Correspondence

Severe dyshidrosis (pompholyx) in two patients with HIV infection shortly after starting highly active antiretroviral treatment

Dyshidrotic eczema is a common skin disease affecting the hands and sometimes the feet. The exact etiology is unknown (1). We report two cases of severe dyshidrosis shortly after introduction of antiretroviral therapy in two HIV seropositive patients.

Case 1
A 53-year-old Caucasian man, living in Nigeria, is diagnosed with HIV infection in November 2002. At presentation he complained of anorexia and weight loss (- 8 kg, <10%). His CD4+ lymphocyte count was 117/µl and his viral load 50.000 copies/ml. In January 2003 he was started on treatment with nevirapine, lamivudine, stavudine and cotrimoxazole. Seventeen days later he developed large pruritic blisters on the soles of his feet. Both feet became oedematous because of secondary inflammation. Walking was very painful. There were no other skin or mucosal lesions. He also developed fever (up to 39°C). No laboratory tests were performed but the fever rapidly disappeared after anti-malaria treatment (atovaquone-proguanil). His physician in Nigeria decided to stop the nevirapine and switched to indinavir. However, the foot lesions persisted. Four months after the start of the antiretroviral therapy in April 2003 he is seen at the outpatient clinic of the Institute of Tropical Medicine in Antwerp, Belgium. His CD4+ lymphocyte count is 341/µl and his viral load is undetectable. On physical examination large bullous and excoriated lesions are still present at both feet. Also small Kaposi’s sarcoma lesions are noted in his mouth and on his penis. He also presents facial seborrheic dermatitis, onychomycosis and a tinea cruris. Using a miconazole cream for the
tinea and a corticosteroid cream for the dyshidrosis finally the lesions on his feet disappear. The indinavir, lamivudine, stavudine and cotrimoxazole treatment is continued, and terbinafine is started for the onychomycosis with good result. The patient had no history of atopic dermatitis, dishydrotic eczema or nickel allergy in the past. A year before the diagnosis of HIV was made he had complained of intense pruritus on both feet but without any visible lesions.

Case 2
A 42-year-old Caucasian man, living in Belgium, is diagnosed with HIV infection in 1997. In May 1998 his CD4+ lymphocyte count is 300/µl and his viral load 145253 copies/ml. He is treated with stavudine, lamivudine and indinavir. Five days later he develops large pruritic blisters on both hands and smaller ones on both feet. Since the age of 26, he experienced similar but much smaller lesions, three to five times a year, but never in relation to the intake of drugs. In April 2002 his antiretrovirals are stopped because of unexplained fatigue. His CD4+ lymphocyte count is 619/µl. In May 2003 antiretroviral treatment (stavudine, lamivudine and nevirapine) is reintroduced because his CD4+ lymphocyte count drops to 272/µl. Three weeks later he develops again pronounced dyshidrosis lesions on the palms of his hands and feet, similar to those he developed in 1998 when he first started to take antiretrovirals.

Discussion
The large bullous lesions that appeared on the feet/hands of our 2 patients suggest a diagnosis of a severe form of acute dyshidrosis (pompholyx). The etiology of pompholyx remains obscure (1). Patients may present severe, sudden outbreaks often with long disease-free periods (2). Recurrences can occur during times of stress (3). The eruption is symmetrical and pruritic, with pruritus often preceding the eruption. The lesions resolve spontaneously over several weeks. Pompholyx
may occur in the presence of a dermatophytic infection or may be a manifestation of an allergic contact dermatitis (4). In none of our patients there was a personal or familial history of contact dermatitis but our first patient presented with a concomitant fungal skin infection.

So far no increased incidence of pompholyx in persons with HIV infection has been described. An association with the use of antiretrovirals has never been reported but pompholyx has been noted after intravenous immunoglobulin therapy and during mycophenolate mofetil treatment (5-6). The fact that in our two patients pompholyx-like lesions developed shortly after the introduction of highly active antiretroviral treatment could be simply a coincidence or could be caused by an immune reconstitution inflammatory syndrome.

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References


