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Childhood Traumatic Infarction Causing Left Ventricular Aneurysm: Diagnosis by Two-Dimensional Echocardiography

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After being struck by an automobile, a 9 year old boy developed transient right bundle branch block followed by electrocardiographic changes of inferior wall myocardial infarction and an increase of serum cardiac enzymes. Two-dimensional echocardiography demonstrated a prominent septal aneurysm. Subsequent cardiac catheterization confirmed septal aneurysm and demonstrated an additional inferior aneurysm. Six weeks after the accident, resection of the inferior aneurysm was performed. At 1 year follow-up study, residual septal aneurysm and cardiomegaly are present, but the boy is asymptomatic.

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Myocardial infarction after blunt trauma is well described in adults (1–4). Post-traumatic ventricular aneurysm has also been described (5–8). However, the two-dimensional echocardiographic findings in traumatic aneurysms have not been emphasized. In this report, we describe the clinical, echocardiographic, angiographic, surgical and follow-up findings in a 9 year old boy who had a traumatic myocardial infarction complicated by aneurysm after being struck by an automobile.

Case Report

History. A 9 year old boy was knocked to the side of the road by a car reportedly traveling at 15 miles/hr. He was taken to the local emergency room where a left anterior sixth rib fracture, left femoral fracture, pelvic fractures and pulmonary contusions were noted. Fluid resuscitation precipitated pulmonary edema, for which the boy was intubated. Peritoneal lavage, skull X-ray films and spine X-ray films were normal. The child was referred to the trauma service at North Carolina Memorial Hospital.

Physical examination and laboratory data. On arrival 4 hours after the accident, he was alert and responsive but intubated, with a blood pressure of 110/68 mm Hg and a pulse rate of 127 beats/min. Pink frothy material was in the endotracheal tube. He had an abraded area over his left anterior thorax with a palpable rib fracture, a swollen, tender left thigh and pain on compression of his iliac crests. His cardiac examination was recorded as unremarkable. His initial rhythm strip showed right bundle branch block. His chest radiograph showed a normal-sized heart and pulmonary edema (Fig. 1, left panel).

Clinical course. The boy's pulmonary edema responded to diuretic drugs and he was extubated within 24 hours. Serial electrocardiograms showed resolution of the right bundle branch block noted on the initial rhythm strip and development of an inferior myocardial infarct (Fig. 2). External traction was applied for the left femoral fracture. The serum creatine kinase (CK) level reached a peak of 2,240 units 10 hours after the accident with an MB fraction of 156 units or 7% (normal ≤5 units, ≤4%). Later chest radiographs showed cardiomegaly (Fig. 1, middle panel).

Diagnosis. Two-dimensional echocardiography performed 24 hours after the accident showed mildly impaired left ventricular function and a large aneurysm of the ventricular septum (Fig. 3). One month after the accident, cardiac catheterization was undertaken in anticipation of aneurysm resection. Left ventricular pressure was 100/18 mm Hg. Angiography documented a septal aneurysm impinging on the right ventricle (Fig. 4 and 5). In addition, a large inferior aneurysm, not initially detected by two-dimensional echocardiography, was found (Fig. 5). Coronary angiographic findings were normal.

Surgery. At surgery a large inferior aneurysm resting on the diaphragm was resected. After resection of the in-

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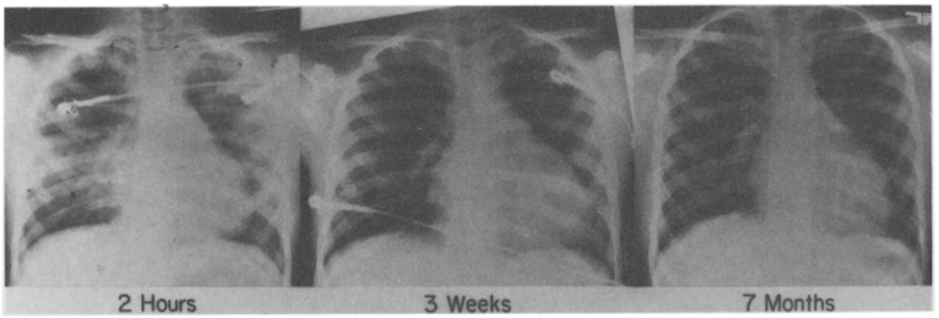

