BLUNT CHEST TRAUMA PRESENTING WITH TRAUMATIC VENTRICULAR SEPTAL DEFECT AND DISRUPTED TRICUSPID VALVE; A CASE REPORT

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ABSTRACT

We present a case of blunt chest trauma in an eighteen year old Thai man. He presented with intra-cerebral hemorrhage and was unconscious on arrival at hospital. Emergency craniotomy with clot removal was done at a near by hospital in his own district. He was discharged after treatment. There was no mention of any other problems in the notes. Symptoms of cardiac failure presented a week after being discharged. The patient came to us with progressive dyspnea, orthopnea, pitting edema and ascites with pan systolic murmur at left lower parasternal border, 3 months after the incident. Echocardiogram and MRI showed muscular type VSD, most probably a traumatic rupture. The late presentation of the patient i.e. more than 3 months after injury and its successful surgical management is presented. Although traumatic VSD is a known complication after blunt chest trauma, in our experience and after review of the literature, this is a not too common occurrence.

INTRODUCTION

Traumatic ventricular septal defect (VSD) is a rare complication of blunt cardiac trauma. There are about 58 cases reported in the English literature since 1970. Traumatic VSD is variable in its presentation, temporal course and severity. It may require emergent repair or be repaired electively. Blunt chest trauma can cause a wide spectrum of cardiac injuries ranging from mild myocardial contusion to disruption of the atrioventricular valves or interventricular septum, to free wall rupture. Serious cardiac injury most commonly results from motor vehicle accidents with steering wheel impact but can occur after any direct blow to the chest (e.g., during sporting activities). Lillehei first repaired a traumatic VSD using a pump and a dog lung as the oxygenator in 1955. A few cases have been reported since then. Of note, in most reported cases is the fact that traumatic VSDs present immediately or within several hours of the traumatic event. Delayed presentation is rare. We report here a case of a traumatic muscular VSD that presented with heart failure 3 months after the accident.

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CASE REPORT

An 18-year old boy, involved in body assault, suffered severe head injury and blunt chest trauma.

He presented with intracerebral hemorrhage and was unconscious on arrival at his district hospital. Emergency craniotomy with clot removal was done, and he recovered satisfactorily. There was no mention of an abnormal heart murmur while he was admitted. Symptoms of cardiac failure presented a week later after being discharged. Progressive dyspnea, orthopnea, pitting edema and ascites got worse and he was transferred to Rajavithi Hospital 3 months after the incident with a presumed diagnosis of right sided heart failure.

Physical examination upon arrival at our center revealed that the patient had a pansystolic murmur at the left lower parasternal border, which did not radiate to the neck or axilla, with an associated thrill. The patient's father confirmed that he had a childhood murmur that he had outgrown.

- Chest X-ray revealed cardiomegaly, right atrium and right ventricle enlargement and pericardial effusion.
- Electrocardiogram (ECG) revealed sinus tachycardia with a rate of 110 beats/

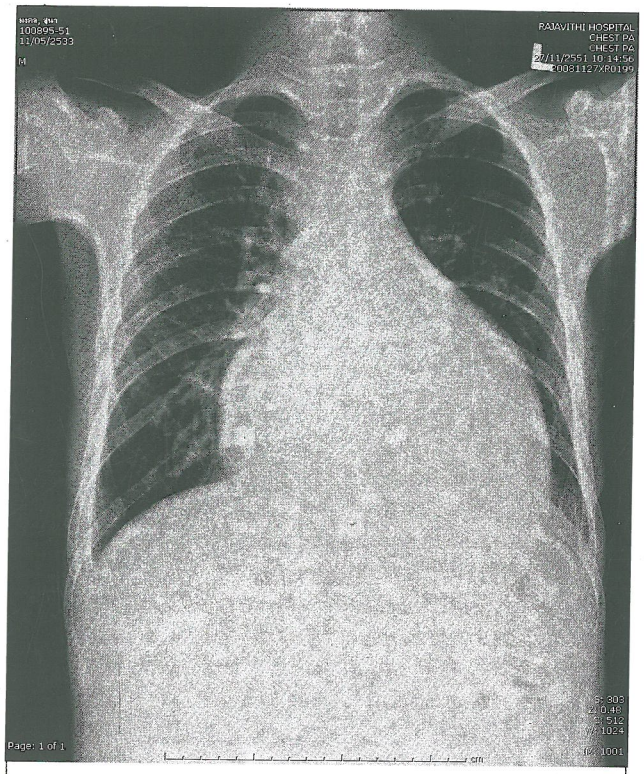

Fig 1.
cardiomegaly, right atrium and right ventricle enlargement and pericardial effusion.

minute, right axis deviation and nonspecific ST elevation.

