

## PERICARDITIS WITH EFFUSION IN CHILDREN

### Case Report

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### INTRODUCTION

Although pericarditis with effusion is uncommon in children, a review of the problem is desirable, since diagnostic errors are common (Friedberg, 1957, Zuidema, 1959, Cayler *et al.*, 1963, Ranti *et al.*, 1965). Jo *et al.* (1969) found no case of pericarditis in his data collected during 1962 to 1967 in Medan. Hanafiah *et al.* (1965) noted two cases of purulent pericarditis out of 459 cases of heart diseases.

The clinical diagnosis of pericarditis with effusion is established by the finding of an auscultatory pericardial friction rub and this is *sine qua non* of pericarditis with effusion, and is found only approximately 50% of cases. When a friction rub is not present, the clinical diagnosis of pericarditis with effusion can be very difficult (Cayler *et al.*, 1963).

The present paper reviews our experience with two patients with pericarditis with effusion at the Child Health Department, Gadjah Mada University Hospital. One case was suspected because the present of pericardial friction rub, while the other was diagnosed by X-ray examination.

#### Case no. 1

A 9-year old Indonesian girl was admitted to the Child Health Department, Gadjah Mada University Hospital on April 26th, 1971. A history of 15 day of fever, cough, shortness of the breath, pain on the chest, especially on pressure or in deep breathing was noted.

On admission the vital signs were: blood pressure 110/80 mmHg, pulse rate 140/minute, respiration 45 per minute, temperature 37,8° C. She was very ill, dyspneic, not cyanotic, and always lying on the right side. The venous pressure was elevated, cardiac dullness shifts to the right and left. The heart sounds were distant and pericardial friction rub was present. Lung auscultation revealed bronchitis signs. The liver was palpable 4 digits below the right costal margin and the spleen was not enlarged. Two days after admission cardiac silhouette was noted to be markedly enlarged. The Mantoux test was: 10 × 11 mm. The white cell count was 13.000 per cubic mm with a marked shift to the left and sedimentation rate 70 per hour. The haemoglobine was 10 mg %.

Urine analysis revealed no abnormalities. ECG examination showed sinus tachycardia, slight ST-segment depression and T-wave changes in almost

all leads. Six days after admission a pericardiocentesis was performed and yielded 300 ml serohaemorrhagic material. Forty two milligram streptomycine in two ml aqua dest. solution was instilled in the pericardial cavity. Laboratory examination of pericardial fluid revealed no micro-organism. The Rivalta test was positive.

Treatment was initiated immediately after admission with penicillin, streptomycine, steroid and digitalis. However, improvement was not seen. Immediately following pericardial drainage the sign of cardiac decompensation has disappeared and she was markedly improved. Digitalis was discontinued, and specific treatment for tbc and steroid was continued. Serial Rontgen films of the chest after pericardiocentesis, demonstrated a progressive decrease in the size of the heart, until there was only a mild cardiomegaly on 54th hospital day and finally discharged after 62 days hospitalization. 6 months after discharge she was checked up, and the result revealed no exacerbation.

### Case no. 2

A two-year old Indonesia boy was admitted to the Child Health Department, Gadjah Mada University Hospital on August 30th 1971.

A history of 9 days of fever, followed by cough, shortness of the breath, vomiting and pain on the chest especially on pressure was recorded.

On admission the vital signs were: blood pressure not recorded, pulse rate 160 per minute, respiration 40 per minute, temperature 38.6°C. He was very ill, dyspneic, and cyanotic. The venous pressure was elevated. A chest examination at the time of admission revealed bilateral broncho-pneumonia. The cardiac dullness could not be determined. The heart sounds soft and distant. A pericardial friction rub was not present. A roentgenogram of the chest disclosed marked cardiomegaly, and showed fluid line in pericardial space and revealed bilateral infiltrate of the lung. Pericardiocentesis was performed immediately after admission and yielded 80 ml purulent material. Laboratory examination and culture of pericardial fluid for micro-organism were not performed.

All laboratory examinations were not performed, because the patient died on the following day.

Penicillin, streptomycine, and steroid were given immediately after admission. Autopsy could not be done.

### DISCUSSION

It is not widely realized that pericarditis was found more frequently at necropsy than during life (Friedberg, 1957, Zuidema, 1959, Cayler *et al.*, 1959, Ranti *et al.*, 1965). Many cases of pericarditis are therefore being missed. In children the main problem, as emphasized by Nadas and Levy, is to differentiate pericarditis with effusion from acute myocarditis in which the cardiac silhouette may suddenly and rapidly increase as a result of cardiac dilatation (Cayler *et al.*, 1963).