Figure 1. Sequential chest radiographs taken at 2 hours, 3 weeks and 7 months after the patient was struck by a car.

ferior aneurysm, bulging of the ventricular septum into the right ventricle could still be felt through the tricuspid valve. The postoperative course was uneventful, and the boy was sent home in a Spiker cast placed for his femoral fracture.

Postoperative course. Two-dimensional echocardiography performed 2 months after surgery documented residual ventricular septal aneurysm that moved paradoxically during systole, and a dilated, poorly functioning left ventricle (Fig. 6, top panel). Repeat cardiac catheterization performed 3 months after the accident showed a left ventricular pressure of 85/9 mm Hg. Angiography documented absence of the inferior apical portion of the aneurysm, residual septal aneurysm (Fig. 6, bottom panel) and normal left ventricular function.

In view of both normal left ventricular pressure and function at postsurgical catheterization and concern over possible harm to the coronary circulation should resection of the residual septal aneurysm be attempted (the residual aneurysm extended into the area of the atrioventricular groove), no further operative intervention was recommended. At 1 year follow-up examination, the boy's physical activity has returned to normal. His electrocardiogram shows evidence of an inferior wall myocardial infarction, and his chest radiograph shows residual but decreasing cardiomegaly (Fig. 1, right panel).

Discussion

Blunt chest trauma can have many different cardiac effects, ranging from clinically insignificant myocardial concussion to free wall rupture with immediate cardiac tamponade and death. Higher energy chest impacts such as those incurred in high-speed automobile crashes can be expected to have more severe adverse cardiac effects; however, seemingly innocuous blunt chest injuries have also proven fatal (9), probably as a result of ventricular fibrillation. Among nonfatal injuries, traumatic myocardial infarction can have the most severe long-term consequences, depending on the size and complications of the infarction and on the viability of the remaining myocardium.

Pathogenesis of traumatic myocardial infarction and aneurysm. The pathogenesis of traumatic myocardial infarction after blunt chest trauma is variable. Traumatic coronary thrombosis has occurred in the absence of preceding coronary atherosclerosis (4,10–14), but this is rare (9). Blunt chest trauma has precipitated myocardial infarction in pa-

