CASE REPORT

Acquired ventricular septal defect: A rare sequel of blunt chest trauma in a 7-year-old boy

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Abstract

Ventricular septal defect (VSD) is the most common congenital cardiac lesion encountered worldwide. Only very rarely is it acquired, and causation through blunt injury in a child is extremely rare. A previously healthy 7-year-old boy suffered blunt chest trauma while at play. He presented 11 days later with features of acute congestive cardiac failure. Two-dimensional echocardiographic examination revealed a mid-muscular VSD. The connection between the defect and the trauma was not initially appreciated. Facilities for required urgent open-heart surgery were not available. Cardiac failure was refractory to antifailure therapy. His clinical condition steadily worsened, and he succumbed after 20 days on admission. We conclude that a diagnosis of traumatic VSD, though rare, should be considered in any previously well child presenting in acute congestive cardiac failure following blunt trauma to the chest. Any such patient should undergo careful echocardiographic evaluation. There is an urgent need for facilities for open-heart surgery to be more readily available and accessible in Nigeria.

Key words: Acquired ventricular septal defect, traumatic ventricular septal defect, blunt chest injury, cardiac failure, echocardiography

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Introduction

Ventricular septal defect (VSD) is the most common congenital cardiac lesion encountered worldwide. Conversely, the isolated acquired defect is rare. Isolated VSD is a rare result of blunt chest trauma[1-3] and is particularly rare in childhood.[4,5] Autopsy data suggests an incidence of approximately 1% for isolated VSD and approximately 5% for VSD associated with other cardiac injuries.[5] The authors are unaware of any previous report in a Nigerian child. Traumatic VSD is variable in its presentation, severity and clinical course. We present the case of rupture of the ventricular septum in 7-year-old boy following blunt chest trauma.

Case Report

A previously healthy 7-year-old boy sustained blunt chest trauma when a wheelbarrow containing cement blocks fell on his chest while he was playing with his peers at school. He had no external injuries or fractures but subsequently complained of chest pains. At a private hospital, a chest X-ray (CXR) revealed no abnormalities; he was given analgesics and discharged. A week later, he developed cough, palpitations and progressive breathlessness.

On presentation to our facility 11 days after the trauma, the child was acutely ill, in respiratory distress (respiratory rate 60/min) although the chest was clinically clear. The jugular venous pressure was not raised. His heart rate was 128/min and blood pressure, 80/50 mm Hg. S_1 was normal, but P_2 was loud. Although initially no murmur was heard, within 24 hours of admission a Grade 2/6 pan systolic murmur was detected at the left lower sternal border and subsequently increased in

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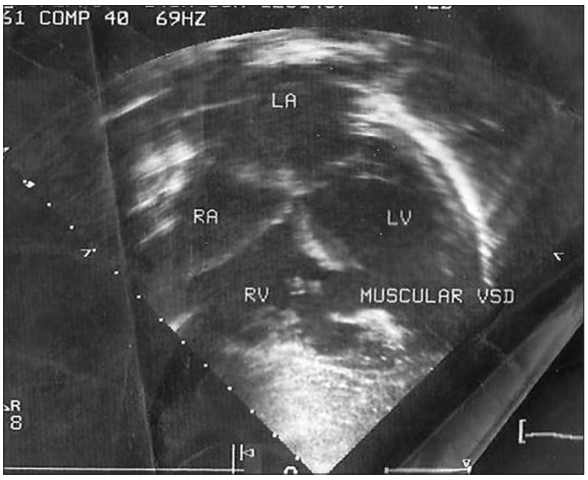

Figure 1: Two-dimensional echocardiography picture of the ventricular septal defect also showing the flap

intensity to Grade 3/6. A tender liver was palpable 5 cm below the right costal margin.

The CXR showed bilateral patchy opacities but no fractures. A 12-lead electrocardiogram (ECG) revealed sinus tachycardia, infant RS progression pattern, suggestive of right ventricular hypertrophy (RVH) for age, with right bundle branch block (RBBB) and tall T waves.

