Intra ventricular septal haematoma and acquired ventricular septal defect following blunt chest trauma.

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Abstract

Septal haematoma and subsequent septal rupture resulting in acquired ventricular septal defect (VSD) is rarely reported as a complication following blunt traumatic chest injury. This case report illustrates the diagnostic approach and management challenge of a septal haematoma and septal rupture that occurred in a young adult. A 17 year old male was admitted to a surgical ward following motor bicycle accident. He was admitted with cardiogenic shock to the emergency department and found to have multiple rib fractures, bilateral pleural effusion and pulmonary contusions. On the fourth day of the intensive care unit stay, a new grade III pan-systolic murmur was found at the left lower sternal edge. ECG showed sinus tachycardia with ST segment elevation in V2 to V6. Blood analysis showed cardiac troponin I value of > 70 mg/L and creatine phosphokinase concentration of 5162 mg/L. Echocardiography showed a hypo-echoic collection with a cavity in the interventricular septum (IVS) and an apically located VSD. The patient had emergency cardiac surgery for septal repair and closure of VSD. He had a rapid recovery following the surgery and postoperative echocardiography showed a small residual collection within the IVS and a residual VSD. Patient was subsequently follow-up with serial echocardiography and found to have gradual expansion of the residual collection. On the 5th week following the septal repair, his cardiac MRI demonstrated an IVS dissection with a cavity that communicated with the left ventricular cavity and a residual small VSD. Conservative management was done for the residual defects and the 18 month follow-up showed no further progression of the size of the IVS dissection and the severity of the residual VSD.

Introduction

Violent blunt chest trauma is reported to cause a wide range of cardiovascular injuries depending on its mechanism [1]. Septal haematoma and acquired ventricular septal defect (VSD) are rarely reported as complications following blunt chest trauma in survivors following such injuries [2]. It is natural to pay less attention and to concentrate on other more apparent injuries in such a patient [3]. However, early detection and timely planned appropriate intervention are extremely important for the survival of these patients with significant cardiac trauma. Therefore, high degree of clinical suspicion and careful evaluation of physical signs are of utmost importance even in the emergency setting. Here we report a case of blunt chest trauma leading to a septal haematoma and subsequent septal rupture resulting in acquired VSD in a young adult.

Case presentation

A 17 year old male was admitted to a general surgical casualty ward following road traffic accident (RTA). The victim was the rider of a motor bicycle and he had a head on collision with a heavy vehicle and subsequently sustained chest trauma due to the impact of the bicycle handlebar. He was admitted with cardiogenic shock to the emergency department. Initial clinical examination showed no external chest injuries but had clinical evidence of multiple rib fractures. Urgent chest X-ray confirmed the left sided multiple rib fractures, bilateral pleural effusions and pulmonary contusions.

He was intubated and resuscitated followed by massive transfusion protocol. Bilateral intercostal tube insertion was performed. There were no other significant traumatic injuries in the rest of the body and he became hemodynamically stable in the subsequent clinical course.

On the fourth day of the intensive care unit stay, he was found to have a new appearance of grade III pan-systolic murmur with the maximal intensity at the left lower sternal edge.

ECG showed sinus tachycardia with ST segment elevation in V2 to V6 (Figure 01). Blood analysis showed elevated cardiac enzymes: Cardiac troponin I was > 70 mg/L and creatine phosphokinase was 5162 mg/L suggestive of cardiac injury. Two dimensional echocardiography showed a hypo-echoic collection with a cavity in the interventricular septum (IVS) (Figure-02 a and b).

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In addition there was a VSD at the apical portion of the septum with left to right shunt (Figure-03). The VSD had a gradient of 68 mmHg and there was no cardiac chamber dilatation or evidence of pulmonary hypertension.

Only a thin collection of pericardial fluid was noted with no tamponade effect. All the cardiac valves and proximal aorta appeared normal.

The patient had emergency cardiac surgery with cardiopulmonary bypass. Left ventriculotomy was done, and during the exploration the hematoma was found over the posterior IVS.

There was a macerated area with very fragile muscle over the IVS just distal to the posterior medial papillary muscle. The rupture and the VSD were closed with a polytetrafluoroethylene patch. He had a rapid recovery following the surgery and postoperative echocardiography showed small residual collection within the IVS and a small residual VSD (Figure-04).

The patient was subsequently followed-up with serial 2D echocardiography and found to have gradual expansion of the residual collection but he remained asymptomatic. On the 5th week following the septal repair, he had a cardiac magnetic resonance imaging (MRI) for further evaluation of the residual collection and the VSD. The cardiac MRI showed a dissection in the intraventricular septum with a cavity within it. The cavity had a communication with the left ventricular cavity with a 13mm size opening (Figure-05 a and b). The residual VSD was located in the apical region with a left to right shunt.
Consultative discussion was made by the cardiologists and cardiothoracic surgeons and it was finally decided to manage the patient conservatively with close monitoring and serial echocardiography.

