

A case of isolated ventricular septal rupture caused by blunt chest trauma

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Abstract: Isolated traumatic rupture of the ventricular septum following blunt chest trauma is rare. We report the case of a patient who sustained blunt chest trauma in a motor vehicle collision. The isolated ventricular septal defect was not discovered by echocardiography until 12 days after accident. Perventricular closure of the defect was performed with occluder. The patient died from a complication of pulmonary aspergillosis. Autopsy findings supported the diagnosis of traumatic rupture of ventricular septum and substantiated a relation between ventricular septum rupture and the accident.

Key Words: ventricular septal rupture, blunt chest trauma, perventricular closure, autopsy.

Isolated traumatic ventricular septal rupture (VSR) is a rare complication of blunt chest trauma. We present an autopsy case in which an isolated VSR was caused by a blunt force impact to the chest during a road traffic accident. Clinically, the discovery of the ventricular septal defect was delayed and closed with occluder using the perventricular technique. The traumatic VSR was diagnosed and attributed to the accident by autopsy.

CASE REPORT

A 20-year-old man sustained blunt chest trauma in an unrestrained motor vehicle collision with a parked truck. The patient was admitted to the local hospital with a complaint of chest and left wrist pain. His initial vital signs and room air oxygen saturation were normal. The computed tomography scan of the chest showed no sternum and rib fracture, lung contusion, hemothorax or pneumothorax. The diagnosis was soft tissue contusion on the chest area. The X-ray examination showed Colle's fracture on the distal end of the left radius. With the orthopedic injury treated, the patient was monitored uneventfully for 5 days and discharged. Twelve days after

discharge, the patient came to a tertiary-care center with exertional dyspnea and tachypnea but no chest pain.

Complete trauma assessment revealed a left pulmonary contusion and small amount of fluid in left pleural cavity. No cardiac murmur was heard on auscultation. Cardiac enzymes were slightly elevated, with a peak troponin I of 152.8 ng/L. Transthoracic echocardiography revealed two ventricular septal defects at the inferior septum with a systemic-to-pulmonary blood flow ($V_{max}=3.06$ m/s, $PG=37$ mmHg)(Fig. 1). The defects were measured 1.3 cm and 0.6 cm in diameter separately. Moreover, the tricuspid regurgitation was observed. The closure of the muscular ventricular septal defect was performed using the perventricular technique. Tracheostomy was performed and ventilator was placed before the surgery. After a median sternotomy, the sheath was placed through the free wall of the right ventricle. Two occluders were implanted under transesophageal echocardiographic visualization with the heart beating. Additional diagnostic information was obtained by the intraoperative transesophageal echocardiography examination. Two chordae tendineae that attached to tricuspid septal leaflet ruptured. Sixteen days after the procedure of defect closure, the patient died from complication

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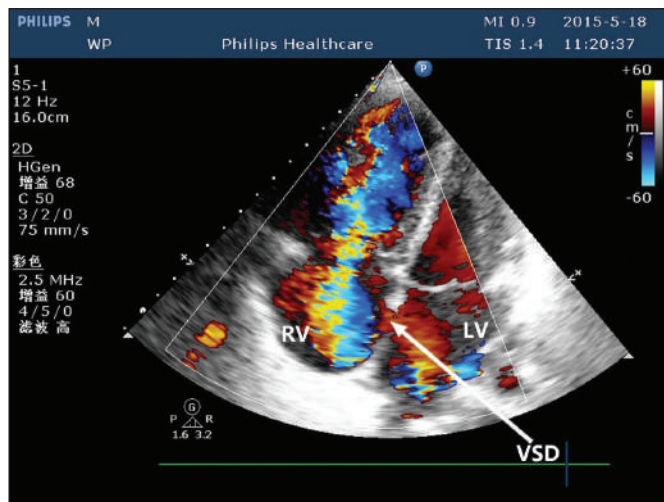


Figure 1. The transthoracic echocardiogram showed ventricular septal defect (arrow) with left-to-right flow by color Doppler imaging. LV, left ventricle; RV, right ventricle.

of pulmonary infection in the intensive care unit.

