Traumatic Ventricular Septal Defect Resulting from a Motor Vehicle Collision

Robert Tonks, David Perkel, Aimee Wehber, Bret Rogers
Department of Cardiology, University of Tennessee Medical Center, Heart Lung Vascular Institute, Knoxville, TN, USA

Abstract

This case report describes a rare case of ventricular septal defect due to non-penetrating trauma in a 43 year old male involved in a motor vehicle collision. The diagnosis was made by echocardiogram and the patient was taken immediately to the operating room for emergent surgical repair of the ventricular septal defect and survived.

Keywords: Blunt chest trauma, blunt cardiac injury, cardiogenic shock, shunt, murmur, ventricular septal defect

Introduction

Posttraumatic ventricular septal defect (VSD) is an uncommon complication of penetrating cardiac injury with an incidence of only 1%–5% and is an even more uncommon complication of nonpenetrating chest trauma. The true incidence is unknown and likely underestimated since this potentially life-threatening condition can rapidly deteriorate and lead to death before the diagnosis is made. In my literature review there are few reports of non-penetrating trauma of non-pediatric patient with an acute presentation of ruptured muscular ventricular septal defect without a known pre-existing septal wall defect. Presenting signs are often masked by concomitant injuries, and the severity, presentation, and course are variable. The mainstay of diagnosing VSDs is the transthoracic echocardiogram. This case report describes a rare case of acquired posttraumatic VSD as a complication of nonpenetrating chest trauma that occurred during a motor vehicle collision.

Case Report

A middle aged male presented to the emergency department after being involved in a motor vehicle crash while riding a motorcycle. He was visibly agitated and complaining of sharp left-side chest pain, severe abdominal pain, bilateral lower extremity, and left upper extremity pain. He was noted to have multiple skin lacerations, decreased breath sounds in bilateral lung fields, and paradoxical movement of the chest wall with a large pulsatile left chest wall contusion. He reported no past medical history. The only family history reported was heart disease in his grandfather, but he was unable to provide any further details. He took no medications. He reported current tobacco use, alcohol use, and illicit polysubstance use including marijuana, cocaine, heroin, and methamphetamine with reported intravenous drug use. He last reported the use of these substances approximately 4 h before the motor vehicle collision, and a large quantity of methamphetamine was found on his person at the scene of the collision.

He was tachycardic and hypotensive, so he was given 2 l of normal saline intravenous fluid and 1 unit transfusion of packed red blood cells. This provided temporary hemodynamic improvement; however, he later continued to develop hypotension with concern for developing shock. Vasopressor support was initiated with norepinephrine for hemodynamic stability with rapidly increasing requirements.

Computed tomography imaging of his chest, abdomen, and pelvis was performed revealing Grade II splenic laceration without active extravasation, midsternal fracture with minimal displacement, bilateral upper lobe pulmonary contusions, and subcutaneous emphysema. The patient was transferred to the operating room emergently for emergent surgical repair of the VSD. He was found to have a large paravascular muscular VSD, with no underlying congenital defect, and was repaired with a patch closure of the defect. He was extubated 12 h postoperatively and was discharged without complications.

Address for correspondence: Dr. Robert Tonks, Department of Cardiology, Heart, Lung, Vascular Institute, University of Tennessee Medical Center, 1924 Alcoa Hwy, Knoxville 37920, TN, USA. E-mail: rtonks@utmck.edu

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Tonks R, Perkel D, Wehber A, Rogers B. Traumatic ventricular septal defect resulting from a motor vehicle collision. J Cardiovasc Echography 2018;28:191-3.
Cardiac enzymes were drawn due to chest trauma and chest pain with initial troponin >50 ng/ml. His initial electrocardiogram showed normal sinus rhythm with a right bundle branch block and ventricular rate of 98 beats/min. There were no ST-segment or T-wave changes concerning for an acute coronary syndrome. He denied having any chest pain before the motor vehicle collision. On examination, the patient was noted to have a large contusion over his left chest wall. He was tachycardic with heart rate >100 beats/min, and there was a harsh, turbulent 3/6 holosystolic murmur on auscultation of his heart at the left sternal border.

Transthoracic echocardiography with color Doppler was performed emergently at the bedside, confirming the presence of a large muscular VSD in the mid-portion of the interventricular septum measuring 16 mm in diameter [Figure 1a-c]. The Qp:Qs ratio was calculated to be 6.6, indicating a very hemodynamically significant VSD. The left ventricle was hyperdynamic with an ejection fraction of 64.3%. The right ventricle was normal in size, wall thickness, and function by tricuspid annular plane systolic excursion. The left atrium was mildly dilated despite pulmonary venous sampling being consistent with normal left atrial pressure. The right atrium was normal in size. There was severe tricuspid regurgitation with calcified chordae. The pulmonary valve was normal, and there was no pericardial effusion.

