Traumatic tricuspid regurgitation

Long-term survival

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Three case histories of patients with isolated traumatic tricuspid regurgitation are reported. Clinical features are compared and contrasted to the 13 cases previously reported. The benign nature of the lesion is emphasized by the long-term follow-up periods of 39, 13, and 31 years respectively. The presence of a normal systolic pressure in the right ventricle may be one reason why isolated severe tricuspid regurgitation can have such a benign clinical course. The unimpressive and atypical systolic murmur led to a delay in the correct diagnosis of these three patients.

Traumatic tricuspid regurgitation is a rare condition, only 13 cases having previously been reported (Parmley, Manion, and Mattingly, 1958; Osborn, Jones, and Jahnke, 1964; Björk, 1965; Aleksandrow *et al.*, 1965; Brandenburg *et al.*, 1966; Shabetai, Adolph, and Spencer, 1966; Jahnke *et al.*, 1967; Shabetai *et al.*, 1969). Two of these cases have a relatively long-term follow-up of 24 years (Brandenburg *et al.*, 1966) and 10 years (Shabetai *et al.*, 1966). The purpose of this report is to document 3 further cases with a long-term follow-up of 39, 13, and 31 years, respectively.

Case reports

Case 1 In 1926, at the age of 16 years, this patient rode his bicycle into the back of a horse which kicked him in the head and chest. He lost consciousness for a few minutes. Soon after this he became aware of unusual breathlessness on exertion and intermittent palpitations. At 18 years a heart murmur was heard for the first time, and at 21 years he had an attack of paroxysmal atrial tachycardia associated with an operation for hernia. From 30 years onwards, he experienced rapid irregular palpitations on the slightest exertion. At the age of 32 years physical examination revealed an enlarged heart and a grade 2/4 intensity systolic murmur at the lower left sternal border. The chest x-ray showed moderate cardiomegaly and a limited electrocardiogram showed a complex in lead I consistent with right bundle-branch block. In the following years, he had recurrent attacks of atrial fibrillation, readily abol-

ished with 300 mg quinidine. In 1958, aged 48 years, he presented to another hospital with symptoms of upper abdominal discomfort and diarrhoea and was observed to be in right-sided heart failure. Subsequently, in addition to palpitations, he complained of breathlessness and fatigue on minor exertion, and discomfort in the right hypochondrium, and he was referred to Green Lane Hospital in May 1965.

Physical examination revealed an anxious, acyanotic 55-year-old man. He was in atrial fibrillation with a ventricular rate of 70 a minute, the blood pressure was 120/80 mmHg, and he had physical signs of severe tricuspid regurgitation and right heart failure. There were prominent systolic pulsations of the neck veins and of the liver, which was palpable 4 cm below the right costal margin. On auscultation there was a grade 1/4 pansystolic murmur at the lower left sternal border, which increased on inspiration. The electrocardiogram showed atrial fibrillation and complete right bundle-branch block. The chest x-ray (Fig. 1B) showed moderate cardiomegaly, but there had been little increase in heart size since an earlier film taken in 1953 (Fig. 1A).

Heart screening revealed a densely calcified rather bullet-shaped opacity within the heart (Fig. 2), which showed considerable movement during the cardiac cycle. Analysis of the motion of this opacity on a cine film showed it to be in the region of the tricuspid valve orifice. The opacity passed to the right ventricle during diastole, to rise slowly to the region of the tricuspid ring. With the onset of ventricular systole it moved back very rapidly into the right atrium. It seemed likely that this represented a calcified detached papillary muscle.

Cardiac catheterization (Table, Case 1) demonstrated severe isolated tricuspid regurgitation

Received 30 November 1970.

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FIG. 1 Chest x-rays of Case 1. (A) 27 years after the accident, and (B) 39 years after the accident, showing a relatively minor increase in heart size over this period.

with prominent systolic 'CV' waves in the right atrium rising to 8 mmHg (Fig. 3A). Dye dilution studies, from dye injected into the inferior vena cava, did not show any early appearance of dye, thus excluding the presence of a right-to-left shunt. During cineangiocardigraphy, the right ventricular catheter recoiled to the right atrium. It showed, however, a large right ventricle and right atrium.

