

CASE REPORT

Ventricular Septal Defect and Mitral Regurgitation Due to Penetrating Cardiac Trauma; a Case Report and Review of Literature

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Abstract: Penetrating cardiac trauma is a fatal condition and can result in the injury of various parts of the heart. Ventricular Septal Defect (VSD) following these traumas occurs only in 1-5% of cases. The patients' conditions depend on location, size, and concomitant injuries. One of the uncommon coincidences with the VSD is Mitral Regurgitation (MR) due to injury to sub-valvular structures. In this study, we report a case of concomitant traumatic-induced VSD and MR in a 14-yearold boy following a stab wound to his chest. The patient was a teenage boy coming to the Rajaei Cardiology Hospital emergency room following a stab wound to the anterior and left part of his chest. Despite primary urgent surgery, his breathlessness had continued for three more months. Evaluations with Transthoracic Echocardiography (TTE) revealed VSD with concomitant MR, but there was no papillary muscle rupture. Cardiac Magnetic Resonance Imaging (MRI) and angiographic evaluation confirmed the provisional diagnosis. The Amplatzer VSD occluder repaired the VSD, and the patient was discharged following the resolution of his symptoms. Although the MR has been present in the follow-up echocardiography, the patient has been asymptomatic. Since the initial presenting symptoms and signs of VSD and MR might be subtle or delayed, imaging modalities such as TTE and Transesophageal Echocardiogram (TEE) are beneficial in determining the diagnosis and the optimal treatment.

Keywords: Ventricular Septal Defect; Heart Injury; Mitral Regurgitation; Transthoracic Echocardiography; Heart Surgical Procedure

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1. Introduction

Penetrating cardiac trauma in pediatrics is very rare (almost 1% of all pediatric penetrating traumas). However, it is a fatal condition with a high risk of mortality before and after arriving at the hospital (30-35% overall mortality) [1, 2]. Following these injuries, left and right ventricles are most commonly involved due to their proximity to the chest wall and larger size [3]. Various concomitant damages might be detected in other parts, including the free wall, interatrial and interventricular septum, valvular and sub-valvular structures, coronary arteries, and cardiac conduction system [4]. A traumatic ventricular septal defect (VSD) is an uncommon complication of blunt and penetrating cardiac trauma [5].

The incidence of this phenomenon in penetrating cardiac in-

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juries is 1-5% [6]. There are two general categories of traumatic VSD, including early and delayed. The early ones are mainly the result of the compression of the filled heart at the end of the diastole when the valves are closed [7]. In contrast, delayed VSDs are caused by the subsequent edema and disruption of blood flow to the septum following injury [8]. The coincidence of mitral valve injury and VSD is uncommonly reported; about one-fifth of patients survived a penetrating cardiac injury, as identified in a study [9]. The mechanism of injury is most commonly due to papillary muscle injury, corda tendinea, and leaflet tearing [10]. This will result in an MV malfunction and an MR. Early diagnosis and treatment should be provided due to the high risk of progression of this condition to congestive heart failure and hemodynamic deterioration [11, 12]. Echocardiography is the goldstandard diagnostic method for traumatic valvular defects [13]. In this report, a 14-year-old with a history of penetrating cardiac injury presented with the final diagnosis of VSD and

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2. Case Presentation

A 14-year-old previously healthy boy presented to an emergency department (ED) in East Iran with dyspnea and fatigue after having a penetrating cardiac trauma in his anterior left lateral chest. The trauma had happened 15 minutes before his arrival with a knife during a street fight. The patients had no past medical or surgical history except a penetrating chest trauma, which will be mentioned below. The patient had been brought in with drowsiness, hemodynamic instability, and pallor. He was drowsy and not fully responsive. The patient's consciousness was evaluated in the physical examination, and the Glasgow Coma Scale (GCS) score was 11 (Eye:3 + Verbal:4 + Motor:4). The pallor was evident in his general appearance. In the vital sign assessment, his blood pressure was 70/40 mmHg, his pulse rate (PR) was sinus and 130/minute, and his temperature was 36.5 Celsius. The chest examination showed a 5 cm laceration with a 4 cm distance from the left sternal border in the fourth intercostal space (between the fourth and fifth ribs). Moreover, a holo-systolic murmur could be detected in his left sternal border. ED specialists inserted a left-side chest tube without hesitation. Volume resuscitation with intravenous 2 liters of normal saline (0.90%) and O-negative blood transfusion was started through two large bores of intravenous accesses. Since the patient was unstable, no time was wasted for more evaluation, and the patient was transferred to the cardiothoracic operation room immediately, and a thoracotomy was performed without delay.

