

PURULENT PERICARDITIS: REPORT OF A CASE*

P. D. BECK,† M.B., CH.B. (CAPE TOWN), M.MED. (PAED.) (CAPE TOWN) AND L. B. KAHN,‡ M.B., B.CH. (RAND), M.MED. (PATH.) (CAPE TOWN), *Departments of Paediatrics and Pathology, University of Cape Town and Groote Schuur Hospital, Observatory, Cape*

Purulent pericarditis is a rare complication of acute bacterial meningitis. The cause of the meningitis and of the pericarditis in these instances is usually *Neisseria meningitidis*. We wish to report a fatal case of *Haemophilus influenzae* meningitis complicated by purulent pericarditis.

CASE REPORT

C.B., a 10-month-old Coloured male infant, was admitted to Groote Schuur Hospital with a 2-day history of restlessness and irritability. On examination, he was found to have a stiff neck. A lumbar puncture was performed and this showed purulent fluid, which had a protein content of 40 mg./100 ml., normal concentration of sugar, and a cell count of 378 polymorphonuclear leucocytes and 42 lymphocytes/cu.mm. *Haemophilus influenzae* was cultured from the cerebrospinal fluid and this organism was sensitive to chloramphenicol, erythromycin, tetracycline and streptomycin. Initially, he was treated with penicillin, chloramphenicol and Gantrisin. One week later, when the results of the culture became known, this regimen was changed to chloramphenicol and streptomycin. A chest X-ray taken 24 hours after admission showed a large cardiac shadow (Fig. 1). The child was extremely ill with a pulsus paradoxus, raised jugular venous pressure and 5-finger enlarged, tender liver. The ECG showed ST elevation in the chest leads.

Under ECG monitoring, 52 ml. of seropurulent fluid was aspirated from the pericardial sac. On the following two days, 30 and 40 ml. respectively of purulent fluids were aspirated. No more fluid was obtained on subsequent attempts at aspiration. At this stage, there was a marked improvement in the child's condition except for the development of a small right-sided pleural effusion on the sixth day. Forty ml. of straw-coloured fluid was aspirated from the pleural sac. The pericardial and pleural aspirates contained pus cells, but no organisms were cultured. Blood cultures were also negative.

Twenty days after admission, another chest X-ray was taken (Fig. 2). This showed a reduction in the size of the cardiac shadow. Two days later the child became extremely dyspnoeic, developed a severe tachycardia with pulsus paradoxus, and died.

Postmortem Findings

The heart (170 G) appeared to be a very large organ (Fig. 3). An extraordinary picture was seen when the pericardial sac was opened (Fig. 4). The visceral and parietal layers of the pericardium were separated by inspissated yellowish-green purulent material measuring between 1 and 1.5 cm. in thickness and causing compression of the heart itself. The myocardium showed generalized pallor. Histologically, the presence of a fibrino-purulent exudate in the pericardial sac was confirmed. The exudate

was undergoing organization adjacent to the myocardium. No organisms were detected on direct microscopical examination, nor were any cultured from the purulent material.

A fairly recent antemortem thrombus about 4 cm. in length was present in the right internal jugular vein.

The right lung (86 G) showed a fibrinous exudate on the pleural surface of the lower lobe. The liver (385 G) showed the effects of severe passive venous congestion, viz. centrilobular zonal congestion with zonal necrosis and fatty change in liver cells. The brain (900 G) showed no evidence of meningitis macroscopically or microscopically.

There was no evidence of spread of infection of the following organs examined microscopically: liver, thyroid, kidneys, brain, pancreas, spleen and adrenals.

The final anatomical diagnoses in this case were: pericarditis causing tamponade following *Haemophilus influenzae* meningitis, right-sided pleurisy, thrombosis of the right internal jugular vein and chronic passive venous congestion of the liver. The meningitis had completely resolved.

