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Meningococcal Pericarditis in the Absence of Meningitis

Summary: A 16 year-old female presented with cardiac tamponade due to purulent meningococcal pericarditis without concomitant meningitis or meningococcaemia. She recovered after aspiration of the pericardial effusion and administration of a high dose of benzylpenicillin via a continuous infusion.

Zusammenfassung: *Meningokokken-Perikarditis ohne Meningitis.* Ein 16 Jahre altes Mädchen wurde mit Herztamponade bei eitriger Meningokokken-Perikarditis ohne Meningitis oder Meningokokken-Septikämie aufgenommen. Nach Aspiration des Perikardergusses und Behandlung mit hohen Dosen Benzylpenicillin über Dauerinfusion trat Heilung ein.

Introduction

Pericarditis is a not uncommon complication of meningococcal meningitis, since it occurs in approximately 19% of the patients with this disease. Cultures of the pericardial fluid usually remain sterile (1–3). Isolated purulent meningococcal pericarditis in patients without meningitis or meningococcal sepsis is, however, exceedingly rare (4). We had a patient with isolated purulent meningococcal pericarditis who was cured by pericardial aspiration and high doses of benzylpenicillin administered via continuous infusion.

Case Report

A 16-year-old female had had an acute otitis media which drained spontaneously two weeks before admission. Seven days later she became nauseated and started vomiting. On the tenth day she developed a temperature of 40°C without chills. She complained of epigastrical pain which was aggravated by breathing and became jaundiced; headache and nuchal rigidity were denied. She was admitted to another hospital for suspected peritonitis. When, however, ultrasound examination revealed a large amount of pericardial fluid, she was referred to our hospital.

On admission, the patient was jaundiced and critically ill but alert and fully oriented. There was no nuchal rigidity or papillary oedema, and on neurologic examination no relevant abnormalities were found. The blood pressure could not be measured, the pulse rate had risen to 130 beats/min and the central venous pressure was greatly elevated. Her temperature was 36.8°C. The right tympanic membrane was dull without signs of active inflammation. The heart was enlarged; no murmur or friction rub could be heard. The lungs were clear. The liver was enlarged 4 cm below the right costal margin and tender. An electrocardiogram showed sinus tachycardia and ST elevation in leads I, II, a V_f and V_2–V_6. Echocardiography revealed a massive pericardial effusion. Since neurologic examination showed no symptoms of meningitis, a lumbar puncture was not performed.

Shortly after admission, 800 ml of pericardial fluid containing 70 g protein/l were aspirated. A Gram stain showed gram-negative diplococci and a culture yielded *Neisseria meningitidis,* serogroup C. Blood cultures remained sterile. Aspiration of the pericardial fluid led to a dramatic improvement in the patient's clinical state. Vomiting stopped and jaundice disappeared. The meningococcal infection was treated with 12 million U of benzylpenicillin per day administered by continuous infusion which resulted in a concentration of 10 U/ml in the serum and 9 U/ml in the pericardial fluid. Initially 2 g of cefotaxime were administered intravenously four times a day, because the results of the Gram stain suggested the possibilities of an infection with a β-lactamase-producing gonococcus. However, when the results of the pericardial fluid culture were available and negative for this microorganism, cefotaxime was discontinued. The patient became afebrile on day 2. The pericardial drain was then removed because fluid production had stopped. However, on day 4 temperature started to fluctuate between 38°C and 39°C (Figure 1).

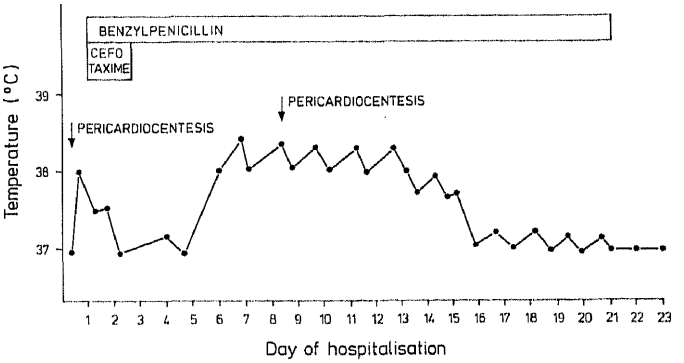

Figure 1. Course of temperature during hospitalisation. Benzylpenicillin was given at a dose of 12 million U/day; cefotaxime at a dose of 4×2 g i.v.

A chest X-ray revealed an increasing amount of pleural fluid. The ESR rose to 50 mm but the leukocyte count was not elevated. C_3, C_4 and CH_{50} levels, which had been diminished on admission, became normal on day 6. During hospitalization, no immune complexes were detected in the serum. Because of increasing pericardial fluid, another pericardiocentesis was performed on day 6, but only a small amount of fluid containing 43 g protein/l could be aspirated; a Gram stain revealed no microorganisms and the culture remained sterile. At this stage we concluded that the clinical picture was most probably due to an immunological process.

Without additional measures the patient became afebrile on day 14, and the pericardial and pleural fluids disappeared. On day 21, the benzylpenicillin was stopped. On day 23 the patient was

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discharged in good condition. During a follow-up of more than one year she remained well without signs of pericardial constriction.

Discussion

Pericarditis caused by *N. meningitidis* is uncommon since fewer than 3% to 4% of the cases of bacterial pericarditis can be attributed to this microorganism (5, 6). Usually it occurs in patients with meningococcal meningitis (1). However, in 1971 a case of purulent meningococcal pericarditis without meningitis or meningococcaemia was reported (7). The primary form without meningitis or meningococcaemia is deemed to be exceedingly rare (4, 7, 8). In our patient pericarditis was possibly due to bacteraemia during otitis media two weeks before admission.

Meningococcal pericarditis is usually caused by *N. meningitidis* serogroup C, as was the case in our patient. The predominance of this serogroup may reflect the inability of the host to clear this microorganism from the pericardial cavity (9).

Cardiac tamponade resulting from meningococcal pericarditis can be treated by pericardiotomy or pericardectomy (9). However, for our patient simple pericardiocentesis together with a high dose of benzylpenicillin administered by continuous infusion proved to be adequate. Continuous infusion was considered necessary in order to maintain a high concentration of penicillin at the site of infection. This regimen yielded drug levels in the pericardial fluid equivalent to serum levels. In addition, continuous infusion compensates for the short half life and the short postantibiotic effect of penicillin (10).

During antibiotic treatment, serous pericarditis developed. This is not an uncommon complication of meningococcal meningitis (1, 11, 12). It has been suggested that such an effusion is initially purulent but is rapidly sterilized by antibiotic treatment (2). An immunological mechanism could also account for this complication (7, 13). Our patient exhibited normal complement levels during the serous pericarditis; no immune complexes could be detected in the serum. However, the relapse of fever and the increasing amount of sterile pleural fluid are compatible with an immunological cause. Opinions on corticosteroid therapy for serous pericarditis are controversial (1, 12). Our patient recovered completely without any further therapy.

Addendum

Recently we observed a second patient with a clinical disease identical to the one described above. This 57 year old man was admitted because of fever, chills and severe abdominal pain. An extensive pericardial effusion was found, that was drained because of pericardial tamponade. From the pericardial fluid a *Neisseria meningitidis* group C was cultured. Blood clutures remained sterile. There were no other signs of meningococcal infection, especially not in the CNS. The patient was treated with 12×10^6 U of benzylpenicillin by continuous infusion for 14 days. On the 5th day he developed a serous effusion in the left pleural cavity. Otherwise the clinical course was uneventful and the patient recovered completely.

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