«plexos» que expresan CD68 y CD163. Este rasgo peculiar se ha descrito previamente en 2 pacientes, pero en menor cuantía[1]. Dada la heterogeneidad inmunohistoquímica y, en general, la baja expresión de marcadores típicos neurales que exhibe esta neoplasia, consideramos la clínica y la morfología como elementos claves para el diagnóstico.

Conflicto de intereses

Los autores declaran no tener ningún conflicto de intereses.

Agradecimientos

Los autores manifestamos nuestro agradecimiento al Servicio de Dermatología y al personal técnico del Servicio de Anatomía Patológica del Complejo Hospitalario de Navarra.

Bibliografía

1. Requena L, Sitthinamsuwan P, Fried I, Kaddu S, Schirren C, Schärer L, et al. A benign cutaneous plexiform hybrid tumor of perineurioma and cellular neurothekeoma. Am J Surg Pathol. 2013;37:845–52.
2. Yamada S, Kitada S, Nabeshima A, Noguchi H, Sasaguri Y, Hisaoka M. Benign cutaneous plexiform hybrid tumor of perineurioma and cellular neurothekeoma arising from the nose. Diagn Pathol. 2013;8:165.
3. Linos K, Stuart L, Goncharuk V, Edgar M. Benign cutaneous biphasic hybrid tumor of perineurioma and cellular neurothekeoma: A case report expanding the clinical and histopathologic features of a recently described entity. Am J Dermatopathol. 2015;37:319–22.
4. Hornick J, Fletcher C. Cellular neurothekeoma: Detailed characterization in a series of 133 cases. Am J Surg Pathol. 2007;31:329–40.
5. Fetsch J, Laskin W, Hallman J, Lupton G, Miettinen M. Neurothekeoma: An analysis of 178 tumors with detailed immunohistochemical data and long-term patient follow up information. Am J Surg Pathol. 2007;31:1103–14.

C. Areán[a,*], A. Córdoba[a], L. Requena[b] y Mª.L. Álvarez[a]

[a] *Servicio de Anatomía Patológica, Complejo Hospitalario de Navarra, Pamplona, Navarra, España*
[b] *Departamento de Dermatología, Fundación Jiménez Díaz, Universidad Autónoma, Madrid, España*

* Autor para correspondencia.
Correo electrónico: carolina.arean.cuns@navarra.es (C. Areán).

http://dx.doi.org/10.1016/j.ad.2016.03.002

Majocchi Granuloma of the Breast: A Rare Clinical Entity

Granuloma de Majocchi: Una rara entidad

Dear Editor,

Deep penetration of the skin by dermatophytic agents may provoke a granulomatous inflammatory skin reaction. This was first described by Majocchi.[1] Majocchi granuloma is characterized clinically by papular, pustular, or nodular inflammatory lesions occurring typically on the limbs or face,[2] though isolated cases affecting the vulva or scrotum have been published.[3] We report a unique presentation of Majocchi granuloma in the breast; there are no previous reports of involvement of this location.

A 28-year-old female presented with a slightly painful, raised reddish lesion that had developed in her left breast over the previous 3 months. She gave no history of trauma and did not describe constitutional symptoms. The lesion had increased in size after the repeated application of mud to the area over 2 months. Treatment with a potent topical corticosteroid (betamethasone valerate, 0.122% wt/wt) was then applied twice daily for a month.

Clinical examination revealed a single erythematous plaque with areas of scarring. A yellowish discharge emanated from multiple openings in an area beneath the left nipple measuring approximately 5 × 4 cm (Fig. 1). On palpation the plaque was firm and indurated and was slightly tender; there was no underlying breast lump. Diascopy findings were nonspecific. Gram stain of the discharge showed the presence of gram-positive cocci and abundant neutrophils. No acid-fast bacilli (AFB) were seen on direct smear. Potassium hydroxide (KOH) mount and fungal culture of skin scrapings and of the discharge was negative. Ultrasonography of the breasts was normal, though a single lymph node of 20 × 8 mm with a preserved hilum was observed in the left axilla. Chest X-ray was normal and the Mantoux skin test produced a wheal of 8 × 6 mm. Routine blood tests were within normal limits. The patient was treated with oral cefuroxime 500 mg twice a day plus topical 2% mupirocin cream but showed no improvement after 2 weeks of treatment. We performed skin biopsy based on a differential diagnosis of hidradenitis suppurativa, lupus vulgaris, or subcutaneous fungal infection. Hematoxylin and eosin staining of the sample revealed a mixed cell,

