

Case report of ventricular septal defect secondary to blunt chest trauma

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Isolated traumatic ventricular septal defect (VSD) is a rare result of blunt cardiac trauma.^{1,2} There are about 58 cases reported in the English literature since 1970.² Traumatic VSD is variable in its presentation, temporal course and severity. It may require emergent repair² or be repaired electively.¹ The case report we present demonstrates key features of a smaller traumatic VSD and a relatively novel interpretation of the injury mechanism that may be applicable in select cases.

Case report

An 18-year-old man sustained blunt chest trauma in an unrestrained motor vehicle collision. The patient was extricated by emergency medical services and had a Glasgow Coma Score of 15. On arrival at the regional hospital, his vital signs were as follows: blood pressure (BP) = 104/47 mm Hg, heart rate = 92 beats/minute, respiratory rate = 24 breaths/minute and room air oxygen saturation = 95%. The blood work revealed an elevated troponin I level of 11.11 µg/L. No cardiac murmur was noted. He was transferred to a tertiary trauma centre with a presumed diagnosis of cardiac contusion.

On arrival at our centre approximately 7.5 hours postinjury, his BP was 106/69 mm Hg, HR was 115 and GCS was 15. Complete trauma assessment revealed the following injuries: left pulmonary contusion, small left pneumothorax, left third and fourth rib fracture, right

ulnar fracture and left talar fractures. Blood work found an elevated creatinine kinase-myocardial fraction at 142 µg/L and a troponin I level of 22.31 µg/L.

On examination, the patient was noted to have a 3/5 holosystolic murmur at the lower left sternal border, which did not radiate to the neck or axilla, with an associated thrill. The patient's mother confirmed that he had a childhood murmur that he had outgrown.

Electrocardiogram (ECG) revealed anterior and inferior ST elevation and non-specific atriovenous conduction delay. An echocardiogram revealed hyperdynamic left ventricular (LV) function with apical hypokinesis and turbulence in the right ventricle, suggestive of a gradient. However, a VSD was not specifically noted. A second echocardiogram the next day revealed a dyskinetic LV apex and akinetic RV apex with a defect in the anterior septum and a high-velocity left-to-right shunt, suggestive of a muscular VSD.

His orthopedic injuries were treated, and he was monitored in the intensive care unit for 5 days uneventfully and discharged with a plan to repair the VSD in 6 to 8 weeks. During the period between his collision and the VSD repair, he did not complain of chest pain but did experience exertional dyspnea.

Fifty-eight days later, open repair of the VSD was performed via sternotomy, cardiac bypass and cardioplegia. The LV apex was opened, and the 0.5- to 1-cm defect in the ventricular septum was repaired with a pericardial patch. During

the procedure, trabeculations and endocardial fibrosis in the area of the lesion was noted. Intraoperative transesophageal echo showed no residual VSD. The patient recovered without incident, and he has been asymptomatic since his injury.

Discussion

For the most part, this case conforms with previous reports of small (< 2 cm) traumatic VSD lesions. The mechanism of injury of unrestrained MVC is typical.³ The holosystolic precordial murmur is also almost universally reported.² ECG findings suggestive of inferior myocardial infarction and elevated cardiac enzymes are often reported and are thought to arise from the concurrent myocardial contusion.¹ Additionally, the appearance of the murmur is commonly delayed,⁴ as in the case of our patient. However, the approximately 1.5-day delay in echocardiographic finding of VSD in this case has not been previously reported. Finally, the clinical course (in and out of hospital) was also fairly typical for VSD injuries of this size.⁴

Two dominant theories concerning the pathogenesis of traumatic VSA are described. The first postulates that the rupture occurs due to acute compression of the heart during late diastole when the ventricles are filled and the valves are closed.⁵ The second proposes that myocardial injury causes a microvascular disruption, leading to infarction and liquefaction of the septum.⁶

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Notes de cas

This raises the possibility of a previously unexplored contributory factor in the pathogenesis of traumatic VSD. In our case, the appearance of the tissue surrounding the defect displayed the gross appearance of a healed wound, and the initial echocardiogram did not demonstrate the VSD. This information, combined with a history of resolved childhood murmur, suggests that the traumatic VSD resulted from the result of a reopening healed congenital lesion in a weakened ventricular septum.

Clinical findings in traumatic VSD cases are relatively nonspecific, can be masked by other injuries and can often be delayed in presentation. The clinician should maintain a high level of suspicion for any new or un-

explained murmur or ECG findings.² This can be particularly challenging in patients with multiple injuries, with other injuries that require immediate attention. The transthoracic or transesophageal echo is the diagnostic test of choice. If promptly diagnosed and treated surgically, these patients enjoy excellent outcomes.

Competing interests: None declared.

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Aggressive angiomyxoma of the pelvis: a case report

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First described by Steeper and Rosai in 1983,¹ aggressive angiomyxoma (AAM) is a mesenchymal tumour found mainly in the pelvis and perineum. There is a strong female predominance, with a female-to-male ratio of approximately 6:1. It is a slow-growing tumour with a high rate of local recurrence. Because of its rarity, it is often initially misdiagnosed, frequently as a gynecological malignancy.

Case Report

A 47-year-old woman presented to a peripheral hospital with a mass in the left labia. Her medical history included a hysterectomy and left oophorectomy. The diagnosis of a Bartholin's cyst was made, and the patient was taken to the operating room (OR) for marsupialization. Further examination revealed that the mass was

not a Bartholin cyst. The operation was terminated and a CT scan was obtained. The CT showed a large pelvic mass (approximately 12 × 9 × 9 cm). A percutaneous biopsy was done, and histology showed a spindle cell tumour. MRI showed a mass 15 × 9 × 9 cm that enhanced well with gadolinium (see Fig. 1). MRI indicated that the mass did not involve the rectal wall or bladder. The mass did not appear to involve pelvic musculature but involvement of the proximal vaginal wall could not be ruled out. The patient was taken to the OR for resection. The mass was completely excised through a perineal incision that incorporated the previous biopsy site and Bartholin's surgery site. A posterior vaginectomy was done to get clear margins. There were no intraoperative complications, and blood loss was estimated at 200 mL. Final

pathological analysis revealed AAM. In 30 months of follow-up, there has been no clinical or radiographic (MRI) recurrence.

Pathology

Microscopically, AAM shows stellate and spindle cells within a loose matrix with some collagen formation and vessels of varying size (Fig. 2). The stroma tends to be quite edematous, but in some fields, differentiation into smooth muscle cells is suggested. Mitotic activity is absent or infrequent. Fetsch and colleagues² reported that all 22 cases stained for desmin were positive; smooth muscle actin was found in 19 of 22 cases, and muscle-specific actin was present in 16 of 19 cases; vimentin was present in all 17 cases studied and CD34 (QBEND-10) in 8 of 16 cases. In contrast, S100 protein was ab-

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