precipitous death of the patient in the face of massive occlusion. As a perfusion defect that persists over a prolonged period with no evidence of a disastrous clinical outcome suggests an anatomic abnormality, rather than an embolic origin. In this instance, the next step in a diagnostic workup would be pulmonary angiography. In the presence of a high-probability perfusion scan, the performance of pulmonary angiography is often met with some reluctance by the radiologist.

Other diagnostic interventions have been proposed. The role of two-dimensional echocardiography in the diagnosis of pulmonary artery thromboembolism has been anecdotal. Computerized tomography may be helpful in delineating nonvascular causes of unilateral perfusion defects, but this application is mentioned only sporadically. Pulmonary angiography remains the diagnostic gold standard.

Pulmonary artery aneurysms are rare. A 1947 retrospective review of postmortem reports found a 7.3 percent incidence in the general population. In the preantibiotic era, tuberculosis and syphilis were often causative agents. In recent years, congenital cardiac abnormalities have been associated with over 56 percent of the cases. Similar dilatations have been reported in patients with Marfan’s syndrome and Behçet’s syndrome.

As the clinical presentation of pulmonary artery aneurysm is nonspecific and may mimic that of a pulmonary embolism, a heightened awareness should prompt a more detailed evaluation. In over 95 percent of the reported cases, proximal pulmonary artery aneurysms may be detected as nodular densities on the chest roentgenogram. More distal lesions may not be as classic in appearance. With a suggestive ventilation-perfusion scan, pulmonary angiography becomes necessary for definitive diagnosis. Given the morbidity and mortality associated with peripheral embolization and rupture, the verification of the presence of a pulmonary artery aneurysm becomes imperative to allow prompt surgery.

The utility of the high-probability ventilation-perfusion lung scan remains invaluable in the correct clinical situation. Its acceptance as prima facie evidence for pulmonary embolism may be inappropriate with unilateral perfusion defects that may be attributable to nonthromboembolic causes. Evaluation for alternate etiologies should be considered when presented with this pattern.

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Tricuspid Valve Regurgitation following Blunt Thoracic Trauma*
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Valvular lesions following blunt thoracic trauma are uncommon. Tricuspid valve regurgitation occurs very rarely. We report a successful tricuspid valve reconstruction for rupture of the chordae tendineae in a young man nine years after a motor vehicle accident. The value of echocardiography and transesophageal echocardiography for the diagnosis and quantification of this valve lesion is stressed.

*Chest 1992; 102:1294-96

ECC = extracorporeal circulation; RA = right atrium; RV = right ventricle; TVR = tricuspid valve replacement

I n this time of high-speed motor vehicle accidents, blunt thoracic trauma is encountered with increasing frequency. Cardiac involvement is infrequent and consists largely of myocardial contusion of varying degree. Valvular lesions are uncommon and traumatic tricuspid valve regurgitation occurs very rarely. Up to now, some 70 cases have

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Tricuspid Valve Regurgitation after Blunt Trauma (Kleikamp et al)
been reported.1-10 Treatment of this acquired lesion was conservative in most cases; some patients required tricuspid valve replacement (TVR). We report a case of valve reconstruction for tricuspid valve regurgitation nine years after blunt thoracic trauma.

CASE REPORT

In 1982, a then 23-year-old man suffered blunt thoracic trauma when crashing against the steering wheel of his car in an automobile accident. Apart from some minor contusions, he seemed to be uninjured. In the following years, he was clinically well until he began to suffer from progressive dyspnea and fatigue in 1989. He sought medical attention that year, but a diagnosis was not made until February 1991.

A systolic murmur was found; there were no signs of cardiac failure. The electrocardiogram showed sinus rhythm and apart from an incomplete right bundle branch block, there were no irregularities. The chest roentgenogram revealed enlargement of the transverse diameter of the heart and slight dislocation to the left.

Echocardiography demonstrated massive tricuspid regurgitation and moderately dilated right atrium (RA) and right ventricle (RV). The end-diastolic diameter of the RV was 43 mm. A paradoxical movement of the interventricular septum was noted. Color Doppler echocardiography confirmed the presence of massive tricuspid regurgitation into the RA and the inferior vena cava. Angiography confirmed the diagnosis; the RA pressure was 155 mm Hg (mean, 8 mm Hg). The V wave was found to be 28 mm Hg. The RV pressure was 220 mm Hg with an end-diastolic pressure of 13 mm Hg. The left atrium, the left ventricle, and the coronary arteries were normal.

An operation was performed through a median sternotomy. After pericardiotomy, adhesions between the apex of the heart and the pericardium were found. A 12-cm-long old tear was visible in the left side of the pericardium, most likely originating from the trauma in 1982. After institution of complete cardiopulmonary bypass, the grossly enlarged RA was opened. The anterior leaflet of the tricuspid valve was found to be insufficient because of the rupture of all chordae tendineae close to their juncture with the papillary muscles. Surprisingly, the chordae were thickened and scarred, but had apparently not lost any length. Therefore, they were sutured to their respective papillary muscles using interrupted, reinforced polypropylene sutures. Intraoperative pressure monitoring after discontinuation of the extracorporeal circulation (ECC) and transesophageal echocardiography, both of which had demonstrated severe tricuspid regurgitation before starting ECC (Fig 1), now showed only mild regurgitation (Fig 2).

The patient made an uneventful recovery; the echocardiography confirmed the mild regurgitation. Six months after surgery, the patient is well and asymptomatic working in his profession as a swimming teacher. He will undergo a reexamination in the months to come.