After the thoracotomy, the surgeon detected a 3 cm laceration on the anterior part of the left ventricle close to the interventricular septum, which had caused a laceration on the muscular anterior part of this wall (about 1 cm). The laceration on LV was sutured successfully with simple, continuous stitches with 5–0 polypropylene sutures. Following the surgery, the patient's condition became stable, and he was observed in the intensive care unit (ICU) under continuous cardiopulmonary monitoring. Routine lab tests were requested, which were unremarkable except for the patient's hemoglobin, which was 11.5 gr/dL (Table 1). The post-surgery recovery time was uneventful, and the patient was discharged one week after the surgery without any symptoms.

Three months after the surgery, the patient presented to the tertiary pediatric cardiology center of Rajaei Hospital in Tehran, complaining of worsening shortness of breath and fatigue following routine physical activities. The physical exam was performed, which revealed a normal general appearance, a stable vital sign, and an unremarkable finding except for an abnormal holo-systolic decrescendo murmur in the left sternal border auscultation. Routine lab tests were requested, which were unremarkable (Table 1). The patient was admitted to the pediatric cardiology ward with the provisional diagnosis of a post-traumatic cardiac structural defect. Inpatient evaluation began with a TTE that revealed the following findings: There was an anterior muscular VSD (Figure

1A) with a size of 7-8 mm. The distance of the VSD to the pulmonary valve (PV) was 13 mm (PPG = 76 mmHg). The mitral valve (MV) was myxomatous with bi-leaflet prolapse. There was a severe MR (Figure 1B) and mild tricuspid regurgitation TR (peak pressure gradient (PPG) = 30 mmHg). Moreover, there was moderate to severe Left Atrial Enlargement (LAE) and moderate Left Ventricular Enlargement (LVE), with left Ventricle Ejection Fraction (LVEF) = 65%. The sizes of the RA and RV were normal. The QP/QS ratio was 2:1. A CT angiography was requested to obtain more information about his heart condition, which confirmed the echocardiography findings (VSD on the anterior and muscular parts of the interventricular wall) (Figure 1C) but no more remarkable abnormal findings.

The attending cardiologists decided to perform an angiography. During this procedure, a VSD was detected in the anterior and muscular parts of the interventricular wall. With the injection of dye, a large anterior muscular VSD (Figure 1D), severe MR, mild pulmonary hypertension, and the enlargement of the left ventricle and atrium were found. Anterior muscular VSD was successfully occluded by the Amplatzer cera liftech muscular occluder (10 mm) (Figure 1E). Following the occlusion procedure, LAO-cranial views (when the camera is rotated to the patient's left side) showed no residual VSD. Then, the patient was kept under observation and continuous cardiopulmonary monitoring for 48 hours. The post-procedure period was uneventful, and the patient's symptoms improved remarkably. The mitral valve and subvalvular repair were planned to be performed later. Two days following the procedure, the patient was discharged without any symptoms and was recommended to continue regular outpatient follow-up until his following admission for the MR repair. In his follow-ups, the patient was asymptomatic and no longer complained of breathlessness. One year later, a cardiac MRI with and without contrast demonstrated the following results: Mitral annulus disjunction (6mm) with bi-leaflet mitral prolapse and severe MR (RF: 42%). Prolapse of the tricuspid valve with mild TR (RF: 8%) was found. Other abnormal findings were an enlarged right atrium (19 cm2/m2), a moderately enlarged left ventricle size and normal ejection fraction, and a high normal right ventricle size and normal ejection fraction. No other remarkable abnormal finding was reported in the patient's laboratory, echocardiographic, and imaging procedures in the patient's follow-ups. The patient was referred to a cardiothoracic surgeon and an interventional cardiologist for further evaluation and treatment. However, the patient's family has not been able to come to the subsequent planned visits due to familial difficulties, and the attempts to help them come for their next steps of treatment are being continued.

