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Psoriatic Plaques Confined to Vitiliginous Skin: Report of Two Cases

Placas de psoriasis sobre piel con vitiligo: reporte de dos casos

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ABSTRACT

Psoriasis and vitiligo are common dermatologic diseases that share autoimmune pathogenesis. Although concomitance of both diseases is occasionally observed in clinical practice, the development of psoriatic plaques over vitiligo depigmented patches is rare. We report the aforementioned phenomenon in two Northwestern Mexican women and give some insight over its possible cause.

KEYWORDS: *psoriasis, vitiligo, autoimmune.*

RESUMEN

La psoriasis y el vitiligo son enfermedades dermatológicas comunes que comparten una patogénesis autoinmune. Si bien su concomitancia es ocasionalmente observada en la práctica clínica, el desarrollo de placa psoriásica sobre áreas de vitiligo es rara. Aquí se reporta el anteriormente mencionado fenómeno en dos mujeres del noroeste de México y se explica sobre su posible causa.

PALABRAS CLAVE: *psoriasis, vitiligo, autoinmune.*

Introduction

Psoriasis and vitiligo are frequently observed on dermatological consultation as they are relatively common, having a worldwide prevalence of 0.5% for psoriasis and 0.5-2% for vitiligo.[1,2] Their high prevalence and the fact that they both possess autoimmune pathogenesis to make it likely for them to converge on a same individual.[1-5] In this sense, the chance of vitiligo patients to develop psoriasis is up to 6%.[6,7] However, developing psoriatic lesions confined to vitiliginous macules is infrequent.[4-8] Growing evidence indicates this phenomenon cannot be attributed just to mere coincidence, as both vitiligo and psoriasis share common autoimmune pathogenesis and genetic components.[1,3,9] Some authors have suggested a putative role for Koebner phenomenon and even neuropeptides genetically predisposed individuals, although there is no current consensus for its etiology.[4,5,8,10]

Here, we describe the clinic-pathological and immunological findings in two Mexican patients with vitiligo that developed psoriasis in concurrent anatomical sites out of a 170 vitiligo patient cohort (1.2% of prevalence). Permission to document the pathology, including biopsy analysis and picture publication, was granted by the patients via written informed consent. Procedures were in accordance with following the 1975 Helsinki Declaration as revised in 1983.

Case reports

Case 1

A 68-year-old female with skin phototype 4 presenting stable vitiligo vulgaris with onset at age 32, without poliosis or leukotrichia, and with repigmentation areas (Vitiligo Disease Activity [VIDA] score=-1). Psoriasis appeared at age 55 consisting in of erythematous-squamous plaques with 1 cm of thickness, sometimes pruriginous, superimposed to vitiliginous lesions on arms and legs (figure 1A), corresponding to plaque psoriasis diagnosis. The anti-TNF-α antibody adalimumab has been administered for the previous 6 months at 40 mg biweekly doses to treat psoriasis symptoms. Moreover, the patient reports a family history of vitiligo, psoriasis, and hypothyroidism. Blood chemistry and hematic biometry show values within normal

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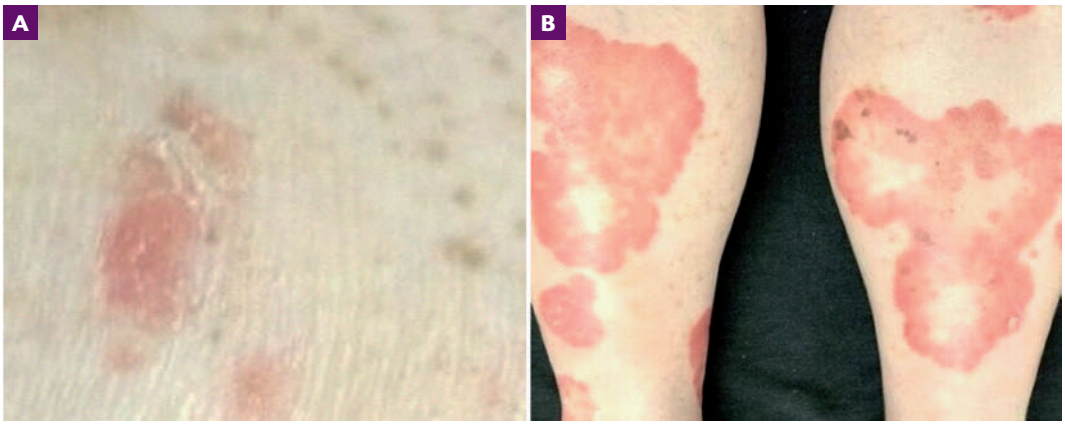

