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Photo Vignette

Granuloma faciale treatment with tacrolimus

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Abstract

We present a 40-year-old woman with a one-year history of a solitary and asymptomatic facial lesion. On physical examination a slightly infiltrated, smooth red to brown nodule was seen at the left malar region. A biopsy established the diagnosis of granuloma faciale. After two-months therapy with topical tacrolimus 0,1%, nodule was resolved.

Case synopsis

A 40-year-old woman presented with a one-year history of a solitary and asymptomatic facial lesion. On physical examination a slightly infiltrated, red to brown nodule was seen at the left malar region (Figure 1). The borders of the nodule were well defined; it had a smooth surface with dilated follicular ostia. The patient's medical history was unremarkable.

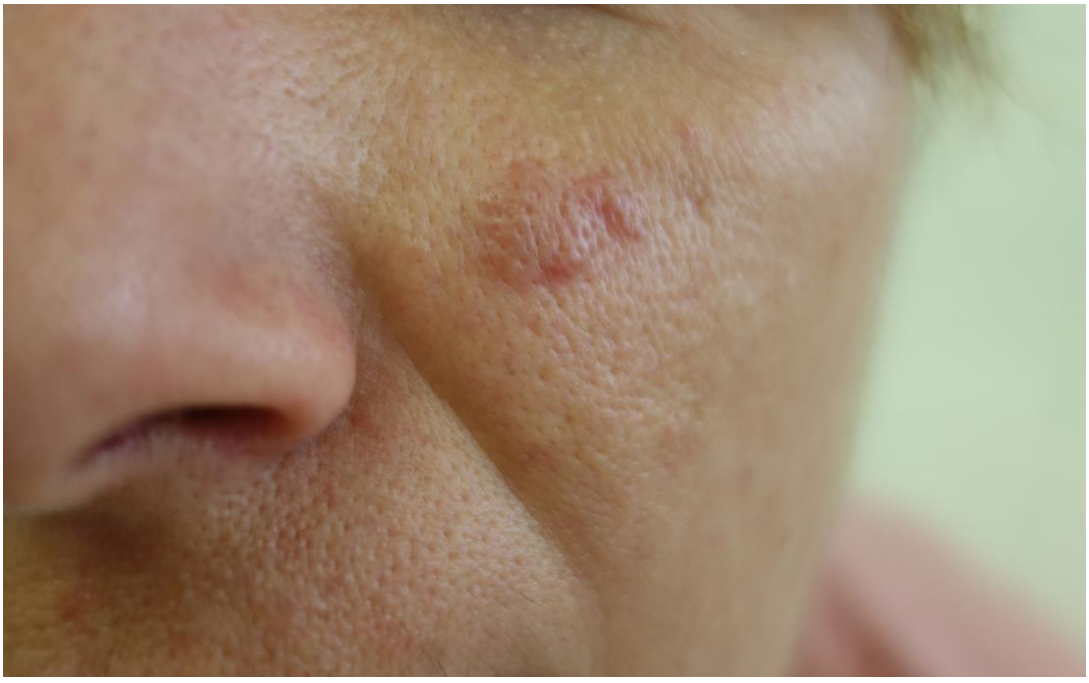

Figure 1. Slightly infiltrated, red to brown nodule at left malar region.

Histopathologic examination demonstrated, a dense, polymorphous, inflammatory cell infiltrate in the mid and deep dermis. The epidermis was spared and a Grenz zone in the upper dermis was present (Figure 2).

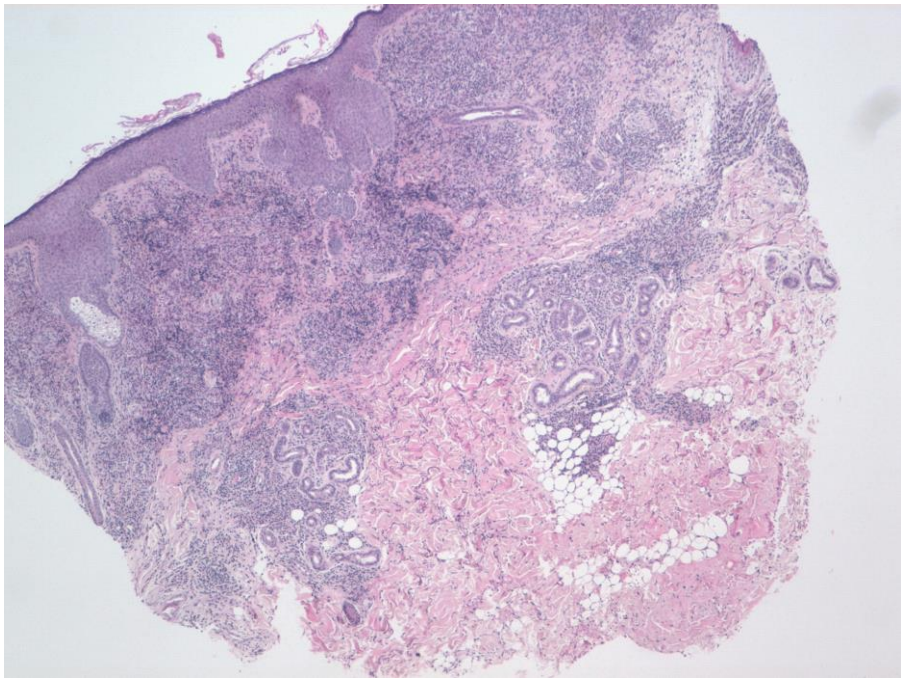

Figure 2. (H&E, original magnification x10). Dense, polymorphous, inflammatory cell infiltrate in mid and deeper dermis. Epidermis was spared and a Grenz zone in upper dermis was present

At higher magnification, this infiltrate had a predominance of eosinophils and neutrophils; some areas of leukocytoclasia and some extravasated red blood cells were present, without true vasculitis (Figure 3).

