Acute Epiglottitis due to Pasteurella multocida in an Adult without Animal Exposure

To the Editor: Pasteurella multocida infection in humans usually involves animal contact, most commonly with a domestic dog or cat (1). Epiglottitis due to human P. multocida infection associated with animal contact is very rare (2-4). We report a case of epiglottitis due to P. multocida not associated with animal contact.

A 44-year-old patient was admitted to the hospital with fever, throat fullness, and drooling. He had been healthy until 12 hours before admission when he noticed difficulty in swallowing liquids; anterior neck discomfort and fever followed, and soon he could not swallow his saliva. When he arrived at the Emergency Department of Montefiore Medical Center on September 23, 1996, the patient was mildly toxic and had an oral temperature of 103.2°F. Pulse was 110 and blood pressure 110/70. He was drooling. He had mild anterior neck tenderness, no cervical adenopathy, no pharyngitis on inspection of the oropharynx, and no palate deviation. The heart, lungs, abdomen, and skin showed no abnormalities. A lateral neck radiograph showed an enlarged epiglottis ("thumb sign"). Indirect laryngoscopy confirmed inflamed and edematous epiglottis and supraglottic structures. A culture of the epiglottis was not performed.

On admission, the patient had a hemoglobin of 1.9 g/dL; hematocrit was 48%; white blood cell count was 14,100/mm³; and platelet count was 170,000/mm³. A machine differential count showed 86% granulocytes, 9% lymphocytes, and 5% monocytes.

The patient was treated with dexamethasone and ceftriaxone. The fever abated rapidly, and all symptoms resolved. Repeat laryngoscopy on day 3 confirmed resolving epiglottitis. Blood cultures taken on admission grew gram-negative, oxidase-positive bacilli that did not grow on MacConkey agar (BBL, Cockeysville, MD) in two sets, both aerobically and anaerobically. The isolate was identified as P. multocida by the Vitek GNI card (BioMérieux-Vitek, Inc., Hazelwood, MO). Kirby-Bauer susceptibility testing demonstrated susceptibility to penicillin. Because of the patient's marked improvement after treatment with...
In the past 15 years, knowledge about the role of Shiga toxin-producing Escherichia coli (STEC) in human disease has expanded rapidly. The most distinctive complication of STEC infection is diarrhea-associated hemolytic uremic syndrome (HUS), a major cause of acute renal failure in U.S. children. Other manifestations of STEC infection can range from mild diarrhea to severe hemorrhagic colitis, thrombotic thrombocytopenic purpura, and death (1). In the United States, O157 is the most common STEC and causes an estimated 20,000 infections and 250 deaths annually. E. coli O157 outbreaks associated with beef have caused concern among public health workers, clinicians, and the public, prompting major changes in clinical and laboratory practice, meat production, and food preparation. However, critical questions remain unanswered. Have prevention measures decreased risk? Are new sources of STEC infections emerging? Is the incidence of O157 infection changing? How much illness is due to STEC of serotypes other than O157?

Diarrhea-associated HUS is associated with Shiga toxin, which is produced in quantity only by STEC and by Shigella dysenteriae type 1; approximately 90% of HUS cases are diarrhea-associated (2,3). In the United States, where S. dysenteriae type 1 infections are very rare, STEC infections are the cause of virtually all diarrhea-associated HUS. The incidence of HUS in North America is about three cases per 100,000 children under 5 years of age per year; the rate among older children is somewhat lower, and the rate among adults is not known (2-6). HUS complicates approximately 5% to 10% of O157 infections and an unknown percentage of non-O157 STEC infections (1). Except for supportive care and hemodialysis, no treatment has been shown to decrease the severity of illness or to prevent complications. The sequelae of HUS—death in 3% to 5% of cases (2,3,5) and long-term renal dysfunction in 10% to 30% of survivors (6)—and the lack of specific therapy make prevention critical.