3. Discussion

Chest injuries can result in cardiac trauma, which is generally categorized as penetrating and blunt trauma. Penetrating traumas uncommonly happen, primarily due to stab wounds

and shotguns. It can be a serious, fatal condition due to the involvement of cardiac structures [14]. Most pediatric cardiac traumas are blunt, and only 10-20% of them are reported to be penetrating, which is primarily due to Gunshot Wounds (GSW) (50-60%) and stab wounds (33%) in the United States [15]. The mortality rate and the outcome of this type of injury largely depend on the location of the heart involved; the highest has been seen in right ventricle injuries (probably due to low pressure and a thick wall) and the lowest in left atrium lacerations. The average mortality of these injuries is higher among children and adolescents due to the proximity of vital organs to their chest walls [16]. Several other factors can be predictors of mortality, such as the mechanism of injury, prehospital care, size and location of the defect, absence or presence of vital signs when arriving at the hospital, arrhythmia in the primary ECG, presence of tamponade, and the early or delayed diagnosis [14, 15]. Since the early diagnosis of concomitant heart injuries and appropriate ontime therapeutic interventions can decrease the risk of death and long-term sequelae such as infective endocarditis, cardiac chambers enlargement, unrepairable valvular dysfunction, and heart failure, these patients should be evaluated immediately and thoroughly [11, 12, 17]. The treatment could be surgical, transcatheter, or even medical, depending on the patient and the available logistics of the center. Emergency department thoracotomy (EDT) is recommended for pulseless patients with or without signs of life, according to the Eastern Association for the Surgery of Trauma (EAST) in 2015 [18]. However, the survival rate of patients undergoing this type of surgery is reported as low as 4.6%, and almost all of them were older than nine years old in a meta-analysis [15]. VSD is one of the rare but severe injuries that occur following both types of cardiac trauma [19, 20]. There are four main types of VSD including perimembranous, muscular, outlet, and inlet types [21]. VSD following blunt cardiac traumas can be caused by any event putting the heart under compression between the sternum and vertebra, primarily seen in motor vehicle collisions, and also might be seen in penetrating injuries, primarily gunshots and stab wounds [11]. Since the clinical presentation of this condition can be very indistinct and delayed, depending on the severity of the injury, the size, and the location of the defect, the diagnosis needs a precise and comprehensive examination [22]. A considerable percentage of VSDs are diagnosed after days or weeks due to the regression of edema and inflammation of the surrounding tissue, resolution of the occluding clot, retraction of the fibrous edges, and ventricular enlargement [23].

Although the best initial diagnostic actions are 12-lead electrocardiography, measuring serum troponin I level, and echocardiography, more advanced procedures such as computed tomography (CT) scan, magnetic resonance imaging (MRI), and cardiac catheterization might be needed for obtaining more detailed comprehension [24, 25]. Any patients with suspicious symptoms and signs, such as an abnormal new murmur, elevated troponin level, or electrocardiogram

(mostly arrhythmia), should be admitted with continuous cardiopulmonary monitoring [26]. The treatment recommended for these defects depends on the size of the defect, hemodynamic condition, and severity of symptoms [27].

Those with severe hemodynamic or CHF symptoms and VSD size 10 millimeters should be treated surgically as soon as possible to prevent deterioration and the advancement of heart failure [14]. In contrast, in stable patients with moderate-size defects and suitable location (such as defects far from the aorta and tricuspid valves that accompany the lower risk of injury to subvalvular tissue), conservative, medical, and less invasive methods such as Amplatzer occluder and transcatheter treatment can be chosen [6, 28, 29]. Patients who undergo transcatheter VSD or ASD closure should be monitored for severe complications such as arrhythmia and bleeding for at least 24 hours [30, 31]. In these cases, the procedural treatment prefers to be postponed by 2-3 weeks because, by that time, the edematous and inflamed location will be more fibrous and solid and consequently more prepared to be enclosed [32].

