VENTRICULAR SEPTAL RUPTURE

Delayed Repercussions of Blunt Trauma: Isolated Muscular Ventricular Septal Defect

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INTRODUCTION

Blunt trauma can lead to various cardiac injuries including stress-induced cardiomyopathy, myocardial infarction, valvular injuries, cardiac contusion, free wall rupture, and ventricular septal defect (VSD). Some of these injuries are self limited, and others are associated with high mortality. We present a case of a young man with isolated large apical VSD that was treated with surgical repair after rapid diagnosis with transthoracic echocardiogram (TEE).

CASE PRESENTATION

A 16-year-old Caucasian male restrained backseat passenger of a side collision motor vehicle accident by a semitrailer was admitted to the hospital with multiple injuries and transient loss of consciousness on the scene. Initial evaluation revealed periorbital hematoma and left forearm fracture. At presentation, he was hemodynamically stable with an unremarkable cardiac examination. Head and cervical spine computed tomography scan did not reveal any acute intracranial or cervical abnormalities. Computed tomography scan of the chest, abdomen, and pelvis with contrast demonstrated right-sided pulmonary contusion with minor laceration and tiny pneumothorax, nondisplaced third rib fracture, and grade 2 (nonsurgical) splenic lacerations.

Within 24 hours of presentation, the patient developed worsening respiratory distress requiring noninvasive positive pressure ventilation with 45% Fio2. He also manifested progressive tachycardia (heart rate 110–120 bpm) and hypotension (blood pressure 98/48 mm Hg). Twelve-lead electrocardiogram demonstrated sinus tachycardia without significant ST or T wave changes. Cardiac telemetry revealed frequent premature ventricular contractions. Serum troponin T level was elevated at 1.7 ng/mL (normal < 0.01 ng/mL). On examination, he had a hyperdynamic precordium, grade IV/VI holosystolic murmur with a thrill at the lower sternal borders, and grade I/IV diastolic rumble at the apex. His jugular venous pressure was 10 cmH2O. Lung fields were clear by auscultation. Urgent bedside TTE revealed a large apical muscular VSD with large left-to-right shunt by color flow Doppler (Figures 1A and B). Left ventricular systolic function was hypodynamic with ejection fraction of 70%. Atrial and ventricular sizes were normal. The right ventricular systolic pressure (RVSP) was elevated at 62 mm Hg. No valvular abnormalities or pericardial effusion were present.

The patient was initially observed without cardiac intervention due to relative hemodynamic stability, with ongoing monitoring of lung injury, splenic laceration, and rib fractures. The possibility of transcatheter device closure was discussed; however, a follow-up TEE further characterized the VSD. The close proximity of the VSD to the cardiac apex did not allow adequate apical rim for consideration of device closure (Figures 2A and B), thus the decision was made to proceed with surgical repair. Four days postaccident, he underwent resection and debridement of the 2-cm VSD (Figure 3A) and subsequent successful patch closure with Teflon patch via left apical ventriculotomy (Figure 3B). A follow-up TTE on postoperative day 3 showed intact patch closure without residual VSD (Figure 3C and D). The apical ventricular septum was akinetic at the patch repair site with no additional wall motion abnormalities. Biventricular size and systolic function were normal. The RVSP normalized at 23 mm Hg, and the diastolic gradient across the mitral valve had normalized. His initial postoperative course was complicated by pericarditis with a small to moderate posterior pericardial effusion and postpericardiotomy syndrome, treated with colchicine, diuretics, and pain control. High-dose nonsteroidal anti-inflammatory drugs were avoided secondary to the patient’s multiple orthopedic injuries. By postoperative day 6, the pericardial effusion was trivial in size and pain had markedly improved; he was discharged home in stable condition with close outpatient follow-up. Six months of follow-up TTE did not reveal residual VSD.

DISCUSSION

Our patient had a rare clinical presentation of apical VSD secondary to blunt trauma. The VSD developed within 24 hours of the impact. Most reported cases of post-traumatic VSD have come from postmortem series. In the largest autopsy series, Parmley et al. demonstrated that following nonpenetrating traumatic injury, cardiac rupture was seen in 353 out of 546 (65%) examined postmortem hearts; 30 of these cases were VSD.1 Another review in 2005 demonstrated a mortality rate of 19% among 58 patients with VSD caused by blunt chest injuries.2 The mechanism of injury has been hypothesized to be related to mechanical compression of the heart between the sternum and spine leading to a concomitant increase in intrathoracic pressure at end diastole/early systole. Subsequently, myocardial contusion, coronary artery dissection, or adventitial hemorrhage can develop.3 Due to the absence of ischemic changes on electrocardiogram in our patient, the most likely primary event seems to have been myocardial contusion predisposing to rupture of the apical septum.

While small traumatic VSDs have been shown to close spontaneously,4 those larger than 2 cm have been associated with a mortality...
Figure 1  (A) TTE apical four-chamber view demonstrates the apical VSD (yellow arrow) (Video 1).  (B) Color Doppler reveals left-to-right shunt through the VSD (Video 2).  (C) Continuous wave Doppler through the VSD illustrating the peak gradient across the defect of 34 mm Hg.  (D) Increased gradient across the mitral valve (mean diastolic Doppler gradient 6 mm Hg at heart rate 114 bpm) consistent with increased flow from large left-to-right shunt.

Figure 2  (A) TEE short-axis view of the ventricular apex further illustrates the large VSD of at least 1.4 cm (yellow arrow; Video 3). (B) Color Doppler demonstrates the large left-ventricular to right-ventricular shunt across the VSD (Video 4). (C) TEE short-axis three-dimensional imaging modality illustrates the depth of the VSD.
rate approaching 71% and if left unrepaired can lead to late cardiovascular complications including pulmonary arterial hypertension. Early surgical repair was undertaken in our case to alleviate congestive heart failure and to prevent potential long-term cardiovascular complications of a large left-to-right shunt.

CONCLUSIONS

Isolated ventricular septal defect is a rare clinical sequela from blunt trauma and can be life threatening. A large percentage of cases are diagnosed postmortem. A high index of suspicion for isolated VSD is required in patients with cardiac arrhythmia, elevated troponin, or hemodynamic instability following blunt trauma, especially in the absence of concomitant valvular injury, pericardial effusion, or wall motion abnormalities. Rapid diagnosis with the aid of echocardiography in our case was necessary to avoid potential late mortality or morbidity related to significant changes in intracardiac hemodynamics.

Figure 3  (A) Gross picture via left apical ventriculotomy illustrates the VSD in close proximity to the apex of the heart (yellow star marks the probe through the VSD opening). (B) Gross image of the repaired VSD using 2-cm Teflon patch. (C) TTE apical four-chamber view and (D) TTE parasternal short-axis view at the level of the ventricular apex illustrates the absence of residual defect or left-to-right shunt after VSD patch closure (yellow arrow; Video 5).

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2016.12.001.

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