Fatal Acute Cellulitis Due to Neisseria meningitidis

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We describe the first fatal evolution of cellulitis due to Neisseria meningitidis serogroup Y involving an 85-year-old woman. She presented with an extensive cellulitis of the left side of the face, neck, and thorax and septic shock. In spite of active antibiotic therapy, evolution was rapidly fatal.

CASE REPORT

An 85-year-old woman was admitted to the Intensive Care Unit of Saint-Germain-en-Laye Hospital with cellulitis of the face and neck and septic shock. She had been bedridden since 1998 following a traumatic vertebral compression (fourth lumbar vertebra) and right cerebral ischemia. She was undergoing corticosteroid therapy (0.5 mg of oral prednisone/kg of body weight/day for 2 years) for polymyalgia rheumatica. She had chronic heart and renal failure. Asthenia associated with pain on the left side of her neck began 8 days before admission. At the time of admission, her temperature was 38.3°C, her pulse rate was 120 (tachyarrhythmia), and her blood pressure was 80/50 mm Hg. She was conscious and alert. There were no symptoms of meningitis. The left side of the neck, the left shoulder, and the upper anterior part of the chest showed a painful erythema and edema and were swollen and warm. Clinical examination showed no angina and the recent partial loss of a left premolar. Otorhinolaryngologic evaluation showed a normal larynx, hypopharynx, and oropharynx and no phlegmon. A tumefaction of the submandibular space was observed; thus, submandibular sialadenitis was suspected. Laboratory findings included a white blood cell count of 2.71 × 10⁹/liter with 46% neutrophils, a hemoglobin level of 7.1 g/dl, a hematocrit of 24.2%, a platelet count of 279 × 10⁹/liter, a C-reactive protein level of 279 mg/liter, a procalcitonin level of 31.11 ng/ml, a lactate level of 6.9 mmol/liter, and a C-reactive protein level of 279 mg/liter (n < 10), a procalcitonin level of 31.11 ng/ml (n < 0.5), and a lactate level of 6.9 mmol/liter (n = 0.5 to 2.5). Computer tomography of the face, neck, and chest, after intravenous (i.v.) administration of contrast material, revealed the stranding of subcutaneous fat associated with skin thickening on the left side of the neck. The thickening of the deep fascia and the enlargement of neck muscles with blurred contours were also observed. Sialadenitis was absent. Neither walled-off, subcutaneous fluid nor deep fluid was visible. No involvement of the vascular structures was found. Blood was drawn for culture (four samples) and inoculated in aerobic and anaerobic blood culture vials (BACTEC 9240; BD Diagnostic Systems, Sparks, Md.). Saline solution was injected into the hypodermis of the submandibular area and then aspirated. This liquid, obtained by needle aspiration, was also incubated in blood culture vials. The treatment of the septic shock was initiated i.v. and included penicillin G (2 × 10⁶ U/6 h) and clindamycin (600 mg/8 h), blood transfusion, fluid expansion, and vasopressors. Oral corticotherapy was stopped on admission, but hydrocortisone (50 mg every 8 h i.v.) was given. However, evolution was rapidly negative, with the extension of the cellulitis, anuria, loss of consciousness, and multiorgan failure, and the patient died on day 2. There were no biological or clinical signs of adrenal insufficiency. Etiology of this cellulitis was assessed on day 3 after the death of the patient: two out of the four blood cultures and the liquid aspirate were positive for Neisseria meningitidis serogroup Y. This strain was susceptible to β-lactams. The MICs, as measured by E-test, were 0.064 mg/liter for penicillin G, 0.125 mg/liter for amoxicillin, 0.008 mg/liter for cefotaxime, and 0.023 mg/liter for pefloxacin.

Cellulitis due to N. meningitidis is a very rare manifestation of meningococcal disease. The international literature concerning this subject is very scarce (13). Cellulitis is an acutely spreading infection of the skin involving the deep dermis and subcutaneous fat, usually due to a group A beta-hemolytic Streptococcus sp. or Staphylococcus aureus. However, cellulitis must be considered a possible clinical manifestation of meningococcal disease. The present case is to our knowledge the first fatal cellulitis due to N. meningitidis (13, 18). However, rates of fatal evolution of meningococcal disease are still very high and have stayed relatively stable over the past 20 years: 9 to 12%, with a rate of up to 40% among patients with meningococemia (16). The most typical manifestations of meningococcal disease are meningitis (50% of patients), meningococcemia, (5 to 20%), and pneumonia (5 to 15%) (14–16). Nontypical manifestations, including conjunctivitis, otitis media, epiglottitis, arthritis, urethritis, pericarditis, and cellulitis, have been described (1, 2, 9, 13, 16, 17). In the United States, the number of cases involving serogroup Y has increased during the last 10 years, and from 1996 to 1998 one-third of the cases were due to serogroup Y (15). In France, the data obtained from the National Reference Center (http://www.pasteur.fr) are very different: in 2001, serogroup B accounted for 52.3% of meningococcemia cases, serogroup C accounted for 38%, serogroup W135 accounted for 7%, and serogroup Y accounted for 2%.

