Tricuspid Regurgitation Due to Blunt Chest Trauma
Report of a Case and Review of the Literature


SUMMARY
A 25-year-old female developed high-grade atrioventricular block and markedly elevated central venous pressure after sustaining a crushing injury to the chest while driving a car. An echocardiographic examination with color Doppler revealed severe tricuspid regurgitation due to a torn papillary muscle. An extensive review of the literature showed the following: 1) correct diagnosis is often delayed because of coexisting multisystem involvement and the subtleness of abnormal physical signs, 2) identification of abnormally elevated right atrial pressure with a prominent “v” wave, and characteristic electrocardiogram appeared to be the key to early diagnosis, and 3) the final diagnosis may be confirmed by echocardiography with Doppler and/or cardiac catheterization. The role of echocardiographic examination with color Doppler technique deserves special emphasis because the final diagnosis can be easily reached during the acute phase at the bedside noninvasively. (Jpn Heart J 34: 361–375, 1993.)

Key Words:
Tricuspid regurgitation  Blunt chest trauma  Echocardiography

Isolated tricuspid regurgitation is a rare clinical condition and is known to be associated with pulmonary hypertension, tricuspid valve prolapse, rheumatic tricuspid valve disease, infectious endocarditis, right ventricular infarction and Ebstein’s anomaly.1,2) It is now recognized more frequently among intravenous drug abusers suffering from tricuspid valve endocarditis.3) Less-well recognized is tricuspid regurgitation due to blunt chest trauma.4) An increase in the number of high speed vehicular accidents makes trauma a potentially important cause of isolated tricuspid regurgitation. However, relative unfamiliarity of the disease, frequent multisystem injury during the acute phase of trauma, in addi-
tion to the subtleness of signs and symptoms, might have contributed to the underdiagnosis of this condition. Increased awareness of the disease and recent advances in diagnostic technology may make the correct diagnosis easier.

In this article, a case of traumatic tricuspid regurgitation, diagnosed in the acute phase with color Doppler, will be presented. Extensive review of the previous literature was performed to define clinical characteristics in this potentially important condition.

REPORT OF A CASE

A 25-year-old female in previous good health was severely injured in an automobile accident while she was driving a car. At the time of collision, she was

Fig. 1. A twelve-lead electrocardiogram showing QRS morphology of right bundle branch block and high grade atrioventricular block. Occasional atrioventricular conduction was suggested by the irregularity of the RR interval.
thrown against the steering wheel and dashboard, sustaining a crushing injury to the chest, upper abdomen and knees.

On admission, blood pressure was 80/40 mmHg, pulse rate 80/minute and respiratory rate 20/minute. The patient was agitated. Bilateral knee joints were severely lacerated, and there were bruise and subcutaneous hemorrhages on the anterior chest wall. The respiratory sounds were diminished bilaterally and an inspiratory crackle was heard over the left lung field. A loud pericardial friction rub was noted and heart sounds were masked by the friction rub. Jugular venous pulsation was difficult to inspect due to the patient’s obesity and short neck, but direct measurement of the central venous pressure was 10 cmH$_2$O.
Fig. 3. A: a color Doppler showing severe tricuspid regurgitation in the right ventricular inflow view. LV=left ventricle; RV=right ventricle; RA=right atrium; TR=tricuspid regurgitation. B: a continuous wave Doppler showing tricuspid regurgitation signal.

Fig. 4. A pressure waveform obtained at bedside, showing prominent "v" wave in right atrium. v=a "v" wave; y=a "y" descent. Other abbreviations are the same as in Figure 3.
An electrocardiogram showed sinus rhythm and right bundle branch block. A chest roentgenogram revealed bilateral pleural effusion which was bloody when aspirated, and cloudy shadows suggestive of lung contusion. An echographic examination of the abdomen revealed moderate ascites, but laceration of the viscera was not identified. Computer tomography of the brain was normal and an aortogram did not reveal traumatic aortic injury.

