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Clinical image

A strange infiltrative plaque on the face



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A 64-year old lady, known to have hypertension and urolithiasis, presented with a 4-month history of a slowly growing, erythematous-edematous and infiltrative plaque with superficial desquamation, involving all the nose skin surface – except for the root – and both zygomatic areas up to the naso-labial folds (Fig. 1).

The patient came from a remote rural area of the south-eastern Sicily, and was a farmer. Personal and familiar anamnesis was negative for autoimmune diseases or skin disorders.

Routine blood exams were within normal limits; serum autoantibodies (antinuclear, anti-ENA, anti-JO1 and anti-Scl70) were negative. Nailfold capillaroscopy and chest X-ray examination were normal.

Histopathologic examination of a cutaneous biopsy revealed a granulomatous infiltrate in the dermis, consisting of lymphocytes, histiocytes, and multinuclear giant

cells with hyperkeratotic overlying epidermis. A touch-imprint preparation of a skin specimen, showed *Leishmania* amastigotes, within the histiocytes as well as extracellularly.

Lupoid leishmaniasis (LL) is a rare form of cutaneous leishmaniasis (CL) showing a striking resemblance with some other granulomatous skin diseases of inflammatory or infectious origin.¹

In fact, LL is characterized by a typical spreading of the initial lesion leading to an infiltrated plaque with undefined borders, whereas some papules and nodules, often with scaling, may become apparent, presenting a lupoid aspect. The involvement of suggestive areas, as in our case, may further complicate the differential diagnosis.²

Histopathological features are that of epithelioid granulomas, and the detection of amastigotes is often hollow, both in microscopy and cultures.³

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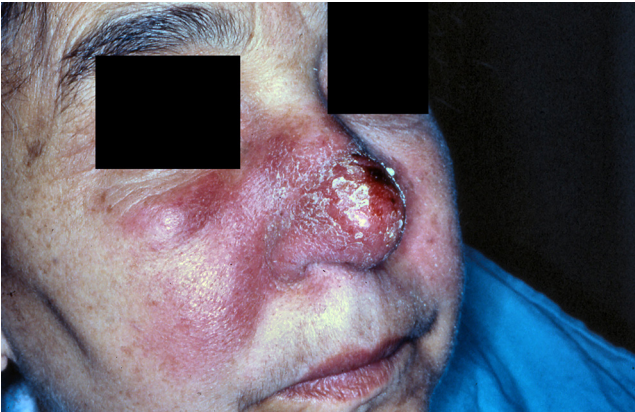


Fig. 1 – Erythematous-edematous-infiltrative plaque involving mainly the right side of the centro-facial region.

It seems that in LL certain strains replicate inside the macrophage, so assuming their ability to evade intracellular destruction or a concomitant defect in the T-cell activation process.^{2,3} *Leishmania infantum*, the most frequent causative agent of CL in our geographic area, have been rarely linked with LL.^{1,4}

The patient received N-methylglucamine-antimoniate, 1 mL twice-a-week intralesionally (total of 7 doses), with progressive improvement.

Conflicts of interest

The authors declare no conflicts of interest.

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