Valvular injuries are among the least common conditions that occur due to cardiac traumas. Blunt traumas mainly occur in the early systole, when ventricles have undergone an abrupt increase in pressure and empty atriums cannot protect them from contusion [33]. Due to the more exposed location, larger size, and more pressure on the left side of the heart, the aorta and mitral valves are more vulnerable to injuries following cardiac trauma [34]. The symptoms of mitral valve injury following penetrating trauma vary based on the severity of the damage, from asymptomatic patients with a recently detected murmur to severe CHF symptoms or hemodynamic instability [35]. The preferred diagnostic tools for this condition are a cardiac CT scan and echocardiography, which are used immediately in patients with predictors of worse outcomes such as abnormal ECG, elevated troponin, or low blood pressure [36]. The early diagnosis and treatment of valvular damage are crucial since, by that time, the likelihood of shortening of the corda tendinea and leaflet damage following the fibrosis progression will be increased [4]. While less common and less severe causes of traumatic MR are corda tending or annular tears, which can be repaired or managed medically in most cases, delayed treatment can cause the progression of lesions to more severe damages such as papillary muscle rupture, and limit the therapeutic options to valve replacement surgeries [34, 37]. Therefore, in cases of simultaneous severe MR and VSD following trauma, the repair should be done as soon as the patient becomes hemodynamically stable to decrease the risk of resulting congestive heart failure, pulmonary hypertension, and the progression of valvular injuries [11, 12]. There are other reports of penetrating cardiac traumas that have been reported, and we mentioned them in Table 3 [1, 36, 38-42].

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4. Strength and Limitations

The presenting study has several strengths. One of the most critical points is recording the patient's history and data and reporting them in detail and as precise as possible. Moreover, the scarcity of reported similar cases and critical and practical clinical learning points are other strength points of the study. The limitation of the study is that it did not record any pictures or media from the initial surgery of the patient in the first hospital in the patient's residing city.

5. Conclusions

This case highlights the complexity of the diagnosis and management of traumatic ventricular septal defects (VSDs), mainly when located in the anterior muscular septum. Other structural defects, such as valvular damage, commonly accompany these injuries. The symptoms of this condition can be very subtle and delayed. Therefore, in any chest trauma, ruling out these conditions through a meticulous physical exam and the utilization of diagnostic devices such as TTE and TEE should be prioritized. The optimal therapeutic method should be determined considering the patient's clinical condition, the size and location of the structural injury, and the availability of devices and expert physicians or surgeons.

6. Declarations

6.1. Acknowledgments

None.

6.2. Funding

No funds have been received for this study.

6.3. Ethical declaration

Written informed consent was obtained from the patient and the patient's guardians to publish this report under the journal's patient consent policy.

6.4. Authors' Contribution

HM contributed to data curation, supervision, and project administration. AT and HP contributed to data collection, analysis, supervision, and the review of the manuscript. MA and PE contributed to data collection, analysis, data curation, analysis of data, writing the initial draft, and revision of the final manuscript. All authors read and approved the final version of the manuscript.

6.5. Conflict of interest

Authors declared no conflict of interest.

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 Table 1:
 Laboratory finding of patients after the thoracotomy surgery (12 hours after the injury) and after presentation to the ED of Rajaei

 Hospital (about 72 hours after the injury)

