Ventricular Septal Defect Secondary to Non-Penetrating Chest Trauma

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A 52-year-old man acquired a ventricular septal defect following non-penetrating chest trauma. Four days after the traumatic accident, he showed signs of congestive heart failure. Imaging techniques using echocardiography and left ventriculography were helpful in diagnosing the condition. Surgical repair by patch closure of the ventricular septal defect was accomplished 7 days after the traumatic accident.

Key Words: Ventricular septal defect; Traumatic accident; Surgical repair

In cases of non-penetrating chest trauma, ventricular septal defect (VSD) is rarely the sole heart lesion. Urgent diagnosis, treatment, and assessment of the need for surgical repair are necessary. We report a case of VSD secondary to non-penetrating chest trauma in which surgical repair was performed.

Case Report

A 52-year-old man was hit on the left side of the chest by a piece of lumber measuring about 12 x 12 x 180 cm while working at a lumbermill on 9 February 1996. He lost consciousness for several minutes and was referred to our hospital. Physical examination revealed no specific findings except an abrasion on his left chest wall. He was prescribed a breast band for chest pain. On 13 February, 4 days after his initial visit, he was admitted to our hospital because of dyspnea. On physical examination, a new harsh holosystolic murmur could be heard, loudest in the fourth left intercostal space, with a thrill detected in the same area. The patient smoked 30 cigarettes and easily consumed 360 ml of rice wine per day. He had suffered a cerebral infarction 6 years earlier, since when he had been treated for essential hypertension. A chest radiograph revealed an increase in the bilateral pulmonary vascular markings. The ECG showed regular sinus rhythm and poor R progression in the precordial lead with no Q wave or ST segment elevation (Fig 1). Echocardiography showed a muscular apical VSD. The diameter of the left ventricular (LV) cavity was increased (left ventricular diameter at end-diastolic phase/end-systolic phase = 60/32 mm, ejection fraction = 0.77). Aortic or mitral regurgitation was not detected. In the short-axis view, the defect on the LV side of the interventricular septum (IVS) was 20 mm long at end-diastole and nearly 0 mm long at end-systole. Serum chemistry showed no specific findings except for a mild elevation of lactate dehydrogenase (LDH; 490 IU, normal range 242–462). The results of cardiac catheterization were as follows. Mean pressure in the right atrium (RA) was 4 mmHg. RV pressure was 69/0 mmHg. The pulmonary artery (PA) pressure was 51/15 mmHg. The pulmonary capillary wedge mean pressure was 13 mmHg. LV systolic/end-diastolic pressure were 117/19 mmHg. The aortic pressure was 111/71 mmHg. The O₂ saturation step-up was noticed at the apex in the RV (RA 72.2%, RV apex 94.0%, RV out tract 87.6%, PA 84.5%). The pulmonary/systemic flow ratio was 2.47 and the left-to-right shunt flow ratio was 59.6% calculated by the Fick method. Left ventriculography revealed opacification of the RV through a muscular apical VSD (Fig 2). Coronary angiography showed a right dominant coronary artery system without any significant organic stenosis.

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Despite drip infusion of dopamine (2-3 μg/kg per min) and oral administration of furosemide (120 mg/day), congestive heart failure was unchanged and hypoxia continued (O₂ saturation was less than 96% on O₂ inhalation). On 15 February, echocardiography showed an increase in VSD size (Fig.3). The size of the LV cavity was not changed. In the short-axis view, the defect on the LV side of the IVS was 24 mm long at end-diastole and 12 mm long at end-systole. Inside the IVS, a large cavity shaped like a spade was seen. It was a maximum of 34 mm long at end-diastole. On 16 February, 7 days after the traumatic accident, an operation was performed. There was no damage to the pericardium and pericardial effusion was not bloody. The VSD was closed by a horse pericardium patch through RV incision. A pathologic specimen showed inflammatory cell infiltration, fibrotic changes, and myocyte detachment in the myocardial tissue around the VSD. There were no findings of atherosclerotic changes in arterioles in that specimen.

**Discussion**

In general, traumatic non-penetrating VSD is caused by the impact of high-speed deceleration during traffic accidents and falls from high places. Few cases have underlying cardiac diseases because the majority of patients are infants and young men. Why the defect is generated only at the ventricular septum is still unknown. Parmley et al. empirically divided the forces
that cause non-penetrating injuries to the heart into 7 broad categories. Stinson et al.\textsuperscript{2} reported that predilection for septal perforation, particularly the muscular portion near the apex, was high. Inkley and Barry\textsuperscript{3} noted in animal studies an increased vulnerability when the heart is struck during the late-diastolic or isovolumic contraction phase. In our case, neither pulmonary contusion nor sternal or rib fractures were recognized. Only VSD occurred. It is important to point out that, following external injury, damage to the thoracic wall and to organs within the thoracic cage, do not always correspond. Rotman et al.\textsuperscript{4} reported that internal thoracic damage was less severe in cases of non-penetrating chest trauma in which skeletal rib fractures occurred. The thoracic wall may have a protective influence, decreasing the force of impact, as Dunseth and Ferguson\textsuperscript{5} reported that only 5 out of 17 patients who suffered traumatic VSD had sternal or rib fractures.

VSD secondary to non-penetrating injury can be diagnosed using echocardiography and cardiac catheterization. The former can exclude the presence of associated cardiac injuries. The left-to-right shunt flow ratio can be calculated by the latter, and coronary angiography should be used in older patients or in those at risk of coronary artery disease. Differential diagnosis of congenital and acquired VSD is dependent on circumstantial evidence. It is reasonable to assume that this VSD was caused by the lumber hitting his chest on 9 February and that the rupture of the ventricular septum occurred some time between 9 and 13 February. There is a possibility of myocardial tissue contusion during those 4 days because of a mild elevation of LDH. This speculation is supported by the pathologic findings that the presence of fibrotic tissue around a defect in the muscular septum suggests a recent inflammatory reaction and aids in the pathologic diagnosis of the traumatic VSD.\textsuperscript{6}

Until recently, surgical repair of acute postinfarction VSD has generally been carried out as soon as possible after establishing the diagnosis. Because of the risk of multiple organ failure following sustained heart failure, the defect-closing operation has usually been performed at an early stage. However, in some patients, such as those presenting a low left-to-right shunt rate and mild congestive heart failure, non-surgical treatment should be attempted first, followed 2–3 weeks later by surgical repair when the myocardial tissue surrounding the VSD shows signs of formation of a fibrous ring. In the same way, traumatic non-penetrating VSD causing medically uncontrolled congestive heart failure requires surgical repair, because only patients followed up medically have been reported.\textsuperscript{7}

References

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