He continued to have worsening hemodynamic instability with cardiogenic shock due to the large VSD, with increasing vasopressor requirements. He was felt to be a poor candidate for transcatheter closure of the VSD with an Amplatzer device which was not immediately available at our institution. The patient was taken to the operating room by the cardiothoracic surgeons. The VSD was 3 cm at the time of surgery and was successfully closed with pericardial patch–bovine reinforced with pledges. Postsurgical transesophageal echo confirmed that there was no flow across the patched VSD. He survived a potentially fatal and rare complication of blunt force trauma to the chest.

**DISCUSSION**

Posttraumatic VSD due to nonpenetrating blunt chest trauma is thought to occur by one of two mechanisms: early presentation, occurring within the first 48 h after trauma due to mechanical rupture or delayed presentation, occurring more than 48 h after trauma due to inflammatory rupture. Mechanical septal rupture may occur as a result of direct cardiac impact or when the heart is compressed between the sternum and the spine. It has also been suggested that a healed congenital VSD with a weakened ventricular septum may be re-opened with significant blunt trauma to the chest. Delayed inflammatory rupture is thought to occur when cardiac injury leads to localized edema, disruption of microvascular flow, leading to infarction, septal liquefaction, and perforation.

Surgical repair is indicated if the defect is large, if the pulmonary to systemic blood flow ratio exceeds 2:1, or if there is evidence of cardiac failure or cardiogenic shock. However, a persisting small lesion with chronic left-to-right shunting may result in right ventricular failure over time. VSDs diagnosed within 48 h after nonpenetrating chest trauma are usually larger, more severe, require emergency surgery and have higher mortality rates than those diagnosed later. In contrast, cases of late diagnosis of posttraumatic VSD (more than 48 h after trauma) rarely required emergency surgery although often are repaired electively and may be managed conservatively since they occasionally close spontaneously.

Posttraumatic VSD is rare and sometimes challenging diagnosis. However, elevated troponin and a new holosystolic murmur should raise suspicion for further investigation. Since cardiac troponin I is only released when there is disruption of the myocardial cell membrane, it is a specific indicator of myocardial damage and of a possible VSD. Echocardiography is the mainstay of diagnosing VSDs.

**CONCLUSION**

Posttraumatic VSD due to nonpenetrating chest trauma is a rare but potentially life-threatening complication. Trauma patients with blunt chest injuries, left chest wall contusion, elevated cardiac troponin I, hemodynamic instability and a newly developed holosystolic murmur, and no history of cardiovascular disease should prompt further investigation with echocardiography. Large or symptomatic defects should be surgically repaired. Smaller, asymptomatic lesions can be managed conservatively but should be monitored closely to detect the possible development of late complications.
Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES
1. Rollins MD, Koehler RP, Stevens MH, Walsh KJ, Doty DB, Price RS, et al. Traumatic ventricular septal defect: Case report and review of the English literature since 1970. J Trauma 2005;58:175-80.
2. Pierli C, Iadanza A, Del Pasqua A, Sinicropi G. Unusual localisation of a ventricular septal defect following blunt chest trauma. Heart 2001;86:E6.
3. Mason DT, Roberts WC. Isolated ventricular septal defect caused by nonpenetrating trauma to the chest. Proc (Bayl Univ Med Cent) 2002;15:388-90.
4. Pruitt CM, Titus MO. Ventricular septal defect secondary to a unique mechanism of blunt trauma: A case report. Pediatr Emerg Care 2007;23:31-2.
5. Genoni M, Jenni R, Turina M. Traumatic ventricular septal defect. Heart 1997;78:316-8.
6. Rootman DB, Latter D, Admed N. Case report of ventricular septal defect secondary to blunt chest trauma. Can J Surg 2007;50:227-8.
7. Amorim MJ, Almeida J, Santos A, Bastos PT. Atrioventricular septal defect following blunt chest trauma. Eur J Cardiothorac Surg 1999;16:679-82.
8. Sawhney J, Patel PH, Blackwell RA. Early progression of an isolated ventricular septal defect after blunt trauma. J Trauma 2008;64:218-20.
9. Zamani J, Amirghofran AA, Moaref AR, Afifi S, Rezaian GR. Posttraumatic coronary artery-right ventricular fistula with multiple ventricular septal defects. J Card Surg 2010;25:670-1.
10. Ryan L, Skinner DL, Rodseth RN. Ventricular septal defect following blunt chest trauma. J Emerg Trauma Shock 2012;5:184-7.