Cardiol Young 2009; 19: 109–110

ISSN 1047-9511
doi:10.1017/S1047951108003223
First published online 18 November 2008

Brief Report

Spontaneous improvement of a haemodynamically significant ventricular septal defect produced by blunt chest trauma in a child

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Abstract We describe the progress of a girl aged 5 years, who suffered blunt trauma to the chest producing a ventricular septal defect of 1 centimetre in diameter. The shunt generated a mild dilation of the left-sided chambers, and exertional dyspnoea. Three months later, therapeutic catheterisation revealed important diminution in the extent of shunting. We decided, therefore, not to close the defect, and she has since remained asymptomatic.

Keywords: Thoracic trauma; cardiac septal rupture; echocardiography

Cardiac injury is a common consequence of major trauma. Common sequels from blunt trauma to the heart include cardiac rupture, disruption of papillary muscles, valvar damage, and dissection of major vessels. In contrast, nonpenetrating traumatic rupture of the ventricular septum is exceptionally rare, with our search revealing less than 60 cases reported in the English literature.[1,2]

Case report

A healthy 5 year-old-girl was a passenger in a violent car crash. On arrival of the emergency services, the girl was found to be alert and responsive, and was taken to another regional centre. On arrival at the intensive care unit, she was conscious, with features recorded at 14 on the Glasgow scale, but presented a systolic cardiac murmur, graded at III from VI, multiple bone fractures, pulmonary contusion, and splenic rupture. Echocardiography showed a traumatic defect in the muscular ventricular septum measuring 1 centimetre in diameter. Within 24 hours of admission, she had respiratory distress, and showed haemodynamic instability, requiring mechanical ventilation and inotropic drugs for stabilization. At that time, echocardiography failed to reveal any significant change in the size of the ventricular septal defect, nor ventricular function. She progressively improved, and was discharged after 26 days of medical treatment in hospital. After 3 further months, she was admitted in a local centre due to exertional dyspnea. Medical therapy was optimized, and she was referred to our centre for consideration of closure of the ventricular septal defect. On physical examination, the systolic murmur remained graded at 3 out of 6, and she still exhibited exertional dyspnea. Echocardiography showed a muscular ventricular septal defect, partly covered by right ventricular muscle bundles, measuring 1 centimetre in diameter, with mild dilation of the left-sided cavities, and a ratio of pulmonary to systemic flows of 2 to 1 (Figs 1 and 2, Videos 1 and 2 – www.journals.cambridge.org/CTY). Investigation using cardiac magnetic resonance imaging revealed the same findings and ratios. We accepted the patient for transcatheter closure of the defect. When the patient was admitted for closure, 3 months later, cardiac catheterisation

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Accepted for publication 2 September 2008

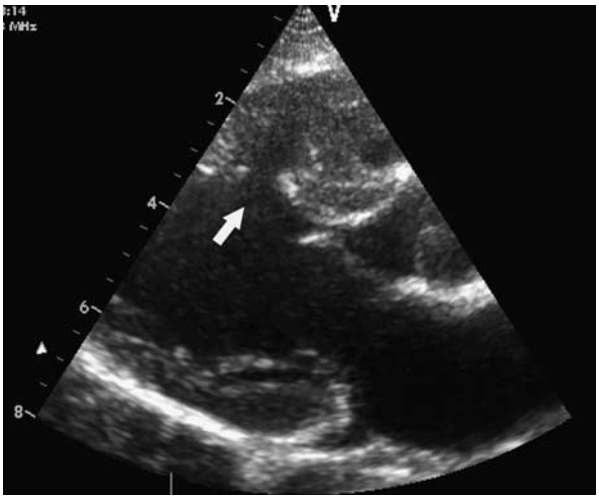

Figure 1.
Cross-sectional echocardiography in the parasternal long axis. The muscular ventricular septal defect is partly covered by right ventricular muscle bundles (white arrow).

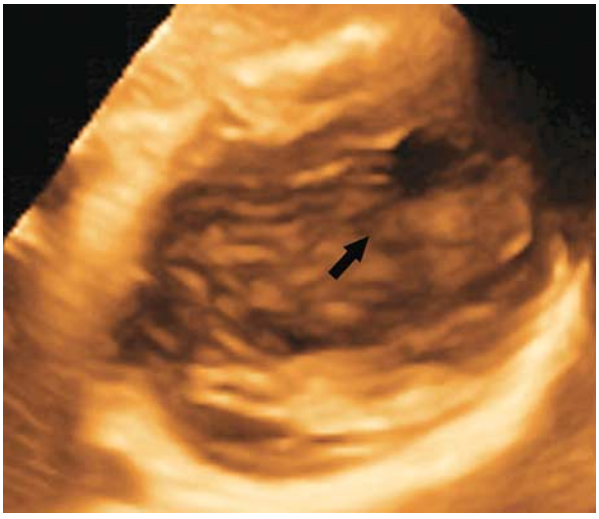

Figure 2.
Three-dimensional echocardiography, illustrating the left ventricular aspect of the septum, and showing the orifice of the traumatic muscular ventricular septal defect (black arrow).

revealed an important diminution in the flow of blood crossing the ventricular septal defect, with the ratio of pulmonary to systemic flows now measured at 1.3 to 1. We decided not to close the defect, and terminated the medical therapy. She has since remained asymptomatic.

Discussion

Rupture of the ventricular septum following blunt chest trauma is believed to be due to compression of the ventricular septum in end-diastole, immediately following atrial contraction, or during isovolumetric systole, when the cardiac valves are closed and the ventricles are filled with blood. The commonest site of rupture is in the muscular portion, near the apex. Some have postulated that the septal injury causes initial oedema, followed by liquefaction necrosis and appearance of a true defect. This sequence of events could explain the delay in appearance of the septal defect.[1,3] In our patient, the septal defect was already present at the first exploration, probably being produced by direct compression.

According to the literature, the decision to close a ventricular septal defect surgically is based on the amount of left-to-right shunting, and the presence of cardiac failure. Transcatheter closure of a postraumatic ventricular septal defect can be an alternative to surgery in symptomatic patients. A conservative approach has been recommended for asymptomatic patients with small traumatic ventricular septal defects when the ratio of pulmonary to systemic flows is clearly less than 2 to 1, the defects being considered to be well tolerated, and likely to close over time.[4] Other authors have recommended delaying surgical closure for 2 or 3 weeks, awaiting the healing of the edges, and thus facilitating the surgical procedure.[5]

Our patient, when first seen in our centre, presented with exertional dyspnea, mild dilation of the left sided chambers, and with a ratio of pulmonary to systemic flows of 2 to 1 as assessed by echocardiography and magnetic resonance. At that time, closure of the ventricular septal defect was indicated due to haemodynamic repercussions, but during the next three months, the defect reduced in size, losing its haemodynamic significance.

In conclusion, we recommend, if the clinical situation allows it, to delay the closure of such traumatic defects, in order to give time for spontaneous reduction in size, as well as healing of its edges.

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