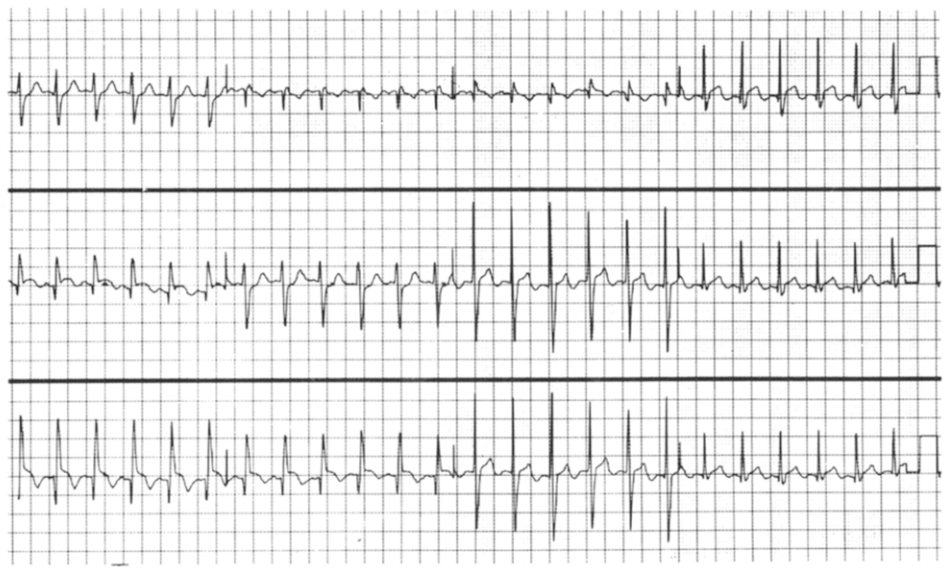

Figure 2. Twelve lead electrocardiogram taken 4 hours after the car accident demonstrates Q waves and ST elevation in leads II, III and aVF, consistent with inferior myocardial infarction. There is also ST elevation in leads V_4 to V_6.

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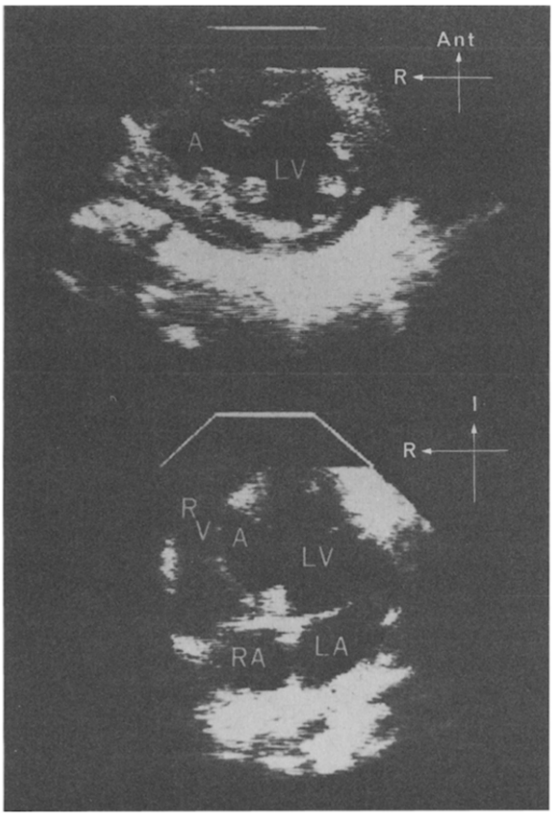

Figure 3. Echocardiograms. **Top panel**, A short-axis view that shows a defect in the ventricular septum communicating with a large aneurysm (A). **Bottom panel**, An apical four chamber view that shows protrusion of the septal aneurysm (A) into the right ventricle (RV). Ant = anterior; I = inferior; LA = left atrium; LV = left ventricle; R = right; RA = right atrium.

tients with coronary atherosclerosis (11,15). Traumatic myocardial infarction despite normal coronary arteries has been reported (3,16,17), leading some investigators (10,11,18) to suggest that coronary spasm may be responsible. However, in these cases coronary arteriography was performed weeks, months or years after the traumatic infarction (3,16,17), and it is possible that recanalization of traumatically occluded coronary arteries could have occurred, thereby giving the appearance of traumatic infarction in the absence of coronary occlusion (10). Finally, embolic coronary occlusion with infarction has occurred after blunt myocardial injury complicated by ventricular thrombus (19).

