
Strongyloides stercoralis Infection as a Manifestation of Immune Restoration Syndrome?

Str—Kim and Lupatkin [1] describe a patient with fever, eosinophilia, hepatitis, and Strongyloides stercoralis larvae in stool, as revealed by microscopy. These clinical features developed after diagnosis of HIV-1 infection and commencement of HAART and are attributed by the authors to immune restoration. Empirical therapy for cerebral toxoplasmosis was also initiated with pyrimethamine and sulfadiazine, as was therapy with dexamethasone. The patient’s condition responded to standard therapy with ivermectin.

A more likely explanation for this case is that the patient experienced an exacerbation of subclinical S. stercoralis infection following the institution of high-dose corticosteroid therapy. Corticosteroid therapy has long been recognized as the major risk factor for development of severe disease and disseminated strongyloidiasis in people with asymptomatic carriage of S. stercoralis [2, 3]. Furthermore, it has been noted that it is rare to develop disseminated strongyloidiasis in the absence of corticosteroid therapy. Although it was initially hypothesized that the immunosuppression secondary to HIV infection would result in an increased incidence of disseminated strongyloidiasis, such a rise in incidence has not been observed. For example, a general lack of correlation between HIV infection and strongyloides hyperinfection has been observed in regions where both are endemic, such as sub-Saharan Africa and Brazil [4]. We, therefore, suggest that the case presented may merely reflect S. stercoralis carriage progressing to clinical disease following the use of dexamethasone.

Acknowledgments


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References


Tropical Pulmonary Eosinophilia

Str—In a recent article, Boggild et al. [1] tackled the problem of imported cases of tropical pulmonary eosinophilia (TPE). However, the diagnostic procedure that was used raised some concerns about the accuracy of the filarial etiology of the reported syndrome. TPE, as underlined by Boggild et al. [1], is characterized by pulmonary infiltrates and blood eosinophilia. This clinical picture can have various noninfectious or infectious etiologies; among the helminthiases, these include ancylostomiasis, strongyloidiasis, and visceral larva migrans (a major form of toxocarasis), which have been recognized as parasitic etiologies of pulmonary eosinophilia [2, 3]. Toxocarasis, a helminthozoonosis found worldwide, appears to be an especially common cause of pneumonitis with eosinophil infiltrates; 9 of 57 Argentine pediatric patients displayed this symptom [4].

How helminthiases other than bancroftian filariasis were ruled out was not reported by Boggild et al. [1]. Moreover, the diagnosis of filarial TPE was dependent on the results of an ELISA, the exact procedure of which was not described. ELISA that uses extracts of heterologous filaria worms is known to cross-react with serum samples from other roundworm diseases [5], but the use of recombinant antigens could resolve this problem [6]. Given these facts, we were surprised that Boggild et al. [1] did not test for circulating filarial antigens to ascertain the bancroftian origin of their TPE cases. Since its first use in the field by the middle of the 1990s [7], detection of the so-called Og4C3 antigen, by either immunochromatography (“card test”) or ELISA, has proven to be a specific and sensitive method for the immunodagnosis of Wuchereria bancrofti infections [8]. It is currently considered a major tool for the control of lymphatic filariasis [9]. We recognize that this test is unable to detect Brugia malayi infections, but none of the patients included in the study by Boggild

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