Primary meningococcal pericarditis with tamponade

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Summary: A case of primary purulent meningococcal pericarditis presenting with chest pain, hypotension and cardiac tamponade followed by oliguric renal failure is reported. Treatment with antibiotics and surgical drainage was successful.

Introduction

Pericarditis is an uncommon but recognized complication of meningococcal septicaemia and meningococcal meningitis (Dixon & Sandford, 1971; Morse *et al.*, 1971). However, purulent meningococcal pericarditis in the absence of clinically evident meningitis is rare. The present paper reports such a case complicated by cardiac tamponade and oliguric renal failure.

Case report

A 63 year old French woman presented with retrosternal chest pain, sweating and hypotension 3 weeks after recovering from a respiratory tract infection. She was found to have a temperature of 100.9°F, blood pressure 90/60 mm Hg and pulse 100 beats/min. The jugular venous pressure and heart sounds were normal. The white cell count was $15.1 \times 10^9/l$ (86% neutrophils), urea 6.2 mmol/l, cardiac enzymes, electrocardiogram and chest radiograph were normal.

Within 72 h she had become drowsy, confused, profoundly hypotensive and oliguric. Clinical features of meningitis were absent. Investigations showed blood urea 44 mmol/l, potassium 6.3 mmol/l, serum creatine kinase 1764 IU/l (normal 24–195), aspartate aminotransferase 3109 IU/l (normal < 40), lactate dehydrogenase 6175 IU/l (normal 230–460). On referral 2 days later the jugular venous pressure was raised at 8 cm H₂O, but there was no pericardial friction rub. The electrocardiogram showed paroxysmal atrial fibrillation and widespread ST elevation. The chest radiograph showed cardiomegaly and mediastinal widening. M-mode and 2-dimensional echocardiography demonstrated a large pericardial effusion compressing the right ventricle. Cardiac catheterization showed mean right atrial pressure

20 mm Hg and right ventricular pressure 40/20 mm Hg.

Pericardiocentesis yielded a small amount of thick yellow purulent material; pericardiotomy and surgical drainage was performed yielding 400 ml of pus and fibrinous exudate. Microscopy demonstrated numerous polymorphonuclear leucocytes with intra- and extracellular Gram negative diplococci, later identified as *Neisseria meningitidis*, serogroup B. It was not grown from blood cultures or nasopharyngeal swabs.

Benzylpenicillin (4 MU) was instilled into the pericardial space and 4 MU given 2 hourly intravenously. Within 24 h, the blood pressure rose to 150/90 mm Hg and the urine output to 200 ml/h. Four days post-operatively the pericardial drains were removed. Two days later she was alert and afebrile; a pleuropericardial friction rub was audible.

Ten days post-operatively she showed evidence of congestive cardiac failure which responded to diuretic therapy. Eight days later there were no signs of cardiac tamponade but a sinus tachycardia (130 beats/min) persisted. Repeat echocardiography demonstrated further pericardial effusion although aspiration was deemed unnecessary. Antibiotics were stopped after 20 days.

Three months later there were no signs of cardiac failure, tamponade or constrictive pericarditis. Echocardiography showed that the pericardial effusion had resolved.

Discussion

Orgain & Poston described the first case of primary meningococcal pericarditis in 1939. Rao *et al.* (1980) reviewed 10 other cases and reported two additional patients. Two further case reports (Jones *et al.*, 1979; Kwa *et al.*, 1981) have since appeared. Of the 16 cases

(including our own), 50% were under 20 y (2.5–63 y), and 9 were male. Chest pain and fever were the commonest complaint although dyspnoea was present in 8 and abdominal pain in 3. Cardiac tamponade developed in all except two cases, being a presenting feature in 3 cases and occurring within 3 days of admission in the remainder. Pericardial friction was audible in only 4 patients at presentation but appeared in 6 others during hospitalization. All patients had leucocytosis with a mean white cell count of $22.2 \times 10^9/l$. Electrocardiograms suggesting pericarditis were seen in 14 cases although the appearances were delayed in 5. Repeat chest radiographs showed an enlarging cardiac shadow in 5 cases although echocardiography was most valuable for assessing the size of the pericardial effusion before and after treatment.

In our case a massive rise in cardiac enzymes without electrocardiographic evidence of myocardial infarction suggests a primary infective myocarditis. Purulent pericarditis, cardiac tamponade, haemodynamic deterioration and renal failure ensued. The resolution of cardiac failure and the related complications after antibiotic therapy supports the concept of a meningococcal myopericarditis. In the reported cases *Neisseria meningitidis* was cultured from blood in 9 (56%) and from pericardial fluid in 10 (60%) suggesting possible pericardial invasion following meningococcaemia. Group C meningococcus is most often

responsible, our patient representing only the second due to serotype B.

Pericardiocentesis is indicated initially for diagnostic and therapeutic purposes. However, because repeat aspiration is often necessary, insertion of an indwelling catheter, pericardial window formation or pericardiectomy may be necessary when cardiac tamponade occurs. Intravenous penicillin is the drug of choice since resistance has not been reported in *Neisseria meningitidis*. Steroids are probably not necessary as long as antibiotic therapy and adequate surgical drainage are effectively maintained.

Finally, all reported patients have survived. This is in marked contrast to that of purulent pericarditis caused by other pathogens where mortality has been reported to be as high as 77% (Rubin & Moellering, 1975). Nevertheless, meningococcal pericarditis may lead to serious complications and death unless recognized early and treated vigorously.

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