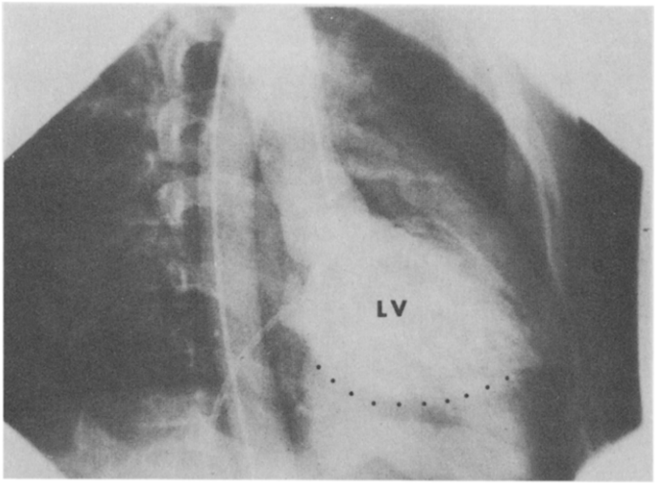

Figure 5. A single frame from a left ventriculogram in the long-axis oblique projection shows the inferior ventricular aneurysm. The **dots** mark the expected inferior border of the left ventricle (LV).

Whatever the pathogenesis of myocardial infarction after blunt chest trauma, aneurysm can be assumed to be a complication of infarction at the time of injury (3,20). Because true aneurysms are unlikely to rupture (21), decisions regarding surgical intervention are based on the clinical and hemodynamic effects of the aneurysm. In contrast, false aneurysms, which result from laceration of cardiac tissue and wall herniation rather than wall thinning, have a high propensity for rupture (21) and require early surgery. Traumatic false aneurysm has been reported during childhood (8).

Cardiac evaluation after blunt chest trauma. Although bedside cardiac evaluation of patients who have had blunt chest trauma is not different from that of patients with heart disease without trauma, signs of tamponade, valve incompetence and air leak should be carefully sought. When hemodynamic compromise (in the absence of internal or external blood loss), regurgitant murmurs or arrhythmias are found, little doubt about blunt cardiac damage should

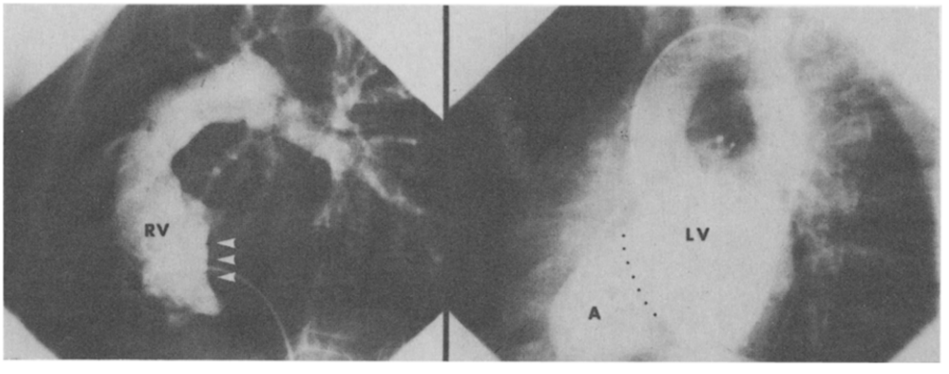

Figure 4. Ventriculograms. **Left panel**, A single frame from a right ventriculogram in the four chamber projection shows impingement of the septal aneurysm (**arrows**) on the right ventricular (RV) cavity. **Right panel**, A single frame from a left ventriculogram in the four chamber projection shows the aneurysm (A) filled with contrast medium. The **dots** mark the expected septal border of the left ventricle (LV).