A review of the clinical findings suggests the only true diagnostic sign is pericardial friction rub (this rub is to and fro murmur with the characteristic *squeaky shoe* pitch) and this at times, may be transient, hard to listen or misleading.

The rub may have three components in the cardiac cycle. These are caused by atrial systole, ventricular systole and ventricular diastole, each of which moves the heart within the pericardial sac. However, only two components may be heard, and sometimes only one, and therefore the rub is difficult to distinguish from common systolic murmurs.

When a friction rub is not present, the clinical diagnosis of pericarditis with effusion can be very difficult. That is why to differentiate it from cardiac dilatation in children, Cayler *et al.* (1963) give the following clinical findings as reliable guides:

1. Marked cardiac enlargement without associated pulmonary congestion (on X-ray).
2. Obliteration of the retrosternal space without evidence of enlargement of the thymus (on X-ray), or right ventricular hypertrophy (on the ECG).
3. Significant respiratory variations in pulse volume (pulses paradoxus).
4. The sudden development of cardiac enlargement (on X-ray) associated with pyogenic lung, bone, or renal infection.

The electrocardiographic signs of pericarditis are characteristically widespread throughout many leads. The earliest signs are ST-segment elevation in many leads. Typically ST-segment elevation assumes a concave curvature in contrast to the usual convex curvature seen in myocardial infarction. In pericarditis with effusion the QRS-complex are of low voltage (Goldman, 1962). But the changes do not occur in a day or two after the appearance of the friction rub and indeed may not occur at all.

To reduce the errors of the clinical diagnosis of pericarditis with effusion a special laboratory and roentgen techniques are needed. These techniques are:

1. Pericardiocentesis
2. Cardiac catheterisation
3. Venous angiocardigraphy
4. Image intensifier fluoroscopy
5. Kymography
6. Radioisotop scanning.

In our first case the diagnosis was suspected by the findings of pericardial friction rub, X-ray examination, electrocardiogram, pericardiocentesis confirmed the diagnosis.

On the other hand for the second case the diagnosis was not suspected clinically until X-ray examination was done, since pericardial friction rub was not present. Pericardiocentesis confirmed the diagnosis.

The etiology of pericarditis may be primary (rheumatic, viral, post-cardiotomy, purulent traumatic or tuberculosis) or an intercurrent manifestation of systemic disease (Friedberg, 1957; Kaplan, 1969).

Pathologically, pericarditis has been classified as:

1. Acute fibrinous pericarditis
2. Pericarditis with effusion
3. Chronic adhesive pericarditis, and
4. Constrictive pericarditis (Friedberg, 1957).

Haemorrhagic pericarditis is a variant of serofibrinous pericarditis, in which the effusion contains blood. It occurs most commonly in neoplastic, lymphomatous and tuberculous pericarditis, but may occur other chronic types (Friedberg, 1957).

Purulent pericarditis is almost always associated with an obvious primary infection at another site, although this primary infection may be quite mild (Friedberg, 1957; Cayler *et al.*, 1963). That arises:

1. by direct extension of intrathoracic infection
2. by hematogenous bacterial dissemination
3. by traumatic introduction of bacteria through the chest, or
4. occasionally by extension or perforation of the diaphragm from a sub-diaphragmatic suppurative focus such as a liver absces.

In Indonesia the disease is commonly as a complication of pneumonia (Zuidema, 1959). The causative organisms are usually the staphylococci, the pneumococci and hemolytic streptococci. Other micro-organisms such as *Hemophylus influenzae* (according to Gotoff and Fousek (1962) is very rare) and *P. tualarensis* are responsible.

Our first case has a serohaemorrhagic exudation with the positive Mantoux test. So the etiological diagnosis was directed to tbc.

The second case was undiagnosed, since the micro-organism of pericarditis material was not examined. However, it was clear that this pericarditis was associated with pneumonia.

## SUMMARY

Two cases of pericarditis with effusion admitted to the Department of Child Health, Gadjah Mada University Hospital were presented.

The suspicion was based on the presence of pericardial friction rub for the first case, and the presence of cardiac enlargement and liquid line (on X-ray) for the second case. Pericardiocentesis was performed to confirm the diagnosis and for treatment.

An accurate etiological diagnosis could not be made due to lack of laboratory facilities. The treatment was based on clinical and pathological findings.

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