An Adolescent Found Unconscious

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Case Report

An 18-year-old male adolescent was found face down and unconscious in the back of his truck by his friends during a weekend party. Close contacts state that he had become increasingly lethargic and confused prior to their finding him unconscious. His parents were then called who brought him to the local emergency department where he was triaged and sent by ambulance to our hospital.

He had been complaining of frontal headaches and rhinorrhea for the last 3 to 4 months and was seen at a local emergency department 2 weeks prior to admission, where he was diagnosed with sinusitis and discharged with a prescription for oral amoxicillin/clavulanate, which was never taken. Then, 2 days prior to admission, he began to have left eyelid erythema and swelling that progressively worsened. The patient denied IV drug use, recent trauma, or memory loss. He had a history of chronic sinusitis dating back as far as 1 year. His braces were adjusted 4 days prior to admission.

On physical exam, he was lethargic, but oriented to time and place. His oral temperature was 101.1°F, heart rate 102 beats/min, blood pressure 124/73 mm Hg, respiratory rate 18 breaths/min, and oxygen saturation 98% on room air. He had moderate left preseptal erythema and edema and moderate pain with extraocular movement. He was also noted to have chemosis of the left eye. The right lower lip was swollen likely from trauma when he passed out, braces were in place, and moderate gingivitis was also noted. The patient's speech was somewhat slurred and he did seem confused, but there were no specific neurological deficits noted on exam. The remainder of the physical exam was unremarkable.

Initial laboratory studies yielded the following results: white blood cell count, 14 600/mm^3 (normal 4900/mm^3-10 800/mm^3); hemoglobin, 12.3 gm/dL (14.0-18.0); hematocrit, 36% (40.0%-54.0%); platelets, 123 000/mm^3 (150/mm^3-350/mm^3); sodium, 138 mmol/L (136-145 mmol/L); potassium, 3.6 mmol/L (3.5-5.1 mmol/L); chloride, 104 mmol/L (97-107 mmol/L); bicarbonate, 23 mmol/L (23-28 mmol/L); blood urea nitrogen, 0.7 mg/dL (6-20 mg/dL); creatinine, 0.6 mg/dL (0.5-1.4 mg/dL); glucose, 111 mg/dL (70-110 mg/dL); calcium, 8.8 mg/dL (8.7-10.5 mg/dL); and magnesium, 2.4 mg/dL (1.6-2.6 mg/dL); a urine toxicology screen was negative. ESR was 78 mm/h (0-15 mm/h), and CRP was 157.9 mg/L (0.1-8.2 mg/L).

Hospital Course

A cranial CT was obtained in our emergency department, which showed a left frontal epidural abscess, and the patient was transferred to the pediatric intensive care unit for management. On admission, he was started on metronidazole, vancomycin, and meropenem. He then had an MRI, which showed a 1.5 × 3 cm^2 left frontal epidural collection of fluid with corresponding mass effect on the adjacent frontal lobe and left frontal lobe edema (Figure 1). The patient was also found to have evidence of severe left frontal sinusitis. The MRI also revealed 2 small abscess collections in the superior lateral left orbit.

Two days after admission, blood cultures from the referring facility were reported to be positive for Gram-negative rods. The organism was not identified until day 7 (1 day after discharge) because of its slow growth in the anaerobic medium, but at this time, the lab confirmed that it was *Fusobacterium necrophorum*.

Subsequent blood cultures during the 6-day hospitalization were negative. The decision was made to manage the patient nonsurgically, and he rapidly improved clinically on the empirical antibiotic regimen. A peripherally inserted central catheter line was placed so that he could be continued on IV metronidazole and meropenem at home with a 6-week course of antibiotics planned. A repeat MRI at 6 weeks showed almost complete resolution of the abscess, and antibiotics were discontinued.

Diagnosis

Epidural abscess and orbital cellulitis secondary to chronic sinusitis caused by *F necrophorum*.

