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CASE REPORT

Traumatic ventricular septal defect

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Abstract

A 26 year old man was admitted to hospital following a traffic accident. He had been sitting in the back of a car without wearing a seat belt. He suffered crush injuries on the anterior chest wall, trunk, and legs. On admission he was awake and cooperative, but restless, and obviously in severe pain. Radiography of the skull, facial bones, chest, spine, pelvis, and legs revealed a shaft fracture of the left femur and tibia and fracture of the 7th and 8th right ribs. The patient was transferred to the University Hospital of Zurich for further assessment and surgical repair of the lower limb fractures three days later. Because of worsening clinical condition with onset of partial respiratory insufficiency and new loud systolic murmur at the left sternal edge, a transthoracic echocardiography was performed, which showed an apical ventricular septal defect. Surgery was performed immediately. The ventricular septal defect was successfully repaired using a Teflon felt patch and interrupted sutures with pledgets, and sealed with glue. At six months' follow up the patient was doing well. Ventricular septal defects after blunt chest trauma occur either because of heart compression between sternum and the spine or because of myocardial infarction. In the present case the ventricular septal defect appeared three days after the accident, probably secondary to a post-traumatic myocardial infarction. Patients with blunt chest trauma and suspicion of cardiac contusion should be monitored carefully.

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Keywords: ventricular septal defect; blunt chest trauma; cardiac injuries; acute myocardial infarction

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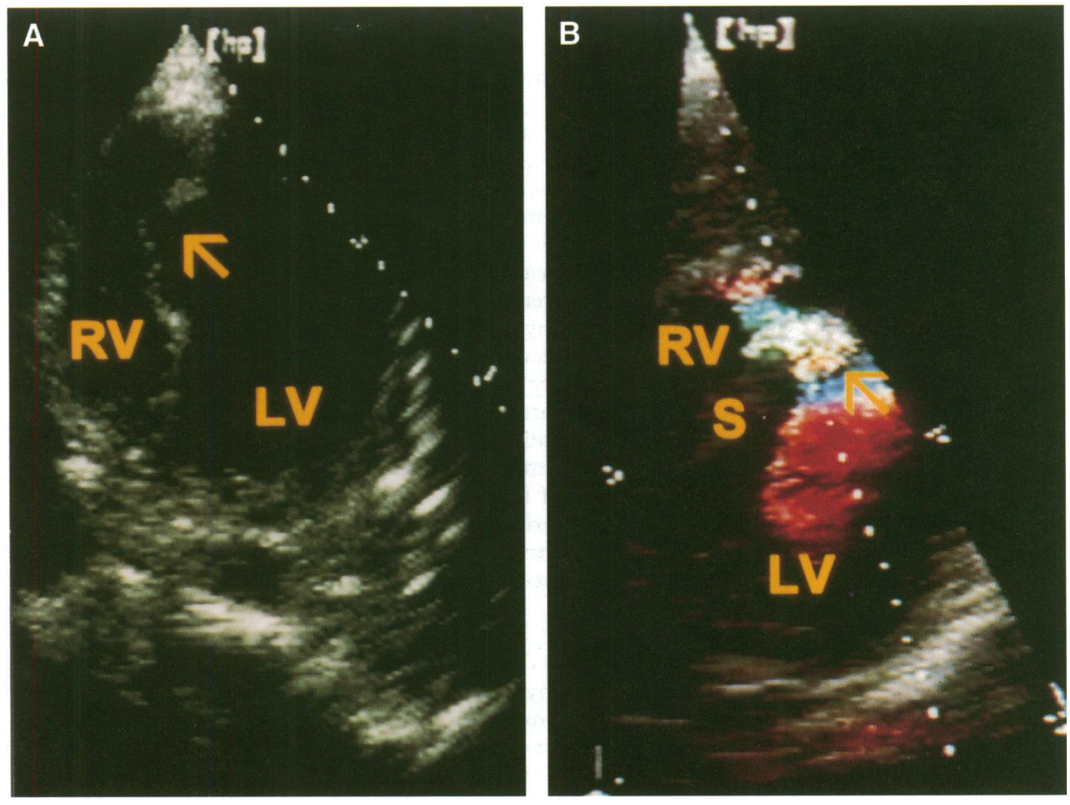
Cardiac injuries from blunt chest trauma are usually the result of high speed motor vehicle accidents; cardiac injuries result in 20% of such accidents.[1] The severity of external injuries does not necessarily reflect the severity of cardiac trauma. Blunt chest trauma can cause a variety of cardiac injuries such as myocardial contusion, myocardial rupture, pericardial trauma, valvar disruption, and acute myocardial infarction. We report a case of traumatic rupture of the interventricular septum caused by a non-penetrating trauma, and its successful surgical repair.

Case report

A 26 year old man was admitted to hospital following a traffic accident. He was sitting in the back of a car without wearing a seat belt. He suffered crush injuries on the anterior chest wall, trunk, and legs. He was said to have briefly lost consciousness; however, on admission he was awake and cooperative, but restless, and obviously in severe pain. There was profound bruising and multiple abrasions on the face, and haematomas around the eyes. There were also multiple abrasions on his back and arms and severe bruising over the anterior chest wall. Haemodynamically he was in stable condition; pulse rate 100 beats/min, blood pressure 120/60 mm Hg. Radiography of the skull, facial bones, chest, spine, pelvis, and legs revealed a shaft fracture of the left femur and tibia and fracture of the 7th and 8th right ribs. The patient was transferred to the University Hospital of Zurich for further assessment and surgical repair of the lower limb fractures three days later.

