









Case Report

Rare Complications of Blunt Thoracic Trauma: Pulmonary Edema and Late Ventricular Septal Defect

Künt Toraks Travmasının Nadir Komplikasyonu: Geç Dönem Ventriküler Septal Defekt ve Pulmoner Ödem

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ABSTRACT

It has been reported that ventricular septal aneurysm and ventricular septal defect (VSD) may rarely develop after blunt thoracic trauma. This case report presents a pediatric case that developed VSD in the follow-up after an in-vehicle traffic accident. A 15-month-old patient had an in-vehicle traffic accident; since no additional problem was detected, he was discharged after a short follow-up. The patient was admitted to the hospital with respiratory distress five days after the trauma, with the symptoms of tachypnea and a 2/6 systolic murmur in the pulmonary focus. In echocardiography, the interventricular septum was intact, sASD and pulmonary hypertension secondary to lung contusion were present. He was transferred to the pediatric intensive care unit and intubated on the sixth day; adrenaline and milrinone were started due to hypotension. On the seventh day, echocardiography revealed a 7 mm VSD with a left-to-right shunt in the apical muscular region, and interventricular septal hypertrophy was detected. In angiography, the right ventricle was filled with two muscular defects of 4.2 mm in the apical region on the interventricular septum and the area near the middle. Direct and indirect mechanisms are well defined in the literature, and our patient's VSD occurred due to a delayed inflammatory response due to liquefaction necrosis following myocardial damage. Similar to the literature, apical muscular VSD was detected in your patient. The patient is followed closely without surgical intervention in the 11th month of follow-up. It should be kept in mind that secondary VSD may develop within days after blunt chest trauma.

Keywords: Blunt injury, thoracic injuries, ventricular septal defect.

ÖZET

Künt toraks travması sonrası nadiren ventriküler septal anevrizma ve ventriküler septal defekt (VSD) gelişebileceği bildirilmiştir. Bu olgu sunumunda, araç içi trafik kazası sonrası takipte VSD gelişen bir pediatrik hasta sunulmuştur. On beş aylık bir çocuk, araç içi trafik kazası geçirmiş ve ek bir sorun saptanmadığı için kısa bir gözlem sonrası taburcu edilmiştir. Travmadan beş gün sonra takipne ve pulmoner odakta 2/6 sistolik üfürüm ile solunum sıkıntısı şikayetleriyle tekrar hastaneye başvurmuştur. Ekokardiyografide interventriküler septum intakt olup, sekonder atriyal septal defekt (sASD) ve akciğer kontüzyonuna bağlı pulmoner hipertansiyon saptanmıştır. Altıncı günde

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hipotansiyon nedeniyle çocuk yoğun bakım ünitesine alınmış, entübe edilmiş ve adrenalin ile milrinon tedavisine başlanmıştır. Yedinci günde yapılan ekokardiyografide apikal musküler bölgede sol-sağ şant gösteren 7 mm VSD ve interventriküler septal hipertrofi saptanmıştır. Anjiyografide ise interventriküler septumun apikal bölgesinde ve orta bölgeye yakın alanda 4.2 mm'lik iki musküler defekt ile sağ ventrikül dolumu izlenmiştir. Literatürde tanımlanmış doğrudan ve dolaylı mekanizmalar arasında yer alan, miyokard hasarına bağlı likefaksiyon nekrozu sonucu gelişen gecikmiş inflamatuvar yanıtın VSD oluşumuna yol açtığı düşünülmektedir. Hastamızda da literatürle uyumlu şekilde apikal musküler VSD geliştiği gözlenmiştir. On birinci ay takiplerinde cerrahi müdahale yapılmaksızın hasta yakın takiple izlenmektedir. Künt toraks travmalarından sonra günler içinde sekonder VSD gelişebileceği akılda tutulmalıdır.

Keywords: Künt yaralanma, torasik yaralanma, ventriküler septal defekt

INTRODUCTION

The most common cause of blunt chest trauma is motor vehicle accidents [1]. Potentially life-threatening complications, such as pulmonary and myocardial contusion, pulmonary edema, coronary injury, pneumothorax, hemothorax, rib fractures, and cardiac tamponade, should be investigated during the initial evaluation. It has also been reported that ventricular septal aneurysm and ventricular septal defect (VSD) may rarely develop after blunt thoracic trauma [2]. This case report presents a pediatric patient who initially developed pulmonary edema and pulmonary hypertension after an in-vehicle traffic accident and then developed VSD in the follow-up.

CASE REPORT

Admission and Initial Evaluation

A 15-month-old male patient had an in-vehicle traffic accident while traveling at 40 km/h in the car's back seat and hit his chest on the door. Since no further abnormalities were identified, he was discharged after a short follow-up. In his medical history, it was learned that the cardiologist followed him up with the diagnosis of a minor secundum atrial septal defect (sASD) and insignificant patent ductus arteriosus.

The patient was admitted to the hospital with respiratory distress five days after the trauma. On physical examination, he had no pathological findings except tachypnea and a 2/6 systolic murmur in the pulmonary focus. Due to the continuation of his tachypneic course and elevated cardiac enzymes (Troponin T: 818.8 ng/L, NT-proBNP: 29744 pg, D-dimer: 3.54 mg/L), he was hospitalized. In echocardiography, the interventricular septum was intact, sASD and pulmonary hypertension secondary to lung contusion were present. ECG was normal. Thoracic computed tomography at admission revealed patchy peribronchovascular and subpleural ground-glass opacities, more prominent in the right lower lobe, with

a cardiothoracic ratio of approximately 50%, while mediastinal and hilar assessment was suboptimal. In the presence of fever and worsening tachypnea, secondary pneumonia, pulmonary contusion, pulmonary embolism, and COVID-19-associated multisystem inflammatory syndrome (MIS-C) were considered.

