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Case Report

Ventricular Septal Defect Following Blunt Chest Trauma in Child: Case Report

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Abstract

Blunt thoracic trauma can lead to cardiac injuries such as myocardial contusion and valvular damage, but the occurrence of traumatic ventricular septal rupture is an exceptionally rare complication, especially in infants. We present an unusual case of an infant who sustained a ventricular septal rupture following blunt thoracic trauma, necessitating surgical repair. The proposed mechanism involves acute compression of the infant's heart between the sternum and vertebral column during ventricular filling, leading to a rapid intraventricular pressure spike that may rupture the interventricular septum. Our findings underscore the importance of prompt surgical intervention for large septal defects and hemodynamic instability arising from such traumatic cardiac injuries, even in infants. Early recognition and timely operative management are crucial for improved outcomes in these rare but life-threatening pediatric cases. We discuss the clinical presentation, diagnostic approach, and surgical treatment of this exceptional condition in an infant, contributing to the limited body of knowledge on this topic.

Keywords: ***Ventricular septal defect, ventricular septal rupture, blunt thoracic injury, pediatric, case report.***

Introduction

Traumatic ventricular septal defect (VSD), also called traumatic ventricular septal rupture (VSR), is a rare complication of blunt chest trauma in adults and children [1].

Thoracic trauma may lead to myocardial injury, including myocardial contusion, ventricular septal defect (VSD), ventricular free wall rupture, or valve compromise [2].

Traumatic VSD has variable clinical presentation, course, and severity, leading to difficulty in diagnosis. Bedside echocardiography is the most rapid and feasible modality for the diagnosis and follow-up of acute and severe VSR cases [3]. Here we report the case of a 6-year-old child who presented with ventricular septal rupture secondary to a 3-m fall at the thoracic point of impact.

Case presentation

This is a case of a 6-year-old child with no prior medical history, who presented with severe trauma following a fall from a three meter height. The child was initially hospitalized in another establishment before being transferred to our structure four days after trauma. At admission, the clinical examination revealed a conscious child with a Glasgow Coma Scale (GCS) score of 15, respiratory distress with a respiratory rate of 50 breaths per minute, 80% desaturation on room air, and signs of respiratory struggle. Upon admission to the intensive care unit, the patient's neurological and hemodynamic condition deteriorated, necessitating intubation, mechanical ventilation, and sedation. Initial assessment showed a biological inflammatory syndrome, hepatic cytolysis, increased urea, and low prothrombin time. Bacteriological assessment isolated an unusual pathogen, Klebsiella pneumoniae, from the blood culture obtained upon admission. Imaging revealed multiple foci of ground-glass opacities, diffuse septal and non-septal line thickening indicative of alveolar hemorrhage, and atelectasis bands. Despite vasopressor support, the patient exhibited signs of shock and ventricular tachycardia treated with intravenous amiodarone. Transthoracic echocardiography identified a rupture of the septal wall extending from the middle to the apical region, forming a neocavity measuring 33.8 x 17 mm [**Figure 1**], and two left-to-right shunts with VSDs measuring 6.2 mm [**Figure 2**] and 12.1 mm, respectively. The patient underwent surgical repair of the interventricular septum defect [**Figure 3;4**].

