of Valsalva (Arrow).
RCC: Right coronary cusp; PA: Pulmonary artery; RA: Right atrium.
(See color figure at http://cardivascular.turkiyeklinikleri.com/)

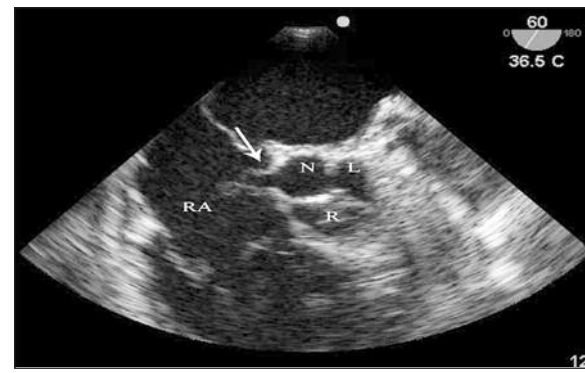

FIGURE 3: Transesophageal Echocardiography of the aortic valve very clearly showing the ruptured noncoronary sinus of Valsalva (Arrow). N: Noncoronary cusp; R:Right coronary cusp; L:Left coronary cusp.

Sinus of Valsalva aneurysms is often asymptomatic unless ruptured. The general presentation of ruptured SVA is congestive heart failure due to acute onset left-to-right shunting.[4-6] However endocarditis, heart block, and myocardial ischemia are also possible, depending on the neighboring structures. Patients having MI after rupture of the SVA without any coronary artery disease are also reported.[7,8]

The incidence and the mechanism of ischemia and MI after ruptured SVA is unclear. Some believe it is the combination of low coronary perfusion pressure and elevated oxygen demand.[7,8]

The presence of a systolic murmur -exact onset unknown-, with a recent anterior MI history, together with echocardiography reports of a shunt, altogether may have led to the misdiagnosis of a post-MI VSD.

Simultaneous coronary artery disease and ruptured SVA are extraordinarily rare. The primary pathology leading to MI in this patient is unclear, whether it was the coronary artery disease or the SVA rupture alone, or maybe SVA rupture accentuating the ischemia in the already stenosed LAD region.

What then would be the clinical implications of differentiating post-MI VSD from a SVA rupture? With acute onset left heart failure, luckily this patient could be hemodynamically stabilized after intubation, sedation and inotropic support. In a more unstable situation, when an intra-aortic balloon pump (IABP) would be considered for myocardial support, we would have two exactly opposite clinical results. Implantation of an IABP would have been life-saving in case of a post-MI VSD, but would have increased the aorto-right atrial shunt and aggravate the left heart failure in a ruptured SVA, thus may have even led to the loss of the patient.

Thus, considering surgery, both cases would be equally challenging for the surgeon, but for the clinician exact differentiation of the two clinical pathologies may be vital.

CONCLUSION

Post-MI VSD and ruptured SVA may clinically mimic each other. Concomitant coronary artery disease in such patients may lead to a false diagnosis of post MI VSD. Exact differentiation of the two pathologies via several different imaging techniques may be vital.

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