Ventricular septal defect due to blunt chest trauma

A case report

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Summary

Blunt chest trauma may cause cardiac trauma, this possibility often being overlooked. Various anatomical structures may be affected. A case of ventricular septal defect due to blunt chest trauma is described and the relevant literature is reviewed.

S Afr Med J 1983: **63:** 660-662.

In cases of chest trauma, and particularly of blunt trauma, injury to the heart is often overlooked.[1] It is sometimes diagnosed at a later stage[2] or only discovered post mortem.[3] It is our aim to draw the attention of physicians and surgeons alike to cardiac trauma arising after blunt trauma to the chest. This commonly occurs in motor vehicle accidents,[4-7] but the cardiac trauma is not always evident[1,2] because of associated injuries. The reported incidence of heart injury following non-penetrating trauma to the chest varies from 16% to 76%.[4,8] Failure to suspect the injury, especially in the face of other more obvious severe injuries, probably contributes to the relative infrequency of this diagnosis.[2,8] Jackson and Murphy[3] discuss the causes and mechanisms of blunt cardiac trauma in an excellent review, and also list 19 types of injury resulting from blunt chest trauma. We are not aware of any South African report of a patient who has sustained an interventricular septal defect following blunt chest trauma. We report a patient in whom this diagnosis was made and who was successfully treated.

Case report

An unmarried, well-built 25-year-old Black labourer was admitted to H. F. Verwoerd Hospital, Pretoria, on 8 September 1981. On admission his main complaint was of having had central chest pain since October 1980 when he had been caught between a reversing 20-ton truck and a huge waste-container, with the rear bumper at the level of his chest. During this he had experienced a sensation as if his chest was cracking. Admission to a nearby hospital was followed by a period of observation, after which he was discharged as healthy and fit. At this time he also became aware of a vibration in his chest. Since his accident he had experienced the pain while working, with improvement on rest. He also noticed shortness of breath when working or walking briskly.

On examination our patient was an intelligent, tall, muscular young man with no evidence of anaemia, jaundice or cyanosis. His pulse rate was 88/min and regular and his blood pressure 120/170 mmHg. His neck veins were not distended. The chest wall showed no sign of previous injury. A left parasternal heave and a pansystolic thrill were palpable. The apex beat was in the 5th intercostal space and well within the midclavicular line. On auscultation a grade 4/6 pansystolic murmur, which was maximal at the 4th intercostal space to the left of the sternum and audible over the whole anterior chest, was heard. The pulmonary component of the second sound was loud. A clinical diagnosis of ventricular septal defect (VSD) with moderate pulmonary hypertension but without heart failure or infective endocarditis was made.

The erythrocyte sedimentation rate was 5 mm/1st h (Westergren), while a full blood count revealed no abnormality. The haemoglobin concentration was 17,4 g/dl and the haematocrit level 0,521 l/l. The blood chemical values were normal. Electrocardiography showed sinus rhythm with right axis deviation, right ventricular hypertrophy and pathological Q waves in the anterior chest leads V2 - V4, consistent with a previous anteroseptal myocardial infarction (Fig. 1). Chest radiography revealed increased vascular markings in the lungs (Fig. 2).

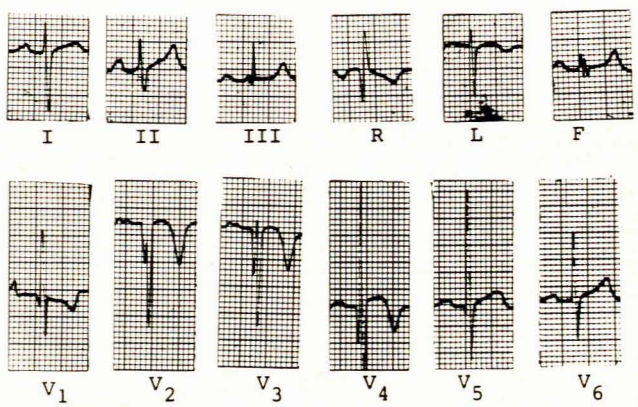

Fig. 1. ECG showing sinus rhythm with right axis deviation, right ventricular hypertrophy and pathological Q waves in anterior chest leads V2 - V4.

On echocardiography the left ventricle was seen to be enlarged, with volume overload. The size of the right ventricle was at the upper limit of normal with hypertrophy of the anterior wall and interventricular septum. Heart catheterization was performed on 9 September 1981 (Table I). A VSD was clearly demonstrated. Oxygen saturation showed a rise from 70% in the right atrium to 86% in the right ventricle. The coronary arteries were of normal distribution and patency.

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Date received: 16 April 1982.