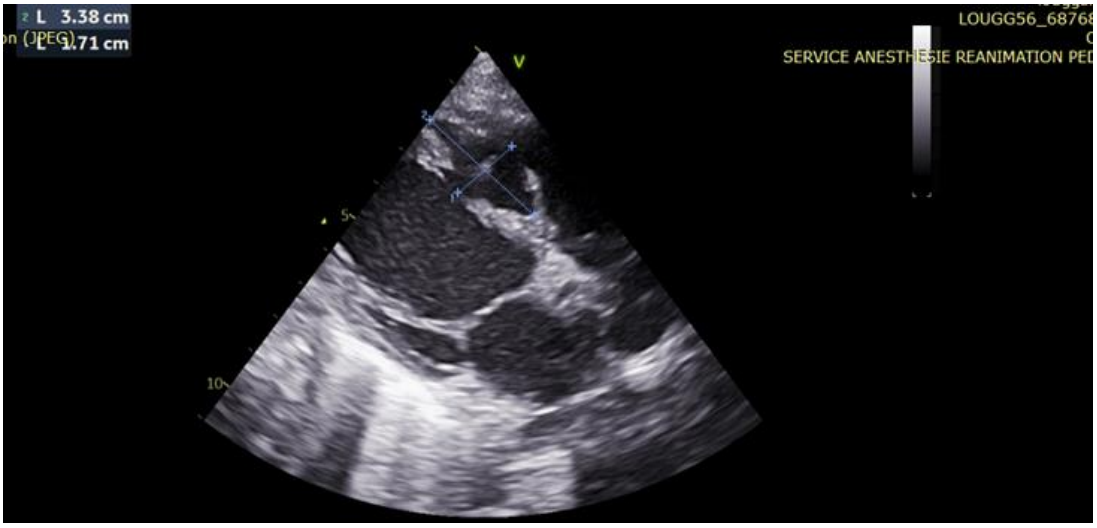

Figure 1: Parasternal long-axis view showing rupture of the interventricular septal wall from the extended middle to the apical part, resulting in a neocavity measuring 33.8*17.1mm.

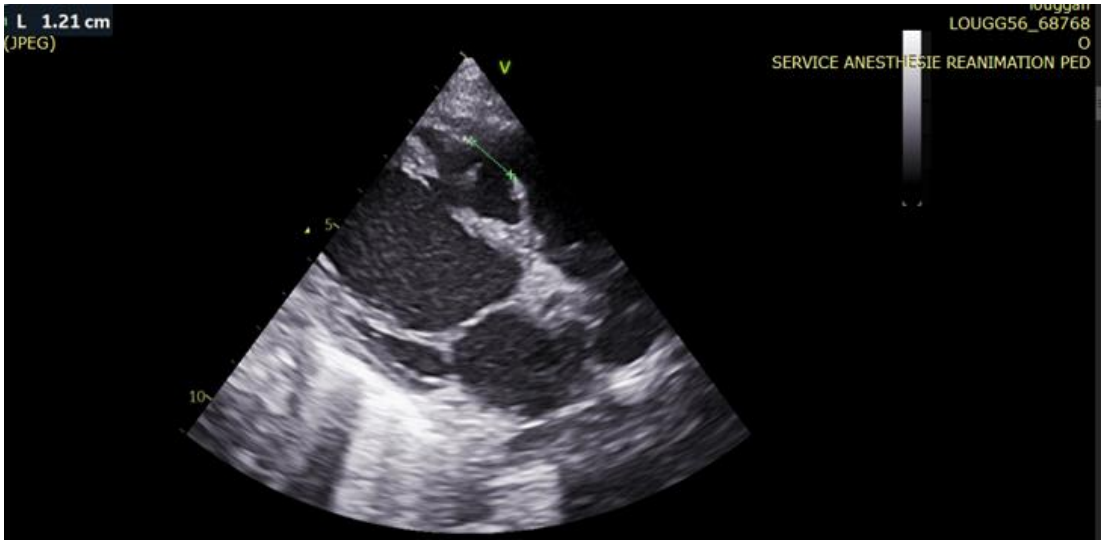

Figure 2: Parasternal long-axis view demonstrating a right ventricular septum defect measuring 12.1 mm

