Published in final edited form as:

J Thorac Imaging. 2012 November ; 27(6): W174–W176. doi:10.1097/RTI.0b013e31823c3ea5.

Traumatic Ventricular Septal Defect: Characterization with ECG-Gated Cardiac Computed Tomography Angiography (CTA)

Carlos A. Rojas, MD¹, Dana M. Cruite, BS², and Jonathan H. Chung, MD³

¹Affiliate Assistant Professor, Radiology Department, University of South Florida College of Medicine, Tampa, FL

²University of South Florida College of Medicine, Tampa, FL

³Assistant Professor, Radiology Department, National Jewish Health, Denver, CO

Summary

Ventricular septal defects secondary to blunt trauma to the chest are infrequently reported in the literature. Additionally, the majority of these cases have been imaged with echocardiography alone. We report a case in which a ventricular septal defect caused by blunt chest trauma was characterized with ECG-gated CT angiography (CTA). A review of the current literature and theories of injury mechanism are also included.

Keywords

thoracic trauma; ventricular septal defect; blunt chest trauma; ECG-gated CTA; cardiac trauma

Introduction

Acquired ventricular septal defects (VSDs) are uncommon. They can be secondary to myocardial infarction, infective endocarditis and trauma. Both penetrating and non penetrating chest trauma are causes of traumatic VSDs. Traumatic VSDs have been described in up to approximately 4% of cases of penetrating heart trauma.¹ VSDs secondary to non-penetrating chest trauma are rare and their pathophysiology is not completely understood.

Case Report

A twenty-eight year old male baseball player collided with another player while trying to catch a ball, receiving a direct impact to his sternum. Afterwards, he felt a new vibrating sensation (thrill) in his chest. His vital signs were stable when paramedics arrived, and he was transported to the closest hospital for evaluation. Echocardiogram performed at that institution showed a muscular ventricular septal defect. He was then transferred to our institution and an ECG-gated CTA was performed for better characterization of this abnormality, evaluation of associated injuries and surgical planning. Cardiac CT was

Name/Address for correspondence: Carlos A. Rojas caranrojas@gmail.com 5225 Bayshore Blvd, Tampa FL 33611 cell (813)220-8191 fax (813)832-1153 .

Conflicts of interest and sources of funding: Jonathan H. Chung (NIH, Siemens AG)

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

performed using a prospectively gated acquisition, with 100kVp and 685mA. Total contrast volume used was 95cc at a rate of 5cc/s. Of the total contrast volume, 20cc was for a test bolus, and 75cc for the CTA, followed by a 60cc saline chaser. The patient was receiving IV metoprolol with a mean heart rate of 56 bpm, and no additional beta blockade was needed.

ECG gated CTA further demonstrated the presence of a partial rupture of the mid to basal anterior walls of the left ventricle and rupture of the basal anteroseptal wall with a ventricular septal defect (Figure 1). Coronary arteries were normal, and despite rupture of the anterior wall of the left ventricle, no pericardial effusion was present. The lungs were normal in appearance and only non-displaced fractures of several left anterior ribs lateral to the costochondral junctions were noted (Figure 2).

The patient was admitted to the cardiac intensive care unit with continued control of blood pressure and heart rate using IV metoprolol, in order to decrease cardiac workload. In addition, myocardial edema was allowed to decrease before surgical repair. Three weeks later the patient underwent successful surgical repair. ECG gated CTA examination was very helpful for surgical planning in determining the location of the partial myocardial rupture and VSD in relation to the coronary vessels (image 1C).

Discussion

A ventricular septal defect (VSD) due to blunt chest trauma is a rare occurrence, with only 58 cases reported in the literature between 1970 to 2005.² The most common mechanism is a motor vehicle accident. The average age reported is 21.4 years.² The presentation following injury varies widely: while some patients arrive immediately afterwards in cardiogenic shock, others initially experience only mild chest pain, subsequently developing heart failure.

The pathogenesis of blunt traumatic VSDs is thought to be due to compression of the heart between the vertebral bodies and the sternum. If this compression occurs during the late diastolic phase, when the atrioventricular valves are closed and the ventricles filled with blood, the increased intracardiac pressure can result in acute rupture of the septum.^{2,4-6,8} However, if the trauma occurs at another phase of the cycle, it may only cause a myocardial contusion. In this setting, formation of the VSD occurs later, after associated injury to the microvasculature has led to infarction, necrosis, and perforation of the interventricular septum.^{2,6,8} This is the proposed mechanism in cases of delayed traumatic VSD, where a defect is not found during the initial echocardiogram, but days to weeks later on a repeat examination. In these instances, the patient often presents with few or no symptoms, but may subsequently develop fatigue, chest pain, and exertional dyspnea.

