Diagnosis and management of purulent pericarditis

Experience with pericardiectomy

Twelve cases of purulent pericarditis seen over 6 years are described. Staphylococcus aureus was the most common causative organism (six patients), and a respiratory infection was the most common preceding illness. The chest radiograph and echocardiogram were useful pointers to the diagnosis, but the electrocardiogram was not reliable. Antibiotics, surgical drainage, and pericardiectomy were used in all 12 cases. There was one death (8.3%), which occurred in a patient who was seen late. A review of the literature dealing with the diagnosis and management of this condition is presented. The importance of early diagnosis before a significant degree of cardiac tamponade occurs is noted. Although there is general agreement that surgical drainage is mandatory, the approach, methods of drainage, and extent of pericardial resection have been the subject of some discussion, and at least seven techniques are available. We conclude that pericardiectomy has a definite place in the management of purulent pericarditis.

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Purulent pericarditis is a rapidly fatal disease if left untreated. Early clinical diagnosis is difficult and the diagnosis may not be made until a significant degree of cardiac tamponade has occurred. Although medical or surgical therapy alone has been used, best results are obtained with a combination of medical and surgical treatment. The most suitable surgical procedure to perform has been the subject of some debate. This article describes our experience with the diagnosis and management of 12 patients seen over a 6-year period.

Patients and methods

Thirteen patients with purulent pericarditis were treated surgically by pericardiectomy at the University Hospital, Kuala Lumpur, Malaysia, between 1982 and January 1988. One case already has been reported and is not included in this series. The findings in the remaining 12 cases are reported in this article. The details are summarized in Table I. Six patients were male, and six were female. There were four adults and eight children (aged 12 years and younger). All were from lower socioeconomic groups, and three were referred from peripheral hospitals.

The most common preceding illness was a respiratory infection that was present in five patients. Four patients had an obvious septic focus (osteomyelitis [patients 8 and 11], septic arthritis [patient 10], and meningitis [patient 5]). There were no cases of pyomyositis. Two patients initially had a vague febrile illness (patients 2 and 12). Four patients complained of abdominal discomfort. One patient (patient 9) was moribund and anuric when transferred from the referring peripheral hospital.

The clinical signs are summarized in Table II. The most common clinical signs were a raised jugular venous pressure, tachycardia, fever, hepatomegaly, and tachypnea. Decreased heart sounds, pericardial friction rub, and pulsus paradoxus were not seen commonly.

An electrocardiogram, chest radiograph, and echocardiogram were performed in all cases. Pericardiocentesis was performed in five cases. In the remaining seven cases urgent operation was performed on the basis of the clinical symptoms and investigations already performed. Specimens were taken from blood and pericardial fluid for microscopy, culture, and sensitivity. Further specimens were taken from cerebrospinal fluid, synovial joint, or osteomyelitic abscess when relevant. Pericardial drainage was performed by either a left thoracotomy approach with a limited pericardial resection (seven patients) or bilateral thoracotomy and anterior interphrenic pericardiectomy (five patients). In all patients both pericardial and pleural drains were inserted.

Results

Investigations. The electrocardiogram showed decreased voltage in two patients and a raised ST segment
Table I. Summary of the main features of the patients and their management