Preoperative preparation included standard monitoring and balanced induction with fentanyl (50 mcg), Preoperative preparation included standard monitoring and balanced induction with fentanyl (50 mcg), midazolam (3 mg), rocuronium (20 mg), dexamethasone (2 mg), and tranexamic acid (200 mg), followed by intubation and ventilation (Cormack grade 1, orotracheal tube size 5.5, fixed at 15 cm with balloon pressure at 25 cmH2O). Controlled ventilatory mode settings were volume-controlled (VT 200 ml, RR 23/min, PEEP 5 cmH2O, FiO2 35%). Intraoperative maintenance anesthesia involved sevoflurane, fentanyl, and rocuronium. Intravenous access was established via the right hand, with central venous and arterial lines in the right jugular vein and right femoral artery, respectively. Antibiotics were administered, and tranexamic acid maintenance was given at a dose of 5 mg/kg/h. Hemodynamic stabilization was achieved with noradrenaline (0.1 mcg/kg/min) and dobutamine (5 mcg/kg/min). The surgical procedure included cardiopulmonary bypass for 70 minutes, aortic clamping for 55 minutes, and assistance for 12 minutes, with transfusion of 1 unit of packed red blood cells and 2 units of fresh frozen plasma. Anticoagulation was managed with heparin (800 IU), followed by protamine (1300 IU) at the end of the procedure. Postoperatively, a VVI-type pacemaker was placed, and the patient was transferred to the intensive care unit, intubated, ventilated, and sedated with vasopressors for 24 hours. Extubation followed drug withdrawal, with non-invasive ventilation (helmet, AI: 12, PEEP: 5, FiO2: 40%). The child was initially stabilized hemodynamically with noradrenaline (0.2 mcg/kg/min) and dobutamine (7.5 mcg/kg/min). However, the child died of septic shock.

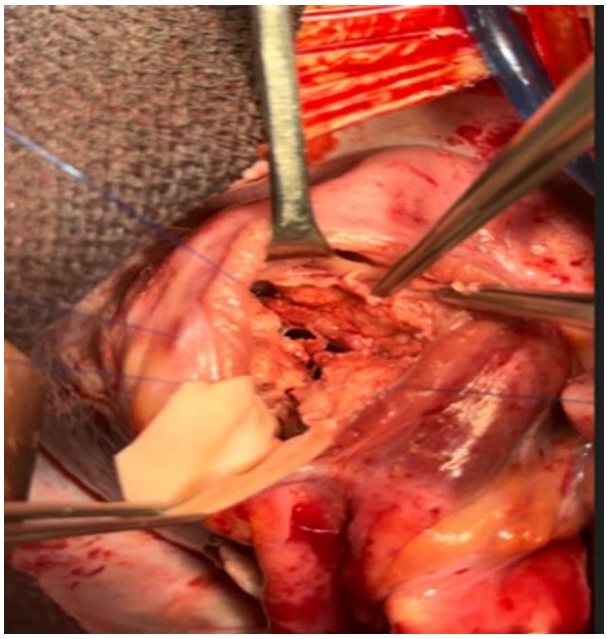

Figure 3: Per-operative image showing ventricular septal rupture

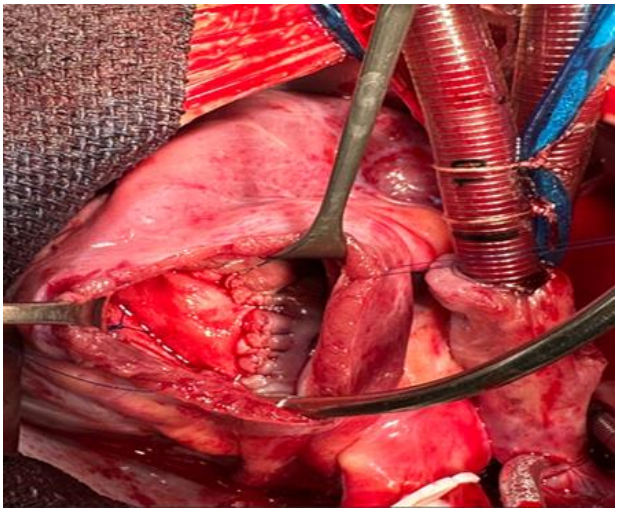

Figure 4: Per-operative image showing the ventricular septal defect sutured with patch placement

