Subacute effusive constrictive epicarditis

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SUMMARY The coexistence of subacute pericardial effusion and epicardial constriction produces a clinical state, subacute effusive constrictive epicarditis, which resembles pericardial effusion with tamponade. However, pericardiocentesis results in only partial and temporary relief, a feature which is of importance in diagnosis. This syndrome was seen in five patients all of whom benefited from early pericardiectomy.

The acute and subacute phases of pericardial effusion may cause cardiac embarrassment by tamponade which, when relieved, results in a rapid restoration of normal ventricular filling. The co-

tuberculosis, with uraemia, after trauma, after radiation, with neoplastic involvement of pericardium, with acute pyogenic pericarditis, and with viral pericarditis. Hancock described the

Table Clinical data in five patients with subacute effusive constrictive epicarditis

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)</th>
<th>Sex</th>
<th>Presenting symptoms and duration</th>
<th>Pulse rate (beats/min) and blood pressure (mmHg)</th>
<th>Jugular venous pressure</th>
<th>Abdomen</th>
<th>Heart sounds</th>
<th>Pulses paradoxus</th>
<th>Kussmaul's sign</th>
<th>Chest x-ray</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>M</td>
<td>Dyspnoea, abdominal distension 1 year</td>
<td>88 100/80</td>
<td>Normal</td>
<td>+ +</td>
<td>Ascites, liver 10 cm, spleen 4 cm</td>
<td>Normal, no 3rd sound</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>M</td>
<td>Fever, dyspnoea, left chest pain 6 wk</td>
<td>88 90/60</td>
<td>Raised</td>
<td>0</td>
<td>Liver 4 cm</td>
<td>Muffled heart sounds, no 3rd sound</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>M</td>
<td>Fever, dyspnoea, 12 wk</td>
<td>84 90/60</td>
<td>Raised, y descent</td>
<td>0</td>
<td>Liver 2 cm</td>
<td>Muffled heart sounds, no 3rd sound</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>F</td>
<td>Fever, cough, orthopnoea, chest pain 4 wk</td>
<td>120 120/70</td>
<td>Raised, y descent</td>
<td>+ +</td>
<td>Liver 4 cm</td>
<td>Quiet heart sounds, no 3rd sound</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>F</td>
<td>Fever, central chest pain, orthopnoea 2 wk</td>
<td>120 105/70</td>
<td>Raised</td>
<td>0</td>
<td>Normal</td>
<td>Normal, heart sounds, pericardial rub</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

PP, parietal pericardium; epi, epicardium. *Serological studies consistent with a viral infection.

existence of acute or subacute pericardial effusion and constrictive epicarditis is unusual. First described from the Mayo Clinic by Burchell and Edwards, it was later recorded in patients with

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beneficial effects of early pericardiectomy.

**Patients and methods**

During the seven-month period between July 1976 and January 1977, five patients were admitted to the Thoracic Unit, Colombo General Hospital, with subacute effusive constrictive epicarditis. Three were male and two were female and the age range was from 16 to 46 years, four of the five being below 30 years. The clinical features of these patients are presented in the Table. Apart from the 16-year-old boy (case 1) who gave a history of one year's duration, the others had relatively short periods of illness varying from two to 12 weeks. In three of the patients (cases 2, 4, and 5) with short histories of two to six weeks, retrosternal or left-sided chest pain was a prominent symptom. All five patients complained of dyspnoea, and two were orthopnoeic. Four were febrile. The pulse volume was poor in all five patients but pulsus paradoxus (defined as an inspiratory fall in systolic arterial pressure greater than 10 mmHg) was not seen. The systolic blood pressure on admission was below 110 mmHg in four of the five patients. Jugular venous distension was present in four patients, only one (case 3) of whom had a positive Kussmaul's sign. A sharp y descent was present in two patients.

<table>
<thead>
<tr>
<th>Pericardiocentesis</th>
<th>ECG</th>
<th>Time interval from admission to operation</th>
<th>Operation</th>
<th>Aetiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Sinus rhythm, low voltage QRS, T ↓ in all leads</td>
<td>1 mth</td>
<td>PP normal, epi 2 mm, epicardectomy only</td>
<td>Probably viral*</td>
</tr>
<tr>
<td>5 x (total 920 ml)</td>
<td>Sinus rhythm, low voltage QRS</td>
<td>2 wk</td>
<td>PP 10 mm, epi 7 mm, pericardiectomy</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>3 x (total 1950 ml)</td>
<td>Sinus rhythm, low voltage QRS, T ↓ in aVF and V6</td>
<td>2 d</td>
<td>PP 8 mm with calcification, epi 8 mm and infiltrating, pericardiectomy</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>2 x (total 400 ml)</td>
<td>Sinus rhythm, low voltage QRS</td>
<td>2 d</td>
<td>PP 4 mm, epi 10 mm, pericardiectomy</td>
<td>Pyogenic</td>
</tr>
<tr>
<td>2 x (total 550 ml)</td>
<td>Sinus rhythm, low voltage QRS</td>
<td>2 d</td>
<td>PP 4 mm, epi 3 mm, pericardiectomy</td>
<td>Probably viral*</td>
</tr>
</tbody>
</table>

The cardiac impulse was impalpable in four, and hepatomegaly, ankle oedema, and ascites were present in four patients, two patients, and one patient, respectively. A third heart sound was not heard in any of the five.

Chest x-ray films showed conspicuous enlargement of the cardiac silhouette in all five (Fig. 1). In one (case 3), a small area of calcification was seen on the left lateral cardiac border. Low voltage QRS complexes were seen in all five electrocardiograms; all five patients were in sinus rhythm and P waves were normal. ST segment abnormalities were not seen, but minor T wave inversions were present in two patients.

