First Reported Case of *Neisseria meningitidis* Periorbital Cellulitis Associated With Meningitis

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**ABSTRACT.** Cellulitis is a rare manifestation of meningococcal disease. We describe the case of a previously healthy 4-month-old female infant who developed periorbital cellulitis associated with meningococcal meningitis. *PEDIATRICS* 2005;116:e874–e875. URL: www.pediatrics.org/cgi/doi/10.1542/peds.2005-0694; meningitis, meningococcal disease.

**ABBREVIATION.** CSF, cerebrospinal fluid.

Periorbital cellulitis is often caused by *Staphylococcus aureus* or *Streptococcus pyogenes* after local trauma. Before universal immunization with conjugate vaccine, *Haemophilus influenzae* type b was responsible for 80% of cases of bacteremic periorbital cellulitis. Here we describe a case of periorbital cellulitis associated with meningitis caused by *Neisseria meningitidis*.

**CASE REPORT**

A previously healthy 4-month-old Amish female was transferred to our hospital with 2 days of fever, fussiness, decreased oral intake, and decreased wet diapers. Her reported rectal temperature was 40.6°C. She had no cough, congestion, or rhinorrhea. On the day of admission, she vomited 3 times but had no change in stool pattern. She had no sick contacts. She had received her first hepatitis B, polio, *H influenzae* type b, and diphtheria and tetanus toxoids and acellular pertussis vaccines at 2 months of age. There was no family history of invasive bacterial infection.

At an outside hospital, the patient had a temperature of 38.4°C, pulse of 170 beats per minute, and a respiratory rate of 42 per minute. She was ill-appearing without localizing signs on examination. A serum basic chemistry was normal except for a glucose level of 155 mg/dL. The complete blood count showed a white blood cell count of 8400/mm³, of which 49% were neutrophils, 7% segmented neutrophils, 33% were lymphocytes, 9% were monocytes, and 2% were atypical lymphocytes; a hemoglobin level of 10.6 g/dL; and a platelet count of 224 000/mm³. Cerebrospinal fluid (CSF) analysis showed a protein concentration of 314 mg/dL; a glucose concentration below the assay range (<1 mg/dL); a red blood cell count of 650/mm³; and a white blood cell count of 6400/mm³, of which 88% were neutrophils, 4% were lymphocytes, and 8% were monocytes. A Gram-stain of the CSF specimen demonstrated many Gram-negative diplococci. Blood and urine cultures were negative. Total complement function (CH50) was normal. Vancomycin and meropenem were discontinued, and the patient was treated with ceftriaxone (100 mg/kg per day). The patient’s left eye and clinical status improved rapidly, but she remained febrile. A repeat MRI on hospital day 9 revealed a small amount of extra-axial fluid along the floor of the middle cranial fossa bilaterally, suggestive of small sterile effusions. Her clinical course was also complicated by bilateral hearing loss. Pediatric neurosurgery and otolaryngology consultations were obtained to assist in the management of these complications; no surgical intervention was deemed necessary. She subsequently became afebrile, and an MRI performed 1 week later revealed a smaller fluid collection in the left middle cranial fossa. She was discharged from the hospital after 17 days of intravenous antibacterial therapy. The family and close contacts received the appropriate prophylaxis.

**DISCUSSION**

Periorbital cellulitis associated with *N meningitidis* has rarely been reported. Typically, periorbital cellulitis is caused by skin flora, predominantly *S aureus* and *S pyogenes*, after local trauma. It can also result from a localized infection such as conjunctivitis. Less often, this disease can also be associated with bactemia, historically with *H influenzae* type b and more recently with *Streptococcus pneumoniae*.

Donahue and Schwartz have described 70 cases of periorbital cellulitis from 1986 to 1996 at their institution. There was no family history of invasive bacterial infection. The patient developed periorbital cellulitis from 1986 to 1996 at their institution. There was no family history of invasive bacterial infection. The patient received intravenous ceftriaxone (100 mg/kg per day) and vancomycin (40 mg/kg per day) and was transferred to our facility for additional management.