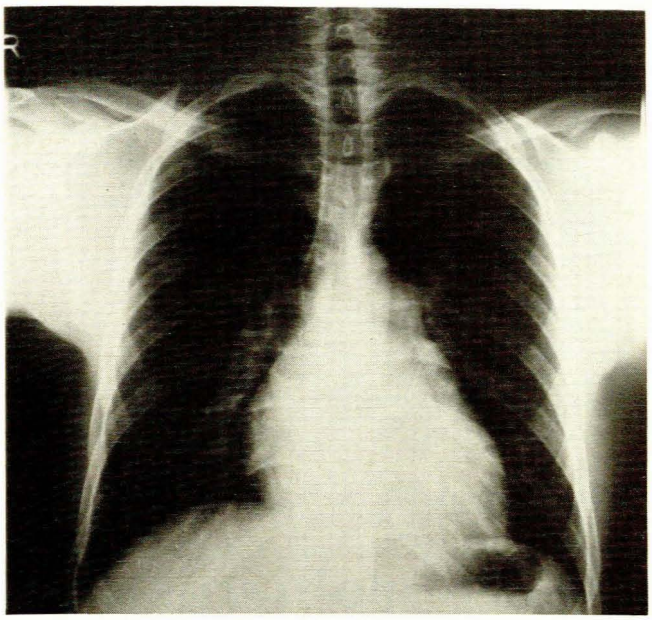

Fig. 2. Pre-operative chest radiograph showing increased vascular markings.

TABLE I. CARDIAC CATHETERIZATION DATA*

Site	Pressure (mmHg)	Oxygen saturation (%)
Superior vena cava		67
Right atrium	Mean 10	70
Inferior vena cava		73
Right ventricle	80/0 - 14	86
Pulmonary artery	80/30	86
Pulmonary wedge	Mean 22	
Left ventricle	150/0 - 15	94
Aorta	150/85	94

*Shunt ratio 1,8:1.

On 23 November 1981 the VSD was closed with a Dacron patch and interrupted sutures through a right ventriculotomy with the aid of total cardiopulmonary bypass and cardioplegic arrest of the heart. At operation a 3 x 5 cm area of adhesions between the epicardium and pericardium in the region of the middle third of the left anterior descending artery (LAD) was seen. Right ventricular hypertrophy was present. A VSD of 1,5 x 1,5 cm was present in the muscular septum just below the middle third of the LAD. Postoperatively the right ventricular pressure was 15/0 mmHg. The postoperative course was uneventful and the patient was discharged on 7 December 1981. On follow-up the patient was found to be asymptomatic and no murmur was present on auscultation. A cardiac flow study performed with radio-isotopes showed no evidence of an intracardiac shunt.

Discussion

In this case the diagnosis of VSD was not made at the first admission. The delayed onset of the typical pansystolic murmur that is frequently noted in the literature[6] is difficult to explain. Perhaps a soft murmur may have been overlooked until it became more obvious with improved cardiac function. Madoff and Desforges[7] suggest the following clinical features as significant diagnostic clues in assisting the physician in diagnosing cardiac trauma: (*i*) fractured sternum; (*ii*) variable cardiac rhythm; (*iii*) tachycardia; (*iv*) changing ECG; (*v*) new cardiac murmurs; (*vi*) recurrent haemothorax; (*vii*) widening of the mediastinum; and (*viii*) haemopericardium.

Rosenthal *et al.*[9] originally described the triad of chest trauma, cardiac murmur and an infarction pattern on the ECG. When our patient was admitted for the second time (to a medical ward) the diagnosis of a VSD was obvious. The question arises as to whether this VSD could have been a congenital lesion. The presence of fibrous tissue around the defect in the muscular septum suggests the recent occurrence of an inflammatory reaction and aids in the pathological diagnosis of traumatic VSD.[2,10]

The ECG in cardiac trauma has been well described,[2,5,11-16] and acute coronary artery occlusion secondary to blunt chest trauma is well documented.[15,16] In our patient there was electrocardiographic and macroscopic evidence of a previous infarction. Pifarré *et al.*[15] recorded a patient with an acute occlusion of the proximal LAD. At operation 5 months later the LAD was patent and fully distended. Much the same could have happened with our patient.

Despite its seemingly protected anatomical position, the interventricular septum appears acutely susceptible to injury secondary to non-penetrating chest trauma.[17] Blunt trauma characteristically produces defects in the muscular septum, as demonstrated in this case.[6,17]

What actually causes the rupture of the septum? Two explanations of the mechanism of rupture of the interventricular septum have been put forward:[18]

1. The initial damage of the nutrient vessels of the septum or papillary muscles results in infarction, which progresses to liquefaction necrosis and perforation or rupture.

2. The heart is compressed during the vulnerable period of late diastole or early systole when the ventricular chambers are full and the valves are closed. This elevation of ventricular tension causes rupture of the septum.

In a patient with a traumatic VSD the prognosis without operation depends largely on the size of the defect, the type of trauma and associated other injuries.[19] The patient who is symptomatic requires repair sooner, occasionally within a few days of injury if the left-to-right shunt is massive. In the case of the small, asymptomatic VSD with a calculated shunt of less than 1,5:1 and with normal right heart pressures, closure is justified primarily to avert the risk of endocarditis.[6] In cases in which the defect was closed using cardiopulmonary bypass the prognosis was extremely good.

Turney *et al.*[6] reported 22 cases in the literature in which surgery was employed without a single death. The indications for surgical repair are:[19] (*i*) evidence of cardiac enlargement; (*ii*) symptoms of congestive heart failure; and (*iii*) right ventricular pressure elevation.

In conclusion, we strongly recommend that in patients who have experienced blunt trauma to the chest the diagnosis of cardiac injury should always be entertained. It is mandatory to nurse such patients in an intensive care unit and daily electrocardiography is essential. Early cardiac catheterization could be beneficial to the patient, remembering that you see only what you look for.

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