Case Reports

Gunshot Wounds

Causing Myocardial Infarction, Delayed Ventricular Septal Defect, and Congestive Heart Failure

Sudeep Kumar, DM, FACC
Nagaraja Moorthy, MD
Aditya Kapoor, DM, FACC
Nakul Sinha, DM, FACC

Penetrating chest trauma can cause a wide variety of cardiac injuries, including myocardial contusion, damage to the interventricular septum, laceration of the coronary arteries, and free-wall rupture. Herein, we describe the case of a 21-year-old man who presented with congestive heart failure, which was secondary to an old myocardial infarction and complicated by the delayed formation of a ventricular septal defect. All of these conditions were attributable to multiple gunshot wounds that the patient had sustained 6 months earlier. Left ventricular angiography showed an apical aneurysm; a large, muscular, ventricular septal defect; and 19 gunshot pellets in the chest wall. Three months after aneurysmectomy and surgical closure of the septal defect, the patient had recovered fully and was asymptomatic.

This case reaffirms the fact that substantial cardiac injuries can appear months after chest trauma. The possibility of traumatic ventricular septal defect should be considered in all multiple-trauma patients who develop a new heart murmur, even when overt chest-wall injury is absent. ***(Tex Heart Inst J 2012;39(1):129-32)***

Cardiac trauma from a penetrating chest injury can produce massive hemorrhage, cardiac tamponade, damage to the myocardial free wall or interventricular septum, laceration of the coronary arteries, and serious damage to the conduction system.[1] Trauma is one of the nonatherosclerotic factors associated with acute myocardial infarction (AMI), particularly in people aged 45 years or younger. Herein, we describe the case of a young man who sustained gunshot wounds that led to delayed symptoms of heart failure.

Case Report

In March 2010, a 21-year-old man presented with progressive dyspnea of 6 months' duration and recent-onset orthopnea with features of congestive heart failure. He reported that he had sustained multiple gunshot wounds 6 months earlier. The pellet wounds, in the anterior aspect of the chest, had caused severe chest pain. He had immediately been taken to a general hospital, where the wounds were dressed and he was given analgesics, but the pellets were not removed. No electrocardiography, chest radiography, or echocardiography was performed. His symptoms had improved afterwards, so he had not sought further treatment. However, a few days later, he noticed gradually progressive dyspnea.

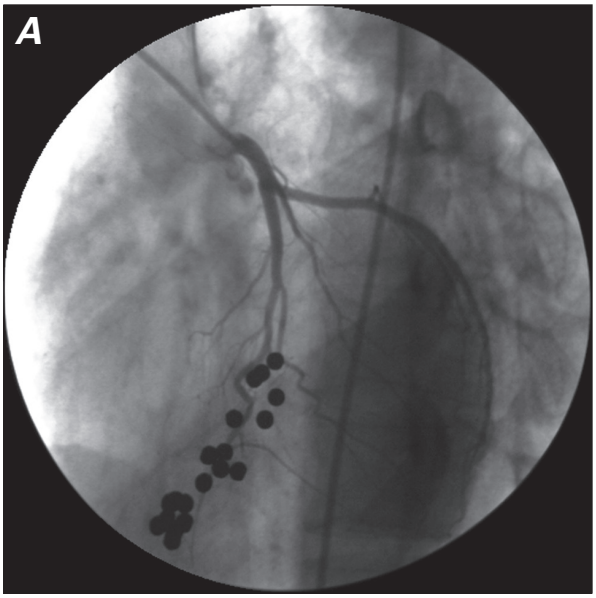
At the current presentation, physical examination revealed signs of heart failure: peripheral edema, raised jugular venous pressure, and a pansystolic murmur with a thrill that was consistent with ventricular septal defect (VSD). A chest radiograph revealed cardiomegaly with pulmonary venous congestion. Electrocardiography showed evidence of an old anterior-wall myocardial infarction. Transthoracic echocardiography revealed a mildly dilated left ventricle (LV) with an apical aneurysm; the LV ejection fraction was 0.40. Fluoroscopy showed 19 radiopaque pellets in the chest wall just over the precordium. Coronary angiography revealed a small-caliber distal left anterior descending coronary artery that bulged paradoxically over the aneurysmal apex and became occluded during each systole (Fig. 1). The other coronary arteries were normal. Left ventricular angiography showed a large apical aneurysm (Fig. 2) and a large, apical, muscular VSD with a left-to-right shunt (Fig. 3). The VSD was within the anteroapical aneurysm of the LV free wall. Oximetric data showed a 14% step-up in oxygen saturation at the right ventricular level. Results of angiocardiography and a hemodynamic examination suggested a hemodynamically significant left-to-right