Figure 1. Image of right leg of patient 1 (A) that shows erythematous-squamous small well defined plaques settled on achromic macule, and (B) erythema and scale that converge on welldefined plaques upon large achromic macules on both legs of patient 2. (This photograph was taken on March 29, 2017, during both interview and dermatological consultation with permission of the patients).

ranges, except for an increase in monocytes (0.7 × 10^3/µL, range: 0.1-0.6 x10^3/µL). The thyroid profile test shows normal values for T3, T4, FT3, and FT4, but thyroid-stimulating hormone (TSH) is elevated: (5.38 mIU/L, range: 0.4-4 mIU/L). Finally, patient is negative for anti-thyroglobulin (anti-Tg), anti-thyroperoxidase (anti-TPO) and antinuclear antibodies (ANAs) and shows low percentage and counts of $CD8^+$ lymphocytes (7%, range: 13-41%; and 194 cells/µL, range: 190-1140 cells/µL, respectively) but normal $CD4^+$ lymphocytes counts/percentage.

Case 2

A 48-year-old female with skin phototype 3 presenting stable vitiligo universalis (VIDA score=0) with onset at age 8, without leukotrichia. Psoriasis emerged 8 months ago disseminating to head and limbs superimposed to vitiliginous lesions. Plaque psoriasis lesions present as erythematous-squamous plaques up to 20 cm, with chronic pruritus (figure 1B) for which 180 mg fexofenadine and 0.1% tacrolimus were administered. She reports a family history of vitiligo, but not of psoriasis or another autoimmune disease so far. For this patient, blood chemistry indicates low glucose levels: 64 mg/dL (range, 74-106 mg/dL) and hematic biometry shows erythropenia (4 × 10^6/µL, range: 4.2-5.4 × 10^6/µL), leukopenia (4.5 x10^3/µL, range: 5-10 x10^3/µL), neutropenia (2.2 x10^3/µL, range: 3-4.5 × 10^3/µL) and basopenia (0 × 10^3/µL, range: 0.3-0.6 × 10^3/µL). Thyroid profile test shows normal values, except for a decrease in FT4 (0.9 ng/dL, range: 0.93-1.7 ng/dL). Moreover, the patient is negative for anti-Tg and anti-TPO, but positive to ANAs. On the other hand, T $CD4^+$ and $CD8^+$ lymphocyte counts/percentages are within the normal range, although $CD8^+$ count tend tends to the lower point of the range (288 cells/µL, range: 190-1140 cells/µL).

Histopathological findings

For both patients, biopsies show thin skin devoid of melanocytes at the dermo-epidermal junction, scanty lymphocytes, acanthosis, elongation, and thickening of crest networks, papillary congestion, confluent parakeratosis and Munro's microabscess in stratum corneum, the latter being predominant in patient 2 (figure 2). The above was corroborated with Fontana-Masson staining and immunostaining for PS100 (images not shown). Also, CD4 (not shown) and CD8 immunostaining was performed (figure 3), finding a scarce distribution of these cells in the skin biopsies of both patients.

