Late Cardiac Tamponade After Open Heart Surgery: Incidence, Role of Anticoagulants in Its Pathogenesis and Its Relationship to the Postpericardiotomy Syndrome

Stephen K. Ofori-Kraeye, M.D., Theodore I. Tyberg, M.D., Alexander S. Geha, M.D., Graeme L. Hammond, M.D., Lawrence S. Cohen, M.D., and Rene A. Langou, M.D.

SUMMARY Cardiac tamponade that occurs late after cardiac surgery (7 days) is relatively uncommon but potentially fatal. We analyzed its incidence, clinical course and relationship to the postpericardiotomy syndrome in 1290 consecutive adult patients who survived surgery. Criteria for diagnosis of cardiac tamponade were (1) elevated jugular venous pressure, (2) hypotension or decreased cardiac index, (3) characteristic hemodynamics at cardiac catheterization, and (4) echocardiographic evidence of pericardial effusion.

Ten of the 1290 patients (0.8%) developed cardiac tamponade. Surgery was for congenital heart disease in five patients, valvular heart disease in two patients, and coronary artery disease in three patients. The onset of hemodynamic compromise ranged from 15-180 days postoperatively (mean 49 days). All patients had echocardiographic evidence of pericardial fluid, eight had a pericardial friction rub at the time of cardiac tamponade, nine had pericardial pain, and all were considered to have a postpericardiotomy syndrome. One patient was receiving coumadin and two patients were receiving aspirin before the diagnosis of cardiac tamponade. Nine patients underwent pericardiocentesis (0.5-11 of fluid). There were no deaths in the group. The syndrome resolved in nine patients with conservative medical therapy and one patient required pericardial stripping for recurrent cardiac tamponade.

In this study, cardiac tamponade occurred in 0.8% of patients who survived cardiac surgery; cardiac tamponade occurred in patients without prior anticoagulation, in marked contrast to previously reported cases; pericardiocentesis and conservative medical therapy were successful in treating the majority of patients; clear symptoms and signs of pericardial involvement were present before cardiac tamponade occurred.

CARDIAC TAMPONADE after surgery is one of many late complications that cause postoperative morbidity and mortality. The incidence of this entity ranges from 0.1-6% in reported series and is considered to be uniformly associated with postoperative anticoagulation, usually in patients undergoing valve replacement. The postpericardiotomy syndrome is characterized by persistence or appearance of fever after the first postoperative week and signs of pericardial, and often pleural, reactions, and has been reported in 20-30% of patients who undergo cardiac surgery.

We attempted to define the incidence of late cardiac tamponade, the role of anticoagulants in its pathogenesis, and its relationship to the postpericardiotomy syndrome in 1290 consecutive patients who underwent open heart surgery at Yale-New Haven Hospital over a 6-year period, from January 1, 1973 through December 31, 1978.

Methods

We analyzed the medical history and clinical course of 1290 consecutive patients who survived cardiac surgery at Yale-New Haven Hospital from 1973-1978. The pericardium was left open in all 942 patients (73%) who had a coronary bypass procedure, in approximately 80% of 296 patients (23%) who had valvular procedures and in 52 patients (4%) who had repair of congenital cardiac malformations.

During hospitalization, all patients were seen daily by the surgical and cardiology teams. ECGs, chest x-rays, and routine blood work were performed every 3-4 days, or more often when necessary. Echocardiograms were not obtained routinely, but rather, as clinically indicated. The echocardiographic diagnosis of pericardial effusion was made using the criteria proposed by Horowitz et al. The usual uncomplicated hospital stay was 10-14 days. Monthly follow-up visits for all 1122 patients (87%) who lived in the area began 1 month after discharge during the first postoperative year and every 6 months thereafter. These patients were followed by both the surgical team and the staff cardiologist responsible for their in-hospital care. The 168 patients (13%) who did not live in the area were followed by their physicians at 1-2-month intervals. Complete follow-up data were collected every 6 months throughout the study, and only two patients were lost to follow-up.