The only patient with a long history (case 1) had another unusual feature: the enlargement of the heart was mainly to the right and the right border showed an angulation the significance of which was not certain (Fig. 2). He also had a firm enlarged cirrhotic liver with splenomegaly. Cardiac catheterisation showed a right atrial pressure of 12 mmHg and the right ventricular pressure record showed the typical early diastolic dip and raised end-diastolic pressure of constrictive pericarditis. Cineangiography showed the heart to be displaced to the right with a small right ventricular cavity and a large pericardial effusion (Fig. 3). In view of these observations this patient was operated on without preceding aspiration.

In the remaining four patients the initial diagnosis was effusion with tamponade; two to five...

![Fig. 1 Chest x-ray film (case 5) showing enlarged cardiac shadow.](image-url)
aspirations were carried out in each of these patients. After each aspiration there was a slight increase in systolic blood pressure lasting only six to 12 hours. The raised venous pressure and hepatomegaly were unchanged. The aspirated fluid was thin in all and blood stained in three. The transient beneficial effect of pericardiocentesis and the overall deterioration was the indication for pericardiectomy, which was done two to 14 days after admission in these four patients.

At operation, the chest was opened through a median sternotomy incision in one while the other four had a left anterolateral thoracotomy. The pericardial effusion amounted to between 200 and 400 ml. The parietal pericardium was normal both macroscopically and histologically in one, slightly thickened in one, and grossly thickened in the remaining three patients. The epicardium varied from a taut white membrane no more than 2 mm thick to 8 mm thick in the two patients who were subsequently shown to have tuberculosis.

There were no adhesions between the two layers of pericardium. In all five there was considerable restriction of ventricular relaxation in diastole. Epicardiectomy alone sufficed in one, and in all five resulted in an obvious improvement in cardiac filling associated with a fall in the central venous pressure. In only case 3 was there any tendency for the epicardium to infiltrate the myocardium. The other four patients also required excision of the parietal pericardium. The immediate results were gratifying; three patients were discharged in excellent condition with normal blood pressure, normal venous pressure, and no oedema, hepatomegaly, or ascites. Two were much improved and when reviewed two years later showed further improvement, with no restriction of activity; residual hepatosplenomegaly in case 1 was attributed to cirrhosis. The histological appearance was that of tuberculosis in two and of acute pyogenic infection in one, and was consistent with a viral infection in the other two patients.

**Discussion**

Constriction is regarded as the culmination of
chronic pericardial disease and has in the past been usually associated with tuberculosis. Sporadic reports have appeared of patients proceeding to constriction after an illness as brief as two weeks, with symphysis of the two layers of pericardium. Though the association of pericardial effusion and concomitant constrictive epicarditis has been infrequently reported, a review of the published cases shows that it occurs in many different diseases. The presenting clinical features with the exception of ascites may easily be confused with those of pericardial effusion with tamponade. Pulsus paradoxus was not observed by us though it has been described as being a feature of effusive constrictive disease.

Hancock analysed the clinical and haemodynamic features of effusive constrictive pericarditis, which were identical to those in our five cases, and drew attention to those features that were of diagnostic significance in distinguishing this condition from chronic constrictive pericarditis without effusion and pericardial effusion with tamponade. In his series pulsus paradoxus was more prominent in both effusive constrictive disease and effusion with tamponade than in chronic constriction without effusion. The characteristic third heart sound that is present in chronic constriction without effusion was rarely heard in the other two conditions, though similar sounds could be recorded on phonocardiograms in effusive constrictive disease, the presumption being that the sound was muffled by the fluid. Low voltage QRS complexes and flattened or inverted T waves were present in the electrocardiograms of both effusive constrictive disease and chronic constriction without effusion, but abnormal P waves were rare in the former. Kussmaul’s sign was present in the jugular venous pulse, but pressure tracings indicated that, while in chronic constriction without effusion there was always a rise in right atrial pressure, in effusive constrictive disease there was an increase in the amplitude of the pulsations not always associated with a rise in right atrial mean pressure. The characteristic y descent of chronic constriction without effusion was not seen in effusive constrictive disease where either a more prominent systolic (x) descent was present or x and y descents were equal. However, in the effusive constrictive group the y descent became more obvious after pericardial aspiration. In four of our patients there was visible elevation of the venous pressure, and in the other (case 1) the right atrial mean pressure was high though the venous pressure was not seen to be raised. In only two of our cases was the y descent seen; an x descent was not seen in any case. No third heart sound was heard.

Hancock advocated simultaneous cardiac catheterisation and pericardiectomy for the diagnosis of combined effusion and constriction. The diagnosis though not easily made initially should be strongly suspected from the poor response to pericardial aspiration and this should lead to pericardiectomy as the next step in treatment.

The constriction layer of epicardium may be deceptively thin and normal in appearance, but the small heart size and restricted range of movement should indicate the presence of constriction. The parietal pericardium may be normal, in which case it should be left alone; however, if it is diseased, then excision of both visceral and parietal layers is necessary. Because of the dominant role of the epicardium in this disorder, the term epicarditis as first used by Burchell and Edwards is preferred to pericarditis. The constrictive epicardium in subacute effusive constrictive epicarditis shows a lesser tendency to infiltrate myocardium than in chronic constriction without effusion, though in both conditions piecemeal removal is usually necessary. The natural history appears to be progression to chronic constriction without effusion and if left alone these cases will show the same tendency to myocardial infiltration as is seen in constriction without effusion. It therefore seems expedient to carry out early pericardiectomy before the development of myocardial infiltration, which further compromises cardiac function and makes operation difficult. The good results obtained in this series confirm this view.

References

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