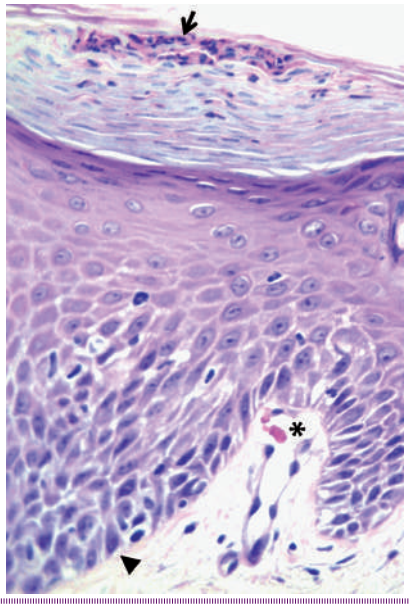

Figure 2. Biopsy sample taken of skin with vitiligo and psoriasis concurrence that shows a lack of melanin pigment at layer basal (arrowhead) with elongation of crest networks, papillary congestion (*) and Munro's microabscess (arrow) (hematoxilin-eosin staining, 400X).

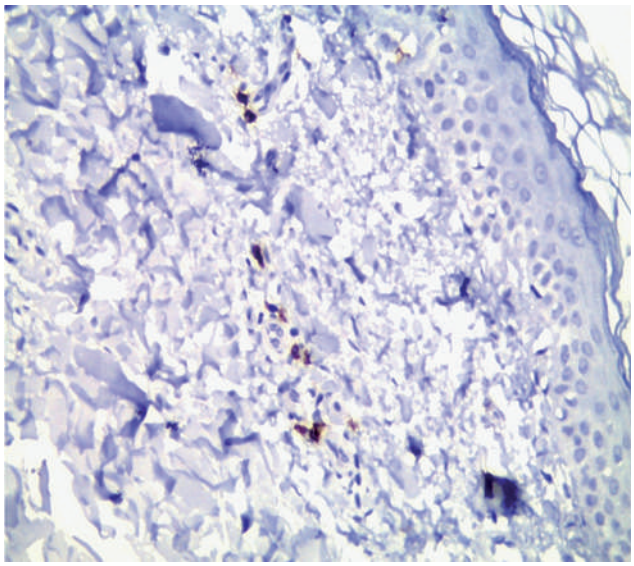

Figure 3. CD8 immunoperoxidase staining showing scanty lymphocytes at dermis (400X).

Discussion

Histopathologically, vitiligo, and psoriasis seemed to act as separate entities each one causing their characteristic skin changes, as no particular feature was observed in concomitant lesions, in accordance with previous reports.[8,10] Vitiligo is stable in both patients and treatment has been focused on psoriasis. Case 1 has had a better outcome than case 2 probably due to anti-TNF-α therapy (adalimumab), further corroborating proposals of high TNF-α levels as a linking factor in the coexistence of vitiligo and psoriasis.[2,4,5]

Although TNF-α is directly involved in the inflammatory process in both diseases, particularly in psoriasis[1,3,11]; recent evidence suggests that the autoimmune response is driven by anti-melanocyte $CD8^+$ lymphocytes: cytotoxic, melanocyte-depleting Tc1 for vitiligo and non-cytotoxic, inflammatory Tc17 for psoriasis.[12,13] In this respect, the wound repairing cytokine TGF-β can convert Tc1 cells to Tc17, thus encouraging the co-localization of both diseases is due to the Koebner phenomenon.[8,14] Nevertheless, the latter process might also be circumstantial due to the shift from Th1 to Th17 immune profile in spreading vitiligo lesions, increasing the risk of developing psoriasis, particularly at lesional sites.[12,15] Moreover, both patients developed psoriasis post menopause which is related to exacerbation of psoriasis but can also be a contributing factor for its development.[16]

Regarding our study, the apparently contrasting observation of low $CD8^+$ lymphocyte levels on circulation and lesional sites in both patients is probably a consequence of treatment, as both adalimumab and tacrolimus exert such effect.[1,11] Nevertheless, case 2 treatment does not seem to be very effective considering her more severe clinical manifestations (figure 1), which might be related to ANAs positivity. Furthermore, other factors should be involved, such as genetics and probably hormones as low estrogen levels typical of postmenopausal women have a stimulatory effect on TNF- α production, thus exacerbating psoriasis.[16]

To our knowledge, this is the second case report of vitiligo-psoriasis concomitance in Mexican patients.[10] We believe it is necessary to keep reporting these associations along with investigations over the etiology of vitiligo and psoriasis to better understand their coexistence.

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