Laboratory Index	After thoracotomy	Rajaei ED	Normal Range
Hemoglobin (g/dL)	11.5	14.5	13.1-17.2
MCV (fL)	85.3	92	81-101
MCH (pg/cell)	27.7	32	27-35
RBC (×106/ ul)	5.17	5.30	4.1-5.7
WBC (×103/ ul)	7.21	5.69	4.4-11
Platelet (/ ul)	158,000	188,000	150,000-450,000
BUN (mg/dl)	16	13	13-43
Creatinine (mg/dl)	0.7	0.5	0.5-1.0
Sodium (mEq/L)	140	136	0.32-3.2
Potassium (mmol/L)	3.9	4.1	3.6-5.2
Calcium (mg/dl)	9.3	9	8.5- 10.5
Magnesium (mg/dl)	1.7	1.9	1.5-2.2
Albumin (g/dl)	4.1	4.8	3.4-5.5
PT (seconds)	14	12	11-14
PTT (seconds)	31	28	25-35
INR	1.1	1.1	0.8-1.2
ESR (mm)/hour	25	8	30
CRP	1+	0	0-1+
Blood Glucose (mg/dl)	100	80	74-126
Urine analysis	Normal	Normal	Normal

MCV: Mean Corpuscular Volume, MCH: Mean Corpuscular Hemoglobin, RBC: Red Blood Cell Count, WBC: White Blood Cell Count, BUN: Blood Urea Nitrogen, PT: Prothrombin Time, PTT: Partial Thromboplastin Time, INR: International Normalized Ratio, ESR: Erythrocyte Sedimentation Rate, CRP: C-Reactive Protein.

 Table 2:
 Penetrating cardiac trauma in pediatrics reported in other literature

Study and Au- thors	Presentation	Physical Exams/ Diagnostic Findings	Treatment and Clinical Progression
1-Penetrating cardiac trauma Lee A. et al. (2023)[38]	male with an anterior chest stab wound/ when he was at a bus stop.	mmHg/ PR:110/ tachypneic/ 3 cm deep laceration on the right sternal border/ unconscious and unresponsive despite massive transfusion/ FAST shows tamponade/lose pulses in the ED/ resuscitation and left thoracotomy happens simultaneously/ intubation/ pericardiotomy/ blood around the heart is evacuated/ pericardiotomy releases torrential hemorrhage/ heart is empty and 3cm laceration is seen on right ventricle/ empty heart/ fibrillating/ resuscitation along with correct anterolateral thoraco-	chloride/ magnesium sulfate/ amiodarone, epinephrine/ rapid enclosure of right ventricle by interrupted 3-0 horizontal mattress sutures, open cardiac massage/ bicarbonate infusion/ al happened in the ED/ transfer to OR for tube and pleural and pericardial drains insertion/closure
trauma: Delayed diagnosis and	old African- American male/ gunshot to left chest/ dyspnea/ nearby hospital inserted nee- dle due to left pneumothorax/ transferred to level 1 trauma	tomy/ Single gunshot wound / the left posterior axillary line/ exit wound in the anterior chest just inferior and medial to the left nipple/ large laceration on forehead No PE in FAST/ splenic laceration in laparoscopy/ laceration in the apex of the left heart.	Chest tube insertion/ diagnostic laparoscopy with 250ml blood in the abdomen/ repair of the diaphragmatic defect and herniation of the omentum / resuscitative left anterolateral tho racotomy in the PICU (pediatric intensive care unit) after the loss of peripheral pulses and rapid bleeding was seen in the chest tube/ repair of the hole in the apex of the LV, pericardium, and lef lung laceration, five days later chest tube was removed, and the patient was discharged while he was symptom-free.
caused by a fall on a pencil with	from stairs with a pencil in his hand and follow- ing penetration	tercostal space/ a piece of the colored pencil detected inside the wound/normal vital signs, breath sounds, and heart tones intrathoracic foreign body with an entrance parasternal left	a Satinsky clamp/defects sutured with a 4-d monofilament non-absorbable suture/ retroster nal and pericardial chest tube inserted/ unevent ful post-surgery/ discharged without any remark
, ,	female was brought to the ED due to ear	A raised erythematous area and a sharp metallic needle-like object were detected in the subcutaneous tissue inferior to the xiphoid process.	able symptom eight days later Subxiphoid incision was made, and the peri cardium opened in the OR. A small caliber sewing needle entering the right ventricle was noted and withdrawn without bleeding or any other complication. Complicated post-surgery due to hy drocephalus, pseudo meningocele, and an occipital skull fracture from her prior head injury Duraplasty + placement of a ventriculoperitonea shunt. On hospital day 65, he was discharged without medication to his grandmother 65 day after surgery (to his grandma's home following proven child abuse by her parents).

