Delayed Mediastinal Tamponade after Open Heart Surgery*

Lee H. Ellison, M.D., and Marvin M. Kirsh, M.D.

Nine cases of delayed mediastinal tamponade occurring between the 6th and 20th days after operation are described. All patients were on anticoagulant therapy at the time tamponade occurred. Tube drainage of the mediastinum and wide drainage of the mediastinum into the pleural space did not prevent this complication. Eight of the nine cases were managed successfully by mediastinal drainage through the subxyphoid portion of the wound or formal re-exploration of the wound. The details of diagnosis and management are discussed.

Materials and Methods

From August, 1967, until November, 1972, 726 adult open heart operations were performed at the University of Michigan Medical Center. All operations were performed with a roller pump and either a disposable bubble or membrane oxygenator. Heparin 3 mg/kg of body weight was administered before cardiopulmonary bypass, with one-third of the dosage repeated every 45 minutes during bypass. The heparin was neutralized with protamine (2 mg of protamine/mg of heparin) in divided doses after extracorporeal circulation. The pericardium was left widely open in all patients. Anterior and posterior mediastinal drainage tubes were used routinely. When the pleural space was entered, a pleural drainage tube was used after a large window was established between the pericardium and the involved pleural space. The drainage tubes were removed 24 hours after bleeding ceased and coagulation studies had returned to near normal values.

Delayed mediastinal tamponade occurred in nine patients. The patients’ age ranged from 21 to 64 years. There were four men and five women. Five patients underwent double-valve replacement, three received single-valve replacement, and one had an aortocoronary bypass (Table 1). A sternal splitting incision was used in seven patients, with a right thoracotomy performed in two patients. Only one patient had marked adhesions from a previous operation. The right pleural cavity was entered in six of the seven patients with a sternal splitting incision. Two patients required operation again for bleeding the same night. Sodium warfarin (Coumadin) was administered to five patients, usually beginning on the fourth or fifth day after the operation. At the time of tamponade the prothrombin times ranged from below 10 percent to 51 percent of normal. The other four patients were receiving heparin. Their total coagulation time (TCT) ranged from 23 to 65 seconds at the time of pericardial tamponade. There was no evidence of bleeding in areas other than the mediastinum in any of the patients.

Table 1—Operations Performed in Patients with Delayed Tamponade

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Patients, No.</th>
<th>Incision Type</th>
</tr>
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<tbody>
<tr>
<td>Single valve replacement</td>
<td>3</td>
<td>right thoracotomy</td>
</tr>
<tr>
<td>Double valve replacement</td>
<td>5</td>
<td>median sternotomy</td>
</tr>
<tr>
<td>Coronary artery bypass</td>
<td>1</td>
<td>median sternotomy</td>
</tr>
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Tamponade appeared from 6 to 20 days after operation (mean 12.5 days). All the patients had the clinical picture of low cardiac output state: hypotension, tachycardia, tachypnea, distended neck veins, cool extremities with diminished peripheral pulses, decreased urine output, and restlessness. A central venous pressure was obtained in only five patients and was elevated in all (range 17 to 26 cm H2O). In eight patients there was a progressive but often subtle increase in the cardiac silhouette on serial chest roentgenograms (Fig 1-4).

Twelve to 24 hours before the detection of the low cardiac output state five patients developed a supraventricular tachycardia, requiring digitalis therapy. Pulsus paradoxicus was not noted in any of the patients. The electrocardiogram was of no diagnostic help in any of the patients.

Results

The correct diagnosis was made in eight of our patients, and the interval from the onset of symptoms to the institution of specific treatment ranged from 1 to 24 hours. Eight of nine patients are alive and well. Paraxiphoid needle aspiration of the mediastinum was successful as the primary treatment in two of three patients. The condition of the remaining patients required exploratory operation on the mediastinum for control of bleeding. A limited retrosternal exploratory operation performed through the lower end of the sternotomy incision was successful in the treatment of tamponade in three of four patients. Three patients were treated...

*From the Department of Surgery, Section of Thoracic Surgery, The University of Michigan Medical Center, Ann Arbor, Mich.

Reprint requests: Dr. Kirsh C 7175, Outpatient Building, University Hospital, Ann Arbor, Mich. 48104

Table 2—Types of Treatment in Patients with Delayed Tamponade

<table>
<thead>
<tr>
<th>Method of Decompression of Tamponade</th>
<th>Patients, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardiocentesis</td>
<td>3</td>
</tr>
<tr>
<td>Opening of lower third of wound plus drainage</td>
<td>3</td>
</tr>
<tr>
<td>Re-exploration of wound</td>
<td>4</td>
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DELAYED MEDIASTINAL TAMPOONADE AFTER OPEN HEART SURGERY

FIGURE 1. Chest roentgenogram before operation demonstrating cardiomegaly, mainly left ventricular.

initially by full reopening of the wound and mediastinal exploration. One patient was initially treated for a pulmonary embolus, and anticoagulation was continued. Since the diagnosis of delayed tamponade was never established, this patient died 24 hours after the onset of symptoms.

DISCUSSION

Because there are few reports in the literature, the true incidence of delayed mediastinal tamponade following open heart surgery is unknown. Whether the complication is not recognized or whether physicians are reluctant to report it is uncertain. Berger reported tamponade in 6 of 186 patients and Engelman reported it in 8 of 310 patients undergoing open heart operations. A similar incidence of 1.4 percent occurred in our experience.

