

Chronic Cutaneous Leishmaniasis Mimicking Sebopsoriasis

Sir,

A 68-year-old man, on pharmacological substitution therapy for 10 years for the removal of a large pituitary adenoma with asymmetric expansion above the sella turcica, presented with chronic dermatitis of the scalp and face with onset referred 1 year earlier. Dermatologists had previously diagnosed it variously as psoriasis, seborrhoeic dermatitis and sebopsoriasis, but the patient claimed no improvement from the various topical and systemic therapies scrupulously followed.

During examination, the patient presented diffused scalp erythema and sparse whitish squamous crusts, especially on the sides. The right ear lobe appeared congested, desquamating and sore, with serous secretion from the retroauricular groove. The face was erythematous-edematous, and the skin of the nose was desquamating. A thin, irregular squamous crust adhering to the underlying tissue was observed on the left ala nasi (Fig. 1). Its removal uncovered a serous-producing surface, while thin cone-like extensions emerged from the inferior surface of the squamous crust. Tiny pieces of tissue were taken from the borders of the oozing lesion, and May-Grünwald coloured slides were prepared. At the same time, a 3 mm diameter tissue sample was punch-removed from the affected right mastoid area.

Microscopic examination of the slides revealed the presence of numerous leishmania inside and mostly outside the macrophages. For this reason, no culture testing was performed.



Fig. 1. Desquamating erythematous-edematous face especially on the nose.

Histological examination of the affected skin showed a granuloma consisting of lymphocytes, plasmacytes and, mostly, histiocytes. The routine blood work-up and the lymphocytic subpopulation assays resulted as normal.

The patient was in poor physical condition and was therefore treated intra-lesionally only, with a weekly administration of 1–1.5 ml meglumine antimoniate distributed in the areas clinically affected. After six sessions of treatment the patient was clinically healed.

After 2 years of total well-being, he returned to our observation, presenting a clinical symptomatology similar to the one already described, but less severe. Histological slides, prepared with the same technique as previously, revealed the presence of parasites outside the macrophages. A subsequent eight-session cycle of meglumine antimoniate infiltration therapy resulted in a complete clinical resolution of the lesions. In the last 6 months, the patient has not had any relapses, but has developed some typical nummular psoriatic lesions on the elbows and lower limbs presently treated with topical therapy and heliotherapy.

It is well known that focal cutaneous leishmaniasis may manifest itself with different clinical expressions from the classical "oriental sore" (1). Recently, some authors have emphasized the rise in sporotrichoid forms with hard, mobile subcutaneous nodules which appear about a month later than the primary lesion as an indication of parasitic diffusion along the lymphatics (2).

We believe that our patient can be defined as being affected by "non-healing" (chronic) cutaneous leishmaniasis mimicking sebopsoriasis, a term used to define the presence of well-demarcated scalp plaques with clinical and histopathological characteristics found across psoriasis and seborrhoeic dermatitis. We cannot exclude that the clinical aspect is influenced by an isomorphic type reaction in a patient predisposed to psoriasis. A case of visceral leishmaniasis presenting as a psoriasiform eruption in a young male patient affected by AIDS has been reported recently; however, neither the lymphocytic subpopulation assay nor HIV investigation was abnormal in our patient (3).

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Carmelo Schepis, MD¹, Maddalena Siragusa, MD¹, Antonino Alberti, MD² and Rosaria Palazzo, MD³

¹Unit of Dermatology and ²Department of Pediatrics, Oasi Institute (IRCCS), Via Conte Ruggero, 73, I-94018 Troina, and ³Unit of Dermatology, Piemonte Hospital, Messina, Italy.