He remained in persistent right-sided heart failure, with symptoms of fatigue and dyspnoea despite full medical treatment. In July 1965, therefore, at the age of 55 years, and 39 years after

the initial blow on his chest, an operation was performed on the tricuspid valve, when the valve was found to be grossly incompetent due to complete rupture of the posterior papillary muscle. There was a flail remnant present, consisting of the matted chordae and papillary muscle which had been converted to a mass of calcified tissue 1×0.5 cm. Calcium was removed from the posterior papillary muscle which was then reattached to the right ventricular wall. Redundancy of the anterior and posterior leaflets was reduced by plication and an annuloplasty performed. He developed the post-pericardiotomy syndrome 4 weeks later, which responded to pericardial and pleural aspirations in addition to treatment with prednisone. After the operation, symptomatic limitations from residual tricuspid regurgitation and right heart failure persisted. In March 1968, two years and eight months later, at a further reconstructive operation, a pericardial insert was placed in the septal leaflet of the tricuspid valve and annuloplasty again undertaken. He again developed the post-pericardiotomy syndrome requiring pericardial aspirations and treatment with prednisone. Now, two and half years after his second operation, he is symptomatically well and active, with mild residual tricuspid regurgitation and atrial fibrillation, but without evidence of right heart failure.

FIG. 2 Left anterior oblique chest x-ray of Case 1 showing a calcified opacity (arrow) in the region of the tricuspid valve orifice.

Case 2 On the 6 April 1957 at the age of 23, this patient had a collision with a car while riding his motor cycle. He was admitted to hospital deeply unconscious. He had a penetrating injury of the left chest just above the nipple and a small left apical pneumothorax. He remained unconscious for three weeks and during this time an increase in heart size was noted, both clinically and on the chest x-ray. A pericardial rub was heard in addition to a gallop rhythm. At this time he developed an atrial tachycardia of 150 a minute.

TABLE Haemodynamic findings

Site	Case 1		Case 2		Case 3	
	Pressure (mmHg) +	Saturation (%)	Pressure (mmHg)*	Saturation (%)	Pressure (mmHg)*	Saturation (%)
Superior vena cava		69		59		62
Right atrium	CV = 8 (mean = 4)	70	CV = 17 (mean = 12)	60	CV = 13 (mean = 4)	63
Right ventricle	15/2		25/11		25/5	
Main pulmonary artery	18/8	70	25/15	60	25/7	65
Aorta	90/60	97	100/76	96	115/80	97
Systemic flow (l./min/m ²)		3.1		2.2		

Zero level sternal angle (+) and midchest (*).

Haemopericardium was suspected, but pericardial aspiration was not performed. He was thought to be too ill for exploration of the chest wound which developed a discharging sinus and from which a piece of shirt was removed six weeks later. An

FIG. 3 Simultaneous right atrial (RA) and right ventricular (RV) pressures recorded at cardiac catheterization, showing prominent CV waves in the right atrium. A = Case 1, B = Case 2, and C = Case 3.

electrocardiogram showed complete right bundle-branch block.

A year later he had only minor symptoms of giddiness and breathlessness on strenuous exertion. Physical examination at this time revealed signs of tricuspid regurgitation. He remained reasonably well with episodic palpitations, fatigue, and effort breathlessness, but was able to continue work in a job involving physical labour. He was known to be in sinus rhythm until 1966. In early 1970 he deteriorated over a period of two months complaining of persistent palpitations, abdominal discomfort, and obvious breathlessness on slight exertion. He was admitted to hospital in May 1970. He had rapid atrial fibrillation and right heart failure which responded partially to treatment with digoxin and diuretics. He remained very limited, however, and was transferred to Green Lane Hospital on 27 May 1970.

