Ventricular Septal Defect as A Consequence of Stab Wound: Two-Year Follow-Up Without Repair: Case Report

Bıçaklı Yaralanmaya Bağlı Ventriküler Septal Defekt: Onarım Yapılmadan İki Yıl Takip Edilen Bir Olgu

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ABSTRACT Many cases with penetrating cardiac injury are later diagnosed to have suffered injury also to one of the intracardiac structures, mostly interventricular septum. Even ventricular septal defect (VSD) was diagnosed preoperatively, immediate repair is not recommended unless intractable heart failure develops as VSD can shrink or even spontaneously close with time. We present a case of traumatic VSD whose VSD was diagnosed on the postoperative fifth day. He was followed up along two years without repair. Although defect size has enlarged, pulmonary-to-systemic blood flow ratio decreased to 0.94 from 1.4. It may be recommended that if a patient is asymptomatic there is no point in insisting on immediate VSD repair.

Key Words: Heart septal defects, ventricular; wounds, penetrating

ÖZET Penetran kalp yaralanmalı birçok olguda, başta interventriküler septum olmak üzere bir intrakardiyak yaralanmanın da olduğu sonradan teşhis edilmektedir. Gerçi ventriküler septal defekt (VSD) preoperatif dönemde teşhis edilse bile, tedaviye cevap vermeyen bir kalp yetmezliği gelişmedikçe acil onarımı tavsiye edilmez, çünkü zamanla VSD küçülebilir hatta tamamen kapanabilir. Burada postoperatif beşinci günde tanısı konulan travmatik bir VSD olgusu takdim ediyoruz. Olgu iki yıl boyunca VSD onarımı yapılmaksızın takip edildi. Bu süre içinde defekt boyutu arttığı halde, pulmoner/sistemik kan akım oranı 1.4'ten 0.94'e düştü. Asemptomatik hastalarda, travmatik VSD'nin acil onarımı için ısrarcı olmaya gerek olmayabilir.

Anahtar Kelimeler: Kalp septum kusurları, ventriküler; yaralar, delici

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Penetrating cardiac trauma due to stabbing may result in a wide range of injuries to intracardiac structures.[1] Because little time and little diagnostic information are available before an urgent operation, patients with penetrating cardiac injuries may require additional surgery to repair injuries not found during the acute stabilization process.[2] Almost 25% of patients are later diagnosed to have suffered injury also to one of the intracardiac structures.[3] The incidence of VSD is about 4.5% among the cardiac traumas.[4] We present a case of traumatic ventricular septal defect after a penetrating cardiac trauma causing pericardial tamponade and cardiac rupture.

CASE REPORT

A 15-year-old male was admitted to the emergency department with a stab wound in his chest just medial to left midaxillary line through the 5. intercostal space. Initial examination revealed dyspnea, tachycardia (130/min), hypotension (80/50 mmHg), increased central venous pressure (20 cmH_2O). A left hemothorax was identified by chest radiographs. Urgent echocardiographic examination revealed pericardial effusion and thrombus-like appearance in the pericardial space. Because of the haemodynamic instability and suspected cardiac tamponade, urgent left anterior thoracotomy was performed during which 1.5-cm perforation in the right ventricular free wall was repaired with direct suture closure using Teflon pledgets. Lung laceration in the left upper lobe was repaired by direct sutures, too.

On the post-operative fifth day, the patient was noted to have a new systolic murmur. Two-dimensional transthoracic echocardiography (TTE) revealed defect in the interventricular septum near the apex, 4 mm wide, and left to right shunt flow. These findings were confirmed by transesophageal echocardiography (TEE). Given the asymptomatic course of the patient and size of the shunt, which had been assessed as non-significant (echocardiographic assessment of Qp/Qs was 1.4), a conservative approach was proposed. He has been followed up with TTE monthly for the first 6 months and then once for every 3 months. The murmur of VSD has been still present on physical examination two years after the trauma. The size of the muscular defect has enlarged to 6 mm, however Qp/Qs ratio has decreased to 0.94 and the patient has no signs of right-heart volume overload or heart failure (Table 1 and Figure 1, 2, 3 representing the TTE and CT ventriculography performed at the second year follow-up).

DISCUSSION

Ventral septal defect is the most common intracardiac lesion found after penetrating cardiac trauma.[3] In most cases any diagnostic intervention may not be performed because of patient's clinical status, hence VSD may not be diagnosed preoperatively. Furthermore many of these lesions become clinically detectable typically in a delayed fashion, sometimes weeks later, when the defect has become larger after resolution of surrounding edema with fibrotic healing and retraction of the

TABLE 1: Last transthoracic echocardiography and transesophageal echocardiography findings.

