Net Letter

Long-term management of HIV/hepatitis C virus associated psoriasis with etanercept

Sir,

Psoriasis is a chronic inflammatory skin disease that, due to its prevalence, can be observed in HIV patients. In such patients, the disease course can be critical and recalcitrant to therapy.[1] A concurrent chronic hepatitis C virus (HCV) infection is a further challenging clinical situation to manage.

A 51-year-old psoriatic erythroderma patient, co-infected with HIV and HCV came to us for consultation of his skin lesions. After failure of conventional treatments, continuous, uninterrupted etanercept therapy was followed by long-term clinical remission without significant infectious episodes.

The patient had a 30-year history of moderate to severe plaque psoriasis without arthritis. He smoked 20 cigarettes/day and his records noted moderate alcoholism and was positive for hepatitis C genotype 2a/2c. Some years before his psoriasis had been treated with intermittent courses of cyclosporine 3 mg/kg/day for 5 years. The patient was "lost to follow-up" for about 5 years. Then the patient was again admitted to hospital because of an erythrodermic psoriasis (PASI score 30) and intermittent fever with leukocytosis of 3 weeks' duration. Patient at presentation showed widespread erythema affecting nearly 100% of his body surface. The patient was treated with prednisolone 20 mg/day intramuscularly for a week. After tapering of steroids and administration of acitretin 0.3 mg/kg/day, the patient experienced a relapse of his psoriasis (PASI score 27.9). At this time HIV seropositivity was discovered, stage (Centers for Disease Control and Prevention) CDC B2 (CD4 cell count 200-499 cells/μL). Antiretroviral therapy was started with fosamprenavir, tenofovir disoproxil fumarate/emtricitabine, and ritonavir with clinical follow-up in the infectious diseases department. Narrow band ultraviolet B phototherapy (starting dose 1000 mJ/cm^2 with increment 10% pre treatment) plus acitretin at a dose of 0.3 mg/kg/day was administered. The latter had to be suspended due to elevated liver enzymes (AST 198 U/l, ALT 655 U/l) after 6 weeks. PASI score was 21. Cyclosporine 3 mg/kg/day provided effective control of psoriasis, but an increase in serum creatinine level (1.41 mg/dL, normal values 0.70-1.20 mg/dL) was noted after 8 weeks. Dose reduction was followed by rapid worsening of psoriasis. PASI score was 13.4 [Figure 1] with persistent elevated creatinine. In view of the lack of response to systemic treatments, a tumor necrosis factor-alfa blocker was considered. The investigations like chest radiograph was normal; the Mantoux test and the Quantiferon TB Gold were negative. The HIV-RNA load was 7,930 copies/mL. Etanercept was given at a dose of 50 mg twice weekly for the 1st 12 weeks, thereafter 50 mg/weekly. At 12 weeks, skin lesions showed marked improvement (PASI score 6.2). The patient continued to take this dose with good clinical remission of psoriasis [Figure 2]. As of his most recent follow-up visit, 132 weeks after the initiation of therapy, the patient remained partially free of psoriasis. Sporadic relapses were controlled increasing the frequency of topical treatment with betametasone/calcipotriol without modifying the schedule of etanercept injections. Lymphocyte count remained stable throughout treatments. The HIV-RNA gradually declined and was negative 18 months after the diagnosis of HIV infection. A decrease of HCV-RNA titres was also recorded. No significant infectious episodes occurred.

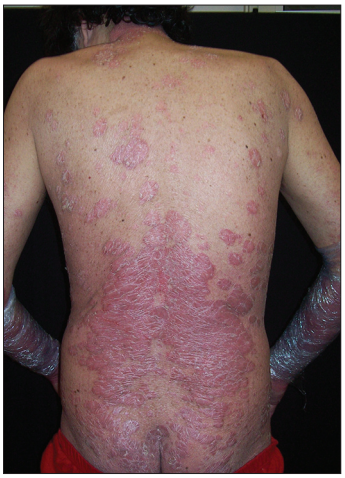

Figure 1: Plaque psoriasis involving extensively the dorsum

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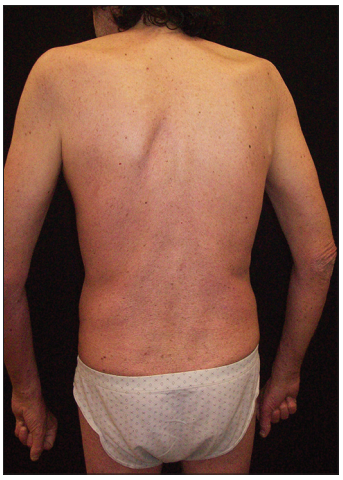

Figure 2: Significant clinical remission (120 week follow-up after treatment with etanercept)

HIV/AIDS is listed among the relative contraindication for anti-TNF-α treatment[2] and HIV infection is usually ruled out prior to initiation TNF-α therapy. However, high levels of TNF-α are associated with all stages of HIV infection.[1] TNF-α stimulates HIV transcription *in vitro* and is thought to be involved in the pathogenesis of fatigue, fever, and cachexia in HIV.[1] Hence, Ting and Koo suggested that etanercept could be used safely in the management of HIV-associated psoriasis.[3] Etanercept or other TNF-α inhibitors have been administered effectively in patients with plaque or pustular psoriasis or psoriatic arthritis refractory to conventional treatments in the setting of HIV infection.[4]

The long-term safety or efficacy of anti-TNF-α agents in patients with chronic HCV is not established at present, but the presence of HCV is not considered a contraindication to therapy with TNF-α inhibitors.[2] High levels of TNF-α are associated also with HCV infection, hence TNF-α inhibitors may be beneficial in moderate to severe psoriasis associated to HCV infection. In particular, etanercept and possibly, other TNF-α blockers have been suggested as second line drugs for patients with psoriasis and HCV infection.[5]

The safety of TNF-α antagonists in the context of HIV infection is unknown. TNF-α-mediated immune responses are of crucial importance in opportunistic infections. Toxoplasmosis is the most common opportunistic encephalitis in HIV-infected patients. A crucial role of TNF receptor type 1 (p55), but not of TNF receptor type 2 (p75), in murine toxoplasmosis has been shown.[6] Etanercept has no activity on p55 receptor, and therefore may have some theoretic advantage in minimizing the potential risk of toxoplasmosis. Since the conceivable risk of opportunistic infections, a firm alliance between dermatologists and infectious diseases specialists remains mandatory in the managing of psoriatic patients with HIV/HCV co-infection.

Vito Di Lernia, Giuliana Zoboli[1], Elena Ficarelli

Unit of Dermatology, and [1]Infectious Diseases, Department of Medicine and Medical Specialities, Arcispedale Santa Maria NuovaIRCCS, 42100 Reggio Emilia, Italy

Address for correspondence: Dr. Vito Di Lernia, Unit of Dermatology, Arcispedale Santa Maria Nuova-IRCCS, Viale Risorgimento 80, 42123 Reggio, Emilia, Italy.
E-mail: vito.dilernia@asmn.re.it

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