After surgical treatment of the knee lacerations, the patient was taken to the intensive care unit. Initial diagnosis included fracture of the sternum and ribs, bilateral lung contusion and hemothorax, bilateral knee laceration, probable liver contusion and probable myocardial contusion with pericardial involvement. Chest tubes were inserted, blood transfusion was performed for anemia and antibiotics were given for possible infection of the knee wounds. Subsequently, the patient's condition seemed to be stabilized.

On the 2nd hospital day, an electrocardiogram revealed advanced atrioventricular block (Fig. 1) and the central venous pressure increased to 20 cmH₂O. The urine output decreased although the vital signs did not change markedly. Pericardial friction rub was still present and made detailed auscultation difficult. An echocardiographic examination of the heart revealed a fluttering structure in the dilated right ventricular cavity, which appeared to be a torn papillary muscle (Fig. 2). Floppy tricuspid valve leaflets and paradoxical motion of the septum were also noted. The amount of the pericardial effusion was small. Severe tricuspid regurgitation was confirmed by color Doppler examination (Fig. 3). Measurement of the intracardiac pressures with a Swan-Ganz catheter revealed the following data: mean pulmonary artery wedge pressure 14 mmHg; pulmonary ar-

![ECG and JVP](image)

Fig. 5. A phonocardiogram and jugular venous tracing. A high pitch systolic murmur (SM), and right ventricular third and fourth heart sounds (S₃ and S₄) were recorded. A prominent "v" wave is characteristic.

JVP=jugular venous pulsation; L=low pitch; H=high pitch; 4LSB=the fourth interspace at the left sternal border.
tery pressure 30/16 (mean 20) mmHg, right ventricular pressure 35/17 mmHg; mean right atrial pressure 16 mmHg with a ‘v’ wave as high as 25 mmHg. The right atrial pressure waveform was similar to that of the right ventricle (Fig. 4). No shunt was detected. The maximum level of creatinine phosphokinase was 936U/dl (<120) with a MB fraction of 11%.

The patient was started on dopamine and adequate fluid management, but did not require pacemaker therapy. Subsequently, the patient’s condition stabilized with conservative therapy, and the patient was discharged from the intensive care unit to the general surgical ward on the 7th hospital day.

One month later, the patient was discharged home without any cardiac medication. At the time of discharge, physical examination revealed an elevated jugular venous pressure with prominent ‘v’ wave, right ventricular heave on precordial pulsation, and an enlarged and pulsatile liver. Cardiac auscultation revealed the third and fourth heart sounds and a grade II systolic murmur on the left sternal border at the 4th interspace (Fig. 5). These were thought to be of right sided origin because the intensity increased on inspiration. An electrocardiogram showed sinus rhythm and right bundle branch block with a PQ interval of 0.2 sec. A chest roentgenogram showed marked cardiomegaly compared with the previous one which was taken several years earlier at the time of a routine health

Fig. 6. A chest roentgenogram before and after the trauma, showing marked increase of the cardiothoracic ratio after the trauma.
examination (Fig. 6).

**DISCUSSION**

An extensive search of previous literature was performed by means of MEDLINE and Index Medicus. Only those articles written either in English or Japanese, with detailed clinical information, were collected for review. Cases reported in an abstract were excluded. Since Todd's historical report in 1948, we are aware of 62 reported cases of tricuspid regurgitation due to blunt chest trauma, and these are the basis for this discussion. Various clinical data obtained from these cases are summarized in Tables I–VI.

<table>
<thead>
<tr>
<th>Table I. Clinical Features in 62 Patients with Traumatic TR</th>
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<tbody>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Mean age of occurrence (years)</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Diagnosis of TR</td>
</tr>
<tr>
<td>Mean interval (years)</td>
</tr>
<tr>
<td>from trauma to the onset of symptoms</td>
</tr>
<tr>
<td>from trauma to diagnosis of TR</td>
</tr>
<tr>
<td>Causes of trauma</td>
</tr>
<tr>
<td>Automobile accident</td>
</tr>
<tr>
<td>Fall</td>
</tr>
<tr>
<td>Industrial accident</td>
</tr>
<tr>
<td>Others</td>
</tr>
</tbody>
</table>

