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/subbacam. A megilumine diatroioate 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 esophagoscope 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. Immunoperoxidase 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 esophagoscope after 1 month of acyclovir treatment showed resolution of the esophageal ulcerations. However, because of persistent fistula patency, a diversionary cervical esophagostomy 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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We postulate that the use of empirical oral corticosteroids in this patient attenuated an already impaired host response to HSV-1 infection and allowed progression of ulceration through the esophageal wall. This case illustrates the need for aggressive evaluation of esophageal symptoms in HIV-infected patients, rather than empirical treatment for presumed aphthous ulceration. Appropriate therapy may then be directed toward the identified pathology.

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