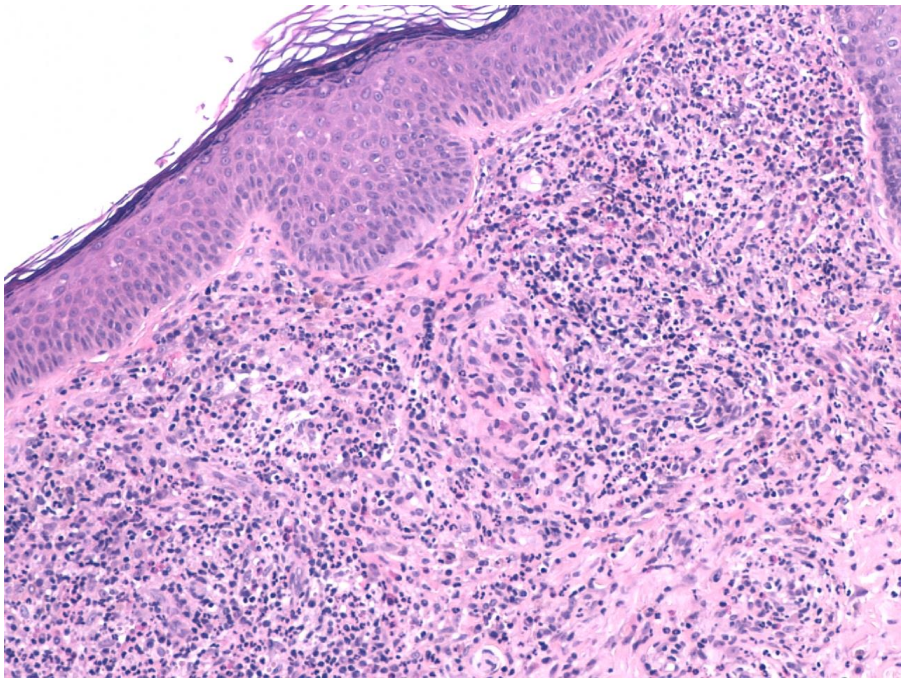

Figure 3. (H&E, original magnification x20). Infiltrate had predominance of eosinophils and neutrophils; some areas of leukocytoclasia and some extravasated red blood cells were present, without true vasculitis signs.

We established diagnosis of Granuloma faciale, and after two-months therapy with topical tacrolimus 0,1% twice daily, the nodule resolved. After six months of follow up there was no recurrence.

Discussion

Granuloma Faciale (GF) most often occurs in middle-aged Caucasian men and typically presents as a single and asymptomatic nodule, but multiple lesions have been described.

The papules or nodules are round or polycyclic, red-brown-purple in color, with a shiny surface that exhibits dilated follicular ostia [1]. Superficial telangiectasias and follicular accentuation can often be observed [2].

The favored sites of GF are the sides of the nose (30%), tip of the nose (7%), preauricular area (22%), cheeks (22%), forehead (15%), and helix of the ear (4%) [3]. Some rare cases of extrafacial GF have been reported and the trunk, upper and lower

limbs, and scalp are the most frequent locations. In fact, it is not unusual for facial lesions to precede the development of extrafacial GF [1].

GF etiology is unknown. GF is one of those misnomers in dermatology, as granulomatous inflammation does not normally occur. Histopathology findings may differ depending upon the lesion stage at the time biopsy is performed. At early stages, a small vessel leukocytoclastic vasculitis pattern is seen. However, it is not a conventional one [4]. Fibrin and neutrophils can be observed at the vessel walls, but nuclear dust is scant and there are few extravasated erythrocytes. The infiltrates become much denser than in conventional leukocytoclastic vasculitis [4].

The changes that make GF more distinctive develop with time [4]. GF is characterized by a dense polymorphic cellular infiltrate with abundant eosinophils, in the upper dermis, with a Grenz zone between the dermis and epidermis and its appendages [4]. The epidermis is often spared, but some reactive hyperplasia can be observed. One may observe previous findings of vasculitis with less cellularity and a new fibrotic component that is often oriented concentrically around small vessels [4]. It is important to note that the histopathology of GF and erythema elevatum diutinum (EED) are very similar [5]. In fact some authors think they are the same entity [6].

The clinical differential diagnosis includes sarcoidosis, lymphoma, tumid lupus, cutaneous fungal infection, or leishmaniasis.

GF lesions are associated with a chronic and progressive clinical course. They usually grow slowly and are very persistent, but without associated systemic involvement. Spontaneous resolution is very exceptional. Moreover, GF can be resistant to treatment and recurrences are frequent. Multiple modalities have been used with variable success including single therapy or combinations of colchicine, dapsone, antimalarials, gold injections, isoniazid, clofazimine, topical psoralen with UVA, corticosteroids, cryosurgery, various laser therapies, surgical excision, and 5-fluorouracil.

Tacrolimus blocks T-cell activation and proliferation, leading to inhibition of secretion of IFN-c. IFN-c is considered to be important in the pathogenesis of GF [7]. There is no consensus concerning the duration of treatment with tacrolimus for GF. After a literature review, authors prefer topical tacrolimus 0.1% used 2 twice daily, from 2 to 6 months, but success is variable. Possible adverse effects of this treatment have been described, such as residual hyperchromia and telangiectasias [8]. Recurrence of lesions after stopping treatment have been described.

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