Physical examination revealed a small plethoric man with a 5 cm scar in the third left intercostal space above the nipple. He was not cyanosed. He was in atrial fibrillation with a ventricular rate of 80 a minute. The blood pressure was 120/70 mmHg. There were signs of severe tricuspid regurgitation with a right ventricular lift, and obvious systolic pulsations of the neck veins and liver, which was palpable 6 cm below the right costal margin. There was a faint systolic murmur (grade 1/4) just audible at the lower left sternal border.

The electrocardiogram was similar to that taken at the time of the accident showing complete right bundle-branch block and left axis deviation but atrial fibrillation was now present. A review of the chest x-rays (Fig. 4A, B, C) showed a mild degree of cardiomegaly in a film taken in 1959, two years after the accident and considerable cardiomegaly in the most recent film, taken 13 years after the accident.

Cardiac catheterization (Table, Case 2) confirmed tricuspid regurgitation, demonstrated by ventricularization of right atrial pressure with systolic waves rising to 17 mmHg (Fig. 3B). There was no evidence of an intracardiac shunt on dye dilution studies or oxygen saturations. Severe tricuspid

FIG. 4 Chest x-rays from Case 2. (A) Normal before the accident. (B) Two years after the accident showing mild cardiomegaly. (C) Thirteen years after the accident showing considerable cardiomegaly.

regurgitation was demonstrated on right ventricular cineangiography.

At operation in May 1970, 13 years after his accident, the anterior leaflet of the tricuspid valve was seen to be flail secondary to chordal rupture, and the valve was replaced with a stent-mounted pulmonary homograft valve. His convalescence was uneventful. Elective cardioversion to sinus rhythm was performed, and at the time of discharge three weeks after the operation there was no evidence of tricuspid regurgitation or right heart failure.

Case 3 In November 1939, aged 26 years, this patient fell 30 feet down the hold of a ship. During the fall, his chest struck a crossbeam which was 15 feet down the hold. He sustained a fractured mandible and a compound fracture of the left elbow. He complained of anterior chest discomfort but there was no external injury and no bone injury to the thoracic cage. No abnormality in the cardiovascular system was noted at the time of the accident. His medical fitness had not been questioned at the time of a detailed insurance examination 3 years earlier. Six months after the accident, however, a heart murmur was commented upon for the first time and again at subsequent examinations. He remained in good health, but in 1960 an abnormality was noted on a routine mass miniature chest x-ray. No further action was taken. In 1963 he underwent hospital assessment because of left inframammary chest pain which was presumed to arise from the donor site of a full thickness skin graft.

He was first seen at Green Lane Hospital in 1963, when he was still asymptomatic. Full examination of the cardiovascular system revealed a grade 2/4 ejection systolic murmur at the lower left border of the sternum, as the only abnormal finding. A diagnosis of idiopathic enlargement of the right atrium was made, to account for the apparent enlargement of this chamber on the chest x-ray.

In September 1970, 31 years after the accident, he returned to his doctor with a history of having been more tired than usual over the preceding year. He was referred back to the Cardiology Unit, Green Lane. *Physical examination* revealed an acyanotic 57-year-old man. Atrial fibrillation was now present, with a ventricular rate of 80 a minute. There were prominent systolic pulsations of his neck veins and of his liver, which was palpable 4 cm below the right costal margin. There was no heart murmur audible at this time. It was clear, however, that he now had important tricuspid regurgitation and right-sided heart failure. Review of the available chest x-rays showed a modest increase in heart size between the years 1956 and 1970 (Fig. 5A, B). Right atrial enlargement was thought to be the main cause of the cardiomegaly.

The electrocardiogram showed partial right bundle-branch block and atrial fibrillation. Digoxin and diuretics were prescribed and the patient had an effective diuresis. On admission to the ward 5 days later, there was no evidence of congestive heart failure. Signs of tricuspid regurgitation were unimpressive. There was a grade 1/4 midsystolic murmur heard at the lower left border of the sternum. *Cardiac catheterization* (Table, Case 3), however, confirmed the presence of important tricuspid regurgitation, as revealed in the right ventricular cineangiogram, and by the ventricularization of right atrial pressure (Fig. 3C). Electrode catheter withdrawal from right ventricle to right atrium was normal. There was no evidence of an intracardiac shunt on dye dilution studies or O₂ saturations. The patient has been advised to continue on medical treatment, as his only symptom is undue fatigue.