Necropsy and diagnostic work-up were carried out at the department of forensic pathology, Sichuan University. The body presented a mild to moderate state of post-mortem decomposition. At autopsy, a median sternotomy was observed. The surface of the heart appeared roughened from the normal glistening appearance by the thin strands of fibrinous exudate. There were two leads entering the right ventricle through the anterior wall. The leads connected with two ventricular septal defect occluders separately (Fig.2A-2B). Two defects, closed by the occluders, were located at the mid and apical portion of the muscular ventricular septum. The occluders, made of nitinol wire, had two concentric discs and a wide waist that accommodated the thickness of muscular portion of the ventricular septum. The disc and waist diameters of the smaller occluder were 1.7cm and 1.2cm, and those of the bigger occluder were 2.9cm and 2.4cm. There was a small thrombus adhered on the occluders. The discs adhered to septum loosely and could be detached easily. After removing the occluders, the defects showed round appearance measuring 0.8cm and 1.9cm in diameter separately (Fig.2C). The edge of ruptured septum was clean-cut and deformed by the compression of occluders. One chordae tendineae of the cusps medialis ruptured and the other one elongated (Fig.2D). Histopathology examination revealed the necrosis of myocardial fiber and formation of granulation tissue on the wall of ruptured ventricular septum. There were small amounts of acute inflammatory cells, macrophages along with numerous collagenization and little capillaries in the granulation tissue (Fig.2E).

Cut surface of the lungs demonstrated patchy areas of tan-yellow consolidation (Fig.3A). On microscopy, there was extensive neutrophilic infiltrate in the consolidating area of lung. The alveolar walls were not clearly seen. The fungus ball composed of blue-

staining hyphal elements of *Aspergillus* was seen amidst the purulent exudate and some hemorrhage (Fig.3B-3C). The patient was diagnosed with pulmonary aspergillosis. The brain, liver, kidneys, stomach, intestines, spleen, adrenals, prostate and urinary bladder appeared unremarkable grossly and microscopically. The cause of death was opined as secondary pulmonary infection of *Aspergillus* following pulmonary and cardiac injury sustained due to blunt force impact to the chest from a road traffic accident.

DISCUSSION

Various cardiac injuries, including myocardial and pericardial contusion or rupture, rupture of valves, chordae tendineae or papillary muscles and septal perforation, may occur after blunt chest trauma [1, 2]. Isolated traumatic VSR is rare [3, 4]. Parmley et al. have reported 5 cases of isolated VSR out of 546 autopsy cases with non-penetrating traumatic injury of the heart. The cases with isolated traumatic VSR accounted for 0.002% of 207548 autopsy cases analyzed [5].

VSR occurring from blunt chest trauma is divided in two mechanisms. The first considers that the rupture occurs due to strong acute anteroposterior compression of the heart between the sternum and the thoracic vertebrae during late diastole and early systole when the atrioventricular valves are closed. Associated intracardiac injuries include damage of valves and the supportive structures (i.e. the papillary muscle or chordae), myocardial contusions, and coronary artery lesions. The second proposes that interruption of coronary blood flow within the ventricular septum secondary to a myocardial contusion causes myonecrosis and subsequently leads to a delayed VSR [6].

The mechanism of injury in the case described here could be explained by a forceful impact that resulted in an extreme change in intrathoracic pressure, heart compression, and excessive intraventricular pressure. The tension rupture and elongation of chordae were the evidences of the sharply increasing of intraventricular pressure during late diastole and early systole. Neither infarction on ventricular septum nor disease of coronary artery was observed in our case. The myocardial necrosis and organized granulation tissue on the wall of ruptured ventricular septum could be the result of the injury itself. Ventricular septal rupture secondary to trauma is commonly low in the septum. The most common location is at the muscular portion of the ventricular septum near the apex [7, 8]. There may be more than one defect [9]. In the case described here, there were two defects at the mid and apical portion of the muscular ventricular septum separately. Although appearance of defects changed under the compression of occluder, the edge of ruptured septum was clean-cut. On microscopy, the necrosis of myocardial fiber and the granulation