TR=tricuspid regurgitation

<table>
<thead>
<tr>
<th>Table II. Initial Presenting Symptoms in Chronic TR (n=58)*</th>
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<tbody>
<tr>
<td>Symptom</td>
</tr>
<tr>
<td>Dyspnea</td>
</tr>
<tr>
<td>Fatigue</td>
</tr>
<tr>
<td>Palpitation</td>
</tr>
<tr>
<td>Cyanosis*</td>
</tr>
<tr>
<td>Syncope</td>
</tr>
<tr>
<td>Leg edema</td>
</tr>
<tr>
<td>Neck pulsation</td>
</tr>
<tr>
<td>Abdominal distension</td>
</tr>
<tr>
<td>No symptoms</td>
</tr>
</tbody>
</table>

TR=tricuspid regurgitation.

* Diagnosis of TR was confirmed in acute phase in 4 patients and these were excluded. 1,13,17,49,53

1 due to right to left shunt.
Clinical presentation

As shown in Table I, the majority of patients with traumatic tricuspid regurgitation were active young males. The leading cause of the chest trauma was an automobile accident followed by falls\textsuperscript{17,20,30,37,45} and industrial accidents such as explosion\textsuperscript{15,23,51,49}. The most characteristic feature of this disease is its chronicity. Patients may be asymptomatic or minimally symptomatic for years\textsuperscript{17,20} and the diagnosis may be delayed even after the patient becomes symptomatic (Table I).

Common initial presenting symptoms in chronic patients included nonspecific cardiac symptoms such as dyspnea, fatigue and palpitation (Table II). Only a small number of patients were aware of neck pulsation\textsuperscript{11,54}, leg edema\textsuperscript{21,22,48} and/or abdominal distension\textsuperscript{16}, which were presumably more specific to the increased venous pressure. Syncope or lightheadedness was noted in 6 patients\textsuperscript{8,11,13,22,27,39}. On the contrary, many of the abnormal cardiovascular findings at the time of diagnosis were directly related to severe tricuspid regurgitation (Table III). An elevated jugular venous pressure with prominent “v” wave, an enlarged liver with systolic pulsation and the murmur of tricuspid regurgitation with respiratory variation were the most common abnormal findings. The tricuspid regurgitation murmur was usually Levine grade I–II, and might have been easily missed without careful auscultation. Actually a murmur might be absent if the tricuspid valve incompetence is severe enough to cause free regurgitation\textsuperscript{39}.

Five patients were asymptomatic at the time of diagnosis\textsuperscript{18,36,44}. In these
patients, tricuspid regurgitation was suspected because of a new murmur, characteristic physical findings and/or abnormal electrocardiogram, and was confirmed either by cardiac catheterization or echocardiography with pulse wave Doppler.

The diagnosis of tricuspid regurgitation was possible during the acute phase in only 4 patients. The key to diagnosis in these 4 patients included shock complicated by elevated right atrial pressure with a prominent “v” wave and right to left shunt or elevated jugular venous pressure with a “v” wave, associated with right side third and fourth heart sounds. In our patient, newly developed high grade atrioventricular block with an elevated central venous pressure prompted an echocardiographic examination, resulting in the final diagnosis during the acute phase.

Interestingly, cyanosis was noted in 12 patients, and in 9 of them it was later proved to be due to a right to left shunt. Cyanosis was one of the initial presenting symptoms in 6 patients. The mechanism of this phenomenon will be discussed later.

**Coexisting cardiac and noncardiac injury**

Usually the patients with traumatic tricuspid regurgitation suffered from various cardiac and/or non-cardiac injuries. The exact incidence of these coexisting injuries was very difficult to estimate, because the collection of accurate information at the time of the event in the remote past was difficult and usually case reports were done by cardiologists who were unfamiliar with the trauma.

The cardiovascular injury coexisting with tricuspid regurgitation included laceration or agenesis of the pericardium, pericarditis, rupture of the free wall of the right ventricle, ventricular septal perforation, aneurysm of the thoracic descending aorta, and traumatic mitral valve prolapse with regurgitation. Contusion of the right ventricular myocardium was not specifically mentioned but seemed to be an inevitable coexisting lesion.