ICU Course

He was transferred to the pediatric intensive care unit on the third day due to respiratory distress and intubated on the sixth day. Adrenaline and milrinone were initiated due to hypotension. Pulmonary embolism was excluded by pulmonary CT angiography. A 4/6 pansystolic murmur in the mesocardiac focus is detected on the seventh day. On echocardiography, a 7 mm VSD with a left-to-right shunt was identified in the apical muscular region, and interventricular septal hypertrophy was detected (Figure 1). In angiography, the right ventricle was filled with two muscular defects of 4.2 mm in the apical region on the interventricular septum and the area near the middle (Figure 2); the coronary arteries were normal.

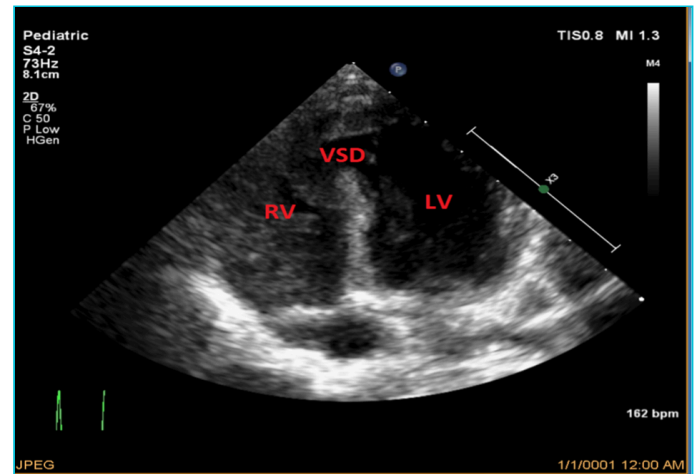


Figure 1. A newly developed ventricular septal defect (VSD) is shown in the echocardiography image taken in the apical 4-chamber plan.



Figure 2. 4.2 mm VSD image with the catheter tip in the main pulmonary artery in the sineangiogram examination

Diuretic and digoxin treatment for heart failure was started during follow-up evaluations, and adrenaline and milrinone support were discontinued. Since myocarditis could not be excluded, intravenous immunoglobulin was administered. Elective surgical repair was planned for the patient, who is still being followed up in the pediatric cardiology outpatient clinic.

DISCUSSION

Blunt cardiac trauma is found in 3% to 76% of trauma patients [3]. Traumatic VSDs can be detected in 2% to 10% of cases of blunt chest trauma resulting from motor vehicle accidents [4]. A direct blow to the chest is considered the most common mechanism, and cardiac injury is most likely to occur when the ventricles are maximally distended at the end of diastole. The indirect cause is due to an increase in preload through the intra-abdominal or extremity vessels, causing a sudden rise in intracardiac pressure, thereby making the heart susceptible to rupture. Bidirectional forces result in cardiac compression between the spine and the sternum. Deceleration mechanisms allow the heart to move freely, resulting in valve, myocardial, or coronary artery rupture [5]. If traumatic VSD occurs, although most of them occur immediately due to mechanical rupture, it may also develop due to a delayed inflammatory response, due to liquefaction necrosis following myocardial damage, and microvascular deterioration hours to weeks after

trauma [6]. As reported in the literature, our patient developed VSD due to necrosis after myocardial injury.

Although the clinic varies according to the size of the VSD, it commonly includes dyspnea, newly developed or altered murmur, cardiomegaly, congestive heart failure, and cardiogenic shock [7]. By location, apical muscular VSDs are the most common, and subpulmonic and infundibular defects are less common [4]. Consistent with previous studies, apical muscular VSD was detected in our patient.

There is no specific criterion for diagnosis, and the clinician should suspect cardiac injury after blunt thoracic trauma. As in our case, patients with tachypnea, new-onset or changed murmur, and signs of heart failure should be followed up with ECG and troponin I, and supported by echocardiography. Patients with abnormal ECG and troponin I levels should be hospitalized for 24-48 hours of hemodynamic monitoring, as life-threatening arrhythmias or heart failure are most likely to manifest during this period [8]. However, in our patient, hemodynamic findings developed after the fifth day. The presence of pulmonary hypertension in the initial echocardiographic examination and obstruction of the left-to-right shunt may have made the diagnosis of VSD difficult.

Traumatic VSDs diagnosed within 48 hours of blunt trauma are associated with increased mortality. Surgical repair is indicated if the defect is significant, the pulmonary/systemic blood flow ratio (Q_p/Q_s) exceeds 2:1, or there is evidence of heart failure or cardiogenic shock. [9]. In contrast, late-diagnosed cases of posttraumatic VSD (more than 48 hours after trauma) rarely require emergency surgery, often require elective repair, and can sometimes be treated conservatively because they close spontaneously [10]. Since asymptomatic VSDs have $Q_p/Q_s < 1.5:1$, the defect is generally well tolerated and can close spontaneously; patients can be carefully monitored [11]. The patient is followed closely without surgical intervention in the 11th month of follow-up.

CONCLUSION

Delayed ventricular septal defect may occur in infants after blunt chest trauma despite an initially negative echocardiogram. New or intensifying holosystolic murmurs and rising cardiac biomarkers in the days following trauma must be thoroughly

investigated, including repeat echocardiography and, when indicated, angiographic confirmation to guide timely management.

Patient Consent Form / Hasta Onam Formu

The parents' of this patient consent was obtained for this study.

Conflict of Interest / Çıkar Çatışması

The authors declared no conflicts of interest with respect to authorship and/or publication of the article.

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