Frequency of Infectious Disease Consultations in the Setting of a Community Group Practice

SIR—I read with interest the letter to the editor by Sexton [1] that described the frequencies of infectious disease consultations at a university hospital (2.2% of discharges), a Veterans Administration hospital (2.4%), as well as two private tertiary care hospitals (4.1%). Dr. Sexton stated that "further data from private practices that examine utilization rates and types of consultations are particularly needed."

In addition to being chief of infectious diseases at a community-based medical school, I am also the infectious diseases specialist for a large multispecialty group practice of 200 physicians, with its own 150-bed hospital [2]. For the sake of comparison, we tabulated all infectious disease hospital patients cared for by me at Straub Hospital (Honolulu) for the 1-year period from October 1990 through September 1991. During this period when Straub Hospital had 6,812 discharges, 848 patients (12.5%) were seen for infectious disease evaluation; approximately one-third were managed as primary (attending) patients.

The most common reasons for an infectious disease consultation were unexplained fever (204 patients), pneumonia (133 patients), complicated urinary tract infection (92 patients), bone and soft-tissue infection (62 patients), bacteremia (60 patients), and postoperative infection (49 patients).

In addition to patients at Straub Hospital, another 199 patients at other Honolulu hospitals were referred to me for consultation during the 1-year study period. During this time I also handled 5,000 clinic visits, including consultations with outpatients and 66 patients receiving intravenous antibiotics at home.

The reasons for such a high volume of infectious disease consultations are several: (1) the long duration of my practice (18 years), (2) the degree to which I make myself available for consultation, (3) the effectiveness of the infectious disease consultation in reducing costs of antibiotics, (4) the complications occurring in patients infected with human immunodeficiency virus, (5) the nature of a multispeciality group practice, and (6) the need for consultations for both medical and legal reasons in serious cases. It should be evident that the infectious disease private practice is not dying [3] but flourishing and that such practices should continue to grow in the foreseeable future.

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Disseminated Coinfection with *Mycobacterium avium* Complex and *Mycobacterium kansasii* in a Patient with AIDS and Liver Abscess

SIR—We present the case of a patient in whom generalized infection with *Mycobacterium avium* complex (MAC) and *Mycobacterium kansasii* was diagnosed simultaneously. To our knowledge, such coinfection has not been described previously in association with AIDS. The resolution of a concomitant mycobacterial liver abscess illustrates a measurable therapeutic response.

A 32-year-old homosexual man from Hawaii was found to be infected with the human immunodeficiency virus in July 1988. Despite treatment with zidovudine, pneumonia due to *Pneumocystis carinii* developed in January 1989. In April 1990 he presented with a fever (temperatures to 40°C) but had no further complaints. The results of physical examination were normal except for slight hepatomegaly. The $CD4^+$ lymphocyte count was 20/μL. Abdominal ultrasonography and computed tomography revealed a hypoechoic mass in the right lobe of the liver that was suggestive of an abscess. A liver biopsy specimen showed acid-fast bacilli on Ziehl-Neelsen staining and fluorescence on auramine-rhodamine staining. Histologic examination demonstrated clusters of neutrophils and eosinophils with areas of epitheloid cells but no granulomas.

MAC was cultured from the liver abscess specimen and blood, *M. kansasii* was cultured from blood and feces, and *Mycobacterium xenopi* was cultured from bronchoalveolar lavage fluid. MAC was multiresistant to isoniazid, rifampin, ethambutol, and amikacin, whereas the *M. kansasii* organisms were susceptible to isoniazid, rifampin, and ethambutol but resistant to amikacin. The antimycobacterial therapy consisted of isoniazid (300 mg/d), rifampin (450 mg/d), and ethambutol (800 mg/d), with the addition of amikacin (1,000 mg/d) to the regimen during the first 5 weeks. After receiving therapy for 3 weeks, the patient became afebrile and his general condition improved remarkably.