In the acute presentation, the patient typically has signs of hemodynamic instability and respiratory distress, including hypotension, tachycardia, and tachypnea.^{2-4,6} Mechanical ventilation may become necessary, as well as positive inotropic agents such as dopamine for worsening hypotension. Physical exam usually reveals a precordial thrill and high grade, pansystolic murmur at the left sternal border^{2,4-7}, but ECG findings are more variable. Either ST segment depression or elevation may be seen, with or without Q wave changes.^{2,6} Chest radiography is often performed, and although it may show cardiomegaly and pulmonary edema, it is frequently normal.^{2,6,8} Echocardiography usually reveals the VSD.^{2-7,9} The defect is most commonly located in the muscular septum, near the apex of the heart.² After stabilization, patients may be evaluated using cardiac MRI, which can detect the VSD itself as well as common associated injuries to the heart such as myocardial infarction, disruption of the aortic, mitral, and tricuspid valves or their corresponding papillary muscles, and myocardial contusion.^{2-4,7,10} Cardiac catheterization, echocardiography, and cardiac MRI

have all been used to calculate the pulmonary to systemic flow ratio (Qp:Qs) of VSDs,^{2,4,5} which can play an important role in determining treatment.

Treatment of the VSD depends on several factors, including the size of the defect, patient symptoms, and degree of left-right shunting. Patients that experience increasing hemodynamic instability, shunts with a Qp:Qs ratio greater than 1.5-2:1, or defects larger than 1.5-2 cm usually require immediate repair of the defect.⁴⁻⁶ This is often done through an open surgical approach, with cardiopulmonary bypass, ventriculotomy, and patching of the VSD. There have also been successful reports of percutaneous repair with closure devices, an alternative option for patients whose critical condition precludes them from undergoing open surgery.⁹ In patients who are more stable, repair is often delayed several weeks, as the tissue surrounding the defect will become fibrotic and promote surgical closure.^{5,8}

Although the echocardiogram is the most common imaging study to diagnose a traumatic VSD, the quality of this examination may be limited due to concomitant injuries to the chest, and additional imaging may be required. While cardiac MRI can be used to confirm a traumatic VSD and assess the degree of shunting, the time required to obtain this study makes it unfavorable in an acute setting. Here we report the further characterization of a blunt traumatic VSD by ECG gated CTA. In this case, ECG gated CTA was very helpful in determining the location, extent and size of the defect. It also enabled identification of the associated partial rupture of the anterior wall, and its relationship to the coronary arteries prior to surgical repair.

References

1. Olsovsky MR, Topaz O, DiSciascio G, Vetrovec GW. Acute traumatic ventricular septal rupture. *Am Heart J*. 1996; 131:1039–41. [PubMed: 8615294]
2. Rollins MD, Koehler RP, Stevens MH, et al. Traumatic ventricular septal defect: case report and review of the English literature since 1970. *J Trauma*. 2005; 58(1):175–180. [PubMed: 15674170]
3. Stahl RD, Liu JC, Walsh JF. Blunt cardiac trauma: atrioventricular valve disruption and ventricular septal defect. *Ann Thorac Surg*. 1997; 64:1466–1468. [PubMed: 9386725]
4. Blasco PB, Comas JG, Munoz RDA. Spontaneous improvement of a haemodynamically significant ventricular septal defect produced by blunt chest trauma in a child. *Cardiol Young*. 2009; 19:109–110. [PubMed: 19014715]
5. Sparrow JG, Miller DWJ. Ventricular septal defect following blunt thoracic and abdominal trauma: a case report. *J Trauma*. 1989; 29(5):690–693. [PubMed: 2724388]
6. Wu JJK, Yu TJ, Wang JJ, et al. Early repair of traumatic ventricular septal defect and mitral valve regurgitation. *J Trauma*. 1995; 39(6):1191–1193. [PubMed: 7500421]
7. Jun SL, Chanani NK, Moore P, et al. Magnetic resonance imaging of a post-traumatic myocardial infarction and ventricular septal defect with a closure device in place. *Circulation*. 2007; 115:e13–e15. [PubMed: 17228006]
8. Kim YM, Yoo BW, Choi JY, et al. Traumatic ventricular septal defect in a 4-year-old boy after blunt chest injury. *Korean J Pediatr*. 2011; 54(2):86–89. [PubMed: 21503202]
9. Cowley CG, Shaddy RE. Transcatheter treatment of a large traumatic ventricular septal defect. *Catheter Cardiovasc Interv*. 2004; 61:144–146. [PubMed: 14696175]
10. Co SJ, Yong-Hing C, Galea-Soler S, et al. Role of imaging in penetrating and blunt traumatic injury to the heart. *Radiographics*. 2011; 31:E101–E115. [PubMed: 21768229]

Figure 1.

Volume rendered CT images of a 28 year old man after blunt trauma to the chest demonstrate a contained rupture of the anterior wall of the LV (arrowheads), extending posteriorly to involve the basal anteroseptum creating an interventricular connection consistent with a traumatic VSD (black arrowhead) (A, B and C). Note the high attenuation contrast entering the “washed-out” RV (A and B), and the relationship of the left anterior descending (LAD) and diagonal (D) coronary arteries with the pseudoaneurysm and VSD (A). Ao= Aorta, LV= Left Ventricle, RV= Right Ventricle, RVOT= Right Ventricular Outflow Tract, PA= pulmonary artery.

Figure 2.
Axial oblique CT image shows a fracture of a left anterior rib (black arrow). LV= Left Ventricle, RVOT= Right Ventricular Outflow Tract.