Left ventricle end-diastolic diameter	5.3 cm
Left ventricle end-systolic diameter	3.55 cm
Thickness of interventricular septum	1 cm
Thickness of posterior wall	0.9 cm
Ejection fraction	70%
Pressure gradient on VSD	82.1 mmHg

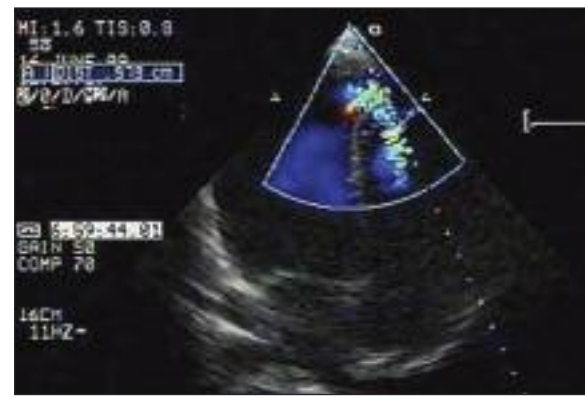

FIGURE 1: Echocardiography image with color doppler demonstrating the ventricular septal defect with 5.73 mm wide.

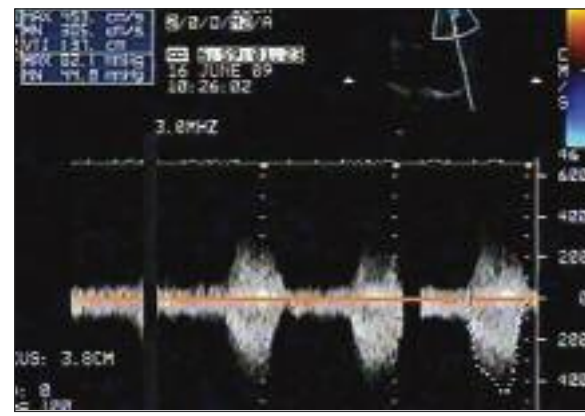

FIGURE 2: Continuous wave spectral doppler showing the pressure gradient in the ventricular septal defect to be 82.1 mmHg.

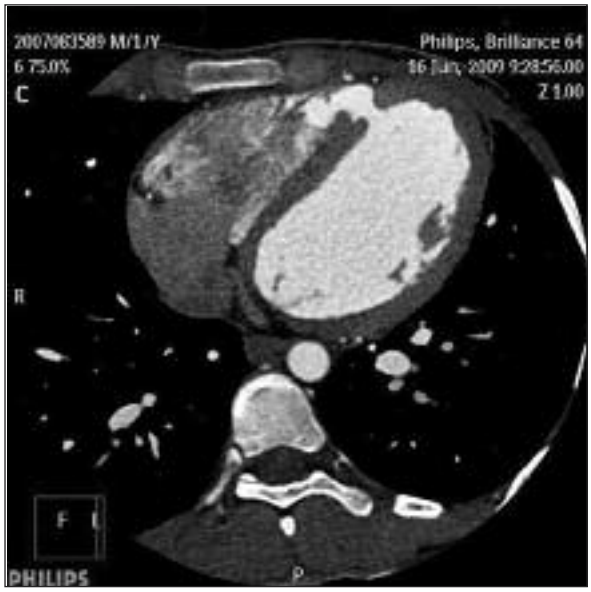

FIGURE 3: Ventriculography obtained by computerised tomography confirms VSD near the apex with little left to right shunt.

intracardiac wound, lyses of an occluding clot, or ventricular enlargement.[4,5] For the same reasons, detection of such defects at the time of initial evaluation can be extremely difficult, even with TEE.[4] The reported mean presentation is 11 months after initial injury.[5]

Likewise, patient's physical examination and TTE performed on arrival to intensive care unit could not cause to be in doubt about VSD. Even if VSD was detected pre/or intraoperatively, we would not carry out urgent repair. Ito and colleagues,[6] does not recommend immediate repair unless intractable heart failure develops. Elective repair may allow time for the margins of the defect to fibrose, thus becoming more favorable to suturing. Besides VSDs can shrink or even spontaneously close with time and not all VSDs need to be treated sugically.[1-7] About 30-40% of patients remain asymptomatic and hence do not require another surgical intervention.[3] A conservative approach has been recommended for hemodynamically insignificant, small traumatic VSDs with a low pulmonary-to-systemic ratio. Follow-up with echocardiography is adequate. Until recently, formal cardiac catheterization with measurement of the shunt was also performed. More recent advances with echocardiography have diminished the role of invasive catheterization.[5]

The decision to perform elective surgical repair is made on the pulmonary-to-systemic flow ratio and presence of significant right-heart failure. However there is controversy about the Qp/Qs ratio. As an indication for VSD repair, some authors advocate Qp/Qs ratio≥1.5,[3,5,8] whereas others agree ≥2.0.[2,6,7] In our case, although the defect size has enlarged, Qp/Qs ratio have decreased to 0.94 from 1.4. This fact may be attributed to the restriction of blood flow through muscular portion of interventricular septum by contraction. Hence we can conclude that given the pulmoner-to-systemic blood flow ratio may decrease with time, especially in muscular defects, if a patient is asymptomatic there is no point in insisting on immediate VSD repair.

An alternative to surgery is transcatheter closure of a posttraumatic VSD, which has been reported more recently for patients beyond infancy with a posttraumatic muscular VSD, provided that the VSD must not be too close to the aortic and atrioventricular valves to allow a safe procedure and no valvular device protrusion.[7,9,10] Hemolysis, which is related to high velocity jet through and around the occluder, has been resolved after complete closure of the defect by placement of a second occluder or simply after endothelialization of the device.

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