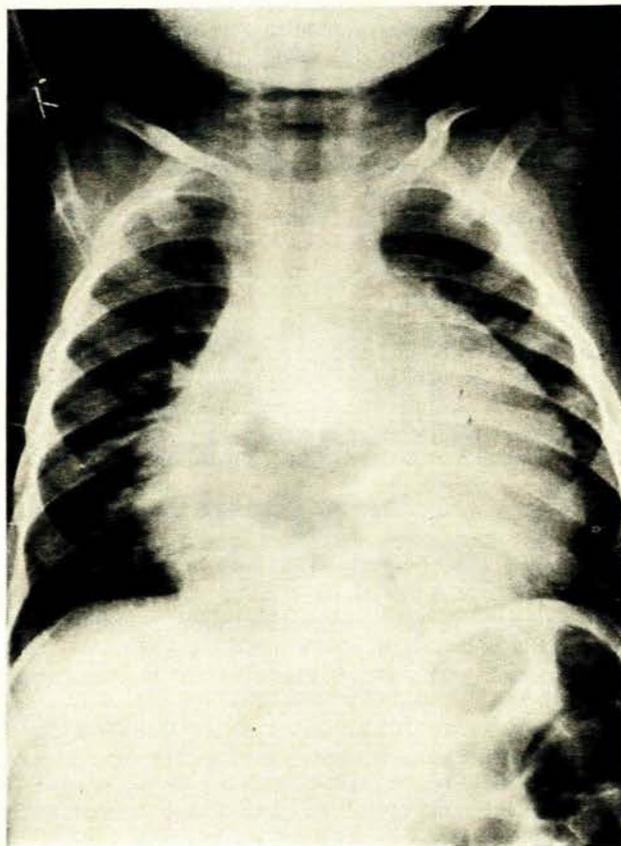


Fig. 1. X-ray taken 24 hours after admission showing cardiac shadow.

*Date received: 28 May 1969.

†Present address: 405 Oasim, Pearson Street, Port Elizabeth.

‡Present address: John J. Cochran Veterans Hospital, St Louis, Missouri, USA.

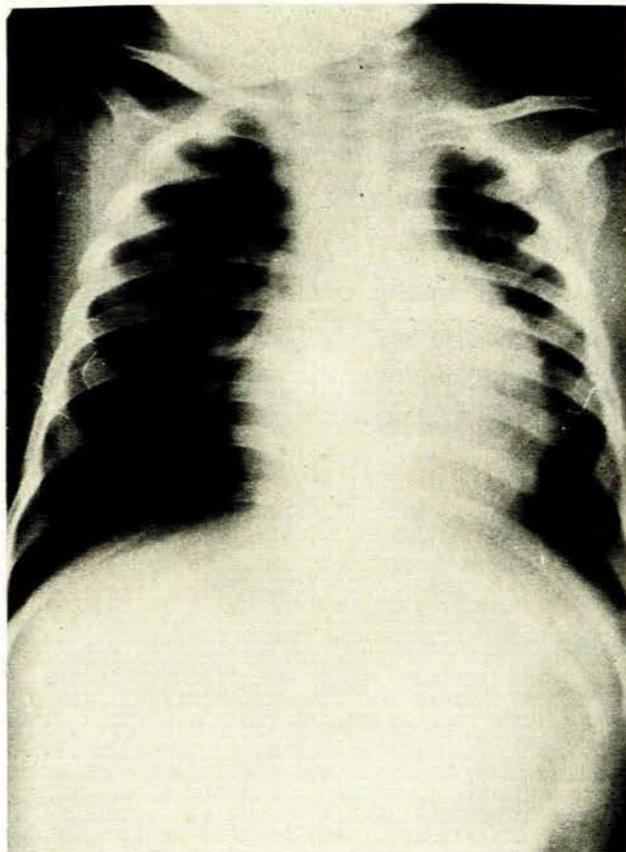


Fig. 2. X-ray taken 20 days after admission showing reduction in size of cardiac shadow.

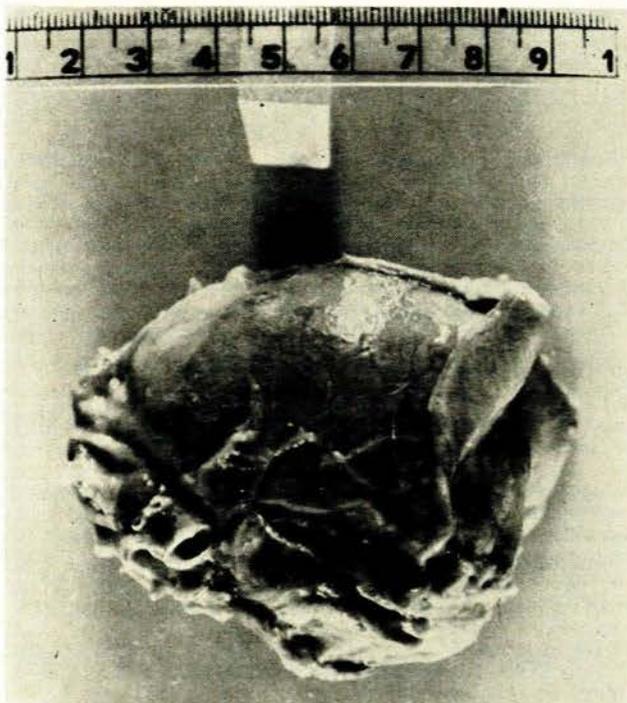


Fig. 3. Unopened pericardial sac showing apparent gross cardiomegaly.

