

Case Report

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# Unusual Cardiac Gunshot Injury Causing Traumatic Ventricular Septal Defect

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## Abstract

Cardiac gunshot injuries are frequently fatal with poor outcome. We report the very unusual survival from cardiac gunshot injuries at multiple sites including the left atrium, left ventricle, right ventricle apex, right ventricular outflow tract and the inter-ventricular septum, creating a large muscular ventricular septal defect (VSD). It shows how rapid transport, proper resuscitation and emergent surgery can improve survival in patients who sustain life threatening cardiac gunshot injuries.

**Keywords:** Gunshot injury; Ventricular septal defect (VSD); Traumatic cardiac injuries

## Introduction

Cardiac injuries can be classified as penetrating and non-penetrating (blunt). Penetrating wounds are usually caused by bullets, missiles or stabbing with a piercing object. The right heart (right atrium or ventricle) is damaged in most cases, because of its anatomical position; making up most of the anterior aspect of the heart. Cardiac trauma from a penetrating chest injury can produce massive hemorrhage, cardiac tamponade, damage to myocardial free wall or inter-ventricular septum, laceration of coronary arteries or great vessels, and serious damage to the conduction system [1,2]. Prognosis and outcome is often not good in cardiac gunshot injuries [3,4].

## Case Report

A 19-year-old African American male presented to an outside hospital after experiencing a gunshot injury to the anterior chest at a gas station. His mother put him in the car, and an ambulance took over on the way to the hospital. There was a small entrance wound in the anterior chest with no exit wound. The patient was intubated in route. When he got to the trauma bay in the emergency room, there was pulseless electrical activity. An emergency left thoracotomy was performed by trauma surgery. The pericardium was incised and pericardial tamponade was relieved. Intracardiac cardiopulmonary resuscitation was started. The patient went into ventricular fibrillation and was shocked several times returning to normal sinus rhythm. The estimated blood loss was 2.5 liters. He was taken to the operating room, where the trauma surgeon extended the prior left anterolateral thoracotomy across the midline in a clamshell fashion. There was a 1-cm laceration to the left atrium which was closed. There was another defect closed; 1-cm hole in the apex of the left ventricle. The cardiothoracic surgeon was consulted emergently to facilitate the mediastinal exploration. An injury to the right ventricle anteriorly overlying the right ventricular outflow tract was suture-repaired. There was another injury, which was noted in the apex posteriorly, between the right and left ventricles overlying the septum that was suture-repaired. The bullet was noted to be in the mediastinal sac and was removed. The patient developed acidosis and coagulopathy during the operation requiring massive blood transfusion and fluid resuscitation.

The patient had a prolonged postoperative course. On post-operative day 4, a holosystolic murmur was noted. An echocardiogram showed a 6 to 10 mm size anterior mid-muscular ventricular septal defect, but no therapy recommended. He was transferred to a rehabilitation center,

where he made huge improvement, being discharged from there with only short-term memory loss.

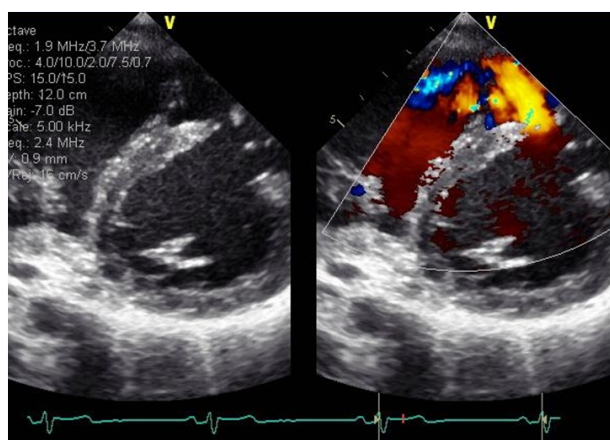
The patient was recently referred to our cardiology clinic for 1 year follow-up and consideration of transcatheter ventricular septal defect closure. He reported doing well, almost back to his baseline status, apart from getting tired easily probably related to deconditioning and having some short-term memory loss mainly with multi-tasking. The patient was not taking any medications. His physical exam was significant for a pronounced precordial systolic thrill, and a loud, harsh 5/6 holosystolic murmur best heard at the left lower sternal border. There was no clinical evidence of heart failure. An electrocardiogram was repeated and demonstrated rightward axis and qr' pattern in V1. Echocardiography during the clinic visit showed [Figure 1-3], a moderate (6-10 mm) muscular ventricular septal defect in the anterior portion of the mid to apical septum with low velocity flow across the septum into the right ventricular apex, then high velocity systolic egress from the right ventricular apical trabeculations toward the tricuspid valve via 2 jets with peak instantaneous pressure gradient of 70 mm Hg (Simultaneous systemic systolic blood pressure 113 mmHg). The study also showed normal left ventricular size and function. A cardiac catheterization was performed prior to our office visit, and showed a pulmonary to systemic blood flow ratio (Qp/Qs) of 1.2:1 with high normal systolic right ventricular and pulmonary arterial pressures (28 mm Hg). Thus, our patient's echocardiography and catheterization findings were consistent with a moderate to large, ventricular septal defect with only minimally elevated right ventricular and pulmonary arterial pressure and minimal left to right shunt. The explanation for this apparent clinical contradiction is that the left to right shunt was restricted by large right ventricular apical trabeculations and brought