Warfarin was started with a target international normalized ratio (INR) of 2.5 to prevent in-situ thrombus formation in the opened cavity in the dissected plane of the IVS. The 18 month follow-up of this patient showed no further progression of the size of the IVS dissection and the severity of residual VSD.

Discussion

This case illustrates the complexity involved in making the diagnosis, possible complications and treatment outcome of traumatic VSD following blunt chest trauma. The diagnosis of such a complication needs a high index of clinical suspicion since there will be other obvious injuries that make this type of injury of secondary importance.

There can be several diagnostic modalities that can help the diagnosis such as ECG, cardiac enzyme markers, myocardial perfusion scans, echocardiography and cardiac MRI[5]. Interestingly, the clinical presentation of septal rupture can have an acute, subacute or late presentation depending on the degree of the damage and subsequent local tissue necrosis[6]. Rotman’s review[7] has shown the detection of cardiac murmur was the initial presentation in few cases, and in the majority it became obvious between 4th to 12th day, whereas multiple ruptures were an uncommon presentation. In our patient, the murmur appeared in the 4th day and the ECG also showed ST changes suggestive of possible associated myocardial damage.

The most common mechanism for ventricular septal rupture following blunt chest trauma is due to violent, high-velocity pressure transmission to the thorax causing acute rupture of the septum by shear stress, most frequently at its posterior insertion[8].
Other explanations emphasized the external compression of the heart during the late diastole or isovolumetric systole when the valves are closed, where there may be little or no relief of pressure, or a possible cause leading to either a laceration at the time of the trauma, or a contusion and subsequent necrosis [9]. The scenario described in our patient is also compatible with septal contusion followed by subsequent necrosis that led to ventricular septal rupture.

Most interestingly, though the internal cardiac injuries are serious, it is still possible to have minimal obvious external manifestations [10] as happened in our patient. The occurrence of these types of abnormalities after blunt chest trauma require careful cardiac evaluation in a periodic manner to identify the subsequent complications.

The 2D echocardiography plays one of the main roles in the diagnosis of myocardial contusions, septal hematomas and septal rupture [5]. The availability and the ability to use as a bed-side test in critically ill patients have made echocardiography an ideal modality in the diagnosis of such conditions. It can be used to identify segmental motion deficit which may be the early evidence of myocardial contusion in addition to estimating the extension of the lesion. It can provide important information on ventricular function and rule out potential complications such as pericardial effusions and free wall rupture [5] as well.

However, considering the technical difficulties such as pneumothorax, subcutaneous emphysema and pneumopericardium the transthoracic study may have limitations. Approximately in 20% of the cases[7] the transthoracic echocardiography may not provide adequate information for a definitive diagnosis. In addition to the transthoracic approach, the trans esophageal echocardiography is also helpful in the visualization of this type of lesion more accurately in the emergency setting where transthoracic echocardiography windows are sub-optimal.

Though nonspecific ECG abnormalities are common after cardiac trauma, the specificity for cardiac pathological entities is less. However, Tsikaderis et al. found that some ECG changes appeared to correlate well with the presence of VSD in the adult population (37/49 cases)[10].

The outcome of a patient with acquired traumatic VSD without surgery depends largely upon the defect size, the nature and the mechanism of the trauma and associated injuries[11]. The patient’s clinical course should guide the ideal time to close the acquired septal defect by weighing the risk and benefit of such surgery. Furthermore, not all patients diagnosed with acquired VSD need urgent interventional therapy. Certainly, a conservative approach is advocated in asymptomatic VSDs located in the muscular septum, where there is no pulmonary hypertension, ventricular dimensions remain normal and patient is stable haemodynamically [12]. Obviously, the patients who have small shunts and no signs of cardiac deterioration may be observed. However, spontaneous closure of traumatic VSDs are not to be anticipated in the majority of patients[11].

Carter et al [13] believe that a time period of four to eight weeks is adequate for the consideration of delayed surgery for these patients. However, emergency repair of this defect may be mandatory in some instances[14]. The macerated tissue segments that were found during the surgery (5th day of the injury)in the affected part of the IVS in our patient also made the surgery difficult and may predisposed to having a residual VSD and have persistent dissection flap in the IVS following surgical intervention.

Cardiac MRI that was performed in the post-operative period clearly elaborated the nature of the residual defect and the hemodynamic significance of the residual VSD aiding a clear decision regarding subsequent management of the patient.

Furthermore, this case illustrates the importance of a multidisciplinary approach when planning interventions for a complex scenario and the place of post-operative re-assessment. In addition it is of paramount importance to ensure vigilant long-term follow-up of these cases as well.

Conclusion

Traumatic septal contusion leading to acquired VSD is a rare complication of traumatic chest injury. Therefore, there should be a high degree of clinical suspicion to diagnose serious cardiac injuries in this category of patients.
As in this case, some complications may present in the sub-acute period depending on the nature of the insult to cardiac tissues. Hence unceasing clinical vigilance, timely use of echocardiography and cardiac MRI may facilitate an accurate diagnosis and assist in making important management decisions.

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