The criteria for the diagnosis of cardiac tamponade were (1) elevated jugular venous pressure (> 16 mm Hg) without a positive Kussmaul's sign, (2) hypoten-
sion or decreased cardiac index, (3) characteristic hemodynamic findings during heart catheterization (an obliteration of the "Y" descent in the right atrial pressure tracing, lack of an early rise in ventricular diastolic dip, elevated diastolic pressures in intracardiac chambers (> 16 mm Hg) and equalization of diastolic pressures in all cardiac chambers), and (4) echocardiographic evidence of pericardial effusion. Pulsus paradoxus greater than 12 mm Hg, electrical alternans, low voltage or ST-T-wave changes on the ECG and persistent tachycardia were not considered essential for the diagnosis of cardiac tamponade. Nine patients who underwent pericardiocentesis improved clinically and hemodynamically, confirming the clinical and catheterization diagnosis of cardiac tamponade.

Late cardiac tamponade was defined as occurring at least 7 days postoperatively. Cardiac catheterization was accomplished using standard techniques, including right-sided heart pressures and thermodilution cardiac output determinations. Pericardiocentesis was performed using the percutaneous subxiphoid approach. No complications occurred during cardiac catheterization or pericardiocentesis.

In the second part of this investigation, all patients with fever persisting after the first postoperative week or those readmitted to the hospital during the first 6 postoperative months with a febrile illness were separated for further study. Our criteria for the diagnosis of pericardiocentesis syndrome were: (1) A prior intrapericardial procedure; (2) unexplained fever* after the first postoperative week that was not related to infection or to drug therapy; and (3) signs of pericardial disease, including pericardial pain or friction rub and echocardiographic proof of pericardial fluid or a new enlarged cardiac silhouette on chest roentgenogram. Leukocytosis, an elevated erythrocyte sedimentation rate and improvement with aspirin and steroids were frequently present, but these findings were not considered diagnostic of postpericardiectomy syndrome.

Statistical Methods

The data were analyzed by the chi-square test or, when expectations were too small to allow the use of chi-square test, by Fisher's exact test. A probability value of less than 0.05 was considered statistically significant.

Results

Ten of 1290 consecutive patients (0.8%) had late cardiac tamponade. These 10 included three males and seven females, ages 16–61 years. Five patients underwent surgery for repair of congenital defects, two for valvular disease, and three for coronary artery disease. Table 1 shows the clinical and operative characteristics of these patients and table 2 shows the electrocardiographic findings.

The onset of hemodynamic compromise due to cardiac tamponade ranged from 15–180 days postoperatively (average 49 days). The duration of illness, defined as persistent fever, pericardial pain, or reaccumulation of pericardial effusion, ranged from 3 days to 12 months (average 36 days). Nine of the 10 patients had pericardial pain and all 10 had fever. Eight of the 10 patients had an audible pericardial friction rub after the first postoperative week. Clinical evidence of pericardial reaction (pericardial rub and pericardial effusion) was present in six patients for at least 2 days before the onset of cardiac tamponade. All 10 patients had a pericardial effusion by M-mode

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*Abbreviations: ASA = aspirin; ASD = atrial septal defect repair; AVR = aortic valve replacement; CABG = coronary artery bypass grafts; Echo = echocardiographic pericardial fluid; ESR = sedimentation rate; F = female; Indomethacin; M = male; MVR = mitral valve replacement; P = pericardium closed or left open; pred = prednisolone; tap = pericardial tap; TET = tetralogy of Fallot repair; + = positive finding; − = negative finding.

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*Onset of tamponade (number of days after operation).

