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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Isolated Ventricular Septal Rupture in a Suicide Jumper

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ABSTRACT

Ventricular septal rupture is an extremely rare sequelae of blunt chest trauma, and is mostly diagnosed postmortem. We present a case of a large isolated traumatic ventricular septal rupture after a suicide attempt by jumping from a height of 5 stories, which was successfully treated with surgical closure. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:1531–1534) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 29-year-old man was brought into our facility after a suicide attempt by jumping from a height of 5 stories. On arrival to the emergency department, the patient had a fluctuating level of consciousness, blood pressure of 60/30 mm Hg, and heart rate of 135 beats/min. Given his multiple injuries and hemodynamic instability, the patient was intubated for further investigation and management.

LEARNING OBJECTIVES

- To be able to suspect and diagnose VSRs in patients with closed chest trauma.
- To be able to decide the optimal timing of repair for traumatic VSRs.
- To recognize the importance of a multidisciplinary approach when managing acute VSRs.

PAST MEDICAL HISTORY

The patient had a history of chronic paranoid schizophrenia and polysubstance abuse. He had a screening echocardiogram performed 4 years previously prior to clozapine initiation, which showed normal biventricular structure and function, and no evidence of intracardiac shunting.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnosis included tension pneumothorax, massive hemothorax, thoracic aortic dissection, cardiac tamponade, and cardiac rupture.

INVESTIGATIONS

A trauma series computed tomography scan revealed numerous injuries, including multiple fractures, small bilateral pneumothoraxes, and hemoperitoneum with lacerations to the liver, spleen, and

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ABBREVIATIONS AND ACRONYMS

TTE = transthoracic echocardiography

VSR = ventricular septal rupture

bladder. An electrocardiogram showed sinus tachycardia with no evidence of Q waves or ST-segment abnormalities (Figure 1). The patient's initial troponin level was significantly elevated at 12,500 ng/L (normal <26 ng/L). Bedside transthoracic echocardiography (TTE) was performed after a harsh systolic murmur was detected, which revealed a large ventricular septal rupture (VSR) measuring 1.2 cm with left-to-right shunt, located at the muscular portion of the posterior interventricular septum (Videos 1 and 2). The left and right ventricles were of normal size and function, which indicates that the VSR was likely acute and not congenital in origin.

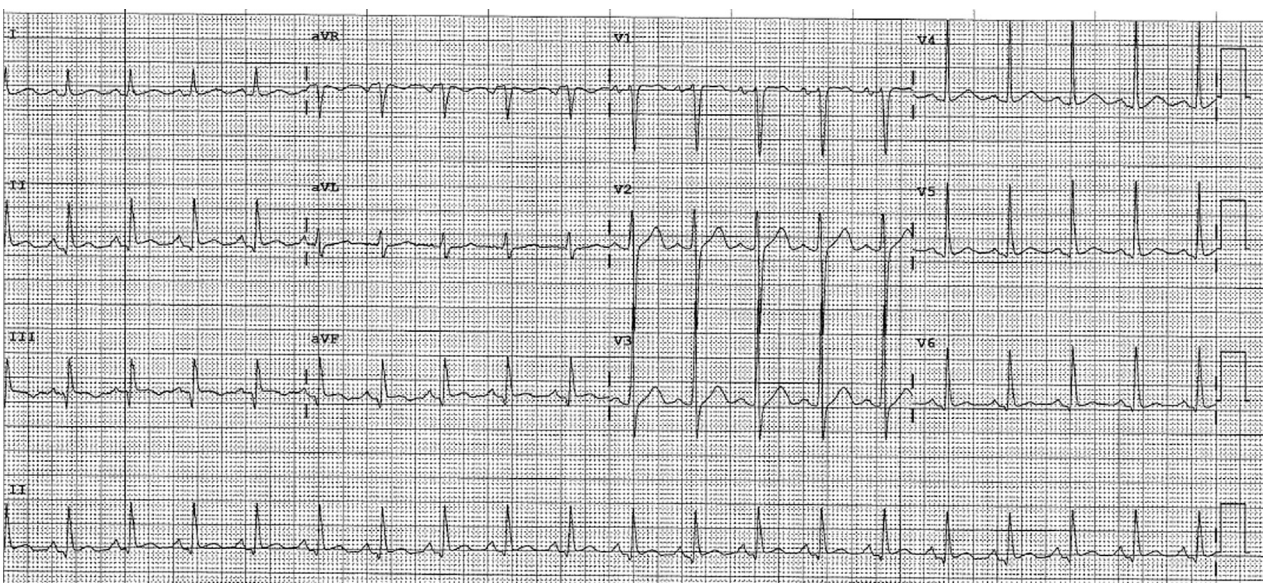
MANAGEMENT

After multidisciplinary discussion with the cardiology, cardiothoracic, and trauma surgical teams, the consensus was that VSR closure should be delayed given the patient's multiple injuries and hemodynamic instability from ongoing intra-abdominal blood loss. The patient underwent 3 emergency surgeries for repair of visceral organs and stabilization of pelvic fractures, and was subsequently admitted to the intensive care unit. Postoperatively, the patient remained hemodynamically stable with progressive reduction of inotrope and ventilator oxygen requirements. Extubation was uneventful on day 6 of admission, and the patient remained in hospital for ongoing recovery and physical rehabilitation. The patient also received daily psychiatric review, with gradual improvement in his mood and mental state throughout his inpatient stay.

