Clinical case: A severe attack of Psoriasis in an HIV infected patient

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ABSTRACT:

Psoriasis, which does not appear to have greater prevalence amongst HIV+ patients, does however present in its progress significant differences in these cases that are not only linked to immunodeficiency, but also to action of the virus itself on factors that aggravate psoriasis, such as the stimulation of keratinocytes, the favoring of skin infections, or the liberation of substance P, which also encourages growth in keratinocytes. A clinical case is presented along with explanatory images.

Key words: Prisons; Psoriasis; HIV Infections; Immunologic Deficiency Syndromes; Keratinocytes; Connective Tissue; Arthropathy; Neurogenic; Keratoderma; Palmoplantar; Spain.

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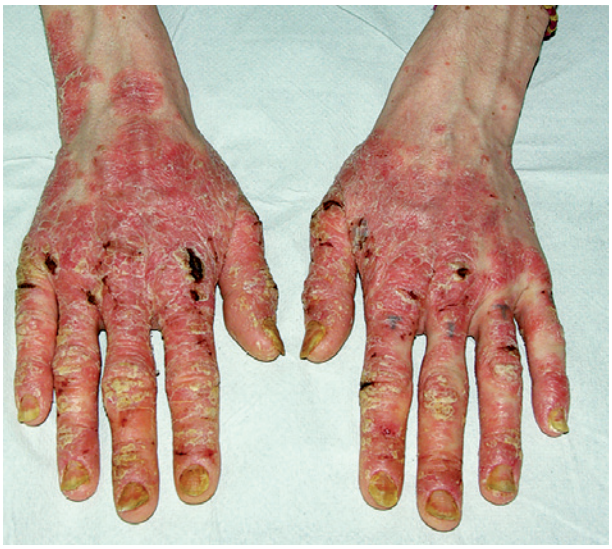
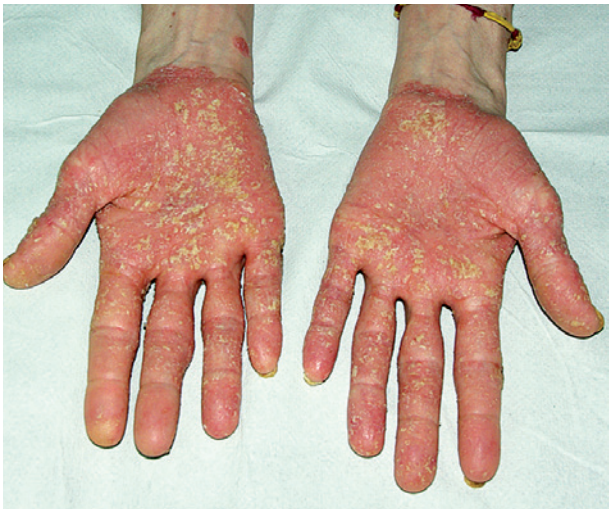
PSORIASIS INCIDENCE IN HIV/AIDS INFECTION

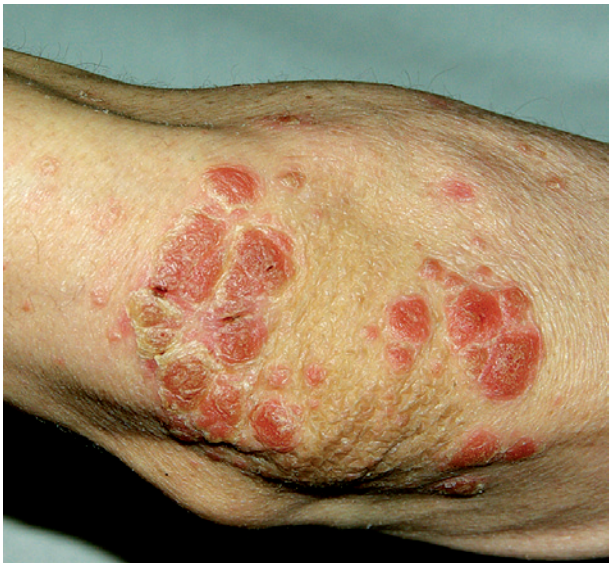
The prevalence of psoriasis among HIV infected patients does not seem higher that in the rest of the population, although differences are found regarding its evolution. Clinical manifestations are usually more aggressive and tend to present arthritis more frequently [1]. Throughout HIV evolution, psoriasis can appear at any point of the disease's history as a new pathology or as a reactivation or a previously existing disease, therefore entailing a higher presentation rate of severe arthropathy and a poorer response to therapy. We must also consider that the later entails that many potentially efficient therapies may involve immunosuppressive agents [2] and the patient's immunodeficiency can therefore worsen.

