



CASE AND RESEARCH LETTERS

Secondary Amyloid Deposition in Discoid Lupus Erythematosus



Depósitos de amiloide secundarios a lupus eritematoso discoide

Dear Editor,

Secondary amyloid deposition is occasionally associated with skin tumors and inflammatory skin disorders, but it is rarely seen in discoid lupus erythematosus (DLE). We describe a

case of secondary amyloid deposition in the lesional skin of DLE.

A 47-year-old woman presented with keratotic erythema on the nose 3 years ago. The erythema had been present for 1 year and the histological features were compatible with DLE. Three years later, she revisited our hospital because the scaly erythematous patches had gradually increased in number. Physical examination revealed several reddish, well-circumscribed plaques with slightly elevated borders on the forehead, nose, cheeks, lip, and ear (Fig. 1A). Laboratory data showed leukocytes within the normal range (8100 mm^{-3} with 32% neutrophils), normal liver and renal

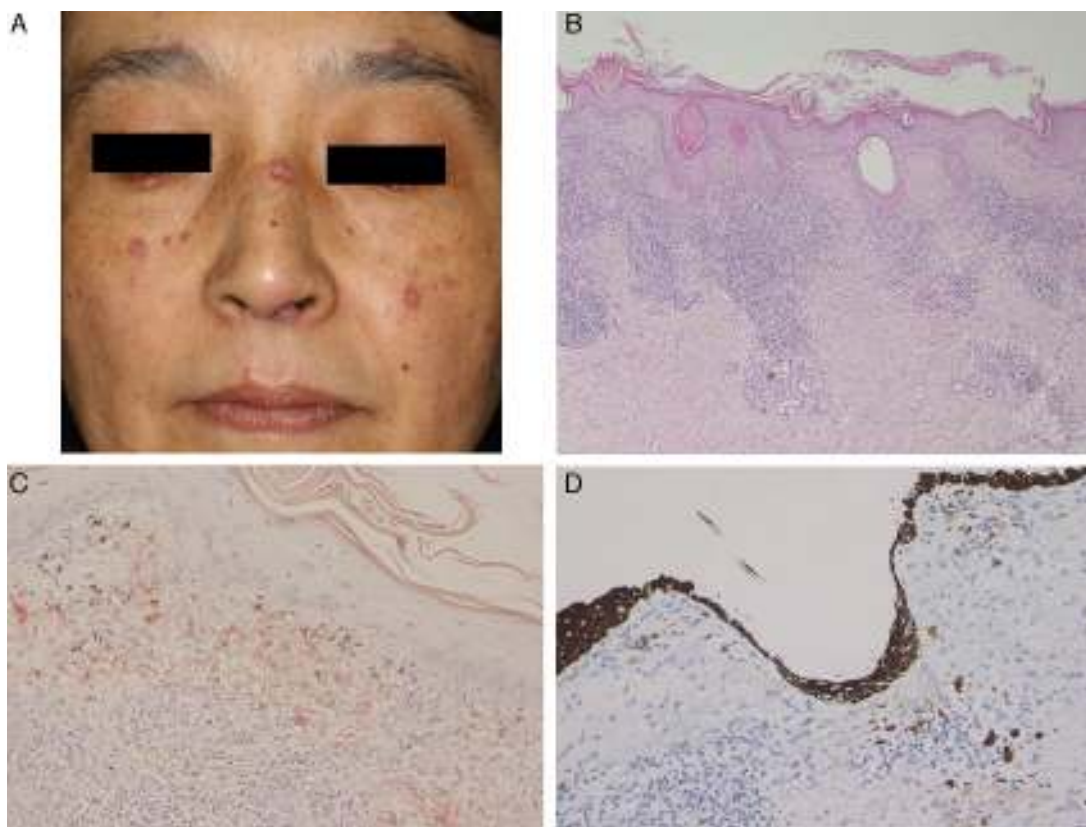


Figure 1 (A) Multiple keratotic plaques scattered on the face. (B) Biopsy showed parakeratosis with focal hyperkeratosis, follicular plugging, liquefaction changes to the basal layers of the epidermis, and cellular infiltrates in the upper dermis (hematoxylin–eosin stain, original magnification $100\times$). (C) Dylon staining revealed amyloid deposition in the upper dermis (original magnification $200\times$). (D) The upper dermis was also positive for keratin (original magnification $200\times$).

function, elevated immunoglobulin (Ig) G (2172 mg/dL), and positive titers of antinuclear antibody (1:1280), anti SS-A antibody (37.0 U/mL; normal, <7.0 U/mL), and anti-centromere antibody (index of 34.4; normal, <10). Serum complement levels and antibodies against SS-B, ds-DNA, and Sm were all within normal limits. A second biopsy taken from a lesion on the dorsum of the nose showed parakeratosis, follicular plugging, liquefaction of the basal layer, and focal cellular infiltration in the dermis (Fig. 1B). There was eosinophilic material in the upper dermis, which was positive with Congo red and Dylon staining (Fig. 1C) and anti-cytokeratin antibody 34 β E12 staining (Fig. 1D). The direct immunofluorescence study revealed IgM deposits at the dermal-epidermal junction, but no amyloid deposits were observed in another DLE specimen taken from the back of the ear. A detailed eye exam revealed decreased lacrimation, but saliva production measured by the Saxon test was normal.