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Even if skin lesions are very common in meningococcal disease, cellulitis is so rare that it has not been mentioned in recent reviews (3, 16). Only two cases of cellulitis of the face or neck involving adults have been published (6, 18). Our present case involved the oldest patient (85 years). The first case was a 76-year-old woman with cellulitis of the face and neck associated with sialadenitis (6). Predisposing factors (diabetes mellitus and corticosteroid therapy) and bacteremia were present. Evolution was favorable after a 10-day course of antibiotic therapy (cefuroxime). The second case was a cervical cellulitis associated with meningococcal supraglottitis (18). The patient, a 44-year-old woman, had no predisposing factors, but bacteremia was present (N. meningitidis serogroup Y). She recovered fully after 16 days of antibiotic therapy (ceftriaxone and clindamycin on day 1 and then ampicillin-sulbactam). Five other case reports of adults with cellulitis due to N. meningitidis involved limbs. Ploy-Song-Sang et al. described a 64-year-old woman with cellulitis of the ankle, without bacteremia (12). Predisposing factors (chronic heart failure, plasma cell dyscrasia, diabetes mellitus, and stasis dermatitis due to venous insufficiency) were present. The absence of bacteremia and the presence of skin lacerations raised the possibility of a primary cutaneous infection of the leg. The patient was discharged after 3 weeks of antibiotic therapy (tetracycline for 2 days, chloramphenicol for 5 days, and erythromycin for 19 days). Another nonbacteremic cellulitis involved a 50-year-old man with necrotizing fasciitis of an arm and a leg (8). A recent nonsteroidal anti-inflammatory therapy was supposed to be the predisposing factor. N. meningitidis serogroup C was isolated from subcutaneous and bulla aspirates. However, involvement of two distant sites suggested hematogenous dissemination. A favorable outcome was obtained after surgery and antibiotic therapy (penicillin G and netilmicin IV). Lin et al. described a 45-year-old woman with meningococcal endocarditis presenting as calf cellulitis (7). The patient had predisposing factors (mixed connective tissue disease, hypocomplementemia, and valvular heart disease). N. meningitidis serogroup Y was isolated from blood cultures. A favorable outcome was obtained after a 6-week course of antibiotic therapy (ampicillin and gentamicin, relayed with ceftriaxone). Porras et al. described an 83-year-old woman with cellulitis of the hand and arm (13). Predisposing factors were absent, apart from the age of the patient and pulmonary and systemic hypertension. N. meningitidis serogroup C was isolated from blood cultures. After 2 weeks of ceftriaxone therapy, the patient was asymptomatic. A cellulitis of the foot due to Neisseria mucosa involving a 33-year-old woman was also described (19). Predisposing factors were present (corticosteroid therapy and lupus erythematosus). Bacteremia was absent, but a skin lesion of the foot was noted. Healing was obtained with lincomycin. Six other case reports involved children presenting with periportal cellulitis (4, 5, 10, 11, 13, 20). Obvious predisposing factors were absent. Bacteremia was present in three cases, and conjunctivitis was present in three cases. Cellulitis was associated with pericarditis in one case and with meningitis in one other case (5, 20). All children were healed.

With only a few cases of meningococcal cellulitis reported in the literature, it is difficult to draw general conclusions. However, it seems that periorbital cellulitis is the usual presenting picture in children. In adults, host predisposing factors are usually present. Possible mechanisms of cellulitis development are hematogenous dissemination, direct inoculation of oral secretions into a preexisting skin lesion, and contiguous spread from another soft tissue infection site.

In summary, this first fatal acute cellulitis due to N. meningitidis underlines the great difficulty in distinguishing the clinical manifestations of meningococcal disease from those of more common but less-serious illnesses. In patients with cellulitis due to N. meningitidis recovery can be expected if the infection is properly diagnosed and treated (13).

REFERENCES