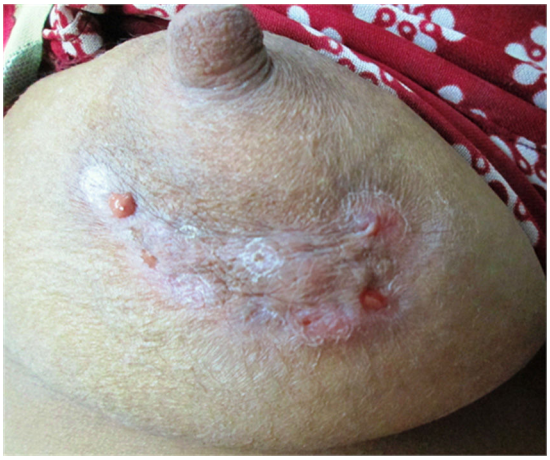

Figure 1 Papular-pustular plaque on the left breast with central scarring and a purulent discharge visible at the periphery.

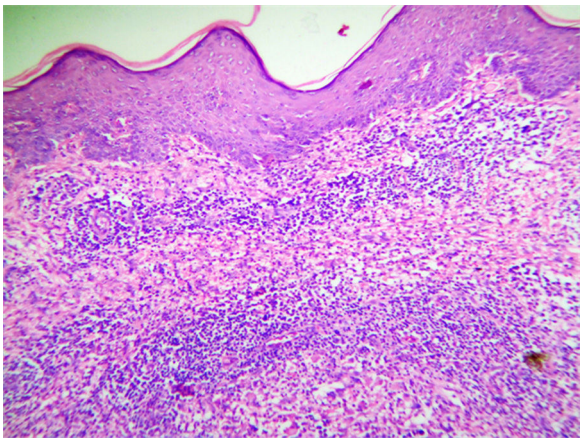

Figure 2 Acanthosis of the epidermis with a mixed cell inflammatory infiltrate in the superficial and mid dermis. Hematoxylin and eosin, original magnification ×100.

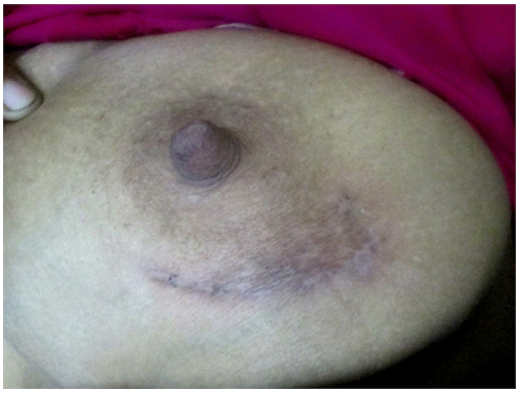

Figure 3 Complete resolution of the plaque after finishing treatment.

granulomatous inflammatory reaction in the dermis (Fig. 2). Ziehl-Neelsen and periodic acid Schiff (PAS) stains to detect AFB and fungi were both negative. Mycobacterial culture of the tissue sample was negative, but fungal culture on Sabouraud dextrose agar with chloramphenicol produced growth of heaped up, folded violaceous colonies of waxy consistency after 3 weeks, suggestive of *Trichophyton violaceum*. Lactophenol cotton blue mount showed the presence of tangled, irregular branched hyphae with chlamydospores. With a final diagnosis of Majocchi's granuloma, the patient was treated with 250 mg of oral terbinafine once daily plus topical 2% sertaconazole cream twice daily. The lesion resolved completely within 8 weeks and there was no evidence of recurrence after 6 months of follow-up (Fig. 3).

In 1883, Majocchi[1] described a phenomenon in which dermatophytes, which usually remain limited to the stratum corneum, become more aggressive and invade the superficial dermis. Majocchi granuloma typically develops when a dermatophyte infection extends down a hair follicle in the setting of localized immunosuppression (most commonly a potent topical steroid) or systemic immunosuppression. The result is a granulomatous response in the dermis.[2] The commonly implicated organisms are *Trichophyton* species (*rubrum*, *mentagrophytes complex*, *violaceum*) as well as some nondermatophyte species such as *Aspergillus* and *Phoma*.[4] Although historically *T. violaceum* has been the most commonly identified organism, Majocchi granuloma now a days is usually due to *T rubrum*.