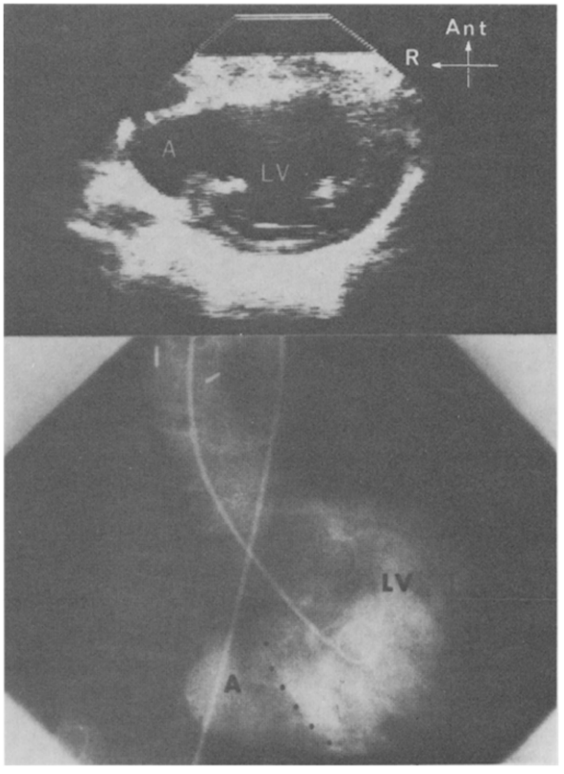

Figure 6. Follow-up findings. **Top panel**, A short-axis view obtained 2 months after surgical resection of the inferior aneurysm seen in Figure 5 shows a prominent residual septal aneurysm (A), which bulged paradoxically during systole. **Bottom panel**, A single frame from a left ventriculogram in the long-axis oblique projection obtained 3 months after surgery shows the residual septal aneurysm (A) filled with contrast medium. The **dots** mark the expected septal margin of the left ventricle (LV); other abbreviations as in Figure 3.

exist, but whether or not such damage is suspected, aortic laceration should be considered in all patients with blunt chest trauma.

In most instances, obvious clinical signs of cardiac involvement are not present, and evaluation of possible cardiac damage after blunt chest trauma will depend on laboratory tests. There is no consensus about the best approach to the laboratory diagnosis of traumatic cardiac injury. This is because the great majority of traumatic myocardial injuries are not fatal, and clinicopathologic correlations to prove the type and extent of myocardial injury associated with a given laboratory abnormality have not been possible. Nevertheless, several laboratory tests can be of value in the cardiac assessment of patients with blunt chest trauma.

Electrocardiogram. Despite conflicting reports (22,23) about the value of the electrocardiogram in patients with blunt chest trauma, its high sensitivity in detection of cardiac damage makes it a very useful cardiac screening test in blunt trauma. Although nonspecific electrocardiographic abnormalities are frequently present in victims without any other evidence of myocardial damage (24–27), all major cardiac complications, such as tamponade, rupture, fibrillation, shock and infarction, occur in patients who do exhibit electrocardiographic abnormalities. Electrocardiographic abnormalities were noted in 20 of 35 consecutive patients with blunt chest trauma reviewed by Jones et al. (2); all 3 patients who developed life-threatening cardiac complications exhibited electrocardiographic abnormalities. In a series of 169 patients, Macdonald et al. (25) found no instance of both a normal electrocardiogram and necropsy evidence of myocardial contusion. In the series of 77 patients of Sutherland et al. (27), each of the three cardiac deaths (all in patients with cardiac contusion confirmed by postmortem examination) was associated with electrocardiographic changes of myocardial contusion (27).

Further, the specificity of the electrocardiogram in the diagnosis of blunt trauma-induced cardiac damage is high when Q waves, heart block, arrhythmias or localized ST-T wave changes are present (28). Because it is noninvasive and relatively inexpensive, the electrocardiogram should be used routinely to screen victims of blunt trauma for cardiac involvement.

Serum creatine kinase-MB. Determinations of creatine kinase(CK)-MB are very useful in detecting significant blunt trauma damage to the heart, notwithstanding rare false positive elevations in patients with cardiogenic shock (29) and certain other types of trauma (30,31).

An old distinction between myocardial contusion, in which cellular injury (but not necessarily infarction) is present, and myocardial concussion, in which electrocardiographic changes may be present but cell damage is absent, has been reiterated by Michelson (28), who simply defined cardiac contusion as an increase of serum CK-MB isoenzyme. In his series of 49 patients who demonstrated electrocardiographic abnormalities among a total of 220 patients with blunt chest trauma, all five potentially fatal cardiac complications and all three deaths occurred among the 26 patients with increased serum CK-MB. Localized electrocardiographic changes were noted in 85% of the 26 patients with increased CK-MB, and in only 26% of the 23 patients without an increase.

