Surgical Repair of Tricuspid Valve Regurgitation Caused by Blunt Thoracic Trauma

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Tricuspid valve regurgitation is a rare complication of blunt thoracic trauma. In addition to the uniqueness of this condition, the diagnosis can be overlooked because of other effects related to the trauma. The literature contains approximately 100 cases of trauma-induced tricuspid regurgitation. Traumatic tricuspid valve regurgitation can be diagnosed in the early or late periods after trauma. Patients who are diagnosed early usually have severe symptoms of tricuspid regurgitation or signs of accompanying cardiac trauma. These cases require immediate surgery. Patients who are diagnosed later usually have isolated chronic tricuspid regurgitation. In most cases, this is well tolerated and the patient typically experiences few or no symptoms in the early post-trauma period. Chronic cases feature progressive dilation of the right ventricle and tricuspid annulus, and right ventricle dysfunction eventually develops. In this article, we document the combination of surgical techniques that was used to repair tricuspid valve regurgitation in a patient who was diagnosed 4 years after blunt thoracic trauma.

CASE REPORT

A 27-year-old man presented with dyspnea and fatigue and was admitted to the hospital. The patient’s history included an automobile crash 4 years earlier. He had been hospitalized for 37 days because of his injuries and had recovered full physical capacity by 4 months after the crash. In the 2 months before presentation, he had developed dyspnea and fatigue; these symptoms had worsened with time. Physical examination revealed a grade III/VI holosystolic murmur in the left parasternal area, a bounding jugular pulse, and hepatic enlargement. An electrocardiogram revealed murmur in the left parasternal area, a bounding jugular pulse, and hepatic enlargement. An electrocardiogram revealed a grade III/VI holosystolic pnea and fatigue; these symptoms had worsened with time. In the 2 months before presentation, he had developed dys- recovered full physical capacity by 4 months after the crash. In the 2 months before presentation, he had developed dys-

phy showed severe tricuspid regurgitation and moderate dilation of the right atrium and right ventricle. Angiography confirmed enlargement of the right heart chambers and demonstrated that the coronary arteries were normal.

Surgery was performed through a median sternotomy. Pericardiotomy revealed adhesions between the heart and the pericardium. Cardiopulmonary bypass was established with aortic and bicaval venous cannulation. Antegrade tepid-blood cardioplegia (20°C) was administered. A right atriotomy was performed to visualize the tricuspid valve. Right ventricular enlargement and severe dilation of the tricuspid annulus were noted. The septal and posterior valve leaflets were intact; however, all of the chordae tendineae associated with the anterior leaflet and one of the papillary muscles were ruptured (Fig. 1). There was also a tear in the anterior leaflet (Fig. 2). The papillary muscles associated with the completely ruptured chordae could not be visualized.

As the first step in repair, the tear in the anterior leaflet was closed with interrupted 6/0 monofilament sutures. The left half of this leaflet was then reconstructed by anchoring three 6/0 expanded polytetrafluoroethylene sutures (e-PTFE, Gore-Tex suture, W.L. Gore & Associates, Inc., Elkton, Md.) (six artificial chordae) to the estimated correct locations of the related papillary muscles. The chorda that was partially ruptured from the papillary muscle was reattached to a suitable location in the ventricle wall. Next, we completely detached the posterior leaflet from the annulus and transposed it, with attached chordae tendineae intact, to the free margin of the flailing right half of the anterior leaflet. The leaflet was fixed in place with interrupted 5/0 monofilament sutures (Fig. 3). One pledgeted 2/0 polyester suture was used to obliterate the portion of the annulus that had been the original site of the posterior leaflet (Kay annuloplasty). Since the annulus was dilated to an extent that precluded adequate leaflet coaptation, a modified version of the De Vega annuloplasty technique was performed. After all steps of the repair had been performed, testing with saline yielded only minimal residual regurgitation (Fig. 4). Intraoperative transesophageal echocardiography (TEE) was also performed this stage, and this revealed mild tricuspid valve regurgitation as well. There were no postoperative complications, and the patient was discharged from hospital on the sixth day after surgery. Repeat TEE at 11 months postoperatively showed that the an-
terior leaflet was closing properly, with only mild central leakage from the valve. At 1 year postsurgery, the patient remains in good health.