Two clinical forms of Majocchi granuloma, follicular and subcutaneous nodular, have been described.[4] The follicular type usually develops after trauma, repeated shaving of hair-bearing areas, or topical corticosteroid treatment, and in cases of long-standing immunosuppression.[5,6] The subcutaneous nodular type occurs in immunocompromised hosts. Our patient developed the follicular type, probably caused by endothrix-type infection due to anthropophilic *T. violaceum* secondary to the use of a potent topical steroid under occlusion, which could have led to deep penetration of the fungus. In modern medicine, systemic antifungals such as griseofulvin,[7] itraconazole,[8] and terbinafine[9] are the mainstays of therapy as they are safe and effective. The duration of therapy should be of at least 4–8 weeks, and treatment should be continued until all lesions have cleared. In the reports in literature, nearly all lesions resolve without scarring within 6 weeks of starting antifungal. The response of Majocchi granuloma to oral terbinafine can be explained on the basis of its pharmacokinetics.[10] Terbinafine is the preferred oral therapy for treating Majocchi granuloma not only for its superior efficacy in eliminating dermatophytes, but also because of its greater selectivity for the skin structures involved in Majocchi granuloma and fewer drug interactions than azole antifungals like itraconazole.[9] We used terbinafine for its good antidermatophyte activity, adequate penetration into common sites of dermatophyte infection (stratum corneum and the hair follicle), lower rates of recurrence, low rate of drug interactions (its metabolism does not involve cytochrome P450), and its cost effectiveness when long-term therapy is warranted to prevent relapse.[8] A diagnosis of Majocchi granuloma should always be considered in papular-pustular plaques, especially when the patient describes factors associated with local or systemic immunosuppression. Negative results of KOH examination or fungal culture of skin scrapings or of the purulent discharge do not exclude a diagnosis of Majocchi granuloma, which should be confirmed either by tissue culture or by PAS staining of histopathology samples. Although the detection of fungal spores or hyphae using special stains on histopathology samples will confirm the diagnosis, these structures may sometimes escape detection, as in our case.[11] Fungal spores and hyphae are usually detected within hairs or hair follicles and in dermal granulomas. Our inability to detect the fungal elements may have been due to the absence of hair follicles and the poorly defined granulomas in our biopsy specimen. To the best of our knowledge, this is the first reported case of Majocchi granuloma of the breast.

Conflict of interests

The authors declare no conflict of interest.

Bibliografía

1. Majocchi D. Sepra una nuova tricofizia (granuloma tricofitico), studi clinici micologici [A new Trichophyton granuloma:

clinical and mycological studies]. Bull R Accad Roma. 1883: 9220–3.
2. Kurian A, Haber RM. Tinea corporis gladiatorum presenting as a majocchi granuloma. ISRN Dermatol. 2011;2011:767589.
3. Ilkit M, Durdu M, Karakaş M. Majocchi's granuloma: a symptom complex caused by fungal pathogens. Med Mycol. 2012;50:449–57.
4. Smith KJ, Neafie RC, Skelton HG, Barrett TL, Graham JH, Lupton GP. Majocchi's granuloma. J Cutan Pathol. 1991;18: 28–35.
5. Steiner UC, Trüeb RM, Schad K, Kamarashev J, Koch S, French LE, et al. Trichophyton rubrum-induced Majocchi's granuloma in a heart transplant recipient. A therapeutic challenge. J Dermatol Case Rep. 2012;6:70–2.
6. Elgart ML. Tinea incognito: an update on Majocchi granuloma. Dermatol Clin. 1996;14:51–5.
7. Novick NL, Tapia L, Bottone EJ. Invasive Trichophyton rubrum infection in an immunocompromised host. Case report and review of the literature. Am J Med. 1987;82:321–5.
8. Gupta AK, Groen K, Woestenborghs R, de Doncker P. Itraconazole pulse therapy is effective in the treatment of Majocchi's granuloma: a clinical and pharmacokinetic evaluation and implications for possible effectiveness in tinea capitis. Clin Exp Dermatol. 1998;23:103–8.
9. Gupta AK, Prussick R, Sibbald RG, Knowles SR. Terbinafine in the treatment of Majocchi's granuloma. Int J Dermatol. 1995;34:489.
10. Faergemann J, Zehender H, Jones H, Maibach I. Terbinafine levels in serum, stratum corneum, dermis-epidermis (without stratum corneum), hair, sebum and eccrine sweat. Acta Derm Venereol. 1991;71:322–6.
11. Gupta S, Kumar B, Radotra BD, Rai R. Majocchi's granuloma trichophyticum in an immunocompromised patient. Int J Dermatol. 2000;39:140–1.