- Echocardiogram revealed RA & RV chamber enlargement. Aneurysm at mid septal level extending to the inferior septum. Rupture of the aneurysm to RV chamber (Muscular VSD); size 1.7-2.5 cm. Left to right shunt was present. There was severe TR with flail anterior & septal leaflets and massive pericardial effusion. No RV collapse.
- MRI of the heart revealed a large muscular VSD, extending from the anterior to the posterior aspect of the interventricular septum. The VSD was covered by a wide necked false aneurysm at the interventricular septum, with bulging to the right ventricular cavity. Mild TR. Large amount of pericardial effusion without evidence of cardiac tamponade. Moderate amount of right pleural effusion and ascites were also present.

The patient was referred to the cardiovascular thoracic department for an emergency operation. At operation, the heart was markedly enlarged and pulmonary pressure was high. There was about a liter of bloody pericardial effusion around the heart. Large muscular VSD, size 2-3 cm with a fibrous neck, extending from anterior to mid interventricular septum was seen. False aneurysm at the interventricular septum was found covering the VSD. There was rupture of multiple chordae of anterior and septal leaflets of the tricuspid valve with severe TR. Right pleural effusion (clear yellow fluid), amount 1000 cc and ascites, amount 2000 cc was also present.

With cardiopulmonary bypass support, bicaval cannulation, and cardioplegic arrest, right atriotomy was performed and the large muscular VSD was visualized (Fig 4, 5, 6). The surrounding necrosed septum was excised. The margins of the defect were closed with interrupted sutures and a PTFE patch, and TV repair (2 Chordal replacements with PTFE sutures, and annuloplasty ring) was performed. Post op the patient made an uneventful recovery with return of heart size to normal, no residual VSD, and good LV systolic function on follow-up echocardiograms.

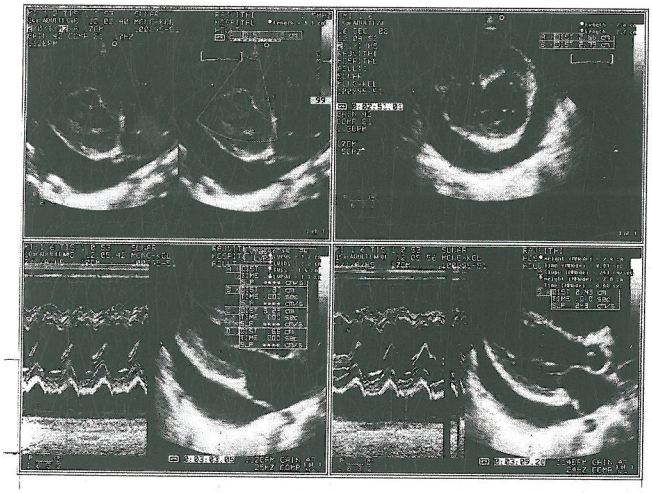

Fig 2.
RA&RV chambers are enlarged Aneurysm at mid septal wall extending to inferoseptal part. Rupture of aneurysm to RV chamber (Muscular VSD) size 1.7-2.5 cm. Left to right shunt present. Aortic valve has three cusps, no AS. Mitral valve shows normal coaptation. PV is normal. Pulmonary artery mildly enlarged. Severe TR. With Flail anterior & septal leaflets.Massive pericardial effusion. No RV collapse.

DISCUSSION

The most common form of blunt cardiac injury is cardiac free wall rupture, which often results in death before help can be delivered. The most common area of cardiac rupture is the right ventricular free wall, but most survivors have injuries to the right or left atria.

