

# Hyper IgE and HIV-2 Infection

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## Abstract

A 36 year old African American female presented with hyper IgE, marked CD4 cell depletion, and extensive pruritic dermatitis. HIV-2 antibody was detected in the absence of HIV-1 RNA. With the administration of anti-retroviral therapy, complete resolution of the pruritic dermatitis and improved CD4 counts resulted. We believe that this association between hyper IgE and HIV-2 infection is analogous to the hyper IgE observed in some cases of HIV-1 infection.

## INTRODUCTION

Hyper IgE associated with chronic pruritic dermatitis has been described in HIV infection (<sup>1,2</sup>). These reports have originated in geographic areas where HIV-2 is uncommon (<sup>3</sup>). In Africa, however, HIV-2 is quite common. Laboratory studies are limited in African countries, and indeed, even a proposed case definition of AIDS (<sup>4</sup>) in this area relies on clinical not laboratory features. In this proposed case definition, the presence of a chronic dermatitis is one clinical feature. Pruritic dermatoses are common in African AIDS patients with HIV-2 infection (<sup>5</sup>). The relationship between these dermatoses and hyper IgE has not been defined. We have recently encountered an African American woman who presented with hyper IgE and chronic pruritic dermatitis, and who had HIV-2 infection.

## CASE REPORT

A 36-year-old African American female was first seen on 2/4/00 for a chronic pruritic rash of the extremities and torso for several months. She denied having ever traveled abroad and was born and raised in the New York City area. In December 1998, she had been hospitalized for phlebitis. Since that time, she noted skin changes of the legs. She had been seen by a dermatologist who had biopsied the rash and diagnosed nummular eczema. She had been treated with prednisone, cetirizine, and corticosteroid creams. As a child, she had allergy testing and was told she was not allergic. She denied a history of asthma, urticaria, eczema, or food/drug allergies.

On physical examination, her skin showed large areas of hyperpigmented lichenified skin on the arms, back, and legs.

Furthermore, the legs showed some superficial varicosities, moderate non-pitting edema, and stasis changes. The face showed allergic shiners and the nose had moderate edema and slight secretions in the inferior turbinates.

Laboratory testing a total leukocyte count of 3500/mm<sup>3</sup> with 18% eosinophils, and 29% lymphocytes. The erythrocyte sedimentation rate was 70. The IgE was 6009 IU/ml. RAST tests were positive to various aeroallergens, including cat, dust mite, mold, and tree pollens. The functional protein S was 40%(decreased).

The patient was again seen on 3/14/00, where T-cell subsets tests were ordered. The CD4 count was 42/mm<sup>3</sup> (4%), CD8 count was 479(46%), and the CD4 to CD8 ratio was 0.09. The patient was asked to have HIV testing through the New York City Department of Health. The results of the tests showed negative PCR to HIV-1 (<50 by branch DNA) and positive PCR and antibody to HIV-2. The patient subsequently developed profound anemia, wasting syndrome, dry cough, fevers and diarrhea. Because of the patient's clinical deterioration, anti-retroviral therapy was started on February 2, 2001 with zidovudine/lamivudine (Combivir), and lopinavir/ritonavir (Kaletra). Because of persistent headaches, the patient's regimen was switched to lopinavir/ritonavir (Kaletra) and amprenavir in March of 2001.

The patient had subsequent resolution and/or improvement of all symptoms. There was resolution of her dermatitis except in the lower extremity areas affected by post-phlebotic stasis dermatitis. There was also some post-inflammatory hyperpigmentation of some areas of her arms. Subsequent

laboratory values showed a CD4 count of 487/mm<sup>3</sup> (11%), a CD8 count of 1904/mm<sup>3</sup> (43%), 5.5% of 8500 leukocytes/mm<sup>3</sup> were peripheral blood eosinophils, and a total IgE of 6910.

### DISCUSSION

We believe that clinicians should be aware of this possible association between hyper IgE, chronic pruritic dermatosis, and HIV-2 infection. Furthermore, we suggest that similar to HIV-1, this syndrome may relate to a TH2 to TH1 imbalance in the setting of HIV associated immunodeficiency. Therapy for HIV-2 infection is not yet well established, and antiretroviral agents available for HIV-1 infection are not all effective against HIV-2<sup>(6,7,8,9)</sup>. Never-the-less, standard anti-retroviral therapy resulted in a marked improvement of both cellular immunity parameters as well as her eczematoid dermatitis. It appears that dual protease inhibitor therapy is effective for HIV-2 infection.

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