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 Table 2:
 Penetrating cardiac trauma in pediatrics reported in other literature (continue)

Study and Au-	Presentation	Physical Exams/ Diagnostic Findings	Treatment and Clinical Progression
thors			
		A 2-cm skin wound at the junction of the left	
,		midclavicular line and fifth intercostal space.	1 *
trauma: Review	,	Conscious, with stable vital signs/ moder-	· ·
and case reports	J 1	ate pericardial effusion, without echocardio-	right ventricle was repaired by a direct suture
Leite L. et al.		graphic signs of cardiac tamponade was de-	1 0 3
(2017) [36]		tected in the ER TTE/ significant pericardial ef-	charged without any considerable symptoms.
		fusion (14 mm) close to the right ventricle and	
		a small left pleural effusion in his thoracic CT	
	minutes before	scan.	
	arrival to the		
	ED/.		
6-Successful		Normal CXR and elevated lactic acid 6.2 (nor-	, ,
0		mal 0.4–2.0) were found. His ABG pH was 7.33	
		(normal 7.35-7.45), and in his IV contrast CT	
		scan, left sixth rib fracture, left lung contusion,	1
· ·		a left hemothorax, splenic injury with (grade 4)	1
		left kidney injury, and perinephric hematoma	
		along with left-side pneumoperitoneum, severe	
thoracic injuries		cardiac contusion, was evident. A post-surgery	0 ,
in a pediatric		echocardiogram showed an ejection fraction of	, , ,
		43%, most probably due to the heart contusion.	
	due to the unilat-		moval from the abdomen, splenectomy, seg
literature review	erally decreased		mental colonic resection at the distal trans
	breathing sound		verse colon, and left chest tube insertion were
(2020) [41]	in the local ED.		performed. Inspection of the heart revealed a
	No remark-		contusion without PE or hematoma. The di
	able abnormal		aphragm was repaired, and two units of packed
	symptom was		blood cells (PRBCs) infusion were also done
	detected.		On the ninth day after surgery, the chest tube
			was removed. Expectant management of the
			cardiac contusion was planned along with brie
			management with inotropic pressors and mon
			itoring.

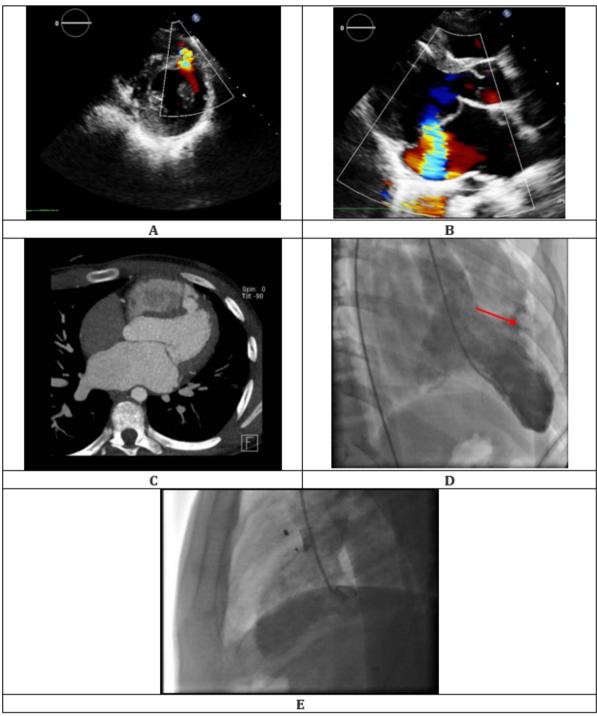


Figure 1: Anterior muscular VSD seen in transthoracic echocardiography (A); Severe MR seen in the echocardiography of the patient (B); Cardiac CT angiography confirmed the presence of a VSD (C); Cardiac Angiography- VSD is shown after dye injection (red arrow) (D); Cardiac Angiography- Occlusion of VSD by an Amplatzer cera liftech muscular occlude (E).