Discussion

The first 12 case reports of patients with traumatic tricuspid regurgitation have been well

FIG. 5 Chest x-rays from Case 3. (A) 17 years after the accident, showing moderate cardiomegaly mainly due to right atrial enlargement. (B) 31 years after the accident, showing further slight increase in heart size. (The translucency in the left lower zone represents the donor site for the skin graft.)

reviewed by Jahnke *et al.* (1967). Characteristically, at a variable time after major chest trauma, the patient presents with tricuspid regurgitation and ultimately with right-sided heart failure. Cyanosis was present in 3 cases owing to an associated right-to-left shunt at atrial level. Physical examination reveals signs characteristic of tricuspid regurgitation. The typical pansystolic murmur, however, may be almost inaudible when there is gross regurgitation. The electrocardiogram usually shows complete or incomplete right bundle-branch block. Chest x-rays show non-specific cardiomegaly.

The three patients described in this report are clearly similar in many respects to those reviewed by Jahnke. They are distinctive, however, in the very long period between the initial accident and final presentation with symptomatic right-sided heart failure. This feature emphasizes the benign clinical course in the face of severe isolated tricuspid regurgitation.

In Case 1, tricuspid regurgitation was not recognized for 16 years after his accident, and in Case 3 not for 31 years. The atypical soft heart murmur may well have delayed the diagnosis in these patients and could account for the failure to recognize this condition in other patients. In Case 3, the absence of a heart murmur and the appearance on the x-ray led to the provisional diagnosis of idiopathic enlargement of the right atrium (Sheldon, Johnson, and Favalaro, 1969). This differential diagnosis may be difficult to establish without cardiac catheterization. The experi-

ence by Jahnke *et al.* (1967) of 5 cases in their own institution indeed suggests that traumatic tricuspid regurgitation may be more common than has been suspected.

The benign clinical course of Case 1 is also surprising in view of the papillary muscle rupture which must have occurred at the time of the accident. His progress contradicts the suggestion by Jahnke *et al.* (1967), that 'the clinical course is most dramatic in those patients with papillary muscle rupture, since this lesion is usually associated with the most flagrant tricuspid insufficiency'. This is further shown by the absence of obvious cardiomegaly on the chest x-ray 39 years after the accident. The presence of a normal systolic pressure in the right ventricle may be one reason that isolated and important tricuspid regurgitation can have such a benign clinical course. This is in contrast to the situation in rheumatic heart disease, when tricuspid regurgitation may be associated with other lesions causing pulmonary hypertension and a high systolic pressure in the right ventricle.

Another interesting feature of Case 1 was the finding at heart screening of the mobile piece of calcium which was strongly suggestive of calcification in a detached papillary muscle of the tricuspid valve. The late development in these 3 patients of atrial fibrillation was presumably a consequence of the long-term effects of tricuspid regurgitation. In Case 1, it became established after 22 years, in Case 2 after 13 years when it was the cause of obvious symptomatic deterioration, and in Case 3 after 30 years. In only one other case report

(Brandenburg *et al.*, 1966) is atrial fibrillation specified, and it is pertinent that this patient had a follow-up period of 24 years from the time of his accident.

The indication for operation in 2 of these 3 patients was the persistence of symptoms of chronic right-sided heart failure. Moreover, in view of the benign clinical course as illustrated by these 3 patients, this seems to be the only indication for operation in the absence of associated intracardiac lesions.

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Addendum

Since submission of this paper for publication, four further case histories of patients with traumatic tricuspid regurgitation have been reported, including three patients with a long-term follow-up (Tachovsky, Giuliani, and Ellis, 1970; Liu, Sako, and Alexander, 1970; Morgan and Faker, 1971).

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