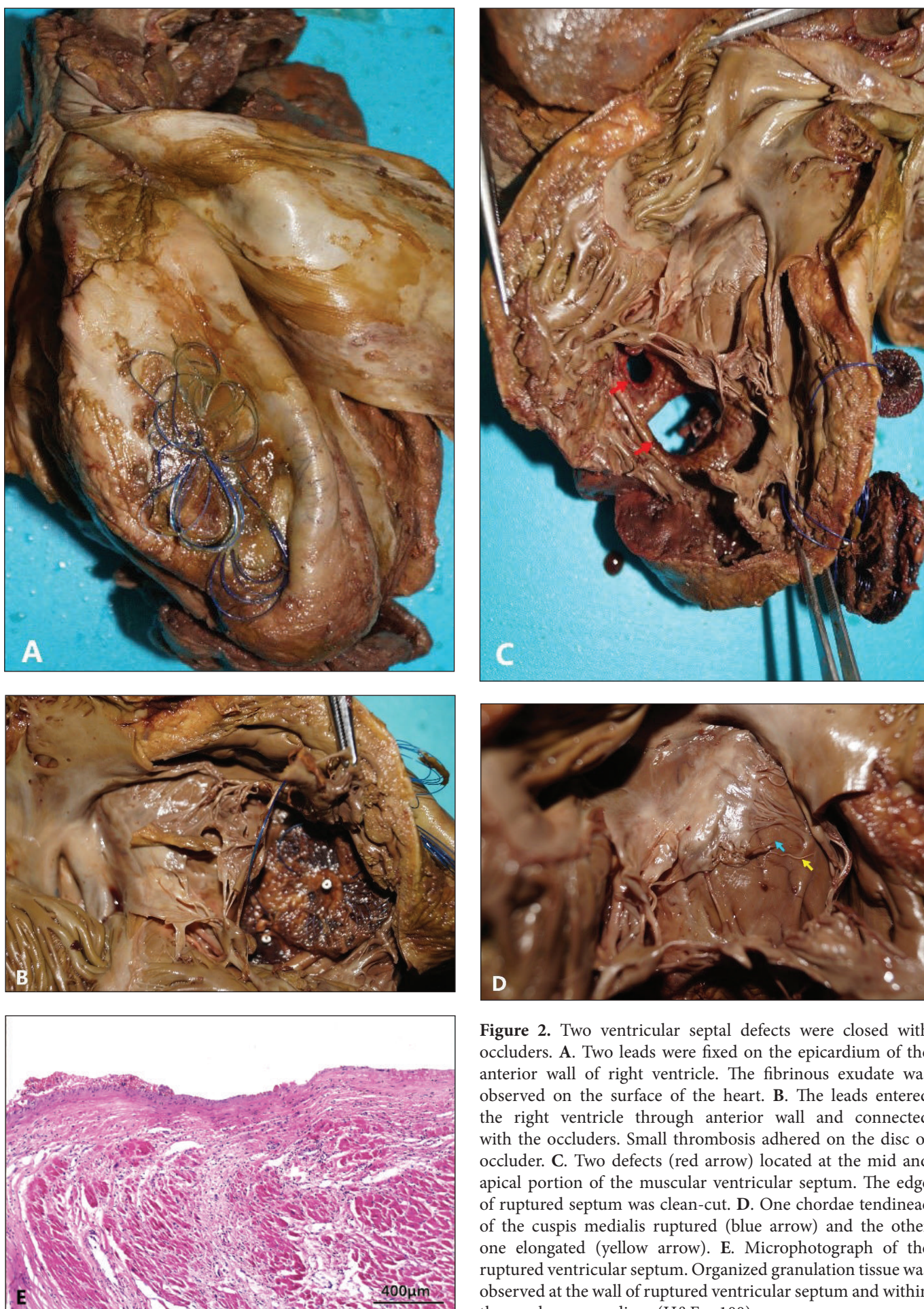


Figure 2. Two ventricular septal defects were closed with occluders. **A.** Two leads were fixed on the epicardium of the anterior wall of right ventricle. The fibrinous exudate was observed on the surface of the heart. **B.** The leads entered the right ventricle through anterior wall and connected with the occluders. Small thrombosis adhered on the disc of occluder. **C.** Two defects (red arrow) located at the mid and apical portion of the muscular ventricular septum. The edge of ruptured septum was clean-cut. **D.** One chordae tendineae of the cuspid medialis ruptured (blue arrow) and the other one elongated (yellow arrow). **E.** Microphotograph of the ruptured ventricular septum. Organized granulation tissue was observed at the wall of ruptured ventricular septum and within the nearby myocardium (H&E, $\times 100$).