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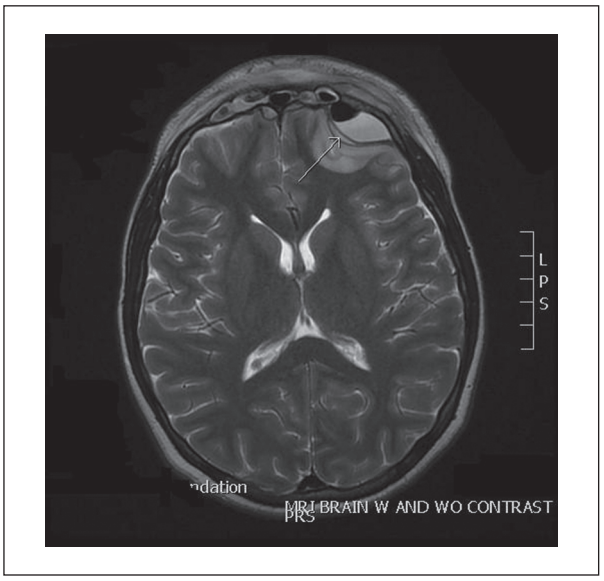

Figure 1. MRI showing a 1.5 x 3 cm left frontal epidural collection of fluid with corresponding mass effect on adjacent frontal lobe and left frontal lobe edema.

Discussion

F necrophorum is a filamentous, non–spore-forming, Gram-negative, anaerobic bacillus that is best known for its association with Lemierre's syndrome, first described in 1936 by Andre' Lemierre. Classically, this syndrome occurs following an oropharyngeal infection, progressing to internal or external jugular venous thrombophlebitis with septic emboli to the lungs and joints.[1] *F necrophorum* is part of the normal human flora of the oropharynx, gut, and female genital tract. Severe infection became rare after the development and widespread use of antibiotics for nasopharyngeal infection. It has been described as a probable cause of persistent sore throat syndrome and also thought to have a close association with infectious mononucleosis.[2] *F necrophorum* has been associated with a variety of infections of the head and neck, including: tonsillitis, peritonsillar abscess, postanginal cervical lymphadenitis, otitis media, and sinusitis.[3]

This case highlights the role of anaerobic bacteria in brain abscesses and emphasizes the serious complications that can result from sinusitis. *F necrophorum* is a relatively frequent cause of brain abscesses, especially in patients with chronic sinusitis, emphasizing the importance of obtaining anaerobic cultures from blood and the anaerobic collection and processing of material from the brain abscess itself. In addition, our patient had stage 2 orbital cellulitis, which has been seen in previous infections with this bacterium.[4]

F necrophorum is a frequent colonizing organism and an important potential pathogen in the head and neck and should be included in the differential bacterial etiology for patients with chronic sinusitis.[5-7] In a case series review, 8 of 251 cases of *F necrophorum* were associated with acute or chronic sinusitis; 3 of these were associated with local spread to the orbit causing orbital abscess, and 4 of the cases resulted in intracranial complications, including meningitis, cavernous sinus thrombosis, and cerebral infarction. In addition, 9 of 251 patients were identified as having cerebral abscesses (patients' ages ranged from 5 to 45 years, with a median age of 17 years). Among these cases 2 were otogenic, and 4 followed throat infections.[8] There has been a recent documented rise in the incidence of *F necrophorum* that has been hypothesized to be a result of the restricted use of antibiotics for sore throat.[8]

Anaerobes and Chronic Sinusitis

Bacterial sinusitis often follows a viral upper-respiratory infection. Chronic sinusitis is defined as episodes of inflammation (cough, rhinorrhea, or nasal obstruction) of the paranasal sinuses lasting more than 90 days. This should not be confused with recurrent acute bacterial sinusitis, which is episodic bacterial infection of the paranasal sinuses lasting less than 30 days separated by at least 10-day asymptomatic intervals.[9] In general, acute bacterial sinusitis is more associated with aerobic bacteria (*Streptococcus pneumoniae, Haemophilus influenzae*, and *Moraxella catarrhalis*). However, anaerobic (especially *Prevotella, Fusobacterium* and *Peptostreptococcus* spp) or resistant aerobic bacteria are more often characteristic of chronic sinusitis.[5] Anaerobic bacteria are an important cause of chronic sinusitis, comprising up to 13% of identified pathogens.[9] Anaerobic bacteria seem to particularly thrive in the frontal sinus during periods of increased inflammation and nasal pressure.[5] Children and adolescents may be especially susceptible to anaerobic colonization because of their immature anatomy. This is important because clinicians need to select antibiotics effective against both aerobic and anaerobic bacteria.