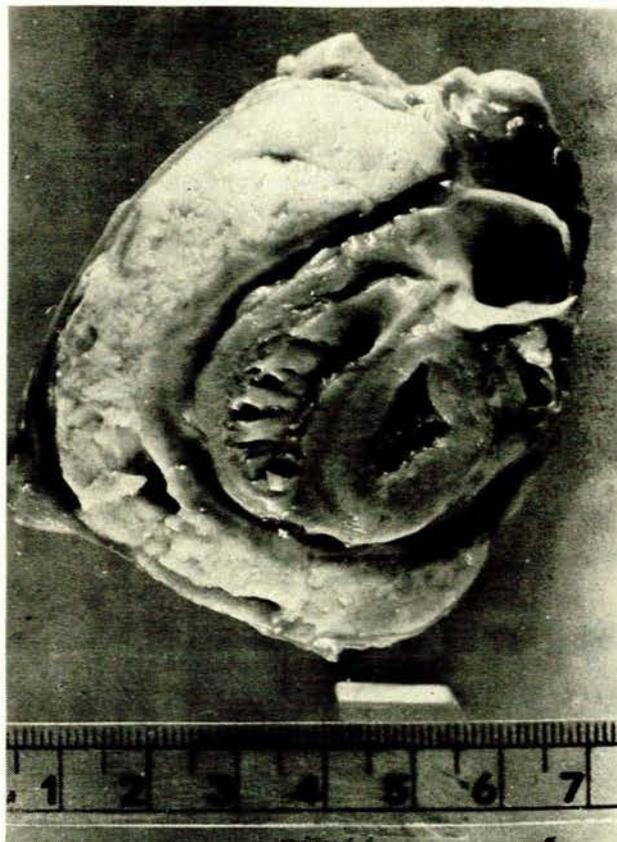


Fig. 4. Vertical section through heart showing separation of the pericardial layers by inspissated exudate.

DISCUSSION

Purulent pericarditis as a complication of acute meningitis is a distinct rarity. In the 8-year period 1959-1966 inclusive, there were 17 cases of purulent pericarditis at the Red Cross Children's Hospital, and 7 in the children's medical wards at Groote Schuur Hospital. Only one case was preceded by acute meningitis. In a review of 425 cases of purulent pericarditis collected from the world literature up to March 1969, Boyle *et al.*¹ found that 4% were the result of infection by neisseria meningitidis. Herrick² and Smithburn *et al.*³ reported meningococcal pericarditis in 4-6% of cases of meningococcal meningitis during epidemics of this disease.

Haemophilus pericarditis accounted for only 9 of the 425 cases in Boyle's review, and in most of these cases the primary focus of infection was in the respiratory tract. In some cases the pericardial involvement was considered to be the result of direct spread from contiguous infected lung. In other cases, haematogenous spread to the pericardium was postulated. In the case reported here, pericarditis developed 3 days after the onset of acute meningitis. The failure to isolate the organism from the pericardial aspirate is not surprising, in view of the antibiotic therapy which had already been administered.

According to Boyle, purulent pericarditis is diagnosed in a low percentage of cases during life. The clinical features include increased heart size, pericardial friction rub, decreased intensity of heart sounds, raised jugular

venous pressure, enlarged tender liver and a pulsus paradoxus. Several newer techniques are now being used and these should increase the percentage of cases diagnosed during life. These techniques include radio-isotope heart scanning with radioactive ^{131}I tagged to human serum albumin or angiocardiology to outline the intracavitary cardiac blood pool and demonstrate its relationship to the radiographic cardiac outline.⁴ The risk associated with these procedures is minimal.

Management of purulent pericarditis is a difficult problem. Despite antibiotic therapy, a high mortality rate still prevails. There were 19 deaths in the 24 cases in children seen at the Red Cross Children's Hospital and Groote Schuur Hospital. Early surgical drainage is important if fatal tamponade is to be avoided in cases which fail to respond to medical therapy.

SUMMARY

An unusual case of suppurative pericarditis complicating *Haemophilus influenzae* meningitis in an infant is described. Despite early pericardial aspirations, a fatal cardiac tamponade developed. There is a significant mortality even with antibiotic therapy. Early diagnosis and adequate surgical drainage is important in cases which fail to respond promptly to antibiotic therapy.

We wish to thank Dr R. McDonald, senior paediatrician, for valuable advice; Prof. J. D. L. Hansen for details of the Red Cross Hospital records; Prof. J. G. Thomson for the pathology report; and Dr J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish the case.

REFERENCES

1. Boyle, J. D., Peake, M. L. and Guze, L. B. (1961): *Medicine* (Baltimore), **40**, 119.
2. Herrick, W. W. (1918): *Med. Clin. N. Amer.*, **2**, 411.
3. Smithburn, K. C., Kempf, G. F., Zerfas, L. Y. and Gelman, L. H. (1930): *J. Amer. Med. Assoc.*, **95**, 776.
4. Folger, G. M. (1966): *Clin. Pediat.*, **5**, 225.