Key words: *Coronary aneurysm/etiology; heart injuries/complications/diagnosis/physiopathology/surgery; myocardial infarction/etiology; treatment outcome; ventricular septal rupture/etiology; wounds, penetrating/physiopathology*

From: *Department of Cardiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226014, India*

Address for reprints: *Sudeep Kumar, DM, FACC, Department of Cardiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226014, India*

E-mail: *sudeep@sgpgi.ac.in*



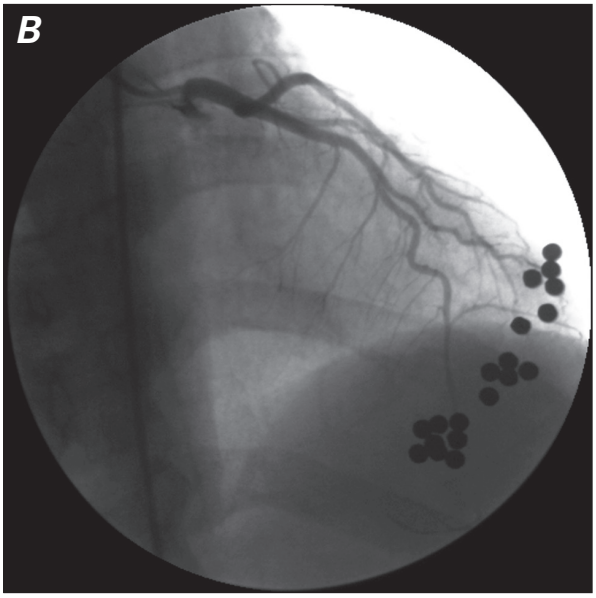


Fig. 1 *Coronary angiograms in **A**) left and **B**) right anterior oblique views show a small-caliber distal left anterior descending coronary artery that bulges paradoxically over the aneurysmal apex. The other coronary arteries are normal.*

Click here for real-time motion image: Fig. 1A.

Click here for real-time motion image: Fig. 1B.

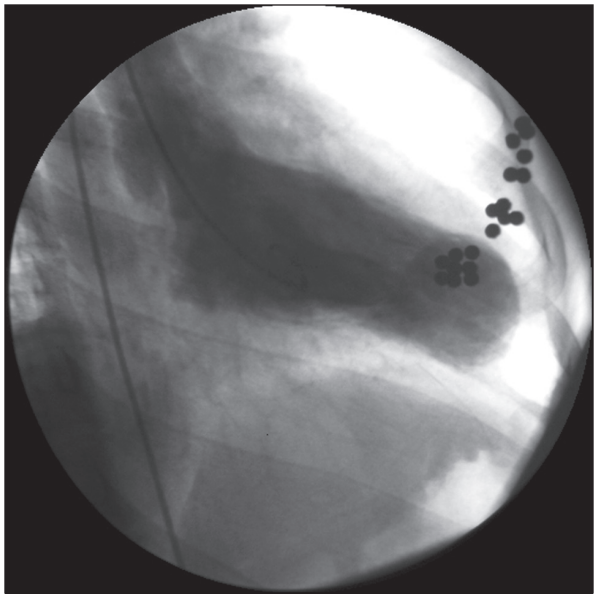


Fig. 2 *Left ventricular angiogram (right anterior oblique view) shows a large apical aneurysm.*

Click here for real-time motion image: Fig. 2.

