Case Report

Delayed Presentation and Repair of Isolated Traumatic Ventricular Septal Defect

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Abstract

The case report of an isolated ventricular septal defect due to blunt chest trauma presenting more than a month after the injury and its successful surgical management is presented.

Introduction

Blunt chest trauma can cause a wide spectrum of cardiac injuries ranging from mild myocardial contusion to disruption of the atrioventricular valves or interventricular septum to free rupture. Serious cardiac injury most commonly results from motor vehicle accidents with steering wheel impact but can occur after any direct blow to the chest (eg, during sporting activities). Traumatic ventricular septal defects (VSDs) may occur as isolated lesions, but they are most commonly encountered in association with other cardiac injuries. Lillehei first repaired a traumatic VSD using a pump and a dog lung as the oxygenator in 1955 [1], and a few cases have been reported [2–8] since then. Of note, in most reported cases, traumatic VSDs present immediately or within several hours of the traumatic event. Delayed presentation is rare. We report here a case of an isolated traumatic muscular VSD that presented with heart failure 5 weeks after the accident.
An 8-year-old boy pulled a large flower box onto his chest and transiently lost consciousness. After a negative evaluation for closed head injury, he appeared fully recovered. There was no clinical evidence for chest injury at that time. However, 3 weeks later dyspnea and a thrill over the left sternal border developed, and he was referred to Children’s Medical Center, Dayton, Ohio.

Physical examination revealed signs of heart failure and a murmur and thrill consistent with a VSD. A chest roentgenogram revealed cardiomegaly and pulmonary venous congestion, and the electrocardiogram showed sinus rhythm with left bundle-branch block and wide Q waves with ST changes in the left lateral precordial leads suggesting possible ischemic injury. Echocardiography demonstrated a large anterior muscular VSD and large left-to-right shunt (Fig 1). Cardiac catheterization, performed to exclude coronary arterial injury, confirmed the diagnosis of a large muscular VSD and demonstrated normal coronary arteries.

The patient was referred to the Cleveland Clinic for repair. At operation, the heart was markedly enlarged and pulmonary pressure was high. Remarkably, there were no pericardial adhesions and only the faintest sign of scar tissue on the right ventricular free wall. On palpation, however, there was an indurated area to the right of the proximal left anterior descending coronary artery and a pronounced systolic thrill. There was no scarring or other external evidence of injury on the left ventricular posterior wall. With cardiopulmonary bypass support, bicaval cannulation, and cardioplegic arrest, a large anterior muscular VSD, partially obscured by trabeculae, was exposed transatrially (Fig 2). The margin of the defect was fibrotic and the left ventricular endocardium, visualized through the VSD, appeared white and thickened, consistent with endocardial fibrosis. The VSD was closed with interrupted sutures and a Dacron patch. The patient has made an uneventful recovery with return of heart size to normal, no residual VSD, and mild regional dysfunction of the posterior left ventricular wall on follow-up echocardiograms.

![Fig 1. Two-dimensional echocardiogram, long-axis parasternal view, demonstrating the ventricular septal defect (arrows). (LA = left atrium; LV = left ventricle; RV = right ventricle.)](91K)

![Fig 2. Intraoperative appearance of the ventricular septal defect (arrows) exposed transatrially.](152K)
Serious cardiac injury can occur in the absence of sternal or rib fracture, particularly when the heart is compressed between the sternum and the spine. Immediate rupture can occur when the heart is compressed in early systole when the ventricles are full and the valves closed. Delayed rupture can occur as a result of contusion that progresses to necrosis and perforation.

We believe that significant myocardial contusion occurred at the time of blunt chest trauma in the case presented, followed by delayed disruption of the muscular septum. An initially small VSD may have gradually enlarged, causing the delayed onset of severe heart failure. With operation performed 8 weeks later, the fibrotic edges of the VSD facilitated suture placement and repair. Residual regional left ventricular dysfunction is likely the result of posterior left ventricular wall myocardial contusion. This case illustrates that significant cardiac injury can occur after blunt chest trauma and that a high index of suspicion coupled with careful follow-up, including physical examination and noninvasive testing, is necessary for its detection and appropriate management.

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