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex, age (yr)</th>
<th>Preceding illness</th>
<th>Pericardial tap</th>
<th>Organism isolated*</th>
<th>Antibiotic agent</th>
<th>Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F, 21</td>
<td>RI</td>
<td>-</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>2</td>
<td>M, 33</td>
<td>Fever</td>
<td>+</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>3</td>
<td>F, 3</td>
<td>RI</td>
<td>-</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>4</td>
<td>M, 20</td>
<td>Abdominal pain</td>
<td>-</td>
<td>S. pneumoniae</td>
<td>Cloxacillin</td>
<td>AP&amp;T</td>
</tr>
<tr>
<td>5</td>
<td>F, 2</td>
<td>Meningitis</td>
<td>-</td>
<td>H. influenzae</td>
<td>Ampicillin</td>
<td>AP&amp;T</td>
</tr>
<tr>
<td>6</td>
<td>M, 2</td>
<td>RI</td>
<td>+</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>AP&amp;T</td>
</tr>
<tr>
<td>7</td>
<td>M, 12</td>
<td>RI</td>
<td>-</td>
<td>No growth</td>
<td>Cefuroxime</td>
<td>AP&amp;T</td>
</tr>
<tr>
<td>8</td>
<td>M, 4</td>
<td>Osteomyelitis</td>
<td>-</td>
<td>Pseudomonas aeruginosa</td>
<td>Cloxacillin</td>
<td>AP&amp;T</td>
</tr>
<tr>
<td>9</td>
<td>F, 1</td>
<td>RI</td>
<td>+</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>10</td>
<td>F, 9</td>
<td>Septic arthritis</td>
<td>+</td>
<td>S. aureus</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>11</td>
<td>F, 2</td>
<td>Osteomyelitis</td>
<td>-</td>
<td>Klebsiella</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
<tr>
<td>12</td>
<td>M, 39</td>
<td>Fever</td>
<td>+</td>
<td>No growth</td>
<td>Cloxacillin</td>
<td>PP&amp;T</td>
</tr>
</tbody>
</table>

RI, Respiratory infection; PP&T, partial pericardiectomy and tube; AP&T, anterior pericardiectomy and tube; CSF, cerebrospinal fluid.

*Organism isolated from pericardial fluid unless otherwise specified.

Table II

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raised JVP</td>
<td>12</td>
</tr>
<tr>
<td>Tachypnea</td>
<td>8</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>10</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>9</td>
</tr>
<tr>
<td>Fever</td>
<td>9</td>
</tr>
<tr>
<td>Decreased heart sounds</td>
<td>3</td>
</tr>
<tr>
<td>Pericardial friction rub</td>
<td>3</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>2</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>3</td>
</tr>
</tbody>
</table>

JVP, Jugular venous pressure.

in three patients. The chest radiograph showed an enlarged “cardiac” shadow in all 12 patients, a pleural effusion in six patients, and pneumonic changes in four patients. Echocardiography in all 12 patients showed the presence of pericardial fluid. In the first two patients M-mode echocardiography was used, and in subsequent patients two-dimensional echocardiography was used. In the five patients who underwent pericardiocentesis frank pus was obtained, and in all five patients pus reaccumulated, as noted on echocardiography.

Operation. At operation thick pus was found in the pericardium in all patients (150 to 600 ml). Five patients had fibrin clots or loculi that were removed. There was an improvement in blood pressure and a decrease in central venous pressure on evacuation of the pus in all cases. One patient (patient 9), who was in a moribund condition on arrival and who was also anuric, died immediately after the operation. The remaining patients electively received mechanical ventilation for 1 or 2 days in the cardiac intensive care unit before being transferred back to the ward. In all 11 surviving patients chest drains were removed from 4 to 10 days after the procedure. Antibiotics were continued for 1 week. Patients were discharged to their home or to the referring hospital 2 to 3 weeks after the operation. There were only minor superficial wound infections in three patients.

Bacteriology. All patients had pus in the pericardium confirmed microscopically. The causative organism was cultured from the pericardial fluid in six patients. In the remaining six patients no growth was obtained on culture of the pericardial pus. In four of these patients the organism presumed to be the causative organism was grown from blood in one patient, from osteomyelitic abscess in two patients, and from blood and cerebrospinal fluid in one patient. In two patients who were already receiving several antibiotic agents, started at the referring hospital, no organism could be cultured.

Histology. All patients showed nonspecific inflammation of the pericardium.

Review. Eight patients were seen as outpatients from 3 months to 4 years after operation and were well. No features of pericardial constriction or recurrence of purulent pericarditis were seen. Three were discharged to the care of the referring peripheral hospital and were reported to be well.