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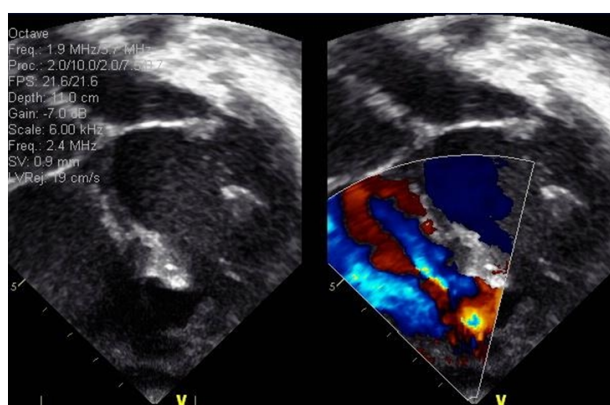
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**Figure 1:** Short axis parasternal view, 2-D echocardiography and color comparison showing a moderate muscular ventricular septal defect in the anterior interventricular septum with low velocity flow across the septum into the right ventricular apex.



**Figure 2:** Modified 4-chamber apical view, 2-D echocardiography and color comparison showing a moderate size muscular ventricular septal defect in the mid to apical septum with low velocity flow across the septum into the right ventricular apex, then high velocity systolic egress from the right ventricular apical trabeculations toward the tricuspid valve via 2 jets.



**Figure 3:** Continuous-wave Doppler of the VSD jet showing a maximum instantaneous gradient of 76.96 mm Hg

together by sutures placed blindly in the right ventricular apex during his emergent surgical salvage at the time of presentation. This has produced a situation where the apex of the right ventricle communicates freely with the left ventricle, but only minimally to the right ventricle body and pulmonary bed. Thus, his ventricular septal defect, although large in size, is not hemodynamically significant. Thus, neither percutaneous nor surgical closure was recommended.

## Discussion

Penetrating cardiac injuries are life threatening emergencies for which the prognosis depends mostly on the prompt transport, efficient resuscitation, and early surgical intervention, as well as the site and the extent of the injury [1]. Injuries to the heart can result in intracardiac injury at various sites: the right ventricle, right atrium, followed by the left ventricle, left atrium and intrapericardial great vessels.

The mechanisms by which a ventricular septal defect can develop after trauma include acute laceration of the septum, deceleration injury that causes myocardial infarction from an intimal coronary artery tear, and cardiac contusion due to compression of the heart between the sternum and the spine [2,5-7]. The contused myocardium can become necrotic and subsequently become perforated and form a ventricular septal defect. A ventricular septal defect, particularly one caused by blunt trauma, can develop or be detected at any time from hours to months after the original insult. In our patient, we believe that the bullet traveled through the interventricular septum and caused the left ventricle, right ventricle and left atrial lacerations by ricochet effect.

Patients with small traumatic ventricular septal defects with restrictive physiology can be managed conservatively with regular follow-up and serial echocardiography. Small traumatic ventricular septal defects can remain hemodynamically stable for years and even close spontaneously over time [2-4]. Large traumatic ventricular septal defects with a pulmonary to systemic blood flow ratio ( $Q_p/Q_s$ )  $>2:1$  should be closed to prevent congestive heart failure and pulmonary hypertension. The timing of surgical or transcatheter intervention depends upon the patient's hemodynamic status. If the patient is hemodynamically stable, closure can be delayed to allow the heart to recover from the contusion and develop fibrosis around the defect, enabling more secure patch or transcatheter occluder placement.

## Conclusions

Survivals of gunshot cardiac injuries are uncommon. Early transport, proper resuscitation and emergent surgery can improve survival in patients who sustain such life threatening injuries. The possibility of a traumatic ventricular septal defect should be considered in all trauma patients who develop a new murmur. The need for surgical or transcatheter device closure depends on the physiologic significance of the intra-cardiac shunt.

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