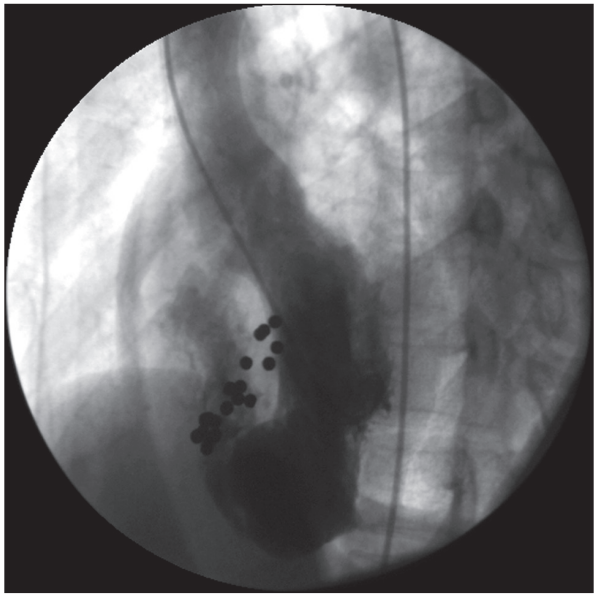


Fig. 3 *Left ventricular angiogram (left anterior oblique view) shows a large apical aneurysm and muscular ventricular septal defect with a left-to-right shunt.*

Click here for real-time motion image: Fig. 3.

shunt at the level of the ventricular septum. The ratio of systemic-to-pulmonary blood flow (Q p/Qs) was 2.2:1.

The patient's condition was stabilized, and he was taken for surgical closure of the VSD with aneurysmectomy. During surgery, an LV apical aneurysm of approximately 3.2 cm in diameter was noted. After cardiopulmonary bypass was instituted, the aneurysm was opened, and a well-circumscribed defect was seen in the anterior part of the septum. The VSD was closed with a double-velour Dacron patch that was sewn onto the left septal surface with running sutures and reinforced with pledgeted sutures. Because the gunshot pellets were numerous and small, no attempt was made to remove them. The patient's recovery was uneventful, and he was discharged from the hospital on the 5th postoperative day. On outpatient follow-up 3 months after the procedure, he was asymptomatic. Echocardiography showed an LV ejection fraction of 0.50 and no residual shunt at

the level of the interventricular septum. After 1 year, he was asymptomatic and doing well.

Discussion

Penetrating injury to the heart can result in intracardiac injury at various sites: the right ventricle is the most common (in 43% of reported cases), followed by the LV (33%), right atrium (15%), left atrium (6%), and intrapericardial great vessels (6%).[2]

Evidence from numerous case studies suggests that trauma is one of the nonatherosclerotic factors associated with AMI, particularly among persons aged 45 years or younger.[3-6] Because the coronary arteries lie superficially on the muscle wall, direct trauma with the resultant acute formation of coronary thrombosis is thought to be a probable mechanism of trauma-related AMI.[7] A shear force applied directly to the coronary artery can cause intimal tearing that could result in platelet aggregation and intracoronary thrombosis.[8] Finally, existing atherosclerotic plaque might predispose the coronary arteries to traumatic disruption.[9] The mechanisms by which a VSD can develop after trauma include acute laceration of the septum, deceleration injury that causes myocardial infarction from an intimal coronary artery tear (resulting in spasm, thrombosis, or dissection), and cardiac contusion due to compression of the heart between the sternum and the spine.[10] The contused myocardium can become necrotic and subsequently become perforated and form a VSD. A VSD, 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 myocardial contusion or a disruption of coronary flow at the time of the initial chest trauma resulted in an AMI and a delayed disruption of the muscular septum. An initially small VSD may have gradually enlarged, causing the delayed onset of severe heart failure.

