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Case Report

Traumatic ventricular septal defect resulting in severe pulmonary hypertension

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Traumatic ventricular septal defect (VSD) is a widely-recognized complication of both penetrating and blunt trauma. Most cases are repaired operatively without the long-term complications of pulmonary hypertension and heart failure that are associated with unrepaired congenital VSD in the pediatric population. To our knowledge, this is the first case report of a patient with a traumatic VSD who declined surgical repair at the time of injury and subsequently developed long-term complications of pulmonary hypertension and heart failure. With nearly 20 years of follow-up, this case demonstrates that the absence of surgical treatment in asymptomatic adult patients at the time of injury can lead to long-term complications associated with VSD. This case also shows that aggressive surgical treatment in patients with severe pulmonary vascular disease and heart failure secondary to traumatic VSD can be performed safely and should be considered in cases refractory to efficacious medical interventions.

INTRODUCTION

Traumatic ventricular septal defect (VSD) is a recognized complication of both penetrating and blunt trauma [1]. The mechanism of injury in traumatic VSD is usually motor vehicle collision or stab wounds, but any force that compresses the heart between the sternum and vertebrae can potentially cause rupture of the ventricular septum. The clinical manifestations and natural course of traumatic VSD vary widely so a high degree of clinical suspicion may be necessary to make the diagnosis. Patients may present acutely with a holosystomlic murmur, non-specific EKG changes, chest pain, pulmonary edema and hemodynamic instability [1]. Prognosis depends on the size of the VSD [2]. Larger defects are associated with greater mortality [3]. One study showed a mortality rate of 56% within 15 days in patients who were managed non-operatively [2]. Early operative repair is usually recommended.

Long-term morbidity and mortality of the non-operative patient is not well described, but may resemble the natural course of congenital VSDs in which chronic pulmonary overcirculation may lead to morphologic changes of the pulmonary artery—such as intimal proliferation and fibrosis, medial hypertrophy and arteriolar occlusion [3]. Necrotizing arteritis and obliteration of the vessels eventually results in elevated pulmonary vascular resistance (PVR), which can lead to reversal of the left-to-right shunt (Eisenmenger syndrome). These patients are at risk for arrhythmia, congestive heart failure, paradoxical embolism, cerebrovascular accidents and severe hemorrhage from pulmonary infarction and rupture of the pulmonary artery [3]. Since 1970, there have been at least 58 case reports of post-traumatic VSD; results of surgical treatment and long-term outcome, however, have not been well described [4].

CASE REPORT

A 50-year-old man was assaulted and stabbed anteriorly near the sixth costal cartilage. The knife penetrated the right ventricle, and he underwent an emergent sternotomy and cardiorrhaphy for tamponade. Post-operatively, a VSD was recognized, but the patient declined further surgery. Eight years later, he sought medical attention for peripheral edema, jaundice and ascites. A large VSD with a left-to-right shunt (peak gradient 50 mmHg and right ventricular systolic pressure 70 mmHg) as

well as a severely dilated right ventricle with free wall/apical hypokinesis was demonstrated by echocardiography. Cardiac catheterization confirmed the VSD and pulmonary arterial hypertension: cardiac index 1.94 l/min/m^2, pulmonary arterial blood pressure (PAP) of 75/36 mmHg (mean 41 mmHg), Qp/Qs 3.05, pulmonary capillary wedge pressure 7 mmHg, PVR 9.43 wood units. The patient underwent repair of the VSD with a Dacron patch and tricuspid annuloplasty with a flexible annuloplasty band. His post-operative course was complicated by mediastinal hemorrhage and an acute parietal stoke. He recovered from both complications without long-term sequelae.

Since his operation, the patient has done well. Six and a half years post-operatively, he was hospitalized for dyspnea on exertion and leg edema due to mild biventricular heart failure and atrial flutter. Restoration of sinus rhythm was initially accomplished with amiodarone and beta-blockers and he ultimately underwent cardioverison. During that hospitalization, an echocardiogram revealed a residual VSD measuring 0.5–0.6 cm with minimal left-to-right shunting, a peak gradient of 23 mmHg, and a right ventricular systolic pressure of 61 mmHg. Subsequently, an outpatient right heart catheterization was performed, which showed a PAP of 62/38 mmHg (mean 46 mmHg), calculated Qp/Qs 1.09, PVR 9.29 wood units. At the time, the cardiac index was measured at 1.36 l/min/m^2 and right ventricular stroke work index (RVSWI) 5.1 g m/m^2 (normal range 5–10 g m/m^2). After stabilization and medical optimization, the patient opted against any further invasive testing including potential surgical management of the residual VSD. Since this visit, he has had two hospitalizations for volume overload and right heart failure as a result of his persistent PAH. His most recent right heart catheterization, performed during one of these episodes, revealed a pulmonary artery pressure of 80/24 mmHg (mean 44 mmHg) and PCWP of 8 mmHg with minimal left-to-right shunting. He is followed in PAH clinic where his pulmonary hypertension is managed with diuretics and sildenafil. His heart failure remains medically managed with fluid restriction, diuretics and digoxin. At last follow-up, he was unfortunately diagnosed with metastatic adenocarcinoma of the lung.

DISCUSSION

This is the first reported case of acquired pulmonary arterial hypertension and cor pulmonale due to a traumatic VSD from a penetrating wound. The traumatic VSD suffered by this patient mimicked the more common congenital cardiovascular malformation and resulted in severe pulmonary hypertension and right heart failure.

Several case reports of small post-traumatic VSD describe spontaneous closure [5], but the defects were likely small and do not result in heart failure. Most data on long-term sequelae of VSD are available from pediatric populations. The Euro Heart Survey on congenital heart disease in adults showed that nearly one in three patients with a congenital VSD develop PAH [6]. Another study of adult patients with congenital heart disease showed that roughly 6.2% of patients with septal defects develop PAH [7].

In traumatic VSD, there are few data regarding indications and results of closure of the defect, particularly after onset of pulmonary hypertension and heart failure. Closure of traumatic VSD is controversial and based on a consideration of hemodynamics, defect size, and heart failure symptoms [8]. Symptomatic and moderate-to-large VSDs with pulmonary-to-systemic shunts greater than 1.5:1.0 should be considered for closure [9]. As an alternative to surgery, transcatheter techniques have recently been introduced as a less-invasive treatment modality for VSD closure in trauma patients [10].

We describe a patient who developed severe pulmonary hypertension and right heart failure due to a traumatic VSD. The patient had an excellent outcome after surgical ventricular septal closure. This case demonstrates the long-term sequela of VSD and shows that aggressive surgical treatment in the adult with severe pulmonary hypertension and heart failure can be performed safely.

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