Culture-Negative Pericarditis Caused by *Neisseria meningitidis* Serogroup C

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We describe a case of primary purulent culture-negative pericarditis caused by *Neisseria meningitidis* serogroup C occurring in an 8-month-old previously healthy boy, which was detected in pericardial fluid by broad-spectrum PCR amplification.

CASE REPORT

A previously healthy infant aged 8 months was admitted to the Emergency Department of Louis Mourier Hospital (Paris, France) with a 10-day history of several daily feverish peaks of between 39 and 40°C. On examination, the patient had normal hemodynamic parameters, and his temperature was 36.6°C. He was pale, with neither spots nor purpura. The examination of the ears and nose was unremarkable. There was no hepatosplenomegaly or local pain. Neurologic and pulmonary clinical examinations revealed no abnormalities. Heart sounds were diminished in intensity. A chest radiograph showed considerable cardiomegaly, with a cardiothoracic index of 0.6. The cardiac echography revealed a pericardial effusion and thickened pericardium. There was no sign of cardiac tamponade. Blood and urine samples were taken for culture. Laboratory studies showed a white blood cell (WBC) count of 21.10⁹/mm³, with 45% polymorphonuclear neutrophils, 11% monocytes, and 44% lymphocytes. The C-reactive protein level was 167 UI/liter. Despite the absence of neurologic signs, cerebrospinal fluid was collected. It was clear, with no WBCs, 2 red blood cells/mm³, and normal glucose and protein levels. The patient was transferred to the cardiac surgery care unit of Necker-Enfants Malades hospital (Paris, France) for management of pericarditis. He had an emergency pericardiocentesis and was then started on a probabilistic intravenous antibiotic therapy including cefotaxime (200 mg/kg of body weight/24 h), and cefotaxime (200 mg/kg/24 h), and ciprofloxacin (30 mg/kg/24 h), and then oral amoxicillin was given for 8 days. The patient was afebrile.

After 2 days of antibiotic therapy, the patient was afebrile. Treatment with intramuscular ceftriaxone was continued for 15 days, and then oral amoxicillin was given for 8 days. The pa-
tient did not receive corticosteroids. Ten months later, the child presented a normal clinical examination, electrocardiography, and cardiac echography, with just a minor echoic pericardium showing no signs of pericardial constriction.

**Discussion.** *Neisseria meningitidis*, a gram-negative diplococcus that colonizes the nasopharynx, can spread to the bloodstream and cause invasive disease. The most common clinical presentations of invasive meningococcal disease are meningococcal septicemia and meningitis (11). Pericarditis is a rare presentation of invasive meningococcal disease. The most common clinical localization of *N. meningitidis*, representing 0 to 14% of cases of purulent pericarditis (2–4, 10), according to the published series. These infections occur primarily in adults. Only three cases of *N. meningitidis* pericarditis in children under 3 years of age have previously been reported (1, 6, 8).

The patient had an atypical medical history without clinical signs of meningitis or other foci of meningococcal infection. Three types of meningococcal pericarditis have been classified by Finkelstein et al. (5) on the basis of clinical and biological criteria: primary, disseminated, and immunoreactive meningococcal pericarditis. Our patient probably presented primary meningococcal pericarditis. In fact, he presented no clinical manifestations of meningitis or other meningococcal localizations, and he recovered quickly after aspiration of pericardial fluid and appropriate antibiotic therapy without corticosteroids (4, 10).

Considering the discordance between the intensity of the inflammatory syndrome and the negativity of Gram stains and cultures of the pericardial fluid and blood samples, we could not exclude a possible antibiotic treatment preceding the hospitalization (even though this was not mentioned in the patient’s clinical history) or the presence of another uncultivable organism. However, the fact that the only sequences amplified by PCR were of *N. meningitidis* makes it highly improbable that inflammation was due to coinfection with an uncultivable bacteria.

To our knowledge, this is the first case of culture-negative pericarditis due to *N. meningitidis* serogroup C that has been diagnosed only by universal 16S rRNA PCR amplification. We think that it may be helpful to perform this technique for patients with clinical evidence of purulent pericarditis with culture-negative septic samples.

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**REFERENCES**