sustained viral response (SVR). The SVR rate among patients who had HCV infections other than genotype 1 was 3 of 7 in the HIV-positive group and 2 of 6 in the HIV-negative group. Although HCV and HIV virus loads were reduced in all patients after induction therapy with high-dose IFN, it is difficult to eradicate HCV genotype 1 in patients also infected with HIV.

Neumann et al. [2] reported a strong link between the degree of virus load reduction during the first phase of treatment and the subsequent second-phase decline slope. There are many reports that SVR is dose-dependent on IFN, even in patients receiving IFN or pegylated (PEG)-IFN plus ribavirin therapy [3, 4]. Although some investigators report inefficacy of induction therapy, 3 times/week injections of IFN after induction therapy are inefficient and show a negative effect on the second phase [5, 6]. For patients infected with HCV genotypes 2 or 3, low-dose induction therapy might be effective, but high-dose induction therapy could make the period of treatment much shorter than 24 weeks. It is necessary to study how short the period of IFN therapy can be made by using induction therapy or high-dose IFN.

All patients could tolerate 2 weeks of induction therapy, although influenza-like symptoms were seen in only a few days. Adverse effects became severe with long term use of IFN therapy. The duration of IFN therapy should be made as short as possible to reduce adverse effects and associated costs.

The CD4 cell count of the HIV-positive patient with SVR of genotype 1 was 514 cells/mL and was the highest cell count in the group. In my small study [1], in which values are represented as medians in the table and as means in the text, the potential increase in CD4 cell counts required for SVR during IFN therapy in HIV-positive patients could not be evaluated. For now, a more conservative approach for starting HIV treatment is recommended. In Japan, hepatitis C has been the leading cause of death among patients with hemophilia and HIV infection, posing a serious problem. Despite failure of SVR, IFN therapy is useful for reducing inflammation progressive to liver cirrhosis or for prevention of hepatic cell carcinoma. In HIV-HCV–coinfected patients, histological response was observed in 25% of IFN nonresponders [7]. In the present study, a high rate of biochemical response was found in the HIV-positive group.

It is necessary to evaluate the dose, frequency, and duration of IFN or PEG–IFN therapy individually, giving consideration to the genotype of the virus, the efficacy of the drug, and the adverse effects of therapy. Some method to detect the eradication of HCV and a combination therapy to strengthen HCV-specific CTL activity are necessary. Whether coinfected patients with minimal-to-mild hepatitis should be treated with current IFN therapy or should wait until new therapy is available is a problem that is not yet settled. IFN therapy should be considered in patients with HCV infection who have not received antiretroviral therapy before CD4 counts decrease and HCV infection progresses. I think the “hit early and hard” approach to therapy is adequate for all HIV-positive patients with hemophilia and active HCV infection at risk of progression.

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References

Antimicrobial Resistance of Salmonella enterica Serotype Typhi in Dakar, Senegal

Sr—Typhoid fever, a systemic infection due to Salmonella enterica serotype Typhi, remains an important public health problem in many countries in the world [1]. In developed countries, sporadic typhoid fever is generally reported in travelers returning from an area of endemcity, whereas, in developing countries, particularly in Africa and Southeast Asia, this disease may be associated with high incidences of morbidity and mortality [1, 2]. In developing countries, poor sanitary conditions appear to be the main risk factor. However, with the emergence of antimicrobial resistance, developing countries are facing alarming problems with treating this condition efficiently [1]. Antimicrobial resistance affects mainly Asia, where nalidixic acid–resistant strains have already been reported in many countries.
but resistance has also spread progressively across Africa [1].

To assess trends in antibiotic susceptibility in S. Typhi, a retrospective study of isolates recovered from patients with enteric fever during 1987–1990 and 1997–2002 was performed at the National Senegalese Enterobacteriaceae Center (CNSE; Dakar, Senegal). Strains that had been conserved in stock culture in glycerol at −80°C were systematically identified (with biochemical testing and serotyping by slide agglutination, in accordance with standard bacteriological techniques) and tested for antimicrobial susceptibility (by the disk diffusion method, in accordance with the guidelines of the Antiibiogram Committee of the French Society for Microbiology [3]).

We isolated a total of 232 isolates of S. Typhi. Most of the isolates were recovered from hospitalized patients (80.6%), and 94% stemmed from patients living in or around Dakar. A total of 135 isolates were recovered during the first period, and 97 isolates were recovered during the second period. Our study showed that 99.6%, 99.6%, 99.1%, and 97.4% of isolates were susceptible to ampicillin, trimethoprim-sulfamethoxazole (TMP-SMZ), chloramphenicol, and tetracycline, respectively. No resistance to nalidixic acid, pefloxacin, and cefotaxime was reported. Only 1 isolate with multidrug resistance (to ampicillin, TMP-SMZ, and tetracycline) was reported. Among hospitalized patients, S. Typhi is the second most common Salmonella serotype isolated (30%), after Salmonella enterica serotype Enteriditis.

Although there is easy access to commonly used antimicrobials in Dakar through pharmacies (where no prescription is required) and street sellers [4], S. Typhi still appears to be very susceptible to antibiotics. These results are very encouraging, especially in comparison with those obtained from a 1997 study from a teaching hospital in Dakar, where, of 21 strains studied, 57%, 76%, and 57% were resistant to ampicillin, chloramphenicol, and TMP-SMZ, respectively [5].

Use of chloramphenicol, ampicillin, and TMP-SMZ, which are widely available and inexpensive drugs, still seems appropriate in Dakar for treatment of uncomplicated cases of typhoid fever, and use of these agents would allow prolongation of the life of powerful classes of drugs, such as fluoroquinolones and third-generation cephalosporins. At the same time, the need to reinforce the national antimicrobial surveillance network appears also obvious to detect the emergence of drug-resistant strains in Senegal.

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Community-Acquired Methicillin-Resistant Staphylococcus aureus

Sir—Salgado et al. [1] recently published a meta-analysis of studies reporting the prevalence of community-acquired methicillin-resistant Staphylococcus aureus (CA-MRSA). The authors note that the greatest risk factor for contracting CA-MRSA is the association with prior visits to a health care facility. Indeed, if patients with health care contact were excluded, then the prevalence of MRSA was 0.2%. This is an important finding, as multiple articles and letters have been published implying that the number of cases of CA-MRSA is on the rise.

To determine the rate of MRSA bacteremia at our private community hospital in the Bronx, New York, we reviewed all cases of S. aureus bacteremia treated at our hospital from October 1999 to November 2000 and from 1 January 2002 to 17 December 2002. A total of 238 cases of S. aureus bacteremia were identified, of which 110 (46%) were due to MRSA. To identify patients who qualified as having CA-MRSA bacteremia, we eliminated patients with bacteremia acquired after 72 h of hospitalization, and we also excluded patients who were receiving hemodialysis or had been admitted from a nursing home. This left only 14 patients who accounted for 13% of all cases of MRSA bacteremia. Furthermore, of these 14 patients, 11 had documentation of being in a health care facility within the 6 month period prior to infection with MRSA. Our data support the low rate of CA-MRSA bacteremia among patients who have not been in contact with a health care facility in the 6 month period prior to developing MRSA.

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