At our hospital she was started on meropenem (120 mg/kg per day), the broad-spectrum antibacterial agent used in our intensive care unit as part of a clinical study protocol, and continued on vancomycin. On arrival, the patient was noted to have developed left periorbital edema and erythema. A computed tomography scan of her head showed normal sinuses and left periorbital edema (Fig 1). An MRI demonstrated left periorbital edema, as well as leptomeningeal enhancement. An ophthalmology consultation was obtained, and the examination was also consistent with periorbital cellulitis. The CSF grew *N meningitidis* serogroup B. Blood and urine cultures remained negative. Total complement function (CH50) was normal. Vancomycin and meropenem were discontinued, and the patient was treated with ceftriaxone (100 mg/kg per day). The patient’s left eye and clinical status improved rapidly, but she remained febrile. A repeat MRI on hospital day 9 revealed a small amount of extra-axial fluid along the floor of the middle cranial fossa bilaterally, suggestive of small sterile effusions. Her clinical course was also complicated by bilateral hearing loss. Pediatric neurosurgery and otolaryngology consultations were obtained to assist in the management of these complications; no surgical intervention was deemed necessary. She subsequently became afebrile, and an MRI performed 1 week later revealed a smaller fluid collection in the left middle cranial fossa. She was discharged from the hospital after 17 days of intravenous antibacterial therapy. The family and close contacts received the appropriate prophylaxis.

**Fig 1.** A computed tomography scan of sinuses and orbits of the patient demonstrates normal sinuses and left periorbital edema.
were 6 positive blood cultures among the 59 cultures obtained. Five cultures grew *Streptococcus* species: 2 cases of *S. pneumoniae* and 3 cases of *S. pyogenes*. In contrast to earlier studies in which *H. influenzae* type B was responsible for 80% of the cases of bacteremic periorbital cellulitis, only 1 culture, obtained from a child in 1987 who was not immunized against it, grew this organism. In a similar fashion, with the introduction of the heptavalent pneumococcal conjugate vaccine, there will likely be a decrease in the incidence of periorbital cellulitis caused by *S. pneumoniae*.

There have been 11 reported cases of cellulitis associated with meningococcus.5–8 Of these 11 cases, 5 were children (age range: 9 months to 9 years). All 5 cases involving children presented as periorbital cellulitis, and the other 6 cases, involving adults, presented as cellulitis of the limbs, neck, face, and thorax. In contrast to our patient, none of these 11 patients had meningitis, although only 1 other child had a lumbar puncture. Meningococcus was isolated from blood, conjunctival exudates, or the area of cellulitis. However, similar to the other 5 children in the reported cases, our patient had no underlying medical conditions.

The prevalence of asymptomatic nasopharyngeal carriage of *N. meningitidis* has been found to be 1% to 2% in infants.9 Our patient did not have conjunctivitis or infection of any structures adjacent to the eye. In addition, her blood culture was negative. A possible explanation for her clinical presentation was that she was colonized with *N. meningitidis*, became transiently bacteremic, and subsequently developed meningitis and periorbital cellulitis. Perhaps meningococcus is a more common pathogen causing periorbital cellulitis than was thought previously. Rarely is a pathogen identified in cases of periorbital cellulitis, and meningococcus is sensitive to most of the antibacterial agents commonly used to treat this disease.

Another possibility is that the conjunctiva served as the portal of entry for our patient’s infection, as has been reported previously.10 Finally, it is possible, although unlikely, that the periorbital cellulitis was not caused by meningococcus but rather by another pathogen that was sensitive to the broad-spectrum antibacterial agents used to treat our patient. For this reason, intravenous antibiotics were continued for 17 days, well beyond the usual 5 to 10 days required to effectively treat meningococcal disease.

Because meningococcus is a rare cause of periorbital cellulitis and remains sensitive to the antibiotics commonly used to manage this disease, it does not seem to be cost-effective to routinely attempt to identify the responsible pathogen. Blood cultures should be obtained routinely; however, the age of the patient, the presence of comorbid conditions, and the clinical appearance of the patient should still dictate the extent of the laboratory evaluation, particularly the need for CSF examination, performed in cases of periorbital cellulitis.

**REFERENCES**


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