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Table 1. Clinical Profile of 10 Patients with Late Cardiac Tamponade

<table>
<thead>
<tr>
<th>Pt</th>
<th>Operation</th>
<th>P</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Aspirin</th>
<th>Anticoagulant</th>
<th>Fever</th>
<th>Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>AVR</td>
<td>Open</td>
<td>31</td>
<td>M</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>ASD</td>
<td>Open</td>
<td>39</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>3</td>
<td>CABG</td>
<td>Open</td>
<td>51</td>
<td>F</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>TET</td>
<td>Open</td>
<td>32</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>MVR</td>
<td>Open</td>
<td>30</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>TET</td>
<td>Open</td>
<td>25</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>CABG</td>
<td>Open</td>
<td>61</td>
<td>M</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>ASD</td>
<td>Closed</td>
<td>16</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>ASD</td>
<td>Open</td>
<td>20</td>
<td>F</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>CABG</td>
<td>Open</td>
<td>36</td>
<td>M</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

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*Fever was defined as a rectal temperature greater than 37.8°C. Fever was considered to be of infectious cause when cultures of blood, sputum, urine, wound swabs or aspirated fluid were positive for bacteria. Fever was considered to be drug-related when a specific drug was clinically suspected as a cause of fever and discontinuation of the drug was followed by prompt disappearance of fever.
echocardiography; this finding was confirmed in all three of the patients who underwent sector-scanner echocardiography. Six of the 10 patients had a pulsus paradoxus > 12 mm Hg, two patients had a pulsus paradoxus < 12 mm Hg, and two patients had no recorded pulsus paradoxus. In the latter two cases, a pulsus paradoxus was not carefully searched for by a staff cardiologist. Nine of the 10 had hypotension (systolic blood pressure < 100 mm Hg), all 10 had typical hemodynamic findings of cardiac tamponade and nine had a decreased cardiac index (< 3.0 l/min/m²). Table 3 shows the hemodynamic measurements obtained before and after pericardiocentesis at the time of the initial diagnosis.

Only one patient in this series had been receiving anticoagulant therapy (coumadin), and two patients had been treated with aspirin, 1200 mg/day to improve coronary bypass graft patency, before the diagnosis of cardiac tamponade.

Nine of the 10 patients (all those with a decreased cardiac index and circulatory embarrassment) underwent pericardiocentesis, and 500–1000 ml of serosanguineous fluid was removed in each case. Bacterial and fungal cultures were negative in every case (table 4). Despite medical therapy, three patients required pericardiocentesis three times each to control recurrent cardiac tamponade. In addition, one patient required pericardial stripping 372 days after the onset of pericardial disease to control recurrent life-threatening cardiac tamponade. Medical therapy was prescribed for nine of 10 patients: one patient received indomethacin alone; one patient, indomethacin and steroids; three patients, aspirin alone; three patients, aspirin and steroids; and one patient, steroids alone. There were no deaths in the group with cardiac tamponade and the syndrome resolved with conservative medical therapy in nine of the 10 cases.

One hundred seventy-one of the 1290 patients (13%) had unexplained fever after the first postoperative week. The final diagnoses in this group were postoperative afebrile in 21 patients (12%), postperfusion syndrome in 32 (19%), postpump syndrome in 17 (10%), wound infection in 46 (27%), endocarditis and other bacteremic infections in 34 (20%), and postpericardiotomy syndrome in 21 (12%).

Twenty-one of 1290 patients (1.6%) were considered...

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**Table 2. Electrocardiographic Profile of 10 Patients with Late Cardiac Tamponade**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Rhythm</th>
<th>AV conduction</th>
<th>QRS voltage</th>
<th>ST segment</th>
<th>T waves</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ V₁-V₆</td>
<td>Flat V₁-V₆</td>
</tr>
<tr>
<td>2</td>
<td>ST</td>
<td>NL</td>
<td>Low limb leads</td>
<td>↓ I, aV₁, V₁-V₆</td>
<td>Flat V₁-V₆</td>
</tr>
<tr>
<td>3</td>
<td>ST</td>
<td>1° AV block</td>
<td>NL</td>
<td>NS depression</td>
<td>Flat all leads</td>
</tr>
<tr>
<td>4</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ V₁-V₆</td>
<td>Flat I, aV₁, V₁-V₆</td>
</tr>
<tr>
<td>5</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ V₁-V₆</td>
<td>NL</td>
</tr>
<tr>
<td>6</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ aV₆, V₁-V₆</td>
<td>Negative aV₆, V₆</td>
</tr>
<tr>
<td>7</td>
<td>ST</td>
<td>NL</td>
<td>Low limb leads</td>
<td>↑ I, aV₆, V₁-V₆</td>
<td>NL</td>
</tr>
<tr>
<td>8</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ aV₆, V₁-V₆</td>
<td>Flat all leads</td>
</tr>
<tr>
<td>9</td>
<td>ST</td>
<td>NL</td>
<td>NL</td>
<td>↑ I, II, aV₆, V₁-V₆</td>
<td>Flat I, V₆</td>
</tr>
<tr>
<td>10</td>
<td>ST</td>
<td>NL</td>
<td>Electrical alternans</td>
<td>↑ NS depression</td>
<td>Flat all leads</td>
</tr>
</tbody>
</table>