Thus, CK-MB determinations enhance the specificity of electrocardiographic detection of significant myocardial injury from blunt chest trauma. Although serum CK-MB isoenzyme increases do occur when the electrocardiogram is normal (in the series of Potkin et al. [23], 2 of 72 victims of blunt trauma with increased CK-MB had a normal electrocardiogram), this group does not appear to be at significant risk for cardiac complications.

Increased serum CK-MB isoenzyme levels appear to occur more rapidly after myocardial contusion than after ischemic infarction, probably because normal coronary arteries usually perfuse the injured area after blunt trauma and cause early appearance of enzyme in the serum.

Technetium-99m myocardial scanning. Myocardial scanning was a more sensitive marker for areas of traumatic

damage than were electrocardiographic or serum enzyme changes according to the study of Kumar et al. (24). However, in that study only 7 of 17 patients with an abnormal electrocardiogram had a positive scan, and 3 patients with an abnormal scan had a normal electrocardiogram. In the absence of pathologic correlations or other clinical or laboratory signs of cardiac damage, the significance of such "positive" myocardial scans remains unknown. Other investigators (23,32,33) have found myocardial scanning to be an insensitive indicator of myocardial damage.

Radionuclide angiography. Initial reports (26,27) have suggested that radionuclide angiography is a very sensitive means for detecting blunt myocardial injury; in one study (26), 42 of 77 patients with multisystem trauma had focal abnormalities in wall motion by electrocardiographically gated scintigraphy. However, although only 11 of the 42 patients with focal wall motion abnormalities had electrocardiographic changes thought to specifically indicate myocardial injury, all 42 patients had electrocardiographic abnormalities, indicating that the electrocardiogram is just as sensitive an indicator of myocardial damage. Electrocardiographic abnormalities were also detected among the 35 patients without focal wall motion abnormalities. Unfortunately, CK-MB levels were not determined in this study.

Two-dimensional echocardiography. To our knowledge, a prospective study of two-dimensional echocardiography in victims of blunt chest trauma has not been reported. Correlation of two-dimensional echocardiographic findings with electrocardiograms, serum CK-MB isoenzyme levels and radionuclide findings, as well as clinical findings and outcome, would be of value. Certainly, two-dimensional echocardiography can provide useful additional information when definite clinical or electrocardiographic signs of cardiac damage are present, as happened in this case. However, routine two-dimensional echocardiographic screening of asymptomatic victims of blunt chest trauma has not been proven to be of value and cannot be advocated, given the expense involved. Further, all diagnostic techniques have limitations; often it is difficult to adequately visualize the inferior surface of the heart with two-dimensional echocardiography. In our case, an inferior aneurysm initially unsuspected with two-dimensional echocardiography was proven by angiography.

Conclusion. Electrocardiographic abnormalities prompted two-dimensional echocardiographic study of a 9 year old boy who had been struck by a car; a tear in the ventricular septum was identified. Serial electrocardiograms showed an inferior myocardial infarction, which was accompanied by elevations of serum CK-MB isoenzyme. Cardiac catheterization confirmed septal aneurysm and demonstrated an undetected inferior apical aneurysm as well. Surgical resection of the inferior aneurysm was successful.

Electrocardiograms and CK-MB determinations are useful in determining which victims of blunt trauma are at risk for major cardiac complications. Two-dimensional echocardiographic evaluation of the heart is indicated when signs of myocardial dysfunction are present or electrocardiographic abnormalities are accompanied by serum CK-MB elevations. Other noninvasive studies, such as myocardial scanning, scintigraphy and radionuclide angiography, add little to identification of subjects at risk for major complications. If repair of traumatic cardiac injuries is anticipated, presurgical cardiac catheterization is indicated.

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