

CORRESPONDENCE

Eosinophilia in Patients Infected with Human Immunodeficiency Virus

To the Editor—Cohen and Steigbigel [1] observed a 62% higher absolute mean eosinophil count in human immunodeficiency virus (HIV)-infected patients with ≤ 200 CD4 lymphocytes/ μL than in patients with > 200 CD4 lymphocytes/ μL [1]. In most of their patients, the etiology of the eosinophilia was not found, and in none of them was it a parasitic infection. Of 24 patients with a total eosinophil count > 1000 cells/ μL , 22 (92%) reported a pruritic skin condition compared with 14 (70%) of 20 noneosinophilic patients with similarly low CD4 cell counts.

In a case-control study among HIV-infected persons seen at the Institute of Tropical Medicine (Antwerp, Belgium), similar results were found. Forty-one patients with > 500 eosinophils/ μL were compared with 41 controls with ≤ 500 eosinophils/ μL . Case-patients had significantly lower mean CD4 lymphocyte counts than controls (226 vs. 283 cells/ μL ; $P = .02$) and more often complained of pruritus (20 [49%] vs. 6 [15%]; odds ratio [OR] = 6, confidence interval [CI] = 1.79–19.81). More case-patients than controls (26 [63%] vs. 14 [34%]; OR = 4, CI = 1.35–11.33) had a history of being in the tropics for > 3 months. Parasitic stool examinations had been done for only 22 (54%) of the case-patients and 14 (34%) controls. In 6 of the patients, parasites known to cause eosinophilia were found: ascaris (2 patients), ankylostoma (1 patient), schistosoma (2 patients), and filaria (1 patient). No such parasites were found in any of the controls. A history of drug allergy was noted more frequently among case-patients than controls (10 [24%] vs. 3 [7%]; OR = 4.37, CI = 0.96–22.5). A papular pruritic eruption of unknown origin (prurigo) was slightly more frequent among cases than controls (5 [12%] vs. 1 [2%]), but this difference was not significant.

The study by Cohen and Steigbigel [1] confirms that eosinophilia in HIV-infected persons is more frequent among those in an advanced stage of infection, but it also shows that eosinophilia occurs more often in persons residing in or frequently traveling to highly parasite-endemic regions. Therefore, investigations for parasitic infections should be done in such persons.

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Reference

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Reply

To the Editor—My colleagues and I thank Dr. Colebunders [1] for his response to our study [2]. It is useful to see that his controlled study also found eosinophilia in people with advanced human immunodeficiency virus infection, perhaps again reflecting increased interleukin-5 production associated with a predominantly Th2 type of T cell response. We are in complete agreement that people residing or traveling in highly helminth-endemic regions should also be evaluated for such infections, which may be the cause of eosinophilia. In our patient population, such travel history was not present.

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References

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2. Cohen AJ, Steigbigel RT. Eosinophilia in Patients Infected with Human Immunodeficiency Virus. *J Infect Dis* 1996;174:615–8.

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Circadian Time Structure of Septic Shock: Timing Is Everything

To the Editor—In 1960, Halberg et al. [1] published the observation that the lethality of *Escherichia coli* endotoxin varied ~10-fold, depending upon when in the circadian (~24 h) cycle the endotoxin exposure occurred. Smolensky et al. [2] were among the first to document that deaths from septic shock were also circadian time dependent. In 1975, Carswell et al. [3] discovered that endotoxin administration induces a serum factor that causes the necrosis of tumors (tumor necrosis factor, TNF). In 1985, Beutler et al. [4] determined that passive immunization against cachectin (TNF- α) protects mice from the lethal effect of endotoxin. In 1994, we found that the lethal effects of TNF- α administration varied 9-fold, depending upon when in the circadian cycle this agent was administered [5]. Pollmächer et al. [6] have recently observed that significant diurnal variations occur in the human physiologic (hormonal and pyrogenic) response to endotoxin. Against the background of large circadian-dependent effects of endotoxin and TNF- α , it might be expected that the efficacy of molecular strategies designed to interrupt the potentially lethal cascade of events associated with septic shock might, in part, vary with the circadian time of their administration.