Noncardiac injuries which were reported to be coexisting with traumatic tricuspid regurgitation included fracture of the sternum, ribs, skull, pelvis and extremities, hemopneumothorax, flail chest, lung contusion and pneumomediastinum.

Abdominal visceral lacerations or contusions were also common. Of note, disturbed consciousness due to shock or head trauma frequently coexisted.

This multisystem involvement requiring immediate treatment in the acute phase might have prevented a physician from considering the possibility of tricuspid regurgitation and performing adequate diagnostic tests. This might have contributed to the delay of the diagnosis, on top of the subtleness and chronicity of the disease.
Laboratory data

Electrocardiogram: Although it has been reported that an electrocardiogram is not a sensitive tool in the diagnosis of myocardial contusion, electrocardiographic findings in patients with traumatic tricuspid regurgitation were rather specific and appeared to be very important (Table IV). The electrocardiogram was abnormal in virtually all patients. Right bundle branch block, right ventricular enlargement and atrial fibrillation were the most common abnormal findings. A mild degree of atrioventricular block was seen in a small number of patients, but the high grade block seen in our patient was a very unusual finding. These electrocardiographic changes are thought to be associated with traumatic injury to the specific conduction systems and/or enlargement of the right atrium and ventricle due to the volume overload of tricuspid regurgitation.

Chest Roentgenogram: The chest roentgenogram frequently showed cardiomegaly secondary to an enlarged right atrium and right ventricle or pericardial effusion but congestion of the lung was uncommon. Chest roentgenographic findings are usually nonspecific and their greatest value exists in the recognition of associated injuries such as hemopneumothorax and fractures of the bony structures.

Cardiac Catheterization: Cardiac catheterization with right ventriculography was the final diagnostic procedure until recent advances in echocardiography. Measurements of the intracardiac pressure usually revealed elevated right atrial pressure with a prominent “v” wave. In almost half of the patients, regurgitation was so severe as to cause very tall “v” waves (Table V). The pressure wave form in these patients resembled that of the right ventricle, resulting in “ventricularization” of the right atrial pressure (Fig. 4, Table V).

Left to right shunt due to coexisting ventricular septal perforation was noted
Table V. Cardiac Catheterization in Traumatic TR (n=60)\(^1\)

<table>
<thead>
<tr>
<th>RA &quot;v&quot; wave &gt;8 mmHg</th>
<th>positive</th>
<th>negative</th>
<th>N/A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricularization of RAP</td>
<td>30/54</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>RVEDP &gt;6 mmHg</td>
<td>32</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>RAP &gt; PAWP</td>
<td>23</td>
<td>10</td>
<td>27</td>
</tr>
<tr>
<td>Shunt</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>left to right*</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>right to left**</td>
<td>10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RA = right atrium; RAP = right atrial pressure; RVEDP = right ventricular end-diastolic pressure; PAWP = pulmonary artery wedge pressure; N/A = not available.

* Two patients did not have catheterization.\(^{36}\) due to ventricular septal perforation.\(^{26,34,40}\) due to patent foramen ovale or atrial septal defect.\(^{7,8,10,15}\) In the presence of a coincidental patent foramen ovale or atrial septal defect, stretching of the intra-atrial septum and elevation of right atrial pressure resulted in a right to left shunt causing "acquired cyanotic heart disease". This complication was present in as many as 10 patients and acquired cyanosis was one of the presenting symptoms in 6 patients (Tables II and V).\(^{7,8,10,15,23,29,32,35,37,41}\) One patient had been erroneously diagnosed for a long period of time as having Ebstein's anomaly.\(^{23}\)

Echocardiography: Echocardiography is a useful noninvasive diagnostic method for the evaluation of cardiac involvement in patients with chest trauma.\(^{56,57}\) Recently, increasing numbers of patients have had echocardiographic confirmation of traumatic tricuspid regurgitation, before undergoing cardiac catheterization.\(^{33-56,38,40,43-55}\) In the past, tricuspid regurgitation was suspected with the demonstration of a dilated right atrium and ventricle, floppy tricuspid valve leaflets, torn chordae or ruptured papillary muscle, and was later confirmed by contrast echogram.\(^{45-47,49,50}\) Eskilsson\(^{36}\) first reported two cases diagnosed by pulse wave Doppler technique. The visualization of the regurgitation jet with color Doppler technique, as in our patient, leads to the direct and final diagnosis and may have further advantages. The noninvasive nature of the test and easy availability at bedside makes echocardiography with color Doppler a valuable diagnostic test, especially in the acute phase.

