Cardiac damage presenting late after road accidents

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ABSTRACT Six examples of cardiac damage secondary to non-penetrating trauma in road accidents are described. In all six cases the lesion was not recognised at the time of the accident but became clinically important two days to 17 years later. As the patients were young or had unusual lesions, the damage could be attributed to the accident. In older patients with common cardiac problems the trauma might not be recognised as the underlying cause.

Non-penetrating trauma to the chest, often sustained in a road accident, can damage the heart. A wide variety of lesions in the valves, myocardium, coronary arteries, and pericardium has been documented.1–5 Most reports have dealt with necropsy findings or have described cardiac injuries presenting immediately after the accident. In this account six examples of cardiac damage, which was unrecognised at the time of the accident but became clinically important later, are described.

Case reports

CASE 1
An 18-year-old man was riding a motor-cycle through a cloud of smoke when he ran into a fire-engine fighting a stubble fire. He sustained multiple injuries including bruising on the left anterior chest wall and a ruptured spleen. No cardiac trauma was detected at the time of admission but two days later he became breathless with pulmonary oedema. An electrocardiogram showed a large transmural anterior infarct. With the help of digoxin and diuretic therapy he made a good recovery. Four years later he is asymptomatic but a third sound and cardiac enlargement persist.

CASE 2
A man of 46 drove his car into a lamp post. The steering wheel hit his chest and he suffered multiple injuries including a ruptured spleen, a ruptured diaphragm, and rib fractures. After operation he made a good recovery, but tiredness and breathlessness were noticed four months later. He was found to have tricuspid regurgitation, and the electrocardiogram showed right bundle branch block. The only abnormality found at cardiac catheterisation 18 months after the accident was gross tricuspid regurgitation. The peak systolic pressure was 28 mm Hg in both the right atrium and the right ventricle. A gradual symptomatic improvement occurred and another catheterisation six months later showed diminished tricuspid regurgitation with a peak systolic pressure of 15 mm Hg in the right atrium and 25 mm Hg in the right ventricle.

CASE 3
A 24-year-old man was involved in an accident while riding a motor-cycle. Few details are available, though he sustained multiple injuries. Immediately after the accident his jugular venous pressure was noticed to be raised but no cardiac damage was detected. He was making a good recovery 10 days after the accident when he suddenly became unconscious and developed a right hemiplegia. An electrocardiogram showed a transmural anteroseptal infarct. A diagnosis of a cerebral embolus secondary to a mural thrombus from the infarction site was made. His recovery was only partial and epileptic fits proved difficult to control. He died two years after the accident. Necropsy showed recent and old cerebral infarction secondary to emboli. The coronary arteries were normal apart from complete occlusion of the left anterior descending artery three centimetres from its origin. Microscopy of the occlusion showed periarterial haemosiderosis and breaks in the external elastic lamina; these findings are consistent with traumatic damage to the artery. An old anterior myocardial infarction was probably the source of the recent and old emboli.

CASE 4
In 1941 a 19-year-old man was in a head-on collision

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with a lorry, the steering wheel hitting his chest. Nine
years earlier he had been seen by a consultant
physician for delayed growth and three months
before the accident he had been passed by a medical
board for the RAF without any heart disease being
detected on either occasion. During his two weeks in
hospital after the accident no cardiac murmur was
noticed but a systolic murmur was heard five weeks
later by his general practitioner. The clinical features
of a ventricular septal defect were present and the
case was reported soon afterwards. He was symptom-
free until 1980 when he developed infective endocar-
ditis (Streptococcus viridans), treated with peni-
cillin and gentamicin. Subsequent cardiac catheteri-
sation demonstrated a shunt, apparently at ventri-
cular level, with a pulmonary to systemic flow ratio
of 2:0:1. Surgical repair was advised and at operation
(Mr TAH English) three small holes were found
between the left ventricle and right atrium. These
defects were closed with some difficulty as the
conducting system was in close proximity.

