Isolated ventricular septal rupture secondary to blunt trauma

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Abstract

A ventricular septal rupture (VSR) is a rare complication of blunt chest trauma. We describe the case of a 25-year-old man who developed a VSR as a result of a high-speed road accident. The rupture was closed by left ventricular remodeling and replacement of the diseased myocardium with a Dacron patch. The patch sutures were reinforced with glue. Redo surgery was necessary at nine months due to patch detachment and embolization of the glue in the right lower lobe. The patient is asymptomatic, at 13-month follow-up.

Keywords: Trauma; Blunt; Myocardial injury; Cardiac intervention; Shunts (cardiac); Wound closure

1. Clinical summary

A 25-year-old man was hospitalized after a high-speed road accident. He was hemodynamically stable, his echocardiogram was normal, troponin Ic had increased to 42 mg/l.

An initial total body computed tomography (CT)-scan showed left thoracic trauma, bilateral lung contusion, left pneumothorax, but no great vessel injury. Two days later, he developed hemodynamic instability with acute respiratory failure. Controlled mechanical ventilation and vasoactive agents were required. A chest tube was inserted into the left pleural space and 1500 ml of blood was drained.

As there was no clinical improvement, arteriography was performed to rule out intra-thoracic or intra-abdominal bleeding. Intra-splenic microaneurysms were detected and embolized. There was a loud systolic murmur over the whole cardiac area on auscultation. Transthoracic echocardiography revealed a septal defect with pseudoaneurysm of the inferior and apical portion of the left ventricle (LV) and significant left-to-right shunt, without pericardial effusion (Fig. 1a). The left-to-right gradient was 81 mmHg, according to the Bernoulli formula.

Coronary angiography showed normal coronary arteries, eliminating ischemic ventricular septal rupture (VSR) due to coronary injury. Left ventriculography showed an infero-apical VSR (Fig. 1b). Pulmonary arterial systolic pressure, right atrial pressure and pulmonary capillary pressure were respectively of 45, 10 and 17 mmHg.

Emergency surgery was performed revealing an apical myocardial contusion associated with septal laceration (Fig. 2a). The VSR was closed using a left ventricular remodeling technique following the principles by Jatene for left ventricular aneurysm repair [1], a Dacron patch was sutured to the limits of contused myocardium, excluding the septal rupture and slightly reducing left ventricular cavity size (Fig. 2b). The patch sutures were reinforced with glue (Cardial®).

Postoperative course was uneventful and echocardiography at hospital discharge showed no residual ventricular septal defect (VSD).

At three-month follow-up, echocardiography revealed patch dehiscence with significant left-to-right shunt. A CT-scan and myocardial magnetic resonance imaging confirmed the presence of an apico-inferior VSD. CT also revealed a right lower lobe pulmonary embolism thought to be due to foreign material (glue).

Redo surgery was performed nine months after the first surgical repair, consisting of reclosure of the VSD with a Dacron patch. Right pulmonary embolectomy was performed confirming embolization of glue. The postoperative course was uneventful and the patient was asymptomatic at 13-month follow-up.

2. Comment

The most common injuries after blunt chest trauma are cardiac contusion, valvular damage, and aortic insufficiency due to a tear in the aortic cusp or avulsion of a commissure. Echocardiography can help to diagnose pericardial effusion, myocardial dyskinesia, or valvular dysfunction [2]. VSRs are uncommon traumatic heart lesions. Cardiac injuries may be more common in the absence of broken ribs or sternal lesions, particularly in younger patients with a less rigid chest wall.
As in our patient, VSR may be delayed for several hours to several days after the trauma [2], but usually occurs within the first week [3], and may be asymptomatic for a long period of time [4].

Myocardial contusion may occur in cases of major chest trauma, due to a squeezing effect on the heart between the spine and the sternum [4, 5], direct and severe compression of the chest, or sudden deceleration. If the trauma occurs during the late diastolic isovolumic contraction phase of the cardiac cycle or during early systole when the valves are closed and the ventricles and septal are in high-pressure state [4], the septal wall can be damaged. The muscular part of the interventricular septum, near the apex of the heart, is most likely to be injured [5]. Alteration of the microcirculation in myocardial contusion can lead to necrosis, and eventually to rupture [4]. At the time of chest trauma, the sudden high pressure [4], sometimes associated with torsion or a pulling effect, may tear the heart [6] or be responsible for a burst-lesion of the ventricle with a septal aneurysmal appearance.

Post-traumatic VSR may also be caused by reopening of a healed congenital lesion. In contrast to congenital VSDs, which are more frequently localized on the membranous septum, the VSD in our case was near the apex, with an aspect of septal contusion never seen in congenital cases.

VSR may occur after myocardial infarction and coronary lesions have to be eliminated. In our case, transthoracic echocardiography revealed features similar to ischemic VSR.

The diagnosis should be made as quickly as possible in cases of hemodynamic instability and emergency surgical closure of the VSR is recommended.

In most cases, when the clinical situation is stable or stabilized with an intra-aortic balloon pump, surgery can be deferred. This allows suturing to stronger fibrous scar tissue and possibly avoids patch detachment due to suture failure on weak myocardial tissue. We choose the Jatene procedure in order to improve ventricular remodeling in this young patient, whereas a double sandwich patch could decrease ventricular function. Another advantage of the left ventricular remodeling procedure is that the high left ventricular pressure apply the patch against the left ventricular muscular wall and reduce tensions on the suture lines. Finally, the sutures were reinforced with glue and the left ventricular part was closed over the patch.
In some cases, transcatheter occlusion may be performed with an Amplatzer device. Lee et al. described closure of patch deinsertion using an Amplatzer device [7]. In our patient, the location of the secondary patch rupture was too close to the apex and free wall of the LV, leaving insufficient margins for insertion of the occluding device [8].

In conclusion, VSR is a rare but serious complication of blunt chest trauma. The diagnosis of VSR may be delayed when major lesions are present. VSR should be considered in cases of chest trauma with increased troponin Ic levels, new heart murmur, or hemodynamic instability. Echocardiography can confirm the diagnosis and should be repeated regularly when myocardial contusion exists, in combination with clinical examination and auscultation.

Management of traumatic VSR is similar to the management of ischemic VSR. If the patient is hemodynamically unstable, surgery must be carried out diligently. Percutaneous closure may be possible depending on the localization, size, and margins of the rupture.

References