Discussion

The spectrum of organisms causing purulent pericarditis differs in various parts of the world and has varied during different eras. In reports found in the Western literature in the preantibiotic era, Streptococcus pneu-
moniae was the most frequent cause, but since the antibiotic era Staphylococcus aureus has become the most important causative agent. More recently a number of series have reported Haemophilus influenzae as an increasingly important cause of purulent pericarditis. Other organisms also are being reported as causative agents in Western countries, and the use of more powerful antibiotic drugs may be partly responsible for this trend. In series from developing countries such as New Guinea, Zimbabwe, and Nigeria, S. aureus remains the most common cause, and no cases of H. influenzae were described. In a series of 28 cases, Weir and Joffe, from South Africa, reported only one case of H. influenzae purulent pericarditis. In Malaysia antibiotic agents are becoming more readily available and probably have a bearing on the spectrum of organisms that we have seen. The main causative organism in this series was still S. aureus, although various other organisms, including gram-negative organisms also were seen. Only one case caused by H. influenzae was seen. In two cases purulent pericarditis was caused by more than one organism. Also important is that we have not seen any cases of tuberculosis.

In this series the predisposing factors were either respiratory infections that progressed to pneumonia or septic foci such as osteomyelitis or septic arthritis. This differs from Nigeria, where pyomyositis is an important cause of purulent pericarditis. Once an established focus of infection is present, the pericardium may become infected by septic emboli or by direct spread from the lungs. Although the diagnosis of purulent pericarditis is ultimately made by demonstration of pus in the pericardium, a number of clinical features may point to the diagnosis, and in this series a raised jugular venous pressure, tachycardia, tachypnea, fever, and hepatomegaly occurred commonly. Also of importance is the presence of an enlarged "cardiac" shadow on the chest radiograph, which was seen in all cases. Bentley and colleagues found this present in all cases, but Sinzobahmyya and Ikeogu have not found this to be a consistent feature. Echocardiography that showed a pericardial effusion was a useful additional investigation and was diagnostic in all of our cases. This confirms the experience of others.

Some features commonly associated with pericarditis were surprisingly uncommon. A pericardial friction rub was uncommonly found, probably because by the time of presentation a large amount of pus in the pericardial cavity had already accumulated and prevented the pericardial and epicardial surfaces from abrading to cause the friction rub. Other authors have reported similar experience, and Cheatham and colleagues and Okoroma and associates found that a friction rub was not a consistent finding. Hardy and colleagues also noted that a loud friction rub occurred in the presence of a small pericardial effusion but was absent when the effusion increased in size. Vigneswaran and colleagues found a large effusion on echocardiography but no friction rub. Berk and colleagues reported that only one of six patients with purulent pericarditis had a pericardial friction rub. Bentley and colleagues found the fleeting presence of a pericardial friction rub in two of seven patients. Pulsus paradoxus was also a sign that was difficult to elicit in the presence of tachypnea, especially in the younger patients.

The classic electrocardiographic feature of pericarditis, a raised ST segment caused by epicardial injury, was uncommonly seen. Berk, Cheatham, and their associates found that the electrocardiogram, although often showing abnormalities, did not frequently demonstrate changes specific for pericarditis, particularly with respect to ST segment elevation. Jaiyesimi and colleagues found that a friction rub was not a consistent finding. Hardy and colleagues pointed out that some patients have an acute fulminant form of illness that is rapidly fatal because of cardiac tamponade. This probably occurs because of a rapid accumulation of pus, and such patients require early treatment. Even recent series continue to report deaths before treatment can be given. In all five of our patients who underwent pericardiocentesis, pericardial pus reaccumulated and was later removed surgically. In the remaining seven patients the presence of clinical features suggestive of tamponade and pericardial effusion on echocardiography suggested the diagnosis of purulent pericarditis, and thus urgent operation was performed without a prior pericardiocentesis. With regard to the second of the twin dangers, septicemia, the choice of antibiotic therapy remains a problem. Ideally, identification of the organism on culture together with the sensitivities should dictate the choice of antibiotic agents, however, not only does this take time but also antibiotic therapy may already have been initiated on an empiric basis by the
referring physician. Cheatham and colleagues found countercurrent immunoelectrophoresis useful for identification of the organism, because it is relatively quick and takes a few hours as opposed to the 24 to 48 hours that Gram's stain and culture and sensitivity take. However, the major disadvantage was the inability to identify Staphylococcus, an important causative organism.