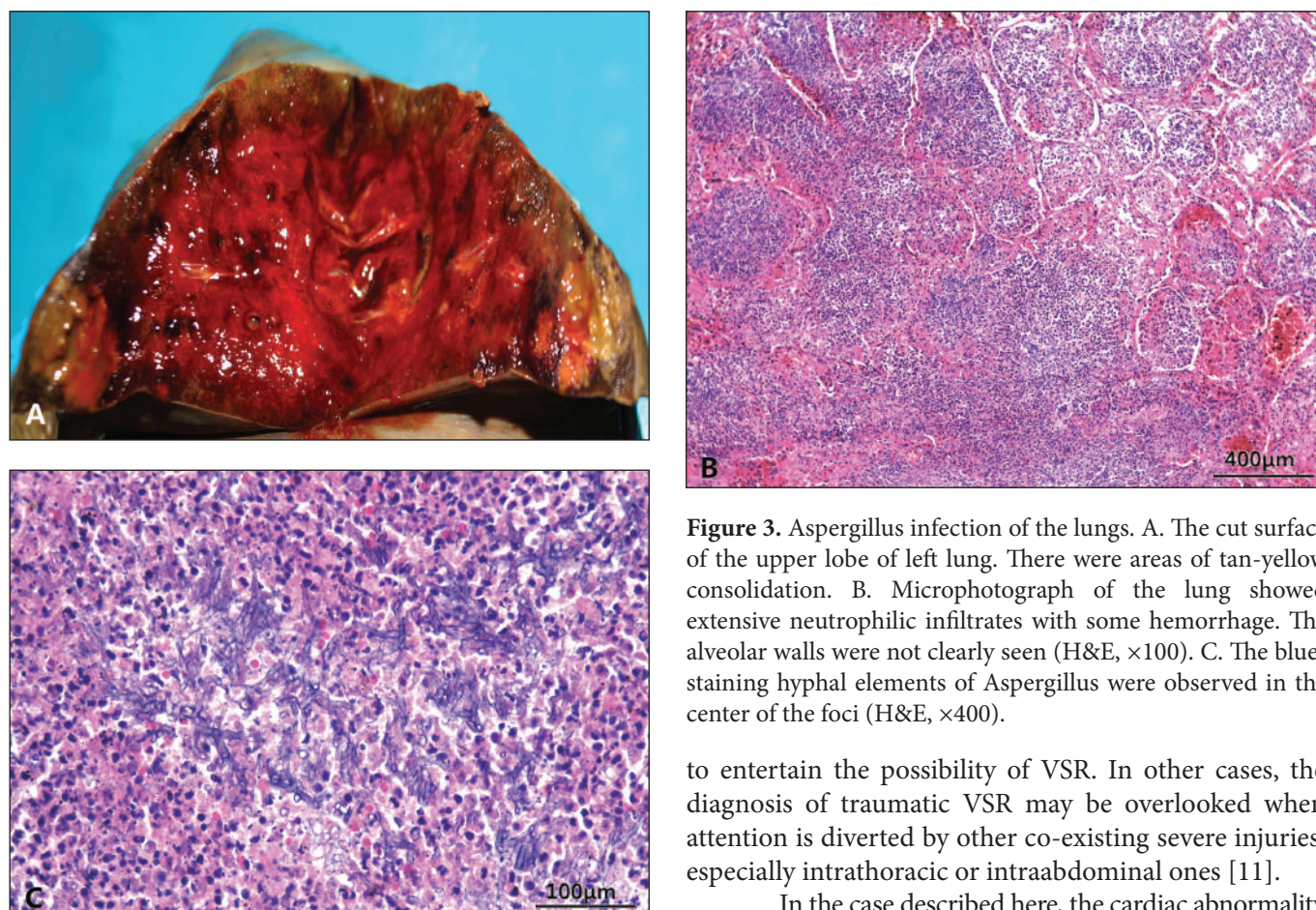


Figure 3. Aspergillus infection of the lungs. A. The cut surface of the upper lobe of left lung. There were areas of tan-yellow consolidation. B. Microphotograph of the lung showed extensive neutrophilic infiltrates with some hemorrhage. The alveolar walls were not clearly seen (H&E, $\times 100$). C. The blue-staining hyphal elements of Aspergillus were observed in the center of the foci (H&E, $\times 400$).

tissue were observed on the wall of ruptured ventricular septum. The findings of gross and microscopic inspection supported the traumatic origin of the ventricular septal defects rather than congenital.

Some traumatic VSR may be diagnosed in the acute setting, or the diagnosis may be delayed such as in this case. The delayed diagnosis of VSR in the case reported here could be explained as follows: firstly, the initial minute fistula could be enlarged over time [10]. Thus, the clinical symptoms could be unrecognised shortly after the accident but became overt days or weeks later. Secondly, the lack of overt chest trauma might lead to underestimation of the degree of injury and failure

to entertain the possibility of VSR. In other cases, the diagnosis of traumatic VSR may be overlooked when attention is diverted by other co-existing severe injuries, especially intrathoracic or intraabdominal ones [11].

In the case described here, the cardiac abnormality was linked to the accident because the mechanism of injury was matched with the accident and the findings of pathologic inspection of the VSR. Moreover, there was no other obvious cause for VSR. In other cases, a delayed VSR might have had other causes, especially if the chest trauma had occurred several months or years before and the patient had common cardiac problems. Early diagnosis is essential to substantiate a relation between VSR and chest trauma. Careful echocardiographic inspection using both 2-dimensional imaging and color Doppler imaging modalities enables an early diagnosis of traumatic VSR. Vigilance is required in any case of blunt trauma to the anterior chest wall to ensure that cardiac damage is not missed.

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