On arrival at the University Hospital of Zurich the patient was pale, was suffering tachypnoea, and tachycardia; pulse rate 129 beats/min, blood pressure 150/50 mm Hg. Blood gas analysis showed partial respiratory insufficiency with an oxygen supply of 6 litres, PO_2 7·43 kPa, PCO_2 5·0 kPa, pH 7·38. There was a pronounced precordial systolic thrill, and a loud 5/6 systolic murmur was heard at the left sternal edge and apex with radiation to the axilla. There was no clinical evidence of heart failure. The chest radiography revealed early signs of pulmonary congestion and fractures of the 7th and 8th right ribs. Cross sectional echocardiography revealed a small pericardial effusion and an apical ventricular septal defect with a diameter of 1·5 cm and shunt < 50% (figure).

Creatine kinase (CK) isoenzymes were raised (CK 1130 U/l, CK-MB 23 U/l), lactate dehydrogenase was 611 U/l, and aspartate aminotransferase 64 U/l. Haematocrit (24·5%) and



(A) Apical four chamber view; arrow, traumatic septal defect. (B) Subcostal view (colour Doppler); arrow, left to right shunt. RV, right ventricle; LV, left ventricle; S, septum.

white cell count (16·54 × 10⁹/l) were the only pathological values of the blood samples.

In view of the clinical deterioration and echocardiographic findings (significant left to right shunt), the patient was immediately prepared for open heart surgery. At operation pericardial effusion was small. The apical portion of the heart had been severely traumatised. After ventriculotomy was performed a defect (1·5–2 cm) was found in the apical portion of the interventricular septum. There was excellent inflow from the left anterior descending coronary artery (LAD). The ventricular septal defect was repaired using a Teflon felt patch and interrupted sutures with pledgets, and sealed with glue. Postoperative recovery was uneventful. The patient had internal fixation of his leg fracture four days after cardiac surgery.

Postoperative echocardiography has shown no residual ventricular septal defect. Left ventricular function was normal with the septum at the position of the Teflon patch repair, apicoseptal being akinetic. At review six months later, the patient was asymptomatic with no abnormal clinical signs.

Discussion

Cardiac injuries from blunt chest trauma usually result from high speed motor vehicle accidents. Blunt chest trauma can cause a variety of cardiac injuries such as myocardial contusion, myocardial rupture, pericardial trauma, valve rupture, and myocardial infarction. Myocardial contusion is the most frequent cardiac injury following blunt chest trauma and occurs in 16–76% of patients involved in motor vehicle accidents.[2] Ventricular septal defects following chest trauma are extremely rare. In 1958, Parmley *et al*,[3] reviewing 5467 cases of blunt chest trauma, reported five cases of isolated ventricular septal defect. Hewett, in 1847, reported the first case, and the first surgical correction was carried out by Lillehei in 1958.[4,5] Rupture of the interventricular septum may occur almost immediately after injury or many days later.

The lesion is believed to occur because the heart is compressed between the sternum and the spine, or as result of extreme intrathoracic pressure during sudden deceleration. It is more likely to occur in late diastole and early systole, the septum near the apex being the most common site of rupture.[4] In such a case the rupture of the interventricular septum may occur immediately after injury and appears with symptoms that resemble those seen in postinfarction ventricular septal defect—dyspnoea, anxiety, chest pain, and cyanosis.[4] In the present case, the clinical deterioration occurred three days after the accident. The cause of the ventricular septal defect was probably post-traumatic myocardial infarction with the well known complication of postinfarction ventricular septal defect, which usually appears two to three after days after infarction.[6] The mechanism of acute myocardial infarction following blunt chest trauma may be related to an intimal tear of the coronary artery with thrombosis, coronary spasm, coronary embolism, myocardial contusion or aortic dissection involving the coronary artery. Coronary artery dissection usually occurs a few centimetres beyond the origin of the LAD

causing extensive anterior wall infarction with the potential risk of left ventricular aneurysm formation.[7] Other mechanisms of acute myocardial infarction (atheromatous disease or embolic occlusion of the distal LAD) are not very likely because of good inflow from the LAD and normal left ventricular function.[8]

The most probable mechanism for myocardial infarction in the present case was the myocardial contusion caused by a local intimal tear, subintimal or adventitial haemorrhage, or direct damage of the myocardium.

Surgical repair is recommended as soon as possible, as refractory cardiac failure may supervene. A conservative approach has been recommended for small traumatic ventricular septal defects (pulmonary:systemic ratio < 2:1), on the basis of follow up observations with cardiac catheterisation demonstrating unaltered haemodynamics up to four years after penetrating injuries.[9]

Conclusion

In conclusion, acute myocardial infarction is a possible complication after blunt chest trauma. In rare cases, ventricular septal defect can occur several days later. Therefore, patients with blunt chest trauma require systematic monitoring of cardiac function to enable timely diagnosis and successful surgical repair.

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