measurable serum itraconazole levels, a topical effect of itracona-
zole was more likely responsible for therapeutic benefit in patients without detectable serum levels of itraconazole.

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References

Group B Streptococcal Meningoencephalitis After Conization in a Nonpregnant Woman

Group B streptococci (GBS), Streptococcus agalactiae, are a major cause of meningitis and septicemia in neonates and pregnant women [1]. GBS can be isolated from genital and/or lower gastrointestinal-tract culture specimens from women (15%–40%) [2]. In nonpregnant adult patients, the most common clinical diagnoses associated with GBS are skin, soft-tissue, or bone infections [3]. To our knowledge, we describe the first case of meningocerehalitides due to GBS as a complication of conization in a nonpregnant woman.

Five days after undergoing conization, a 43-year-old nonpregnant and nonpostpartum woman developed a severe frontal headache, rapidly increasing in intensity, and fever (temperature, to 41°C), followed by nuchal rigidity and pain. On admission to the hospital, the patient became increasingly stuporous. A CT scan of the head was obtained and showed no cerebral lesions. A lumbar puncture was performed in the emergency department, yielded pale-yellow, turbid CSF that contained 9,600 WBCs/mm³. The levels of glucose and protein in the CSF were 71 mg/dL and 103 mg/dL, respectively.

Because meningococcal meningitis was suspected, empirical treatment with ampicillin, 4 g q.i.d., and dexamethasone, 4 mg b.i.d., was initiated. Gram staining of CSF obtained during a second lumbar puncture revealed gram-positive cocci that were identified as GBS by standard laboratory methods. Therapy with ampicillin was continued and resulted in a complete neurological recovery without complication.

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Apparent GBS infections are an increasing cause of invasive disease in nonpregnant women, with an annual incidence among adults of 4.4 per 100,000 [4]; pneumonia, urinary tract infection, and endocarditis are noted as the most common focal sites. GBS meningitis, however, remains rare in adult patients. The characteristic clinical manifestations of GBS meningitis are similar to those of other types of bacterial meningitis, with acute onset of fever, headache, and nuchal rigidity. The majority of patients experience alteration in mental status comparable in severity to that in cases of meningitis due to Haemophilus influenzae B (HIB) or Neisseria meningitidis. The mortality of 18% associated with GBS meningitis is higher than that for meningitis due to HIB or N. meningitidis but similar to the mortality among cases of Streptococcus pneumoniae meningitis [5]. The prognosis for GBS meningitis is similar to that for major types of bacterial meningitis with a high cure rate, but neurological deficits or hearing impairment may occur in a few patients.

As reported in a recent review about the use of corticosteroids in cases of meningitis, clinical trials suggest that treatment with dexamethasone reduces the incidence of long-term neurological sequelae in children and animal models; the same should hold true for adults [6, 7]. Therefore, in the present case, we administered dexamethasone, 4 mg b.i.d., together with ampicillin. The patient had become increasingly stuporous on admission to the hospital, but she had a complete neurological recovery. However, currently there are insufficient data available concerning the use of corticosteroids in adults with bacterial meningitis to allow a reasonable analysis of the efficacy of such therapy.

There are reports of GBS infections occurring after diagnostic procedures [8, 9]. Pregnant women who are known to carry the organism in their vaginal tracts should receive intrapartum antibiotics to prevent GBS disease [10]. The case we describe might lead to the conclusion that to prevent GBS infection in women undergoing conization, use of a vaginal swab for screening for GBS prior to the procedure must be considered. However, because this is the first reported case of GBS meningoencephalitis after conization in a nonpregnant woman and because GBS infections...
are rare in adults, routine screening for GBS, as done before delivery to prevent GBS infection in neonates, is not economically feasible.

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References

Boerhaave’s Syndrome Due to Herpes Simplex Virus Type 1 Esophagitis in a Patient with AIDS

Herpes simplex esophagitis (HSE), a well-recognized cause of esophageal ulceration in patients with HIV infection [1–4], has not been associated previously with esophageal rupture. We describe the first case of Boerhaave’s syndrome, spontaneous esophageal rupture, resulting from herpes simplex virus type 1 (HSV-1) infection in a patient with AIDS.

A 31-year-old male with AIDS and a most recent CD4 cell count of 25/mm³ was referred to the University of Connecticut Health Center (Farmington, Connecticut) for management of dyspnea and fever. He had a longstanding history of odynophagia with esophageal ulcerations that had been documented by barium swallow. The ulcers were diagnosed presumptively as aphthous and resolved in response to prednisone therapy. Two months before presentation, he again reported the onset of odynophagia. Prednisone, 20 mg/qd, was administered without significant improvement. Thirteen days before admission, he noted increasing dyspnea and left-sided pleuritic chest discomfort. A mild cough with minimal sputum production was reported. He denied any history of vomiting. The day of admission, he became increasingly febrile and markedly dyspneic.

On admission, he was febrile (temperature, 107°F), his blood pressure was 98/50 mm Hg, and respiratory rate was 36. An oral examination demonstrated no lesions. A chest examination revealed tympany over the left upper chest, dullness to percussion over the left lower chest, and the absence of breath sounds over the left hemithorax. The remainder of the examination was unremarkable. A chest radiograph revealed a left tension hydropneumothorax. Laboratory studies revealed a WBC count of 11,700/mm³ (62% neutrophils, 28% band forms).

A chest tube was inserted, resulting in drainage of 2 L of foul-smelling purulent fluid. Gram staining of the fluid specimen revealed large numbers of mixed flora, and cultures of the specimen yielded mixed aerobes and anaerobes. Initially the patient was treated with cefazidime and clindamycin; however, upon return of culture results, therapy was changed to that with ampicillin/subactam. A meglumine diatrizoate swallow (figure 1) revealed extravasation of contrast material from the esophagus into the left pleural space.

The patient underwent exploratory thoracotomy, but the site of esophageal perforation could not be located. Eight days after thoracotomy, fiberoptic esophagoscopy revealed multiple ulcerations with a fistula tract 30 cm from the incisors. Histopathologic evaluation of esophageal biopsy specimens demonstrated multinucleated giant cells and inclusion bodies, consistent with HSV infection. Immunoenaprizedase staining was positive for HSV, and viral cultures yielded HSV-1. Studies for other pathogens, including cytomegalovirus (CMV) and fungi, were negative. Acyclovir therapy, 10 mg/kg q8h, was initiated. Repeated fiberoptic esophagoscopy after 1 month of acyclovir treatment showed resolution of the esophageal ulcerations. However, because of persistent fistula patency, a diversionary cervical esphagostomy was required.

The causes of esophageal symptoms in patients with HIV infection are varied and include acute retroviral syndrome [5], infection with CMV [1–3], candidiasis [1–3], HSV infection [1–4], idiopathic (aphthous) ulceration [1–3, 6], pill-induced ulcers, reflux disease [2, 3], and malignancies [1, 2, 7]. Systemic corticosteroid treatment may predispose to invasive HSV infection. In the largest reported series, 21% of HIV-infected patients with HSE had received recent antecedent treatment with corticosteroids [4].

Esophageal perforation is an unusual complication of AIDS. In patients with HIV infection, Mycobacterium tuberculosis may cause esophageal fistulas [8], HSE has been implicated as a cause of tracheoesophageal fistulas [9], and esophageal lymphomas may

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