U. Khanna*, T. Kumar Dhali, P. D'Souza, S. Chowdhry

Department of Dermatology, ESIPGIMSR Basaidarapur, New Delhi, 15, India

* Corresponding author.
E-mail address: urmi23khanna@gmail.com (U. Khanna).

http://dx.doi.org/10.1016/j.ad.2016.02.005

Sífilis secundaria simulando liquen plano en el paciente con infección por VIH

Secondary Syphilis Mimicking Lichen Planus in a Patient With HIV Coinfection

Sra. Directora:

La sífilis representa una infección de transmisión sexual, con una incidencia creciente en los últimos años. En España, afecta sobre todo a hombres que tienen sexo con hombres, y muchos de los afectados tienen, además, infección por el virus de la inmunodeficiencia humana (VIH)[1]. La coexistencia de estas 2 enfermedades afecta a la historia natural de las mismas. La infección por el VIH hace que las manifestaciones clínicas de la sífilis puedan progresar de forma más rápida. Además, son más frecuentes las formas agresivas y atípicas de sífilis[2].

Dos varones homosexuales con infección por VIH acudieron al dermatólogo por presentar lesiones genitales. El primero de ellos, de 35 años de edad, con lesiones discretamente pruriginosas en genitales de un mes y medio de evolución, refería haberse aplicado corticoides tópicos, sin mejoría. Su infección por VIH presentaba un buen control inmunológico desde 2009, sin tratamiento antirretroviral. En la exploración se observaban placas eritematosas de aspecto liquenoide, con tendencia a la confluencia, localizadas en la cara dorsal del pene (fig. 1A y B). El paciente aportó un análisis sanguíneo (incluyendo bioquímica, hemograma y coagulación), sin hallazgos significativos. Se realizó una biopsia cutánea que reveló una dermatitis liquenoide con borramiento de la capa basal, exocitosis neutrofílica y un infiltrado linfoplasmocitario en banda (fig. 2A). Se observaron, también, numerosas células plasmáticas en la pared y alrededor de los vasos de la dermis (fig. 2B). La tinción mediante técnicas de inmunohistoquímica para *Treponema pallidum* resultó positiva (fig. 2C). Se solicitó una serología para sífilis, obteniéndose unos títulos de anticuerpos no treponémicos reagina plasmática rápida (RPR) de 1/128 y anticuerpos treponémicos positivos.

El segundo caso, de 29 años de edad, con diagnóstico de infección por VIH desde el 2011, con buen control inmunológico, y sin tratamiento farmacológico, consultó por la presencia de lesiones escrotales con escaso prurito de un mes de evolución. Había sido diagnosticado de eczema, y tratado con corticoides tópicos, sin mejoría. En la exploración presentaba numerosas placas rosadas con aspecto liquenoide a nivel escrotal y en base del pene (fig. 3). Se realizó un análisis sanguíneol (bioquímica, hemograma y coagulación) que no mostró alteraciones, así como una serología para sífilis. Se realizó una biopsia cutánea en la que se objetivó una dermatitis liquenoide, con estudio inmunohistoquímico positivo para *Treponema pallidum*. El RPR fue positivo a título de 1/64, con ELISA y hemaglutinación positivos.

En ambos pacientes se realizó el diagnóstico de sífilis secundaria, simulando un liquen plano, con afectación exclusivamente genital. La evolución fue satisfactoria con resolución completa de las lesiones tras tratamiento con $2{,}4 \times 10^6$ U de penicilina G benzatina intramuscular, y descenso en los títulos serológicos.

Las manifestaciones cutáneas de la sífilis secundaria aparecen entre las semanas 3-12 después de la aparición del chancro, aunque pueden desarrollarse meses después o antes de la desaparición del mismo[2]. Las sifílides son con frecuencia maculopapulares y eritematodescamativas, aunque de manera menos frecuente pueden manifestarse como sifílides liquenoides, nodulares o ulcerativas[3,4]. La infección por el VIH favorece la progresión más rápida de la sífilis, por la alteración existente en la inmunidad celular y, en ocasiones, se observan manifestaciones clínicas atípicas[4].