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Short reports

Tricuspid regurgitation caused by blunt chest trauma in association with pericardial agenesis: surgical correction after eight years

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Tricuspid regurgitation (TR) caused by nonpenetrating trauma of the chest is rare. It has been seen more frequently in recent years, presumably because of the increase in automobile accidents and the availability of better diagnostic facilities.

Since the first description by Williams in 1829, 31 cases have been reported, but surgical or necropsy proof of the condition was obtained in 23 only.[1-11] In 10 cases the cause was rupture of papillary muscles; in the remainder it was rupture of the chordae tendineae or laceration of the valve leaflets. Associated tear of the pericardium was reported in three cases. TR caused by rupture of the papillary muscles is the most severe variety.[7-10 12]

We report here a case of isolated TR caused by rupture of the anterior papillary muscle after blunt chest trauma. Partial agenesis or, less probably, tearing of the pericardium and herniation of the right ventricle were also found at the time of operation.

Case report

An 18-year-old healthy male student suffered blunt chest trauma during a car accident in 1971. Admitted to the orthopaedic division of a nearby hospital, he was found to have fracture of the fourth to sixth left ribs and a pleural effusion on the same side. The ECG showed sinus rhythm and incomplete right bundle branch block.

After discharge, the patient continued to enjoy good health. In 1972, during a medical examination for military service, a cardiac murmur had been heard, but the patient was asymptomatic at that time and practised sport vigorously. In 1973 he began to complain of occasional nocturnal palpitation and was admitted to the University of Padua Hospital for investigation.

The physical examination revealed moderate cardiac enlargement, weak first and second heart sounds, and a high-pitched 2/6 mid-systolic murmur in the second right intercostal space at the sternal border, during inspiration. The liver was not enlarged. The chest radiograph showed an enlarged heart with a prominent convex left border. Right atrial hypertrophy was evident on the ECG. Right heart catheterisation revealed a right atrial v wave of 30 mmHg. Normal pressures were found in the other cardiac chambers explored. He was not considered a candidate for surgical correction at that time, but he was kept under periodic review during the next three years.

In July 1977, he was readmitted to the University of Padua Hospital for a second haemodynamic investigation. There was now a significant pressure increase in the right cardiac chambers; the right atrial v wave was 38 mm Hg, the right ventricular systolic pressure was 45 mm Hg and the right ventricular end-diastolic pressure 9 mm Hg. A right ventriculogram showed massive tricuspid regurgitation.

In July 1978 he had bouts of paroxysmal atrial fibrillation with rapid ventricular response and was treated continuously with digitalis, diuretics, and verapamil. Sinus rhythm returned. At this time he was found to have an enlarged liver. Surgical correction was advised and was performed two months later.

At operation the heart appeared grossly enlarged and partially herniated through a left pleuro-pericardial "window", 4 × 8 cm in size, and completely free from adhesions. Under cardiopulmonary bypass, with moderate hypothermia and cold cardioplegia, the dilated right chambers were explored through a vertical atriotomy. The right ventricle had a thin parietal wall and an area of fibrosis near the apex. The anterior papillary muscle was detached and the tricuspid valve was massively regurgitant. After excision of the valve, a 36 mm Hancock bioprosthesis was inserted with a continuous monofilament suture. The pericardial window was enlarged by incising the superior and inferior edges.

The postoperative course was uneventful and the patient was discharged, taking digitalis and a diuretic. At the last review, 18 months after operation, he was in good health. The chest radiograph showed the cardiac silhouette to be back to normal size and the ECG revealed disappearance of the right atrial hypertrophy.

Discussion

There seems to be no doubt about the traumatic origin of the tricuspid lesion. However, some uncertainty exists

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about the pericardial lesion. The absence of pericardial and pleural adhesions is suggestive of partial pericardial agenesis, but it is not sufficient to rule out a traumatic cause. Four cases of TR with pericardial tearing after nonpenetrating chest trauma have been reported.[6 9 10 13] Our patient also had a left-sided traumatic pleural effusion: this could substantiate the possibility of the tear. The operative findings were convincing for direct injury to the right ventricular wall. Herniation of the right ventricle through a pericardial laceration has also been described.[6] The sudden increase in right ventricular pressure, especially during systole, is considered responsible for the rupture of the papillary muscle. If the blow happens to coincide with late systole, when the atrioventricular valves are closed and the chordae and papillary muscles are under tension, laceration of the valve cusps and rupture of the chordae and papillary muscle could ensue.[2 14 15]

It has in the past been noted that TR caused by rupture of the anterior papillary muscle has a severe prognosis and most of the cases had either a fatal outcome or required early surgical repair.[7 16] Our experience is different as the patient survived eight years after the trauma, before surgical correction. We agree with Astori[7] that surgical treatment is indicated only when medical treatment fails to relieve the symptoms, unlike Jhanke *et al*[14] who advocate early surgical correction.

While the right bundle branch block is a constant ECG feature, the presence of right atrial hypertrophy has rarely been observed. A remarkable feature was the recurrence of paroxysmal atrial fibrillation and flutter, with a rapid ventricular rate, as previously observed also by Morgan,[10] Croxon,[17] and Astori.[7] The persistent incomplete right bundle branch block could be attributed to the damaged right ventricular myocardium. Such a view is further supported by the operative observation of a thin right ventricular wall with areas of fibrosis.

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