Gonococcal infection can be associated with septic shock leading to multiple organ failure and death.

Gonococcal infection is rarely associated with septic shock. We describe the first case of septic shock related to confirmed disseminated gonococcemia, and discuss briefly the possible reason for this unusual presentation.

A 53-year-old woman was found unconscious in her apartment. The temperature inside was low and the patient had deep hypothermia (temperature, 28°C). She had a history of alcoholism that had led to liver cirrhosis and severe malnutrition. She was not known to have any risk factors for sexually transmitted disease. On admission to the intensive care unit, her blood pressure was 55/32 mm Hg, and her heart rate was 40 beats/min. Her Glasgow Coma Score was 9. There was no sign of meningism. Examination of the skin showed mottling on the patient’s knees. Laboratory values were as follows: WBC count, 21,000 cells/mm³ (82% neutrophils); hemoglobin level, 3.5 g/dL; platelet count, 131,000 cells/mm³; prothrombin time, 18%; proaccelerine level, 29%; and fibrinogen level, 1.9 g/L. The patient had acute renal failure with oliguria and deep metabolic acidosis. A chest radiograph appeared normal. Three samples of blood were obtained for culture.

The patient underwent mechanical ventilation. Gradual rewarming, fluid resuscitation, and administration of fresh frozen plasma and RBCs led to temporary improvement of the patient’s hemodynamic status. Endoscopic gastroscopy revealed multiple gastroduodenal ulcers, which suggested that shock was related to gastroduodenal bleeding. No antibiotic therapy was administered. However, severe shock recurred 12 h after admission, with diffuse mottling of the skin, necrosis of the extremities, and disseminated intravascular coagulation, but without recurrence of a bleeding event. There was neither petechial rash nor evidence of acute synovitis. After lumbar puncture was done and new blood samples were obtained for culture, empiric antibiotic therapy with cefotaxime and amikacin was initiated.

Cultures of 2 blood samples drawn before administration of antibiotics grew gram-negative diplococci by the fifth day after admission to the hospital. The isolate was strictly aerobic and grew only on chocolate agar with 10% CO₂; results of catalase and oxidase tests were positive. The isolate produced propyl-p-nitroanilide and acidified glucose when tested by means of the RapID NH kit (Biotech), whereas it only acidified glucose when tested by means of the Neisseria 4H kit (Diagnosis Pasteur). This biochemical profile is specific for the species Neisseria gonorrhoeae, and identification was confirmed by the Neisseria French Laboratory (Institut Pasteur). The strain produced β-lactamase, as determined by the chromogenic cephalosporin disk test (bioMérieux), and was susceptible to amoxicillin-clavulanate.

The results of cultures of CSF samples and of vaginal secretions were normal, and the results of serologic tests for HIV and hepatitis C virus were negative. A sample of ascitic fluid was cultured and results were normal, although a WBC count of ascitic fluid found 1230 cells/mm³ (75% neutrophils), suggesting primary peritonitis. Nevertheless, ascitic fluid and CSF specimens were obtained and cultured 4 days after antibiotic therapy was begun. Specimens from the urethra, throat, and rectum were not obtained. Transesophageal echocardiography revealed that no vegetations were present. Left ventricular function was hyperdynamic. Levels of all components of complement were extremely low. Over the next few days, multiple organ failure developed, leading to refractory shock, and the patient died 1 week after admission to the hospital.

We could identify only 1 previously published case report of septic shock related to gonococcemia [1]. Because this association is extremely rare, the identification of the Neisseria strain in that report was questioned [2]. For our case, the various biochemical tests performed on the isolate confirmed the identification of the strain as a gonococcus. Furthermore, the strain was β-lactamase positive, a common feature of gonococcus isolates in France, whereas β-lactamase–producing meningococci
are extremely rare (only 4 reports, of which none were from France).

Why disseminated gonococcal infection is rarely associated with septic shock remains unexplained. Variation in the clinical expression of gonococcemia may be due both to phenotypic features of the infecting strain and to host-dependent factors [3]. The resistance of gonococci to complement-mediated killing is due to sialylation of the lipopolysaccharide, which prevents bacterial antibody from reacting with target sites [4], and could prevent the cytokine activation associated with septic shock. In the case we describe, a large amount of inoculum in an immunocompromised host may in part explain the severe infection with a microorganism that has “less potent” lipopolysaccharide. Host complement deficiency, which is reported in patients with cirrhosis, also predisposes to gonococcal and meningococcal bacteremia [5]. The low levels of complement components in our patient may have been secondary to sepsis. Finally, we hypothesize that earlier administration of antibiotics might have improved the outcome for the patient.

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References