DISCUSSION

Traumatic tricuspid regurgitation is a rare sequela after blunt thoracic trauma. Up to now, some 70 cases have been reported.1-10 The most likely cause is compression, decompression, or deceleration of the thorax. These mechanisms result in intraventricular pressure peaks, which in combination with a closed valve and the systolic pressure in the RV, most often affect the chordae tendineae and the papillary muscles.3,4 Trauma of the valve leaflets has very rarely been reported. The forces applied to the thorax in our patient were so enormous that they caused a large pericardial tear.

Some patients are asymptomatic for long periods of time.5,7-10 In symptomatic patients, the spectrum extends from acute failure of the RV to mild degrees of fatigue and dyspnea on exertion.2,3,8-10 Diagnostic clues are a systolic murmur at Erb's point, a right bundle branch block, and/or absolute arrhythmia with atrial fibrillation in the surface ECG. Two-dimensional echocardiography and Doppler echocardiography are the most important diagnostic tools when cardiac involvement is suspected.3,11-12 Not only do these methods provide information on ventricular performance in general in the more common cases of myocardial contusion and transient myocardial dysfunction, but they also help to diagnose and semiquantify noninvasively valvular involvement. This will undoubtedly lead to earlier diagnosis and more adequate treatment of patients following blunt cardiac trauma. Intraoperative transesophageal echocardiography helped to quantify the regurgitation fraction and thus the decision to reconstruct or replace the valve.

To our knowledge, this is the first case in which reconstruction of the chordae tendineae and annulorrhaphy of the tricuspid valve were used to treat traumatic regurgitation. Valve reconstruction has been widely advocated in other forms of acquired heart valve disease34-15 and has even been used in traumatic mitral incompetence.16 In comparison with valve replacement and the lifelong anticoagulation

Figure 1. Intraoperative transesophageal echocardiogram demonstrates massive systolic regurgitation into the right atrium.

Figure 2. Following reconstruction of the valve, only a mild regurgitation is left.
needed, we think that valve reconstruction, whenever possible, should be tried, even if later on valve deterioration should make valve replacement unavoidable.

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Interleukin-5 Levels of Pleural Fluid and Serum Samples in a Patient with PIE Syndrome*

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An increased production of IL-5 was detected in the pleural fluid (7.2 ng/ml) and in the serum samples (53 pg/ml) of a patient with PIE syndrome. Following steroid therapy, pleural fluid disappeared, eosinophilia improved and serum IL-5 concentration became undetectable. These results suggested that eosinophilia in the PIE syndrome is a consequence of increased production of IL-5, especially in the lung. (Chest 1992; 102:1296-97)

ELISA = enzyme-linked immunosorbent assay; IHES = idiopathic hypereosinophilic syndrome; IL = interleukin; PBS = phosphate-buffered saline; PIE = pulmonary infiltration with eosinophilia; mRNA = messenger ribonucleic acid

Interleukin-5, a T-cell-replacing factor, shows eosinophil colony-stimulating activity in humans. We and others recently observed that transgenic mice expressing IL-5 have severe eosinophilia. These reports suggest that eosinophilia in some patients with allergy or parasite infection may be due to an enhanced production of IL-5.

Because the concentration of IL-5 can be determined with an ELISA method in mice and humans, we examined the concentration of IL-5 in the serum samples and pleural fluid of a patient with PIE syndrome.

MATERIALS AND METHODS

Serum Samples and Pleural Fluids

Serum samples were obtained from a patient with PIE syndrome and from ten normal volunteers. Pleural fluid was obtained from a patient with PIE syndrome and from five other patients with carcinomatous pleurisy who showed no eosinophilia in their peripheral blood or pleural effusion. The pleural fluid samples were centrifuged to remove cells and debris. These test samples were stored at −20°C until use.

Reagents

Recombinant human IL-1, IL-2, IL-5, IL-6 and TGF-β were analyzed. Recombinant human IL-3, IL-4, G-CSF and M-CSF were obtained from Genzyme Corporation (Boston, Mass). Furthermore, TB13, an anti-mouse IL-5 monoclonal antibody which can react with human IL-5, was purified as previously described.

ELISA Assay to Detect Human IL-5

For the assay of human IL-5, we slightly modified the ELISA method established to detect mouse IL-5. Briefly, polystyrene plates were coated with TB13 (5 μg/ml) in PBS (10 mM phosphate, 140 mM NaCl, pH 7.4) overnight at 4°C. Nonspecific binding sites were blocked with PBS containing 2 percent bovine serum albumin for 2 h at room temperature. After washing the wells with PBS containing 0.05 percent Tween 20 (PBS-Tween), test samples were applied to the wells and incubated overnight at 4°C. Then, after washing again, polyclonal rabbit anti-human IL-5 IgG antibodies were added and incubated again overnight at 4°C.

Horse radish peroxidase-coupled goat anti-rabbit Ig (Bio-Rad Labs, Richmond, Calif) was added, followed by an hour’s incubation at room temperature. After washing, 0.5 percent 3-(4-hydroxy phenyl) propionic acid in phosphate buffer (10 mM, pH 7.0) / 0.03 percent H2O2 was added and incubated 10 h at room temperature.

The reactions were stopped by adding 0.25N NaOH containing 1.5 percent NaN3, and the fluorescence was measured at 405 nm (excitation 320 nm) on a fluorescence spectrometer (F-3000, Hitachi). Figure 1 shows a standard curve of this ELISA assay using rIL-5 (sensitivity: 2 pg/ml). The fluorescence of 2 ng/ml of recombinant IL-1, IL-2, IL-3, IL-4, IL-6, G-CSF, GM-CSF or TGF-β.