If surgical treatment is combined with medical therapy, the mortality rate of purulent pericarditis decreases to 20% or less. Although there is general agreement that drainage of the pericardium is necessary, controversy still surrounds the method of surgical treatment that should be used. Questions revolve around surgical approach, method of drainage, and whether pericardiectomy is necessary. The surgical approach is closely tied to the drainage procedure that is to be performed. If a tube drain alone is planned, a subxiphoid approach gives good access. If a pericardial window or limited pericardiectomy is planned, a left thoracotomy can be used. If an anterior interphrenic pericardiectomy is to be performed, either a bilateral submammary thoracotomy or a median sternotomy can be used.

The methods of drainage include the following:

1. Subxiphoid tube drain
2. Subxiphoid tube and irrigation
3. Pericardial window and pleural drain
4. Pericardiectomy with tube
5. Partial pericardiectomy with pericardial tube
6. Anterior pericardiectomy or anterior interphrenic pericardiectomy
7. Total pericardiectomy

Insertion of a subxiphoid drain is a relatively small procedure that can even be performed under light anesthesia with the patient sedated. It is more suitable for removal of thin pus and has been used with good results. Its disadvantage is that loculi or fibrin clots may not be adequately drained. Irrigation of the subxiphoid tube has been described and various agents including povidone-iodine and streptokinase have been used successfully to prevent tube blockage and to break down fibrin clot. The technique of pericardial window and pleural drainage has been used by others when loculi can be lysed and fibrin clots removed at thoracotomy. Drainage from the pericardial cavity occurs through the pericardial window into the pleural space to be removed by a pleural drain. Blockage of the pericardial window by fibrinous adhesions can occur, necessitating reoperation. The technique of partial pericardiectomy with drainage of the pericardium with a pericardial tube drain also necessitates thoracotomy. Evacuation of the clot and lysis of loculi can be performed at operation, and more direct drainage of the pericardium with the pericardial tube coupled with the partial pericardiectomy makes reaccumulation of pus less likely. Anterior pericardiectomy, anterior interphrenic pericardiectomy, and total pericardiectomy, though they complicate the procedure, confer the advantage of preventing late pericardial constriction.

Advocates of the larger procedures refer to the need to evacuate thick clots, which in the case of H. influenzae, have been graphically described as having the consistency of scrambled eggs. We also found that the presence of thick fibrin clots is not restricted to H. influenzae pericarditis alone but was also seen in our patients with S. aureus and other organisms. In one case a large clot was found posterior to the heart, a location accessible only by thoracotomy. In addition to the presence of fibrin clots, loculation also occurs in staphylococcal purulent pericarditis and purulent pericarditis caused by other organisms, and our experience has been similar. Thus we believe that more complete evacuation of pus and breakdown of loculi can be achieved by open operation. The removal of some pericardium decreases the likelihood of further tamponade and constriction. Although the theoretic possibility of septicemia with pericardiectomy has been raised by Cameron, this has not been the general experience, as described in case reports and in one series of eight patients. We confirm that pericardiectomy is a satisfactory method of treatment for purulent pericarditis. Our experience has been improvement in all but one of our patients. In that one case we believe that death was related more to the moribund condition of the patient on arrival than to further septicemia. Although we have performed both partial pericardiectomy and anterior interphrenic pericardiectomy, both appear to give good results, and it is not possible to state which method is preferred.

Thus, although clinical features diagnostic of purulent pericarditis are lacking, the triad of fever, an infective focus, and some features of cardiac tamponade should alert the physician to the possibility of purulent pericarditis. Once the diagnosis has been considered, a chest radiograph that shows an enlarged "cardiac" shadow is a useful pointer to the diagnosis, and echocardiography can then be performed to demonstrate the presence of pericardial fluid and exclude other causes of an enlarged cardiac shadow. Pericardiocentesis is helpful for diagnostic and temporizing purposes. Surgical drainage should be performed; a pericardiectomy enables the physician to remove fibrin clots and loculi and prevents the possibility of late pericardial constriction.

REFERENCES