A TTE repeated on day 32 of hospitalization for surveillance showed a persistent VSR, and a shunt calculation revealed a pulmonary-to-systemic flow ratio of 3:8:1. There was severe pulmonary hypertension, and the right ventricle was now moderately dilated with severely impaired systolic function (Video 3). After further multidisciplinary evaluation, a decision was made for surgical closure of the VSR, given a pulmonary-to-systemic flow ratio of more than 2:1 and progressive right ventricular dysfunction.

The patient underwent surgery on day 47 of hospitalization. A transesophageal echocardiogram performed intraoperatively demonstrated the large posterior VSR with left-to-right shunt (Video 4). Surgical repair was performed via median sternotomy, and closure with a Dacron patch (Invista). Intraoperatively, the VSR appeared trabeculated with fibrosis of the edges. Postoperative transesophageal echocardiography showed successful closure of the VSR with small residual left-to-right shunt only

FIGURE 1 Presenting Electrocardiography



Sinus tachycardia with no signs of ischemia.

(Videos 5 and 6), and the patient came off cardiopulmonary bypass with no complications.

DISCUSSION

To our knowledge, we are the first to report a VSR resulting from a suicide attempt by jumping, which was subsequently successfully treated with surgical closure. Blunt chest trauma has been reported to cause serious cardiac injuries, but VSR remains extremely rare. This may be due to nonsurvivable trauma, or small, clinically insignificant, ventricular septal defects that remain undiagnosed (1). A study conducted by Parmley et al (1) found that of the 5,467 cases examined following nonpenetrating traumatic injury of the chest, only 5 cases had evidence of isolated ventricular septal rupture.

Traumatic VSRs more commonly occur at the muscular interventricular septum near the cardiac apex (2) but can also occur de novo at the mid septum, as illustrated in our case. The mechanism of VSR after blunt chest trauma remains unclear, but it is hypothesized that the heart is mechanically compressed between the sternum and spine, causing an increase in intrathoracic pressure at end-diastole or early systole, resulting in interventricular septal rupture (3). Other possible causes include traumatic damage to coronary arteries, although defects owing to myocardial necrosis usually happen between 2 and 6 days after the initial trauma (4). Given the presence of the defect on day 1 of presentation and the absence of any ischemic changes on our patient's electrocardiogram, the most likely cause of the VSR is an increase in intracardiac pressure with compression of the heart on impact.

Patients with VSRs present in a wide variety of clinical states, with some being completely asymptomatic, while others are in clear hemodynamic instability. Clinical examination may reveal a harsh systolic murmur, although this may present late. Although many polytrauma patients often have nonspecific elevations in cardiac troponin levels (5), extremely low troponin levels are highly sensitive in excluding significant VSRs, with very high levels increasing the suspicion of significant myocardial damage. TTE remains the diagnostic tool of choice for VSR. If diagnosis remains unclear, cardiac catheterization with an oximetry run remains gold standard in identifying small or complex defects (3).

Currently, there are no clear guidelines for the treatment of traumatic VSRs, and therefore, a multidisciplinary approach is key. Conservative management is recommended in small, asymptomatic, muscular VSRs, as these defects often close spontaneously (6). Closure of the VSR via surgical or percutaneous means is considered if the defect is large, as a shunt measuring more than 2 cm is reported to be associated with a mortality rate of close to 71% (7). The timing of VSR closure often depends on the hemodynamic status of the patient and the size of the defect, although emergent closure increases the risk of device or patch failure as the myocardium is often friable soon after rupture (8). Therefore, in patients who can be stabilized, closure of the shunt should be delayed to allow for a fibrous ring to form around the shunt to increase the chances of a successful repair (3).

FOLLOW-UP

A follow-up TTE performed on postoperative day 6 showed an intact ventricular septal patch with 2 small residual left-to-right shunts. The right ventricular systolic function had improved when compared with his preoperative TTE. The patient was subsequently discharged home on postoperative day 12 in a stable condition, with ongoing outpatient medical and psychiatric follow-up.

CONCLUSIONS

VSRs remain an extremely rare complication of blunt chest trauma. It should be considered in patients following blunt trauma with significantly elevated troponin, cardiac arrhythmia, or hemodynamic instability with no other obvious cause. Bedside TTE is a quick and useful tool to help diagnose VSR and exclude any other cardiac sequelae of trauma. As in the case of our patient, VSR repair should be delayed if possible to increase the chances of successful closure, but this will depend on the patient's hemodynamic status.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS cardiac surgery, echocardiography, trauma, ventricular septal rupture

APPENDIX For supplemental videos, please see the online version of this paper.