



Letter

Chronic lymphocytic lymphoma presenting with recurrent demodicidosis

Demodex folliculorum is a common obligatory human ectoparasite commonly seen in areas of high sebum production that does not commonly present with clinical symptoms in the immunocompetent patient. Demodicidosis presents clinically as a rosacea-like rash when a high density of *Demodex* mites are present in the pilosebaceous glands. This clinical manifestation of *Demodex folliculorum* has been associated with primary, acquired, and malignant immunocompromised states. Although most commonly reported in patients with AIDS and pediatric patients with leukemia, it is not often seen in adults with chronic lymphocytic leukemia as the presentation before the initiation of chemotherapy. We report a case of recurrent demodicidosis that led to a diagnosis of CD38⁺ chronic lymphocytic leukemia and small lymphocytic lymphoma.

A 69-year-old, white man initially presented with an ongoing, intensely pruritic rash. The patient noted a 6-month history of pustules and papules that involved the face, scalp, chest, and arms. He stated that he first noticed the rash shortly after arriving in Panama. Scrapings and biopsy of the lesions were initially unrevealing. The patient was treated with triamcinolone injections, which temporarily cleared the rash, but it returned within a month. Another skin biopsy revealed folliculitis with greater than 5 *Demodex* mites per square centimeter. The patient was given 5% permethrin cream, 2.5% selenium sulfide lotion, sulfur soap, and 2 doses of 0.2 mg/kg of oral ivermectin, which revealed impressive treatment efficacy.

After a recurrence of the rash 1 year later, the patient presented for an immunologic evaluation. Serum immunoglobulin tests revealed normal levels (IgG, 654 mg/dL; reference range, 620–1,400 mg/dL) with a deficiency in subclass 1 (342 mg/dL; reference range, 620–1400 mg/dL) and subclass 3 (34 mg/dL; reference range, 35–135 mg/dL). Lymphocyte evaluation and lymph node biopsy were performed given the patient's cervical and axillary lymphadenopathy noted on examination. The results revealed a small population (approximately 2% of all events analyzed) of κ clonal CD5⁺ CD23⁺ B-lymphocytes most consistent with chronic lymphocytic CD28⁺ leukemia and small lymphocytic lymphoma.

Demodex folliculorum is the most common ectoparasite of humans and is frequently found in facial sebaceous glands in patients aged 50 to 70 years.¹ The clinical manifestation of demodicidosis is rarely seen in children because of decreased sebum production; however, cases have been reported in human immunodeficiency virus (HIV)–positive children.^{2–5} There have also been several cases reported in the literature of HIV- and AIDS-positive adults who developed facial demodicidosis shortly after beginning antiretroviral therapy, likely because of an inflammatory

reaction with improved immune status.^{6–8} Immunodeficiencies in general will permit infectious agents to invade the host, depending on the immunologic limb involved. In a study comparing 29 individuals with demodicidosis with 13 age- and sex-matched healthy individuals, Akilov et al¹ found that the absolute CD3⁺, CD4⁺, CD8⁺, and CD16⁺ cells and the functional activity of leukocytes were significantly lower in individuals infested with *Demodex*. In addition, there is an increased readiness of leukocyte apoptosis that is parallel to the increasing density of *Demodex* mites. This is likely a result of the *Demodex* mites causing local immunosuppression, which thus enhances their survival in the human host.¹

In healthy or immunocompromised patients, 5% permethrin cream, 1% lindane cream, selenium sulfide, topical retinoids, metronidazole cream, erythromycin, and oral ivermectin have all been used to varying degrees of success in the treatment of demodicidosis.^{2,9,10} Invariably, successful eradication of the infectious agent will depend on a normal host response and appropriate antiorganism therapy. In a reported case of a 56-year-old HIV-seropositive man who developed facial demodicidosis 2 months after initiating highly active antiretroviral therapy, there was successful treatment with oral ivermectin and 5% permethrin cream.⁹ The absence of immunocompetence or treatment may result in a persistent or recurrent infection, which may lead to the clinical suspicion of an immunocompromised state. This case represents a unique presentation of recurrent demodicidosis in an adult that led to the diagnosis of chronic lymphocytic leukemia before the initiation of chemotherapy.

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