Acute Right-Sided Heart Failure Caused by *Neisseria meningitidis*

GUILLAUME TALDIR,1 PERRINE PARIZE,2 PHILIPPE ARVIS,3 AND CHRISTOPHE FAISY1*

Medical Intensive Care Unit,1 Department of Microbiology,2 European Georges Pompidou Hospital, Assistance Publique–Hôpitaux de Paris, Paris–Descartes University, Paris, and Department of Emergencies,3 Argentan Medical Center, 61200 Argentan, France

Correspondence: Christophe Faisy, Medical Intensive Care Unit, European Georges Pompidou Hospital, 20 rue Leblanc, 75015 Paris, France. Tel: +156093201, fax: +156093202.

E-mail address: christophe.faisy@egp.aphp.fr
Meningococcal myocarditis is a rarely diagnosed affection and could be the consequence of primary invasive infection or late immunologic complications. An unusual presentation of meningococcemia in an immunocompetent adult is described and specifies Neisseria meningitidis to cause elective right-sided heart failure in case of acute myocarditis.
A 47-year-old man was admitted to the hospital with a complaint of chills, nausea, vomiting and diarrhea for 24 h. The patient took no medications, denied tobacco, alcohol, or illicit drug use, and no cardiovascular risk factors were present. Vital signs on admission were as follows: temperature 38.5°C; heart rate, 120 beats/min; blood pressure 78/45 mmHg; and respiratory rate 30 breaths/min. Examination revealed the following: chest, clear bilaterally; cardiac, tachycardic and regular, no murmur, no peripheral edema, and flat neck veins; abdomen, non-tender, normal bowel sounds, without hepatosplenomegaly; neurologic, normal state of consciousness, and no sign of meningism nor sensorimotor impairment; cold extremities, without skin rash. Laboratory investigations revealed a level of C-reactive protein of 114 mg/liter and a white blood cell count of \(27 \times 10^9\)/liter. Cardiac biomarkers, kidney and liver function, coagulation study and urinalysis were normal. Blood and stool cultures were also performed. The first 12-lead electrocardiogram (ECG) demonstrated isolated sinus tachycardia. Chest X-ray and abdominal ultrasound failed to show any pathology.

The patient was initially treated with fluid resuscitation and IV cefotaxime (3 g/day) and ciprofloxacin (400 mg twice day). Systemic hypotension initially resolved. Seven hours after admission, the patient developed acute onset of orthopnea and his exam was significant for a jugular veins distention. The next ECG revealed acute concave upwards ST-segment elevation in posterior and lateral leads, without any mirror in the opposite leads. Cardiac troponin level dramatically raised (55 ng/ml). A transthoracic 2-dimentional echocardiography showed a posterolateral dyskinesis, normal inferior kinetic and impaired right ventricular contractility, dilation of the inferior vena cava and no pericardial effusion. A cardiovascular magnetic resonance imaging (MRI) was performed and demonstrated impairment of right ventricular function associated with non-ischemic damages (Fig. 1A). The signs of heart failure and the ST-segment elevation resolved within 24 h without development...
of Q weave. On day two, *Neisseria meningitidis* was isolated from initial blood culture (BacT/Alert® blood culture system, identification by biochemical kit API® NH, BioMerieux, Craponne, France), establishing the diagnosis of meningococcemia. The phenotypic determination, based on the antigenic formula (serogroup, serotype, serosubtype) revealed a serogroup C isolate. The strain was susceptible to antibiotics of clinical interest (minimum inhibitory concentration tested for penicillin G, amoxicillin and cefotaxime). The clinical exam revealed then few purpuric lesions and the absence of neurological impairment. No cerebral spinal fluid sample was collected given the absence of meningeal signs.

The patient received high dose parenteral amoxicillin (14 g daily) intravenously for 10 days. Cardiac troponin normalized within 7 days. Serological results for atypical and fastidious organisms (including *Brucella* species, *Yersinia* species, *Coxella* species, and *Treponema pallidum*) and viruses (including human immunodeficiency virus, influenza viruses, cytomegalovirus, parvovirus, herpes viruses, and hepatitis A, B, and C viruses) were negative. Dosage of complement was within normal range. A second MRI was performed at day 11 and showed a slight expansion of the myocardial damages towards the lateral wall (Fig. 1B). A third MRI at day 32 demonstrated a regression of both myocardial edema and non-ischemic late gadolinium enhancement in T1-weighted (Fig. 1C) images whereas moderate posterolateral wall motion abnormalities and right ventricle dysfunction persisted. Finally, a MRI performed 5 months later confirmed recovery of the right ventricle (Fig. 1D) and clinical follow-up, including an exercise testing, was satisfactory.