Other Laboratory Data: An increased level of the MB fraction of creatinine phosphokinase reflects damage to the myocardium and appears to be useful in the diagnosis of myocardial contusion.\(^{57,58}\) However, a negative creatinine phosphokinase does not necessarily exclude myocardial involvement.\(^{58}\) A radionuclide study may be more sensitive to demonstrate right ventricular wall motion abnormalities,\(^{58}\) but it may not be easily available during the acute phase and its value is limited in many Japanese hospitals.
Table VI. Anatomical Findings in Traumatic TR

<table>
<thead>
<tr>
<th>Involved leaflets (n=36)</th>
<th>TVR</th>
<th>TVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>anterior</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>anterior+posterior</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>posterior</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>septal</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>anterior+septal</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>all</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Involved part (n=43 surgical patients)

<table>
<thead>
<tr>
<th>Component</th>
<th>TVR</th>
<th>TVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chordae tendineae</td>
<td>18</td>
<td>15</td>
</tr>
<tr>
<td>Papillary muscle</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Leaflets</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Papillary muscle+leaflets</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Not specified</td>
<td>10</td>
<td>6</td>
</tr>
</tbody>
</table>

TVR=tricuspid valve replacement; TVP=tricuspid valve plasty; TR=tricuspid regurgitation.

**Anatomical information**

The leaflets involved were specified in 36 patients (Table VI). The anterior leaflet was most commonly involved. Involvement of the posterior and septal leaflet was rare.

Of 43 patients treated surgically, the torn chordae tendineae was the most common site of involvement. Less frequently, rupture of the papillary muscle was the cause of the regurgitation, while laceration of the leaflet itself was rare. In our patient, rupture of the septal papillary muscle with a resultant floppy tricuspid valve appeared to be the cause of tricuspid regurgitation.

**Therapy**

Some patients remained in relatively good condition for a long period of time without surgery. The slow clinical course of the disease has been attributed to the low pressure present in the right side of the heart, as contrasted with tricuspid regurgitation secondary to pulmonary hypertension. However, the choice of therapy after the development of symptoms was surgical in the majority of cases. Some authors suggested that patients with a ruptured papillary muscle might receive earlier surgical intervention compared with those patients with torn chordae.

Among 43 surgically treated patients, tricuspid valve replacement was the most common treatment. The Starr-Edwards ball valve prosthesis had been used until mid 1970. A disc valve prosthesis or a porcine bioprosthesis was used in more recent cases. Tricuspid valve plasty was performed in a smaller number of patients.

The choice of the procedure did not seem to be different among different in-
volved sites (Table VI).

Although the long term prognosis after surgical therapy has been seldom reported,\(^{31}\) the short term outcome after surgery appeared to be relatively benign. Only one perioperative death was reported among 43 surgical patients.\(^{21}\) Two patients required permanent pacemaker implantation for postoperative bradyarrhythmia.\(^{27,49}\)

**Concluding remarks**

Because of an increase in the number of high speed vehicular accidents, blunt chest trauma is potentially an important cause of isolated tricuspid regurgitation. In the acute phase following the trauma, a correct diagnosis may be difficult because signs of the disease may be subtle and masked by coexisting multisystem injury requiring immediate treatment. The role of the echocardiography with color Doppler deserves special emphasis in the early diagnosis of the disease.

In chronic patients, usually a careful physical examination reveals specific signs attributable to severe tricuspid regurgitation, although initial presenting symptoms are usually nonspecific. The electrocardiographic findings are rather specific and include right bundle branch block, right ventricular enlargement, and atrial fibrillation. Final diagnosis can be achieved by echocardiography with color Doppler, and this may be further confirmed by measurement of the intracardiac pressure. Surgical treatment is usually necessary once the patient becomes symptomatic.

**References**

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