Abbreviations: AV = atrioventricular; NL = normal; NS = nonspecific; ST = sinus tachycardia; ↑ = elevation; ↓ = depression.
to have a postpericardiotomy syndrome. The onset of their illness ranged from 7–186 days postoperatively (average 36 days), and the duration of their syndrome was 3–372 days (average 41 days). All 21 patients had pericardial pain and fever, and 17 patients had a pericardial friction rub. In 12 patients, pericardial pain was associated with nausea and vomiting. Echocardiography documented the presence of pericardial effusion in 20 of 21 patients and a new enlargement of the cardiac silhouette in 16 of 21 patients.

Of the 21 patients with clinically diagnosed postpericardiotomy syndrome, 10 developed late cardiac tamponade. Six patients had prominent clinical features of postpericardiotomy syndrome before they showed clinical evidence of late cardiac tamponade. Five patients had a prolonged clinical course with frequent recurrences; three developed late cardiac tamponade each time they manifested evidence of renewed pericardial inflammation, and one patient required a total pericardial stripping 372 days after her initial illness to treat recurrent life-threatening cardiac tamponade. None of the 1290 patients developed constrictive pericarditis during the follow-up period.

When we analyzed the clinical course of patients with postpericardiotomy syndrome to identify descriptors that may anticipate the development of late cardiac tamponade, we found that patients with cardiac tamponade had larger echocardiographically measured pericardial effusions than those who did not develop cardiac tamponade (table 5).

**Discussion**

The reported incidence of late cardiac tamponade after cardiac surgery is 0.1–6%.11–13 Almost all of the approximately 50 patients reported in the literature were recovering from valvular surgery and nearly all were on anticoagulant therapy with heparin or coumadin. Occasionally, cardiac tamponade complicating postpericardiotomy syndrome has been reported, usually in the pediatric age group and frequently when no anticoagulants were used.14–18 We reviewed 1290 consecutive patients who survived cardiac surgery at Yale-New Haven Hospital over a 6-year period and found 10 cases of late cardiac tamponade, an incidence of 0.8%. Only patients with clinically severe late cardiac tamponade were included in this report. The high range of mean right atrial pressure (16–26 mm Hg), the low range of arterial pressure (75–105 mm Hg) and the presence of circulatory embarrassment indicate that these 10 patients had severe cardiac tamponade. It is unlikely that patients with milder degree of cardiac tamponade were unrecognized and therefore not included in this report. The follow-up was strictly monitored to assure that any patients with suspected symptoms or signs of late cardiac tamponade had appropriate workup.

Late cardiac tamponade was reported to be less frequent after open heart surgery for congenital heart disease. However, in our series, five patients developed late cardiac tamponade after repair of congenital cardiac malformations. Although the etiology of late pericardial effusion and cardiac tamponade is not clearly understood, previous studies suggested a relationship with postoperative anticoagulation. Only one of our 10 patients with late cardiac tamponade had been on anticoagulant therapy (coumadin) and two other patients were taking aspirin. The remaining seven patients were not taking anticoagulants at the time of cardiac tamponade, which suggests, in contrast to previous reports,16–19 that late cardiac tamponade does occur in patients not taking anticoagulants.