Complications of Chronic Sinusitis

Epidural and brain abscesses are known complications of chronic sinusitis. This case highlights the clinical ambiguity of initial presentation and importance of early diagnosis and treatment. There are several complications of chronic sinusitis in children, including orbital cellulitis, subperiosteal abscess, orbital abscess, brain abscess, subdural empyema, meningitis, facial osteomyelitis, and thrombosis of the cavernous sinus and cortical vein in up to 5% of patients hospitalized for sinusitis.[6,7] Our patient presented with orbital cellulitis, a clinical clue that more severe disease was involved.

In the United States, brain abscesses in children most commonly follow infection in the paranasal sinuses, the

Table 1. Leading Pathogens in Epidural Abscesses[8-10]

Organism	Percentage of Brain Abscess Infection
Staphylococcus aureus	63%
Gram-negative bacilli	16%
Streptococci (especially *S milleri*)	9%
Coagulase-negative *Staphylococcus*	3%
Anaerobes	2%

Table 2. Likely Organisms by Source of Infection in Brain Abscesses[13,14]

Source of Infection	Organisms
Paranasal sinuses	*Streptococci* spp (especially *S milleri*), *Haemophilus* spp, *Bacteroides* spp, *Fusobacterium* spp
Odontogenic	*Streptococcus* spp, *Bacteroides* spp, *Prevotella* spp, *Fusobacterium* spp, *Haemophilus* spp
Otogenic	*Enterobacteriaceae, Streptococcus* spp, *Psuedomonas aeruginosa, Bacteroides spp*
Lungs	*Streptococcus* spp, *Fusobacterium* spp, *Actinomyces* spp
Urinary tract	*Pseudomonas aeruginosa, Enterobacter* spp
Penetrating head trauma	*Staphylococcus aureus, Enterobacter* spp, *Clostridium* spp
Neurosurgical procedures	*Staphylococcus* spp, *Streptococcus* spp, *Pseudomonas aeruginosa, Enterobacter* spp
Endocarditis	*Viridans streptococci, Staphylococcus aureus*

middle ear, and dental abscesses, whereas in developing countries, meningitis and congenital heart disease are more common antecedent events.[10] Abscesses caused by chronic sinusitis are most frequently found in the frontal lobe resulting via transport of organisms through valveless emissary veins. In recent years, anaerobic bacteria have become increasingly more common, whereas *Staphylococcus aureus* has become less prevalent.[10] Although there are a diverse number of organisms associated with epidural abscesses in the immunocompetent host, there are several aerobic and anaerobic bacteria that are the more frequent pathogens (Table 1).[11-13] Anaerobic bacteria from brain abscesses include *Fusobacterium, Bacteroides, Peptostreptococcus, Veilonella, Propiono bacterium, Prevotella*, and *Actinomyces*.

Early treatment of epidural and brain abscesses is critical to prevent disease progression and sequelae. Surgical excision is generally considered the definitive treatment, and patients should immediately be evaluated by a neurosurgeon. However, many patients, particularly those with small abscesses, are candidates for medical therapy alone as seen with our patient. There are current antibiotic recommendations for brain abscesses according to the primary source of infection. Specific organisms are associated with different sources of infection (see Table 2), and antibiotic regimens should be tailored accordingly. The recommended initial empirical antimicrobial therapy for brain abscess associated with sinusitis is vancomycin, metronidazole, and a third-generation cephalosporin or carbapenem. Metronidazole is particularly recommended when the source of infection is a VP shunt, otitis, mastoiditis, or unknown infection.[10] The case presented above highlights the importance of using metronidazole for anaerobic coverage in patients with brain abscess caused by sinusitis.

Conclusion

Untreated chronic sinusitis in children and adolescents can lead to serious complications, including brain and epidural abscesses. *F necrophorum* and other anaerobic bacteria are important pathogens in these serious infections. Clinicians need to have a high suspicion for brain or epidural infection in a patient with chronic sinusitis and acute mental status changes and alter culturing techniques and antibiotic coverage accordingly.

Declaration of Conflicting Interests

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