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CASE REPORT

Meningococcal pericarditis and tamponade

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ABSTRACT

We report the case of a 37-year-old female with a complex manifestation of serogroup C meningococcal disease. The patient presented with symptoms and signs of pneumonia, sepsis and diffuse intravascular coagulation. Moreover, she suffered from a culture-proven pyogenic pericarditis that deteriorated into cardiac tamponade. Immediate pericardiocentesis was successful and eventually the patient recovered.

INTRODUCTION

Meningococcal disease is caused by *Neisseria meningitidis*, a gram-negative diplococcus. Humans are the only natural hosts in which meningococci are pathogenic.[1] The most common clinical manifestations of meningococcal disease are meningitis, sepsis and concurrent pneumonia. Much less frequent manifestations are conjunctivitis, arthritis, urethritis, pericarditis, otitis media, sinusitis and epiglottitis. A rare manifestation is chronic meningococcaemia.[2-4] We report a case of a 37-year-old female with a complex manifestation of meningococcal disease, including pyogenic pericarditis. In the months before her illness the incidence of meningococcal disease, caused by *N. meningitidis* serogroup C, had increased markedly in the Netherlands.[5]

CASE REPORT

A 37-year-old female with a history of borderline personality disorder was suffering from dyspnoea, dry cough, fever, episodes of palpitations and nausea. Her general practitioner suspected a pneumonia and prescribed amoxicillin 500 mg orally three times a day. The patient had no other medication. Her condition, however, did not improve and on the 4th day of her illness she was referred to our hospital.
On arrival the patient's condition was critical. She was tachypnoeic and cyanotic. Her blood pressure was 90/50 mmHg, pulse 130 beats/min and temperature 38.2°C. External jugular veins were distended and the central venous pressure was 23 cm of water. At cardiac auscultation S1 and S2 were soft, neither murmurs nor friction rub were heard. Dull percussion and bronchial breath sounds were found at the base of both lungs. The liver was slightly enlarged and tender. The extremities were cool and pale with mottling. Petechiae were absent at this stage. Neurological examination was normal.
The electrocardiogram demonstrated sinus tachycardia, low QRS voltage and slight negative T waves in leads V_1 to V_3. There was no electrical alternans. Chest X-ray showed an enlarged cardiac silhouette, consolidations in the inferior lobes of both lungs and pleural effusion. Laboratory investigation revealed a C-reactive protein of 278 mg/l, a leucocyte count of 26 x 10^9/l with left shift, a haemoglobin of 6.9 mmol/l, and a thrombocyte count of 106 x 10^9/l. INR was 2.50, fibrinogen 6.7 g/l, and D-dimer >2000 μg/l. The plasma creatinine was 761 μmol/l.
The differential diagnosis consisted of pneumonia and sepsis of unknown origin, complicated by renal failure and diffuse intravascular coagulation (DIC). Antibiotic therapy was started, consisting of amoxicillin, clavulanic acid and tobramycin intravenously.
Pericardial effusion was suspected because of the enlarged

cardiac silhouette, low-voltage ECG and elevated central venous pressure. Echocardiography confirmed a large volume of pericardial effusion and showed compression of both the right ventricle and the right atrium. Pericardiocentesis was performed which yielded 700 ml of viscous, straw-coloured fluid. Gram stain of the pericardial fluid showed small gram-negative diplococci and the leucocyte count was 128.8 x 10^9/l. The presumptive pathogen was *N. meningitidis* and antibiotic therapy was changed to penicillin G, 4 million units intravenously every six hours. After the pericardiocentesis the patient improved at once, the mottled aspect of her skin disappeared and diuresis recovered. A drainage catheter was left in the pericardium for two days.

Culture of the pericardial fluid and immunological reactivity identified *N. meningitidis*, serogroup C, serotype 2a, with good antibiotic susceptibility for both penicillin G (MIC 0.032 mg/l) and amoxicillin. Blood cultures taken before the initiation of the parenteral antibiotic therapy remained negative. A lumbar puncture yielded normal liquor and no pathogens on gram stain and culture. Cultures of expectoration did not reveal a specific pathogen. Serological examination showed a *Mycoplasma pneumoniae* IgM antibody titre greater than 1:20480. Cultures from urethral and vaginal smears excluded *Neisseria gonorrhoeae*.

The day after pericardiocentesis the patient's condition worsened because of ongoing sepsis and aggravation of the DIC (INR 3.11, thrombocytes 60 x 10^9/l), and petechiae and ecchymoses developed on the lower extremities. Low-dose heparin, extensive fluid and inotropic therapy were given. Unfortunately necrosis of both lower extremities developed, which resulted in amputation of both her right lower leg and three toes of her left foot. Eventually she recovered well.

DISCUSSION

Our patient presented with a clinical picture of sepsis due to meningococcal disease. The site of infection appeared to be a pericarditis. Invasive meningococcal disease can present in different ways. The most benign presentation is the transient meningococcaemia, the worst presentation is called the fulminant meningococcal sepsis.

Primary meningococcal pericarditis (PMP) is a rare presentation of meningococcal disease. PMP should not be confused with the reactive, immune complex-mediated pericarditis, which occurs in 10 to 20% of patients during the convalescent phase of invasive meningococcal disease.[6] PMP is mainly caused by *Neisseria meningitidis* serogroup C. This bacterium also caused the PMP in our patient.[7]

Interesting in our case is the way the patient presented. DIC was present on the day of admission and resulted in limb ischaemia for which amputation was necessary. This presentation of PMP is quite exceptional. PMP usually presents as a febrile disease without severe sepsis or DIC and usually runs a relatively benign course with an excellent prognosis, provided that appropriate antibiotics are given and, if necessary, pericardiocentesis or surgical release is performed. The meningococcus reaches the pericardium via the haematogenous route and the pericarditis is usually the only infectious site. Blood cultures are negative in more than half of the patients. Our patient also had negative blood cultures but she was treated with amoxicillin orally for four days before hospital admission.[7-10]

Serological examination indicated that the patient had recently suffered from an *M. pneumoniae* infection. This infection probably facilitated meningococcal invasiveness by diminishing the barrier function of patient's respiratory mucosa.[11,12] Caution should be taken when interpreting the serological test for *M. pneumoniae* because cross-reactive antibodies appear to play a role in positive results found in patients with other bacterial infections.[13]

CONCLUSION

This case report shows that PMP can present as a severe ongoing sepsis complicated by DIC that can even lead to severe ischaemia of the extremities.

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