Examination of the pericardial fluid in our nine patients revealed straw-colored fluid in two, neither of whom was taking anticoagulants and bloody, nonclot-
ting fluid in seven, including the three who were receiving anticoagulants or aspirin. This suggests that some of the accumulation of pericardial fluid may be caused by perioperative bleeding into the pericardial space.

Cardiac tamponade occurred 2 weeks to 6 months postoperatively. Three patients had recurrent cardiac tamponade, and one of these patients required pericardial stripping. Previous studies suggested that thoracotomy and surgical drainage of the pericardium was the procedure of choice to relieve late cardiac tamponade; however, nine of our 10 patients treated only by pericardiocentesis and medical management required no subsequent surgical procedures. Recently, Becker et al. strongly questioned the role of pericardiocentesis in treating patients with cardiac tamponade, arguing that pericardiocentesis has a high potential incidence of complications. However, we agree with Wei et al. that pericardiocentesis is the procedure of choice in treating cardiac tamponade when an experienced physician is performing the procedure with appropriate hemodynamic monitoring in the catheterization laboratory, and after clear echocardiographic demonstration of pericardial fluid. Wong et al. confirmed that pericardiocentesis could be done with minimal risk. Further, we showed in our patients that late postoperative cardiac tamponade is a self-limiting process, in contrast to early postoperative cardiac tamponade, which is caused by bleeding and requires reexploration. If surgery is performed,
pericardial stripping is the procedure of choice, because a simple pericardial window may not relieve loculated pericardial fluid or may close soon after being performed, giving no protection to patients with recurrent cardiac tamponade. The pericardium was left open in nine of the 10 patients with late cardiac tamponade. The fact that they developed cardiac tamponade suggests that adhesion may close the pericardium soon after operation. Pericardial stripping may carry a high risk in patients with late cardiac tamponade. Therefore, we propose that pericardiocentesis should be attempted before a more aggressive approach is undertaken.

Reports in the literature link cardiac tamponade to the postpericardiotomy syndrome. In our study, all 10 patients who developed late cardiac tamponade had a postpericardiotomy syndrome. We stress this finding because no patient had late cardiac tamponade in the absence of postpericardiotomy syndrome. However, we identified only 21 cases of postpericardiotomy syndrome, an incidence of 1.6%, which is well below the incidence reported in the literature.\(^5\) This low incidence may be due to the diagnostic criteria used, and because the postoperative follow-up was structured to detect only severe cases of postpericardiotomy syndrome. Nevertheless, the development of a postpericardiotomy syndrome may effectively identify patients at risk of developing this extremely dangerous and reportedly fatal late complication.

The information contributed by echocardiography was extremely useful. Large pericardial effusion (> 500 ml) by echocardiography predicted late cardiac tamponade accurately (seven of 10 cardiac tamponade vs one of 11 without cardiac tamponade \(p < 0.05\)). Because M-mode evaluation and quantification of pericardial fluid are difficult after cardiac surgery, careful interpretation of echocardiography is necessary. Sector-scanner echocardiography has helped to differentiate between pericardial and pleural effusion, but does not contribute significantly in identifying patients with large pericardial effusions. These patients were accurately identified by simple M-mode echocardiography.

In conclusion, cardiac tamponade may occur 2 weeks to 6 months after cardiac surgery, and should be suspected in any patient who presents with fever, pericardial pain, pericardial effusion and signs of decreased cardiac output, regardless of whether the patient has been taking anticoagulants. In six of our 10 patients, clear symptoms and signs of pericardial involvement were present for 2 days before cardiac tamponade actually occurred. We do not know if medical intervention at the time a large pericardial effusion is recognized would alter the patient’s subsequent course by preventing full-blown tamponade. Pericardiocentesis and medical management of the postpericardiotomy syndrome appear to be sufficient to control this self-limiting process. Late cardiac tamponade is easily detected if a high degree of suspicion exists on the part of the clinician.

References
Late cardiac tamponade after open heart surgery: incidence, role of anticoagulants in its pathogenesis and its relationship to the postpericardiotomy syndrome.
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