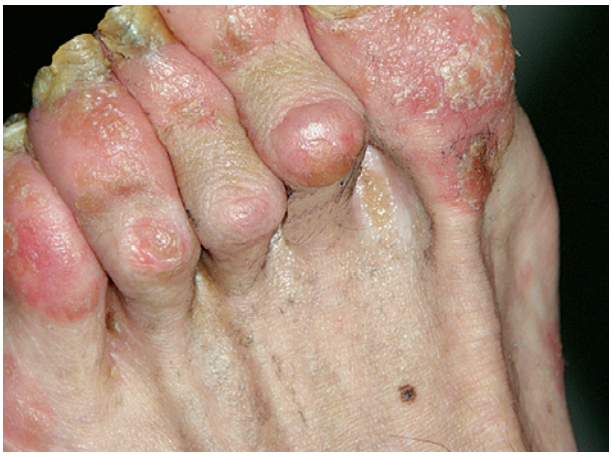
As far as the features of lesions, these can be: a) the most common manifestation involves reddish plaques with thick silvery scales on extensor surfaces; b) as a guttate psoriatic flare; or c) most frequently as widespread exfoliative erythodermic disease with palmoplantar keratoderma.

It is mainly an unaesthetic disease with a potential danger: bacterial infections secondary to scratching as pruritus is a very common manifestation. Infections are more common among people with poor hygiene habits, a frequent feature among prison inmates.