Results of abdominal ultrasonography after 4 weeks of therapy were completely normal. After 5 months, therapy with ethambutol was discontinued because a skin rash developed. While receiving isoniazid and rifampin, the patient remained well and afebrile. In March 1991 the patient again developed fever, although his general condition remained good and the results of abdominal ultrasonography remained normal. Blood cultures

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again revealed MAC. *M. kansasii* was not detected. Because of the ongoing fever with diarrhea, clofazimine and clarithromycin were added to the regimen. The patient's condition deteriorated rapidly and he died in June 1991.

Although disseminated infections with MAC or *M. kansasii* have been described in patients with AIDS, we are not aware of any previous reports of a concurrent disseminated infection with both mycobacteria. A mixed infection with MAC and *Mycobacterium simiae* in an African man with AIDS has been reported, although the clinical significance of both isolates could not be evaluated [1]. Of 47 blood cultures of patients with AIDS that yielded MAC, only one yielded both MAC and *M. kansasii* [2]. Among mycobacterial cultures, only 1.3% were mixed; MAC and *M. kansasii* accounted for 13% of all mixed cultures [3]. Most of the organisms were isolated from bronchial secretions, urine, and stool. Therefore, the occurrence of a mixed blood culture is rare, and its clinical significance has not been evaluated [3]. In the case of our patient, who presented with a prolonged febrile illness and a liver abscess, MAC was cultured from the liver abscess specimen and from blood; *M. kansasii* was cultured from blood and feces. Among all organisms that cause infections affecting the liver parenchyma in patients with AIDS, MAC has been shown to be the most frequent cause [4].

The role of MAC and *M. kansasii* as causative agents is difficult to separate. In view of the fact that MAC was repeatedly detected in the blood cultures and that the liver appeared normal on follow-up abdominal ultrasonograms, we regard MAC to be more likely responsible for the disease. However, the therapeutic response suggests an additional pathogenic role for *M. kansasii*, which was susceptible to the therapeutic regimen. As expected with a multiresistant disseminated infection due to MAC, the disease reappeared after a 10-month period of good clinical response [5, 6].

In conclusion, atypical and dual infections with nontuberculous mycobacteria can occur in patients with AIDS, and adequate culture techniques are required to identify possible coinfections with different mycobacteria. Microbiologists and clinicians must be aware of the rare possibility of concurrent mycobacterial infections in such patients.

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Myonecrosis Due to *Aeromonas hydrophila* Following Insertion of an Intravenous Cannula: Case Report and Review

SIR—We report a case of fatal, rapidly spreading, crepitant myonecrosis caused by *Aeromonas hydrophila* following insertion of an iv cannula (Nipro, Japan) into the lower limbs of a patient who had cirrhosis. To our knowledge, this is the first reported case of a gas gangrene–like presentation due to *A. hydrophila* following insertion of an iv cannula.

A 50-year-old man who had diabetes and cirrhosis was admitted to the emergency department following an episode of hematemesis and melena. Diagnosis of cirrhosis was first made in November 1990, when a biopsy of the liver revealed moderately active cirrhosis. Serologic tests for detection of antibodies to hepatitis B and C were negative. Since then, the patient had sclerotherapy on two occasions.

The patient was initially managed conservatively. After failing to gain iv access in one leg, we were able to successfully introduce the same iv cannula into the long saphenous vein in the other leg. The patient was resuscitated, and urgent upper gastrointestinal endoscopy revealed grade IV lower esophageal varices that were actively bleeding.

The varices were injected, and hemostasis was achieved. Within 24 hours the patient had another episode of massive bleeding and was taken to the operating room. The patient underwent splenectomy, esophagogastric transection, and pyloroplasty, following which he was electively ventilated in the intensive care unit. Despite the use of central and arterial catheters, the iv cannula in his leg was not removed because of the poor condition of his peripheral veins.

Within the next 24 hours, the patient's fever started spiking to a high-grade fever, and he developed multiple-organ failure and disseminated intravascular coagulation. He also developed bilateral edema below the knee as well as subcutaneous gas and bullae. Fluid was aspirated from both legs, and gram stains, cultures, and susceptibility tests were performed. Staining revealed moderate to large numbers of gram-negative rods whose size and shape were not compatible with *Clostridium perfringens*. The

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