When cardiac injury is suspected, structural damage should be researched by means of echocardiography—with examination repeated after several days, due to the possibility of delayed rupture. When an echocardiographic evaluation is not definitive, cardiovascular magnetic resonance can help to delineate structural damage to the ventricular free wall and septal structures. Initially, our patient was managed conservatively with analgesics and clean dressing of the superficial chest wounds. Because he was apparently hemodynamically stable and his symptoms improved, cardiac injury was not suspected, and he did not undergo noninvasive imaging or the measurement of cardiac biomarkers.

Patients with small traumatic VSDs (Qp/Qs <2:1) can be managed conservatively with regular monitoring and serial echocardiography. Small traumatic VSDs can remain hemodynamically stable for years and even close spontaneously over time.[11,12] Large traumatic VSDs (Qp/Qs >2:1) should be surgically closed to prevent congestive heart failure and pulmonary hypertension. The timing of surgery depends upon the patient's hemodynamic status. If the patient is hemodynamically stable, surgery can be delayed to allow the heart to recover from the contusion and develop fibrosis around the defect, enabling more secure suture placement.[13] Newly developed VSD occluder devices might offer alternative therapeutic option with significantly less morbidity and very low mortality rates.[14]

Conclusion

This case reaffirms the fact that substantial cardiac injury can develop months after chest trauma, and that suspicion coupled with thorough investigation—including physical examination and noninvasive testing—is necessary for detection and appropriate management. The possibility of a traumatic VSD should be considered in all multiple-trauma patients who develop a new murmur, even when overt chest-wall injury is absent. Early diagnosis enables the optimal management of these patients.

References

1. Mulder DG. Stab wound of the heart. Ann Surg 1964;160:287-91.
2. Karrel R, Shaffer MA, Franaszek JB. Emergency diagnosis, resuscitation, and treatment of acute penetrating cardiac trauma. Ann Emerg Med 1982;11(9):504-17.
3. Calvo Orbe L, Garcia Gallego F, Sobrino N, Sotillo J, Lopez-Sendon JL, Oliver J, et al. Acute myocardial infarction after blunt chest trauma in young people: need for prompt intervention. Cathet Cardiovasc Diagn 1991;24(3):182-5.
4. Chun JH, Lee SC, Gwon HC, Lee SH, Hong KP, Seo JD, Lee WR. Left main coronary artery dissection after blunt chest trauma presented as acute anterior myocardial infarction: assessment by intravascular ultrasound: a case report. J Korean Med Sci 1998;13(3):325-7.
5. Atalar E, Acil T, Aytemir K, Ozer N, Ovunc K, Aksoyek S, et al. Acute anterior myocardial infarction following a mild nonpenetrating chest trauma--a case report. Angiology 2001;52(4):279-82.
6. Sinha AK, Agrawal RK, Singh A, Kumar R, Kumar S, Sinha A, et al. Acute myocardial infarction due to blunt chest trauma. Indian Heart J 2002;54(6):713-4.
7. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958;18(3):371-96.
8. Stewart JS. Primary traumatic coronary thrombosis. Br Med J 1967;1(5542):739-40.
9. Marcum JL, Booth DC, Sapin PM. Acute myocardial infarction caused by blunt chest trauma: successful treatment by direct coronary angioplasty. Am Heart J 1996;132(6):1275-7.
10. Genoni M, Jenni R, Turina M. Traumatic ventricular septal defect. Heart 1997;78(3):316-8.
11. Pirzada FA, McDowell JW, Cohen EM, Saini VK, Berger RL. Traumatic ventricular septal defect. Sequential hemodynamic observations. N Engl J Med 1974;291(17):892-5.
12. Ilia R, Goldfarb B, Wanderman KL, Gueron M. Spontaneous closure of a traumatic ventricular septal defect after blunt trauma documented by serial echocardiography. J Am Soc Echocardiogr 1992;5(2):203-5.

13. Cowgill LD, Campbell DN, Clarke DR, Hammermeister K, Groves BM, Woelfel GF. Ventricular septal defect due to nonpenetrating chest trauma: use of the intra-aortic balloon pump. J Trauma 1987;27(9):1087-90.
14. Hijazi ZM. Device closure of ventricular septal defects. Catheter Cardiovasc Interv 2003;60(1):107-14.