Two-dimensional echocardiography subsequently revealed a large VSD in the muscular portion measuring 2.57 cm [Figure 1]. At the time, the connection between the defect and the trauma was not recognized - the defect was assumed to be congenital and the history of trauma a red herring.

The child was commenced on hydrochlorothiazide, spironolactone, frusemide and enalapril. Despite these measures, his condition steadily deteriorated. Repeat echocardiography revealed the VSD being partially walled off by a flap-like structure. Facilities for the required urgent open-heart surgical intervention were not available.

The child developed features of acute renal failure on the 18th day of admission and died 2 days later during a session of hemodialysis. The parents refused postmortem examination.

Discussion

Two dominant theories concerning the pathogenesis of VSD following blunt trauma have been described. The first postulates that the rupture occurs due to acute compression of the heart between the sternum and the thoracic column when the ventricles are filled, and the valves closed with a resultant rise in intra-cardiac pressure.[2,6-8] The second proposes that the myocardial injury causes microvascular disruption leading to infarction and liquefaction of the septum.[2,6,8]

The second option seems to be the more plausible one in our patient in view of the time lapse between his injury and his presentation and his subsequent course.

The clinical presentation of traumatic VSD may be acute, subacute or late, depending on the extent of the injury and subsequent local necrosis. In general, those with larger defects, as in our case, tend to be more symptomatic with hemodynamic compromise. The appearance of the murmur is also commonly delayed, as happened in our patient, and should have alerted us to the diagnosis. The clinician should, therefore, be alert to the presence of any new, changing or unexplained murmur following chest trauma. In retrospect also, the absence of clinical symptoms suggestive of a large VSD prior to the trauma in our patient, should have suggested that the lesion was unlikely to be congenital. Furthermore, the appearance on echocardiography of the tissue flap surrounding the defect also depicted the gross appearance of a ragged wound, also suggesting an acquired rather than congenital lesion. Similar features have also been observed in other reports.[2,8] The ECG findings of infant RS progression pattern, RBBB and tall T waves were initially interpreted to be evidence of RVH, but in retrospect, could have been consequent on myocardial injury, especially as there was no evidence of RVH on echocardiography. Elevation of cardiac troponin I has been shown to be both a sensitive and specific indicator of myocardial cell necrosis following blunt chest trauma[9] and its measurement would also have been helpful in the diagnosis.

Two-dimensional echocardiography is the investigation of choice for diagnosis[10] and for guiding further management. The presence of the facility in our center aided the diagnosis of this rare condition. Unfortunately, there was no opportunity for echocardiographic evaluation prior to the patient's presentation - which would have been the incontrovertible proof that the VSD indeed was subsequent to the trauma sustained, and not congenital.

This case is similar to others previously reported in children.[2,5,11,12] The key to a favorable outcome is early suspicion of such a defect in the presence of heart failure following a period of relative cardiovascular stability.[4,10] Unfortunately, in our case, because of its rarity, the diagnosis of traumatic VSD was not initially suspected. Even when the diagnosis was eventually made, facilities and funds for urgent life-saving open-heart surgery were not readily available, as is the case in many resource-poor countries of the world.

Large lesions associated with hemodynamic instability require urgent surgical closure, but, unfortunately, facilities and funds for open heart surgery were not available for our patient.

Renal failure is a known complication of severe, intractable heart failure, which was the experience in our patient.

Many such patients, without the required intervention, would chart a downhill course, culminating in multi-organ failure and eventual demise, which was the likely case in this patient.

There are reports of some cases being successfully managed by delayed surgical closure[2,6] or even by percutaneous device closure.[12,13] If promptly diagnosed and treated, such patients should enjoy excellent outcomes.

Conclusion

A diagnosis of traumatic VSD, though rare, should be considered in any previously well child who sustains blunt trauma to the chest and presents in acute congestive cardiac failure. Facilities for open-heart surgery need to be more readily available, accessible and affordable in Nigeria. Regionalization where centers of excellence with such facilities exist is an option.

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