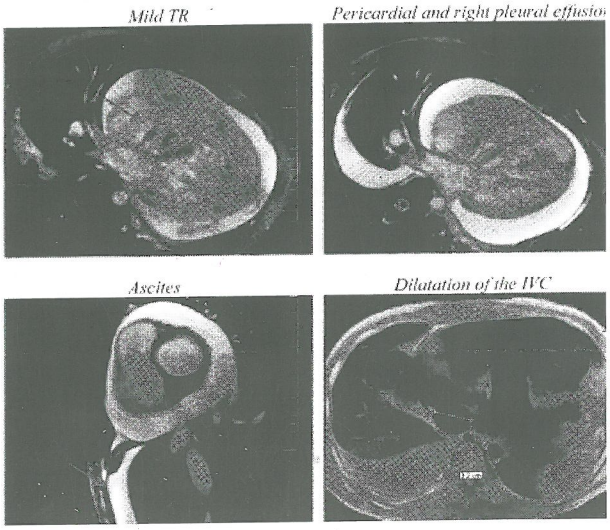

Fig 3. Large muscular VSD, extending through the whole (thickness), anterior to posterior aspect of interventricular septum. Coronary MRA reveals no high grade stenosis of the LMA and LAD. VSD covered by wide necked false aneurysm at the interventricular septum, with bulging to the right ventricular cavity. Mild TR. Large amount of pericardial effusion without evidence of cardiac tamponade. Moderate amount of right pleural effusion and ascites.

Mechanism of traumatic VSD: With an anteroposterior compression of the heart against closing AV valves, traumatic VSD occurs most commonly in the muscular portion of the interventricular septum near the cardiac apex.

Other Mechanisms are where an indirect or hydraulic force resulting from a crush injury to the abdomen or lower extremities acts upon the heart, acceleration/deceleration forces, blast forces and penetration of a cardiac chamber by a fractured rib or sternum are all known to cause cardiac injuries.

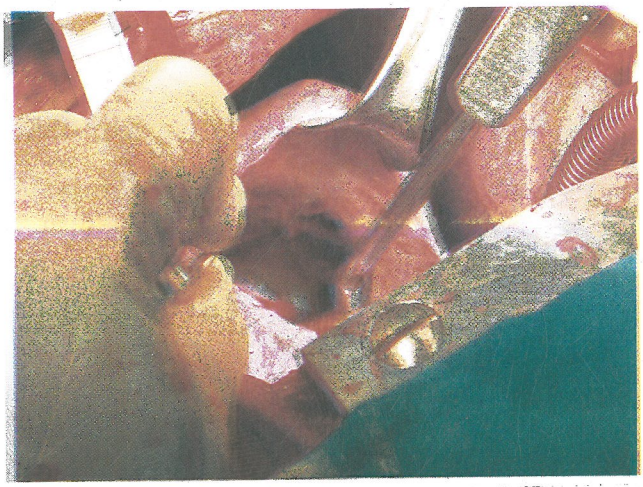

Fig.4

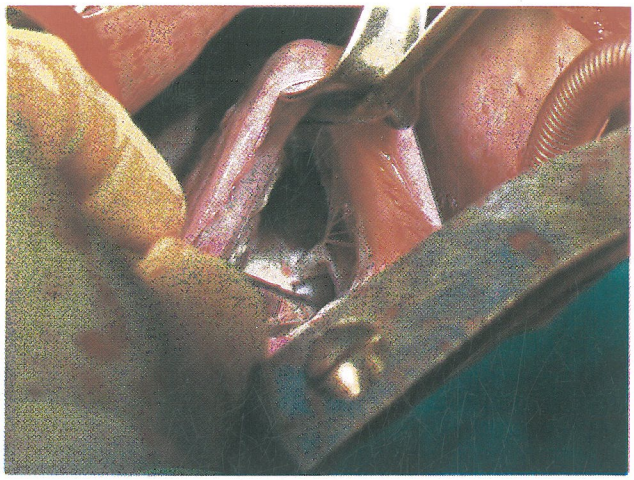

Fig.5

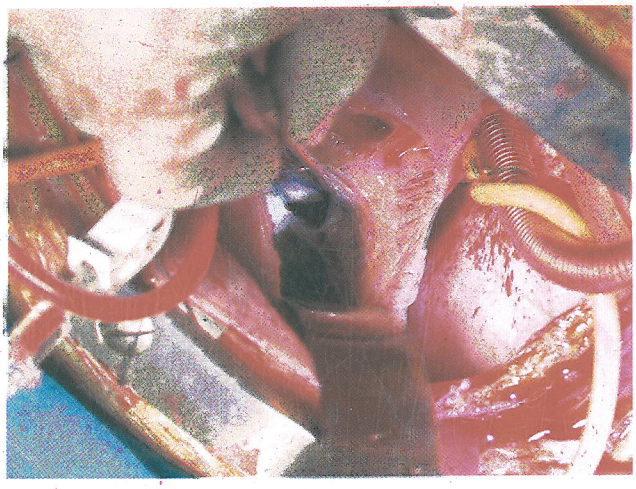

Fig.6

Onset of ventricular wall rupture is either immediately at the time of injury, or delayed for several days, when it occurs secondarily to myocardial devascularization in areas of myocardial contusion, with subsequent necrosis and perforation (Similar to our case). The highest risk of rupture from myocardial necrosis is between 2 - 6 days post injury.