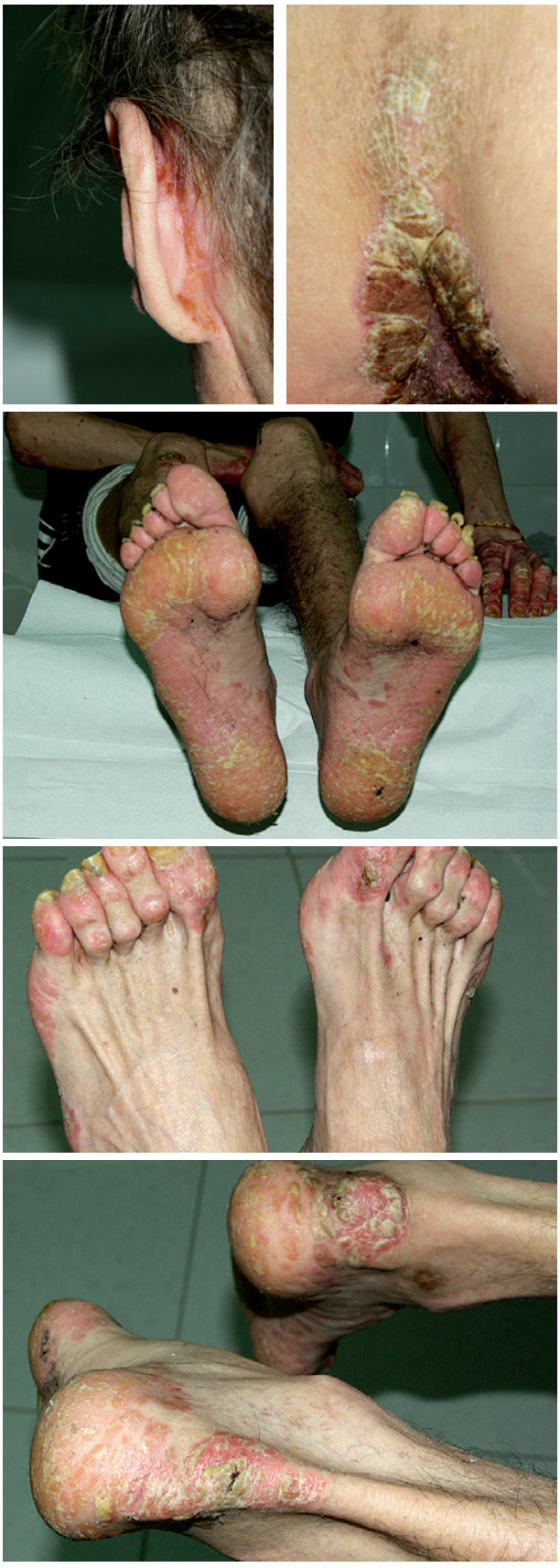
CLINICAL CASE

The case involves a 40 year old patient with a history of drug abuse from adolescence, IDU, who first entered in prison when he was 20 years old and later underwent reiterated imprisonment periods. In 1993 HIV and HCV coinfection was identified. His medical history refers to a first ART in 1998 denoted by constant therapy noncompliance, especially during freedom periods. Other relevant pathologies:

- pneumonia (*pneumocistis jirovecii*, before known as *carinii*) in 1998
- Extrahospitalary repeated pneumonia
- Repetitive oropharyngeal, and probably esophageal candidiasis
- Pulmonary TB and cerebral involvement due to toxoplasmosis and /or TB
- Almost constant seborrheic dermatitis

In August 2009, he presented scaling scablike lesions without pruritus which initially were found on the scalp, the auricula and the axilla. These lesions later spread towards the chest and limbs, including palmoplantar involvement. Similar lesions were also found on the intergluteal cleft and genitals.

The dermatologist diagnosed psoriasis and instituted therapy with Calcipotriol and Betamethasone, Salicylic Vaseline and Tracolimus 0.1%. This regimen was not initiated until re-imprisonment occurred at the beginning of 2010 (see Figures 1 to 9). Slight improvement has been observed throughout the last weeks. At the beginning the amount of CD4 cells was 64/ml with a HIV viral load of 1,090,000 copies/ml (6.04 log). Important cachexia was also present (BMI<16) and anemia (Hb 10g/dl; HCT 30%). The



patient has refused to undergo ART. Currently he is in hospital so that etiologic analysis of cerebral lesions can be performed.

DISCUSSION

Some studies have concluded a lower prevalence of psoriasis in the post HAART-3 era. It must be recalled though, that clinical manifestations are more aggressive in strongly immunodeficient patients, and certain correlation has been observed between CD4 cell count and the severity of psoriasis. The key – or at least one of them- to understanding this fact would be that memory T cells CD8+ and particularly the subtype CD45R0+, play an important role in the proliferation of keratinocytes in psoriatic patients.

It has equally been observed that in actively inflamed psoriatic lesions, keratinocytes are induced by IFN-γ to synthesize and express HLA-DR, an antigen especially present in psoriasis in immunodeficient patients [4].

Nevertheless, other factors have also been pointed out regarding potential exacerbation of psoriasis in immunodeficient patients [5]:

a) The presence of HIV in keratinocytes and Langerhans cells could stimulate the proliferation of the first ones.
b) Another hypothesis points out that psoriasis gets worse in AIDS due to an enhanced colonization of the skin by Gram-negative germs, due to impaired immunity and exacerbated infections. Moreover the liberation of bacterial lipopolysaccharides (LPS) acts as a proliferation factor for keratinocytes, which releases TNF-α (Tumor Necrosis Factor) giving rise to deteriorating immune deficiency, therefore completing the disastrous circle.
c) Finally HIV infected limphocytes, release Substance P (associated to connective inflammatory processes), leading to keratinocyte proliferation and therefore causing psoriatic plaques to appear.

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