Discussion

Several investigations have documented the incidence of ventricular septal defect (VSD) in the pediatric population following blunt thoracic trauma, highlighting that although relatively uncommon, VSD can manifest as a severe complication [1,7]. Motor vehicle collisions represent the predominant etiology although alternative mechanisms such as falls from substantial heights or direct thoracic trauma have also been implicated [5]. Blunt force applied to the chest wall may precipitate myocardial injury, potentially culminating in myocardial contusion, VSD, ventricular free wall rupture, or valvular dysfunction [2]. The purported mechanism underlying the development of traumatic VSD secondary to blunt thoracic trauma typically involves anteroposterior compression [6], whereby elevated intraventricular pressure ensues following atrioventricular valve closure, with the sudden pressure spike induced by the impact rendering the interventricular septum vulnerable to rupture [2].

Diagnosing traumatic VSD can prove challenging or delayed because of the variable symptomatic presentation and timing, which may manifest acutely following the inciting event or even hours to months later [4]. In this infant case, septal rupture occurred on the fourth day after the injury. Clinical manifestations span a broad spectrum, ranging from mild symptomatology to overt cardiogenic shock [8], underscoring the critical importance of promptly and accurately evaluating the severity of thoracic trauma. Furthermore, traumatic VSD may be obscured by concomitant injuries such as intra-abdominal trauma, rib fractures, or extremity fractures. Akin to congenital VSD, pansystolic murmurs are typically auscultated along the left sternal border, whereas chest radiography may appear normal or demonstrate mild cardiomegaly or pulmonary edema [9]. Cardiac enzyme biomarkers generally lack sufficient sensitivity and specificity for diagnosing cardiac lesions [10], although troponins I and T have utility in risk stratification based on potential complications [11].

Transthoracic echocardiography remains pivotal for diagnosing septal rupture and associated lesions, providing a rapid and accurate assessment of the defect dimensions, magnitude of left-to-right shunting, and overall cardiac function, thus guiding therapeutic decision-making [14]. Other reported cardiac sequelae include mitral and aortic valvular lesions, ventricular aneurysms, and pericardial pathology [12]. The management of traumatic septal rupture necessitates an individualized approach that considers the patient's hemodynamic status, concomitant complications, and echocardiographic findings. Patients with VSDs exceeding 1 cm typically warrant surgical correction to mitigate the risk of progression to congestive heart failure secondary to elevated pulmonary blood pressure [9]. Early surgical intervention may be indicated for severe congestive heart failure or a substantial intracardiac shunt, whereas those with mild symptomatology or a modest left-to-right shunt may initially be managed medically, with surgical consideration deferred for 2-3 weeks [15]. Smaller defects coupled with hemodynamic stability may be amenable to conservative management with vigilant monitoring, as spontaneous closure has been documented [15,16]. More recently, percutaneous closure of traumatic VSD has been reported as an effective alternative to open surgical repair [13].

Conclusion

In this report, we present a rare case of traumatic ventricular septal defect (VSD) resulting from blunt thoracic trauma with delayed clinical manifestation. Among patients sustaining traumatic VSD, the symptomatology and clinical signs can be highly variable and potentially masked by concomitant injuries. Therefore, clinicians must maintain a high index of suspicion for cardiac involvement in patients with significant thoracic trauma. It is imperative to recognize that the absence of apparent myocardial lesions on initial evaluation does not preclude their subsequent development; thus, serial cardiac assessments via echocardiography, computed tomography, and/or magnetic resonance imaging are strongly recommended.

Abbreviations

VSD: Ventricular septal defect,
VSR: Ventricular septal rupture,
GCS: Glasgow coma scale

Conflict of interest statement

The authors declare no conflicts of interest.

Sources of funding

No funding was received for this study.

Ethical approval

As this is a case report, ethical approval was not required.

Informed Consent

Written informed consent was obtained from the patient's legal guardian.

Registration of research studies

Not applicable, as this is a case report and not an interventional or observational study.

Provenance and peer review

This manuscript was subjected to an external peer review before publication.

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