**COMMENT**

Most tricuspid valve injuries associated with blunt trauma result from transmission of a sudden compressive force to the heart during late diastole or the beginning of systole, when the heart is full and the valves are closed. The patient in this case presented with symptoms of right heart failure several years after he suffered blunt thoracic trauma in a car crash. A combination of surgical techniques was used to treat his tricuspid regurgitation: primary closure of a defect in the anterior leaflet, anchoring of artificial chordae to this same...
leaflet with e-PTFE sutures, reattachment of partially ruptured papillary muscles, posterior leaflet transposition, and Kay and modified De Vega annuloplasties, respectively.

Trauma can lead to tricuspid regurgitation in various ways, including rupture of chordae tendineae or papillary muscle, torn leaflet, or simple contusion of a papillary muscle. With leaflet tears or ruptured chordae, the onset of symptoms is often insidious. In contrast, most patients with ruptured papillary muscles develop symptoms rapidly. In our case, the patient began to show symptoms 4 years after trauma, and his heart damage included ruptured chordae and papillary muscles, as well as a defect in the anterior leaflet. One report in the literature describes a patient who needed tricuspid repair 37 years after trauma. A few series of trauma-induced tricuspid valve regurgitation cases have been published, but most information about this condition is in the form of case reports. The early reports discuss valve replacement as the most common surgical approach. However, tricuspid valve replacement with mechanical valves is known to be associated with a high incidence of thromboembolic events, and long-term outcome is reportedly poor. Other available prostheses include bioprosthetic valves, but these devices have shown structural failure in the tricuspid position. Because of such problems with valve replacement, surgical repair is the treatment of choice for correcting post-traumatic tricuspid regurgitation.

One of the largest series of traumatic tricuspid regurgitation published to date is from the Mayo Clinic. In this study, 13 patients were followed during a 29-year period. Eight of the patients underwent valve replacement and five were treated with surgical repair. The authors stated that it was not possible to repair the valves in patients who were operated a long time after the trauma because the valvular structures (papillary muscles, chordae tendineae, and involved leaflet[s]) had contracted and atrophied. In our case, we operated approximately 4 years after the initial trauma. Although we were unable to clearly visualize the papillary muscles from which chordae had ruptured, the quality of the anterior leaflet tissue was adequate for repair.

Alfieri et al. described five cases of traumatic tricuspid regurgitation in which they successfully performed a procedure called the “Clover technique.” This method is similar to the “Alfieri technique” that the same group developed for mitral valve repair. Briefly, the Clover technique consists of suturing together the middle points of the free edges of the tricuspid leaflets, producing a clover-shaped valve. However, the long-term results of this surgery are not yet known.

In traumatic tricuspid regurgitation, the anterior leaflet is the one most often affected. As described above, the trauma can cause a tear, or damage the subvalvular structures related to the leaflet, or both. The anterior leaflet is the widest of the three; thus, this leaflet and its related structures are under more pressure than the others during blunt trauma. In traumatic tricuspid regurgitation, if the subvalvular structures for half of the anterior leaflet are ruptured but those associated with the other half are intact, the leaflet can be made competent by transposing a segment of the neighboring septal or posterior leaflet to the incompetent half. The free margin of the anterior leaflet is longer than the others. If this leaflet is so badly damaged that all related chordal structures are ruptured, then transposition of the septal or posterior leaflet to the free margin of the anterior leaflet may not provide adequate support. In cases of serious anterior leaflet damage, as in our patient, combined repair techniques (artificial chordae, leaflet transposition, and annuloplasty) can be attempted before the less preferred option of valve replacement.

This case of traumatic tricuspid regurgitation is important because it demonstrates that, in patients with massive damage to the subvalvular structures of the anterior leaflet, a combination of repair techniques can achieve valve competency and avoid valve replacement.

REFERENCES