Few papers have reported on secondary amyloid deposition in DLE, but it has been suggested that this phenomenon might be underreported.¹ Powell et al.¹ reported 3 cases of DLE on the head and neck with amyloid deposition. They also retrospectively examined 18 cases of DLE, and detected amyloid deposition in 1 case. In a Japanese series, Khan et al.² detected amyloid deposition in all 4 cases of hypertrophic DLE analyzed but in just 1 of 12 cases of nonhypertrophic DLE. They speculated that cutaneous amyloid material might have been deposited secondary to abnormalities in the basement membrane zone following repeated sunlight exposure for long periods. Our case was not hypertrophic-type DLE, but amyloid deposition was detected on the nose, which had an evident keratotic appearance. The fact that amyloid deposits were not detected in the DLE lesion on the back of the ear suggests that UV radiation may play a role in inducing basement membrane impairment, resulting in secondary amyloid deposition. The amyloid material also stained positively for 34 β E12, suggesting that the origin of the amyloid was degenerating epidermal keratinocytes, which may be precursors of cutaneous amyloid. The presence of lamina densa-like substances or disturbed keratinization in the basal layers might also be involved in amyloid production.³ Our patient also developed Sjögren syndrome, although

she did not show any symptoms of dry mouth. Because systemic sclerosis was ruled out, the presence of serum anticontromere antibody was considered to be associated with Sjögren syndrome, although coexistence of this condition with DLE is rare.⁴ Anticentromere antibody has been detected in 10% of Japanese patients with primary Sjögren syndrome⁵; these patients had lower mononuclear cell infiltration in the minor salivary glands, possibly explaining the lower frequency of dry mouth in this subgroup of patients. This hallmark symptom of Sjögren syndrome was also absent in our patient. In conclusion, there have been few reports on secondary amyloid deposition in DLE, but there may be more cases, especially in hyperkeratotic lesions in sun-exposed areas.

References

1. Powell AM, Albert S, Bhogal B, Black MM. Discoid lupus erythematosus with secondary amyloidosis. *Br J Dermatol.* 2005;153:746–9.
2. Khan MAK, Maruno M, Khaskhely NM, Uezato H, Nonaka S. Amyloid deposition is frequently observed in skin lesions of hypertrophic lupus erythematosus. *J Dermatol.* 2002;29:633–7.
3. Horiguchi Y, Fine JD, Leigh IM, Yoshiki T, Ueda M, Imamura S. Lamina densa malformation involved in histogenesis of primary localized cutaneous amyloidosis. *J Invest Dermatol.* 1992;99:12–8.
4. Matsui S, Kitabe S, Itoi S, Kijima A, Murota H, Tani M, et al. A case of disseminated DLE complicated by atopic dermatitis and Sjögren's syndrome: link between hypohidrosis and skin manifestations. *Mod Rheumatol.* 2011;21:101–5.
5. Nakamura H, Kawakami A, Hayashi T, Iwamoto N, Okada A, Tamai M, et al. Anti-centromere antibody-seropositive Sjögren's syndrome differs from conventional subgroup in clinical and pathological study. *BMC Musculoskelet Disord.* 2010;11:140.

Y. Hanami, T. Yamamoto*

Department of Dermatology, Fukushima Medical University, Fukushima, Japan

* Corresponding author.

E-mail address: toyamade@fmu.ac.jp (T. Yamamoto).

<http://dx.doi.org/10.1016/j.ad.2015.05.014>

Milia en placa en la cara posterior de ambos pabellones auriculares secundaria a radioterapia



Milia en plaque on the posterior surface of both auricles following radiation therapy

Sr. Director:

La milia son quistes epidérmicos benignos, que pueden presentarse en forma aislada o asociados a otros hallazgos clínicos. Si estas lesiones se encuentran agrupadas sobre una base eritematosa se denominan milia en placa (MEP).

Presentamos el caso de un varón de 48 años de edad, con antecedentes de adenocarcinoma de pulmón con metástasis cerebrales, que recibió tratamiento paliativo con radioterapia holocraneal que incluye a los pabellones auriculares a dosis de 30 Gy en 10 fracciones (2 campos laterales y opuestos conformados de fotones sobre el sistema nervioso central; la dosis total que reciben los pabellones auriculares se calcula que está entre 20-25 Gy). Acudió por presentar lesiones en la cara posterior de ambos pabellones auriculares de 3 meses de evolución, asintomáticas. Había padecido alguna lesión similar aislada desde la juventud, pero habían aparecido múltiples lesiones un mes después de recibir radioterapia. A la exploración clínica se observaron múltiples pápulas blanquecinas milimétricas, de superficie brillante, agrupadas sobre una base eritematosa, bilaterales