*Neisseria meningitidis* remains a worldwide human pathogen responsible for epidemic meningitis and rapidly progressing fatal shock. The virulence of this Gram-negative diplococcic is mainly related to capsule expression and six capsular groups are associated with the majority of invasive diseases (A, B, C, W-135, X, Y) (1). The meningococcal disease has an annual
incidence ranging from 0.35 in the United States to 1 case per 100,000 population in Europe, marked by the occurrence of outbreaks (1). Serogroups B and C are responsible for the majority of cases of meningococcal disease in Europe; serogroups B, C and Y predominate in the Americas, and serogroups A, C and W135 cause most disease in Asia and Africa. Asian epidemiology of the meningococcal disease is highly heterogeneous depending on varied ethnicities, population density, socio-economic conditions and healthcare availability, and reveals an increasing gradient of incidence from poorer to more developed countries and a particular trend to serogroup A outbreaks (2). A large spectrum of clinical presentations are described, the two most frequent being meningitis and fulminant meningococcal septicemia. Nearly one-third of patients present with initially paucisymptomatic forms ("flu-like") or abdominal syndromes without signs of meningitis leading to erroneous diagnosis (1). Fulminant meningococcemia is characterized by a rapid proliferation of bacteria in the circulation with very high concentration of meningococci and endotoxin, causing shock with multiple organ failure, severe coagulopathy and few signs of meningitis. Other atypical presentations of meningococcal disease include pericardial infection, arthritis, conjunctivitis, panophthalmitis, pneumonia or infections of the urogenital tract (1-3). Bacteremia is the key step preceding any form of primary invasive infection or late immunologic complications.

Myocarditis is a rarely diagnosed affection in meningococcal disease. To our knowledge, a quasi-elective right ventricular impairment due to meningococcal myocarditis has never been described. However, the frequency of myocardial failure during meningococccemia is probably underestimated. This was suggested by autopsy series which have highlighted that myocarditis (defined by histologic evidence of myocardial infiltration with inflammatory cells) was found in 27% to 57% of children who died of meningococcal infection (4). In the present case, the diagnosis was made on the basis of the combination of clinical manifestations of myocardial dysfunction, increased serum cardiac troponin level and typical
echocardiographic and MRI abnormalities. MRI is a non invasive method to diagnose myocarditis without the risk of endomyocardial biopsy. We chose, here, not to perform endomyocardial biopsy because we estimated the benefit/risk balance unfavorable and MRI criteria for myocarditis were in accordance with the Lake Louise International Consensus (5).

Myocardial dysfunction is known to be a central component in the pathophysiology of septic shock occurring during fulminant meningococcal septicemia. The involvement of cytokines, and more particularly interleukin-6, has been suggested to explain the pathogenesis of myocardial dysfunction in meningococcemia (6). Moreover, meningococcal pericarditis which is described in 3% to 19% of patients with neisserial infection is also thought to be mediated by an immunologic process as suggested by high levels of cytokines in the pericardial fluid (3, 7, 8). Our case of meningococcal myocarditis was probably primary but we cannot exclude a delayed immunologic process in view of the progression of the morphological and functional abnormalities revealed by MRI.

We did not identify any risk factor for meningococcal disease for the patient, particularly no previous travelling history, but it is noteworthy that he wasn’t immunized against any serogroup of *N. meningitidis*. Several meningococcal vaccines are available, except for serogroup B which development of broadly effective vaccines remains a major issue as long as this serogroup is responsible for endemic disease and outbreaks (9). In many industrialized countries, routine meningococcal immunization is recommended for children and people in high-risk groups or traveling to countries with highly endemic or epidemic disease, with marked impact on serogroup C disease in particular (10).

This first documented report of acute elective right-sided heart failure caused by *N. meningitidis* emphasizes the need for getting blood culture results in case of acute myocarditis and illustrates the usefulness of MRI for evaluating the myocardial damages of this complication of meningococcemia.
ACKNOWLEDGEMENTS

We thank Eric Brugières (Department of Radiology), Morad Djebbar (Medical Intensive Care Unit) and Jean-Luc Mainardi (Department of Microbiology) from the European Georges Pompidou Hospital, Paris, for assistance during the manuscript preparation.

REFERENCES


FIGURE LEGENDS

FIG 1 Cardiovascular magnetic resonance imaging performed initially (A), at day 11 (B), day 32 (C) and day 180 (D) after meningococcemia and showing extended thickening and intense hypersignal (T2-weighted image) of the right ventricular free wall (red arrows), corresponding to myocardial edema, partially regressing at days 11 and 32 (T1-weighted images), and late gadolinium contrast enhancement of the left ventricular posterolateral segment (white arrows), expensed at day 11 and persisting at one month. Recovery was confirmed at day 180. RV: right ventricle; LV: left ventricle.