The occurrence of tamponade is higher in patients who require anticoagulant therapy. All of Berger's and Engelman's patients, six of Hill's seven patients, and all of our patients were receiving anticoagulant therapy at the time delayed tamponade occurred. The occurrence of tamponade is higher in patients who require anticoagulant therapy. All of Berger's and Engelman's patients, six of Hill's seven patients, and all of our patients were receiving anticoagulant therapy at the time delayed tamponade occurred. There was no correlation between the degrees of anticoagulation and the development of this syndrome since it occurred in some patients whose anticoagulant therapy was under good control and in therapeutic range. This is substantiated by the report of Fell et al who show that anticoagulant

FIGURE 2. Portable chest roentgenogram 72 hours after operation demonstrating increase in cardiac silhouette.

FIGURE 3. Portable chest roentgenogram seven days after operation demonstrating marked increase in cardiac silhouette and fluid within minor fissure.

FIGURE 4. Portable roentgenogram demonstrating marked decrease in cardiac silhouette following drainage.
therapy with sodium warfarin increases the risk of hemopericardium in patients with myocardial infarction and traumatic or idiopathic nonspecific pericarditis. A relationship between excessive bleeding after operation and pericardial tamponade is suggested by the fact that two of our patients required exploratory operation again for bleeding the night of the operation. It is possible that the retained mediastinal clot or hematoma may expand by absorption of fluid much in the same manner as delayed subdural hematoma, causing cardiac compression.

The single most important factor in making the diagnosis of delayed mediastinal tamponade is a suspicion of this entity in any patient who develops a low cardiac output state, particularly if the patient is receiving anticoagulant therapy during the first three weeks after operation. This is especially true if the patient has had a period of hemodynamic improvement in the period immediately after operation. The clinical findings are those described in mediastinal tamponade: hypotension, with evidence of peripheral perfusion, tachycardia, tachypnea, and decrease in urine output. The onset of these symptoms is often insidious and may be subtle, leading to delay in diagnosis. The physical examination may be unrewarding except for cervical venous distension or muffled heart tones. Although these signs and symptoms are highly suggestive of delayed mediastinal tamponade, they are not diagnostic because they may occur in myocardial failure and pulmonary embolism. If there is hypovolemia or if the clot is localized in the vicinity of the left atrium, the central venous pressure will not be elevated. Pulsus paradoxicus or inspiratory swelling of the neck veins has not been recognized in most patients with delayed tamponade.5

An increase in the cardiac silhouette on serial chest roentgenograms has been the most valuable diagnostic sign of delayed pericardial tamponade. This occurred in the roentgenograms of eight of our nine patients after operation. Other symptoms reported in delayed pericardial tamponade are chest pain, mental disorientation, anorexia and malaise, and a marked drop in the hematocrit level.5

Delayed pericardial tamponade must be differentiated from congestive heart failure, the entity with which it is most often confused. Differentiation is most difficult in the period after operation. The sudden onset of hypotension and elevated central venous pressure without pulmonary edema is more often indicative of tamponade than myocardial failure. Paradoxical pulse and inspiratory swelling of the neck veins are of little value in differentiating tamponade and myocardial failure. Angiocardiography may be used to demonstrate a pericardial collection or a pulmonary embolus. Because of the time-consuming nature of this examination, it should be reserved for equivocal instances or those in which pulmonary embolus is the most likely diagnosis.

If the diagnosis is suspected, immediate mediastinocentesis should be performed, with the patient in the upright position (45°), using the subxyphoid approach. As much fluid as possible should be removed. The removal of as little as 15 to 25 ml of blood may be sufficient to decompress the mediastinum temporarily. When thick blood or clot are present it may not be possible to aspirate any fluid. This “negative” mediastinocentesis should not result in a false sense of security or further delay in establishing the diagnosis.

Mediastinal tamponade following operation is a surgical emergency and must be treated as such. Delay in treatment is usually due to a lack of awareness of this entity and can be fatal. This was true in one of our patients mistakenly treated for a pulmonary embolus. Although temporary improvement may occur with mediastinocentesis, we agree with Hill5 and Engleman3 that it is, as a rule, an ineffective treatment of delayed pericardial tamponade. Decompression at operation is the preferred method of treatment. In most cases this can be accomplished by a limited retrosternal exploration through the infrayphoid portion of the sternotomy wound.1 This may be performed, if necessary, in the patient’s bed or if time permits, in the operating room. In addition to the removal of the fluid blood with an aspirating tip, manual evacuation of as much clot as possible should be accomplished. A large bore mediastinal tube should be left in place for several days. This approach was successful in four of our nine patients. In those patients who are not improved by this limited retrosternal approach, the wound should be reexplored in the operating room. The sternotomy wound is then fully opened and the mediastinum explored thoroughly. This was necessary in three of our patients.

References
1 Beck CS: Acute and chronic compression of the heart. Part 1: compression of the heart. Am Heart J 14:515, 1937
3 Engleman RM, Spencer RC, Reed GF, et al: Cardiac tamponade following open heart surgery. Circulation (Suppl 2) 41:165, 1970