able to induce a net proabsorptive effect at the intestinal level.

Interestingly, in an in vivo animal model of diarrhea, zinc was unable to interfere with oral rehydration solution (ORS) efficacy in reducing dehydration [10]. Finally, in a recent clinical study, it was reported that ORS supplemented with zinc was effective in reducing the severity of acute diarrhea without reducing ORS intake [11].

We believe that zinc should be considered as an addition to the composition of the new universal ORS [12]. This addition would not only enhance ORS efficacy but also increase its use, by introducing an active component capable of reducing water loss, rather than relying on components that only replace fluid loss, as with the standard ORS. This could be an even more important aspect of adding zinc to ORS than its direct ion proabsorptive/antisecretory effects, given that ORS is still now largely underused throughout the world.

References


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HIV and Leprosy in the Eastern United States

To the Editor—The management of patients with coexisting HIV and Mycobacterium tuberculosis infections is complicated by multiple issues, including the timing of the initiation of highly active antiretroviral therapy (HAART), potential complex drug interactions and additive adverse effects, and the possibility of developing an immune reconstitution inflammatory syndrome (IRIS) that may be severe. According to the current recommendations from the Department of Health and Human Services (DHHS), HAART (which is usually indicated for patients with a CD4+ cell count of <200 cells/mm³ or a history of an AIDS-defining illness) should be withheld from HIV-positive patients with concomitant tuberculosis—with the possible exception of patients with a CD4+ cell count of <50 cells/mm³—until 4–8 weeks after initiation of the multidrug regimen for tuberculosis [1]. The recent retrospective study by Dheda et al. has suggested that HAART reduces the risk of death and new AIDS-defining illnesses in HIV-positive patients requiring treatment for tuberculosis and that delaying HAART in patients with a CD4+ cell count of <100 cells/mm³ may increase the risk of death or developing an opportunistic infection [2].

Perhaps the DHHS recommendations for the initiation of HAART in patients with HIV and M. tuberculosis coinfections should also apply to patients with HIV and coinfection with another mycobacterium, such as M. leprae. In the past, HIV-1 infection was thought to put a patient at no additional risk for the development of leprosy, and coinfection has not been reported to alter the clinical manifestations or course of leprosy [3–5]. However, a number of recent reports from Brazil, Europe, and the Caribbean have identified leprosy as an acute presentation caused by the development of IRIS after the initiation of HAART [6–9]. We now report the first case, to our knowledge, of coinfection with HIV-1 and M. leprae in the eastern United States.

A previously healthy 22-year-old Sudanese refugee presented to the dermatologist at Wake Forest University Baptist Medical Center with saddle nose deformity, malar rash, and diffuse infiltration of his ears and cheeks. The patient also exhibited generalized xerosis and pruritis as well as multiple annular, poorly demarcated, infiltrated, hypopigmented plaques on his trunk and upper arms and several soft, dermal nodules on his arms. He had evidence of glove and stocking peripheral sensory neuropathy and no motor neuropathy. A positive Fite-stain biopsy of 1 skin lesion was diagnostic of lepromatous leprosy. Polymerase chain reaction of DNA extracted from the skin biopsy amplified M. leprae heat-shock protein 65, and the results of the subsequent restriction frag-
ment–length polymorphism analysis (figure 1) were consistent with those in a study published elsewhere [10]. Serological studies performed at the time of the diagnosis of leprosy also indicated infection with hepatitis B and HIV-1. The patient had significant immunosuppression, with a CD4+ cell count of 270 cells/mm³ in blood (with 26% helper lymphocytes) and a plasma viral load of 52,730 copies/mL (HIV-1 Ultraquant; Roche Diagnostics).

The patient was treated with rifampin, dapsone, and clofazimine for leprosy. Approximately 6 weeks later, the patient developed symptoms of erythema nodosum leprosum. He complained of significant swelling and stiffness of his hands and feet, generalized arthralgias, increased scaling of his extremities, fevers, chills, diffuse infiltration of his face and ears (leonine facies), and multiple nodules on his thighs. Rifampin, a potent cytotoxic P450 inducer, was discontinued, and the patient was initially treated with prednisone and later with thalidomide. HAART was started 1 month after the initiation of therapy for erythema nodosum leprosum. His current treatment includes thalidomide, dapsone, clofazimine, minocycline, tenofovir disoproxil fumarate, emtricitabine, and efavirenz.

Given the influx of emigrants from countries where leprosy is endemic and the high prevalence of HIV, it is important for physicians in the United States to recognize clinical manifestations of leprosy that may coexist with HIV infection or arise after the initiation of HAART. The issue of when to initiate HAART in patients coinfected with HIV and mycobacteria is a subject of controversy, and prospective studies are needed for further clarification. However, physicians should be aware that HIV and M. leprae infections can coexist; leprosy is emerging as a presentation of IRIS in HIV-positive patients, and some patients are also at risk for leprosy reactions. This diagnosis must be considered for HIV-positive patients from countries where leprosy is endemic who present with typical tuberculoid or lepromatous skin lesions. In patients who present with coexisting HIV and M. leprae infections, delaying HAART or the initiation of a modified regimen without rifampin should be considered to prevent reactions, complex drug interactions, and additive adverse effects.

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Reply to Lu et al.

To the Editor—We thank Lu et al. for their interesting report [1] and for highlighting the complex treatment–related issues for patients coinfected with HIV and Mycobacterium tuberculosis who commence highly active antiretroviral therapy (HAART). The challenge is to reduce treatment–related complications, including paradoxical reactions, while at the same time maximizing the benefit of HAART, to improve survival and prevent opportunistic infections [2]. However, as Lu et al. point out, prospective studies are needed to determine the optimum timing of the initiation of HAART in patients coinfected with HIV and mycobacteria. This is particularly important,