Clinical presentation ranges from the asymptomatic to uncompensated hemodynamic shock.

The following signs may be present or not. (On auscultation: harsh PSM in the 3rd, 4th or 5th ICS at the left sternal border with an associated precordial thrill, Lung: pulmonary edema with defects larger than 2 cm, jugular venous distention may become apparent, CXR: 1/3 are normal, but may demonstrate pulmonary edema or cardiomegaly, EKG: often abnormal with nonspecific findings; ta-

chycardia, Q waves, ST-segment or T-wave changes, conduction abnormalities and complete heart block).

Diagnostic tools; echocardiography is the most effective screening tool along with cardiac catheterization which remains the "gold standard". It is recommended that both right and left ventriculography as well as coronary arteriography be performed.

Mattox et al. recommended that cardiac catheterization be performed in those patients with abnormalities on echocardiography post trauma, or with a missile overlying the cardiac silhouette on chest radiography.

Associated injuries: Intrathoracic (diaphragmatic rupture, aortic disruption, and pulmonary vein injuries), cardiac (myocardial contusion, mitral, tricuspid, aortic valve injuries, ventricular aneurysms, coronary artery injuries, pericardial injury, and acute myocardial infarction), perimembranous rupture associated with tricuspid valve injury and multiple traumatic defects of the septum, may exist simultaneously.

Prognosis is related to the size of the defect and concomitant injuries (25% mortality rate if VSDs <2 cm, 71% mortality rate if VSDs >2 cm in size), CHF is present often with defects larger than 1 cm. There is a 56% mortality rate within 15 days of injury, secondary to CHF, in those patients managed nonoperatively and 10% mortality rate in those undergoing surgical repair.

Cases of traumatic VSD since 1970: There were 11 deaths caused by VSD (19%) (4 had the VSD discovered at autopsy, 5 underwent emergency surgery, while 2 died after delayed surgical repair of the VSD).

Mechanisms of injury were vehicular crashes (41.4%), crush injuries and falls (25.9%), abuse and assaults (8.6%), sport and motorcycle crashes (6.9% each), automobile-pedestrian collisions (3.4%) and other various blunt mechanisms (6.9%).

Pathology: Isolated VSDs constituted 67.2%, while concomitant cardiac injuries 32.8% of the patients. Male patients accounted for 86.2%. Average age of the patients was 21.4 years (range: 6 months 75 years). In only one case cardiac catheterization was used as the primary diagnostic modality, whereas 85% used echocardiography. The remaining cases were diagnosed at autopsy.

Patients not receiving operative repair were 7 of 58 (12.1%), (4 of these patients died as a result of their injuries and the VSD was discovered at autopsy while 3 survived) **In 10 of 51 patients (19.6%) that underwent surgery immediately, there was a** 50% mortality rate. The deaths occurred with defects >2 cm, or multiple cardiac injuries. Survivors usually had isolated VSDs, with an average size of 1.9cm.

In the 51 patients managed surgically, 29 (57.0%) underwent surgical repair within the first 4 weeks post injury, 11 (21.5%) between 4 and 8 weeks, 11 (21.5%) greater than 8 weeks after injury. Mortality rates for these subsets of patients were 21%, 10%, and 9%, respectively.

Conclusion

A High index of suspicion remains the cornerstone for the diagnosis of VSD after blunt trauma. Nonoperative management is reasonable in the hemodynamically stable patient with a small septal defect. Larger VSDs, or those with a compromised hemodynamic profile, may benefit from early operative repair.

Timing of surgery : Many authors recommend delayed surgical intervention if the patient is stable hemodynamically. Modern techniques, including the intra-aortic balloon pump, and patch grafts for the septum may allow for earlier repair, **while** echocardiography provides a reliable, rapid, noninvasive diagnostic tool for blunt traumatic VSD.

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