Case 5
At the age of 9 years a boy was run over by a tractor
trailer, sustaining injuries to his chest. He made
an uncomplicated recovery. Seventeen years later he
was found to have constrictive pericarditis with gross
pericardial calcification. No history or evidence of
tuberculosis could be obtained from the patient or
his family. Cardiac catheterisation confirmed the
diagnosis and pericardiectomy was performed
(Mr BB Milstein) with a good result.

Case 6
A 46-year-old taxi-driver drove into a brick wall. He
sustained multiple fractures of the right arm, right
leg, and ribs. The admission notes recorded the
absence of any murmurs. The following day an early
diastolic murmur was detected but no attention was
paid to it. Two days after admission he developed
pulmonary oedema but the possibility of cardiac
damage was not considered. His subsequent course
was stormy and he needed artificial ventilation for
several days. Three weeks after the accident an
electrocardiogram showed left ventricular hyper-
trophy. A diagnosis of aortic regurgitation was made
and he was started on therapy for left ventricular
failure. When seen for the first time in the cardiac
department a year later, he was symptom-free but the
chest radiograph showed increasing cardiomegaly.
Cardiac catheterisation demonstrated severe aortic
regurgitation, and an atrial septal defect with a
pulmonary to systemic flow ratio of 2:0:1. At
operation (Mr TAH English) fine pericardial
adhesions and two posterior pericardial defects
were found. The non-coronary cusp of the aortic
valve had a large traumatic perforation. The edges of
the atrial septal defect were thickened and fibrous;
the lesion could only have been produced by trauma.
The septal defect was closed and the aortic valve was
replaced with a Björk-Shiley prosthesis.

Discussion

Many examples of cardiac trauma secondary to
non-penetrating injury have been described in the
last 200 years and the subject has been extensively
reviewed. At times certainty that the lesion did not
pre-date the injury is missing. Nevertheless cardiac
rupture, septal defects, myocardial infarction,
coronary artery damage, valve lesions, pericarditis,
complete heart block, and many arrhythmias have
been described as occurring secondary to non-
penetrating injuries.

The cases described here reflect this diversity of
lesions and also contain some unusual features. Case 3
is an example of trauma producing complete
occlusion of an otherwise healthy coronary artery.
The possibility of such arterial damage has been
questioned in the past and this case is one of the few
clear examples. The tricuspid regurgitation found in
case 2 is an uncommon traumatic lesion. Valve
replacement is often required for severe regurgita-
tion, but in this patient spontaneous improvement
occurred and was documented by cardiac catheteri-
sation. As far as we are aware the left ventricular/
right atrial commnication found at operation in
case 4 has not been described before as a complica-
tion of non-penetrating trauma. Ventricular septal
rupture secondary to trauma is commonly low in the
septum. The traumatic atrial septal defect found in
case 6 is a rare lesion in accident survivors: most
elements have been found at necropsy.

Cardiac trauma may be missed after road accidents.
In none of these six cases was the cardiac damage
recognised immediately after the accident. This
delayed appearance of cardiac damage has not been
commented upon in previous reports though late
presentation of aortic rupture as an aneurysm is well
recognised. Apart from some relevance to the
selection of hearts from road accident victims for
transplantation, these findings suggest the traumatic
origin of a cardiac lesion may not be appreciated. In
the cases described here the cardiac abnormality was
linked to an injury because the accidents were severe
and the patients were young or developed a lesion
which was unlikely to have any other aetiology. In
older patients the defects might be ascribed to other
causes, particularly if the chest trauma was not severe
and had occurred several months before. Vigilance is
required after road accidents to ensure that cardiac
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damage is not missed because more superficial injuries have healed.

References


The International Society for Heart Transplantation

This Society has recently been founded and will be publishing a Bulletin called Heart Transplantation. For further information please contact Dr JG Losman MD, Michael Reese Hospital and Medical Center, 29th Street and Ellis Avenue, Chicago, Illinois 60616, USA.
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