Case reports

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Traumatic aortic incompetence following road traffic accident

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Summary
This case report describes the presentation and treat-
ment of a case of aortic incompetence, resulting from
a road traffic accident. The relevant literature is
briefly reviewed.

Aortic incompetence due to trauma has been de-
scribed following non-penetrating chest injuries, such
as kicks from horses (Barie, 1881), falls from heights
and crushing accidents (Kissane, Koons and Clark,
1948; Levine, Roberts and Morrow, 1962). Despite
the frequency of road traffic accidents, there have
been no recent reports of traumatic aortic valve
damage.

Case history
The patient, a 44-year-old transport manager,
was admitted to hospital in September, 1972. He
gave a history of increasing breathlessness on exer-
tion over the previous 15 months. For the 2 months
before admission he had complained of nocturnal
dyspnoea. The symptom of breathlessness as well as
episodes of dizziness had dated from May, 1971,
when he had been involved in a serious road accident.
The nature of the accident was such that, on impact,
he had been thrown through the back window of
his car, sustaining injuries to the upper thoracic
spine and occipital part of the skull. He was un-
conscious for approximately 10 min and, on admis-
sion to the casualty department, the principal
abnormality was of tenderness, swelling and marked
angulation of the thoracic spine. X-rays revealed a
compression fracture of the 7th thoracic vertebra.
No murmurs were noted and blood pressure was
normal. Systolic and early diastolic murmurs were
first noted 5 days after admission.

Following discharge the patient noted that he was
breathless on climbing stairs and that he suffered
quite marked light-headedness on more severe
exercise.

In the past history, he had a myocardial infarction
in 1968. During that admission only a soft systolic
murmur was noted. There were no signs of aortic
insufficiency, the heart was of normal size and the
electrocardiogram showed normal left ventricular
voltages. He was said to have had rheumatic fever
at the age of 28, treated at home. Subsequent medical
examinations in the Services were satisfactory.

On admission to hospital, he had signs of left
ventricular failure and of gross aortic incompetence.
Good progress was made on standard therapy but
shortly after discharge he was re-admitted in acute
pulmonary oedema and subsequently transferred
for more specialized investigation.

Examination
The patient was pale, mildly dyspnoeic with marked
Corrigan pulsations in the neck. Jugular venous
pressure was elevated and a third sound was audible
on auscultation. There was a loud aortic systolic
murmur and long aortic diastolic murmur, both
radiating to the neck and maximal over the upper
left sternal border. Pulse was collapsing, sinus in
rhythm, and blood pressure was 180/50. There were
bilateral fine crepitations in the chest.

Investigations
Chest X-ray showed cardiomegaly and pulmonary
venous congestion and the ECG showed marked
left ventricular hypertrophy. At cardiac catheteriza-
tion, the left ventricular end diastolic pressure was
15–45 mmHg and a supravalvular angiogram showed
severe aortic incompetence. Coronary angiograms
were normal except for minor narrowing of the left
anterior descending artery. WR was negative.
Following these investigations and further treatment with digoxin and diuretics, the patient underwent bypass surgery. At operation the aortic valve was replaced with a Bjork prosthetic valve and the operation note stated: 'The ascending aorta was a little enlarged and there was a marked systolic and diastolic thrill in it. The aortic valve was tricuspid. All the cusps were thickened but there was no calcification. The left coronary cusp prolapsed and there appeared to be a tear in it just at the junction between it and the non-coronary cusp. The macroscopic picture did not support either a diagnosis of chronic rheumatic fever or acute traumatic rupture of a cusp'.

The report on the microscopy of the valve stated: 'Section of the leaflets shows dense fibrosis with focal myxomatous areas of collagen degeneration. No diagnostic features of rheumatic fever seen'.

The patient made an uncomplicated postoperative recovery and returned to work after 8 weeks.

Discussion

The evidence that the aortic incompetence was due to the accident relies on the clinical history. The macroscopic and microscopic appearances were not clearly diagnostic. The alternative explanation, that the valve was diseased from chronic rheumatic fever, is unlikely as the valve was known to be normal in 1968 and the evidence that he had definite rheumatic fever is doubtful. It would be unusual for rheumatic fever to occur at the age of 28 and secondly to cause aortic incompetence as the only lesion.

The delay from the accident to the onset of left ventricular failure is not evidence against this diagnosis. Howard (1928) reviewed 113 cases in the literature. While death was common within days or even hours of the rupture of the valve, some patients lived for 10–15 years after the trauma. The case described by Barie (1881) was symptom-free for several years. Patients may complain of a purring sensation in the chest (Kissane et al., 1948; Leonard, Harvey and Hufnagel, 1955; Sainani and Syatkowski, 1969) following the acute event, but frequently they are preoccupied with other injuries.

Traumatic rupture of aortic valve cusps was probably first described in 1830 by Plenderleath. There have been several reports since then, a frequent cause being a kick from a horse. Since horses were replaced by the motor car there have been no reports of ruptured aortic valves in road traffic accidents, despite the frequency at which they occur. This is surprising and it is possible that damage to valves is overlooked because of the presence of multiple injuries.

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References


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