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Scattered Hypopigmented, Atrophic, and Folliculocentric Papules on the Trunk

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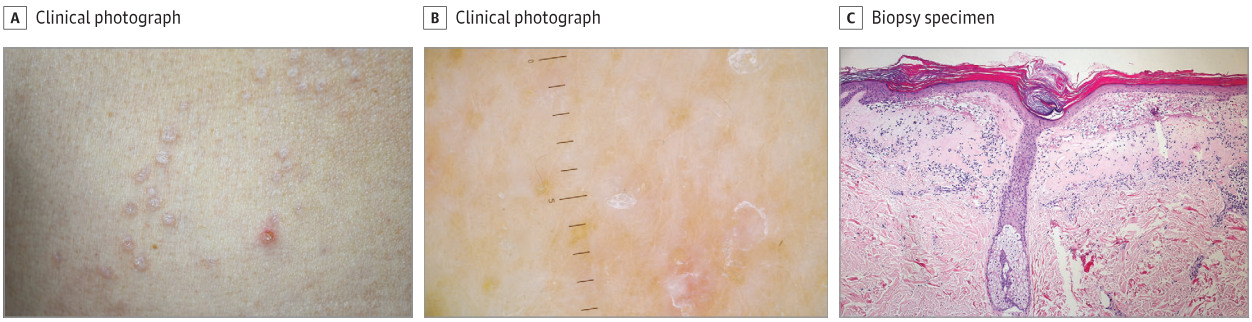

Figure. A, Multiple hypopigmented, atrophic, keratotic, and folliculocentric papules are seen on the trunk. B, Dermascopic findings included 2 foci of central keratin plugs and another ill-defined, whitish, and homogenous area with surrounding erythema. Scale bar corresponds to 1 mm. C, Histopathologic examination of a biopsy specimen (hematoxylin-eosin, original magnification ×100).

A woman in her 60s presented with numerous whitish papules on the back of more than 10 years' duration. The lesions previously resolved with topical corticosteroids but flared soon after treatment discontinuation. Some papules became pruritic and extended to the waist and anterior trunk in the past month. No family history of similar lesions was recorded. Physical examination revealed multiple hypopigmented, flat-topped papules on the back, waist, and inframammary area without genital involvement (**Figure**, A). Most lesions were folliculocentric under close inspection. Dermoscopy revealed central keratin plugs and some foci of structureless, whitish, and homogenous areas with surrounding erythema (Figure, B). The results of laboratory tests for antinuclear antibody and hyperglobulinemia were negative. A biopsy specimen was obtained from the back for histologic analysis (Figure, C).

WHAT IS YOUR DIAGNOSIS?

- **A.** Lichen amyloidosis
- **B.** Folliculocentric lichen sclerosus et atrophicus
- **C.** Guttate vitiligo
- **D.** Eruptive tumors of the follicular infundibulum

Diagnosis

B. Folliculocentric lichen sclerosus et atrophicus

Microscopic Findings and Clinical Course

Histopathologic examination revealed a central follicular plug and epidermal atrophy covered by orthokeratotic stratum corneum. The papillary dermis was markedly thickened with homogenized collagens, areas of edema, and sparse bandlike lymphocytic infiltration (Figure, C). There were some eosinophils in infiltrate and red blood cell extravasation. Congo red and mucicarmine stains produced negative results for amyloid and mucin. After the biopsy, fluocinonide cream, 0.05%, was prescribed, and the itchy papules resolved but still recurred after intermittent treatment.

Discussion

Lichen sclerosus et atrophicus (LSA) is a chronic inflammatory dermatosis first described by Hallopeau in 1887.[1] The cause is unclear, but LSA is linked clinically to autoimmune diseases, such as vitiligo, autoimmune thyroiditis, alopecia areata, pernicious anemia, and scleroderma.[2,3] The association with genetic factors, specific HLA types, estrogen deficiency, human papillomavirus and spirochete *Borrelia burgdorferi* infections, and trauma is also implicated.[2,3] Classically, LSA manifests as porcelain white, polygonal papules that often coalesce into plaques over the anogenital area. Approximately 15% to 20% of LSA involves only extragenital regions, among which the most common locations affected are the trunk, proximal extremities, buttocks, breast,

inframammary area, and neck.[2] Typical histopathologic findings include hyperkeratotic, thinned, and effaced epidermis with a wide band of hyalinization in the upper dermis and a lichenoid infiltrate below.

The folliculocentric variant of extragenital LSA has only been published in the literature twice. Mann et al[4] described a woman in her 70s, and El Habr et al[5] described a 10-year-old girl. Including the current study, all 3 patients were female from different ethnic groups and had bimodal age distributions, with 2 peaks occurring before puberty and after menopause.[2] The affected areas were generalized over the trunk and/or extremities without genital or facial involvement.

Dermoscopy may aid in the diagnosis of LSA with the homogenously whitish to yellowish areas and other minor features at various stages. Keratotic plugs and comedonelike openings correlate with histopathologic findings of follicular plug in the early phase.[6,7] Different from typical extragenital form, folliculocentric LSA demonstrates the keratotic plug standing out at the center of the expected follicle in the present case. Surrounding erythematous halo is the vascular pattern found in some active lesions. In older lesions, chrysalis structures correspond to the increased homogenized collagen and fibrosis in the upper dermis.[7]

The differential diagnosis of hypopigmented, atrophic, and keratotic eruption includes LSA, lichen amyloidosis, morphea, anetoderma, mycosis fungoides, eruptive tumors of the follicular infundibulum, guttate psoriasis, pityriasis versicolor, verruca plana, lichen planus, and pityriasis lichenoides chronica. Histologic findings were essential for the diagnosis. Special staining and laboratory tests exclude other causes. The folliculocentric feature is rare and specific for identification of the subtype.

Potent topical corticosteroid is the mainstream treatment for LSA, and topical calcineurin inhibitors are also recommended for maintenance.[2] Phototherapies, including psoralen plus UV-A, UV-A1, and narrow-band UV-B therapy, are the alternatives for extragenital LSA in the refractory condition.[2] Topical corticosteroid was used as the first-line option in all 3 reported cases with variable results, and pimecrolimus cream, 1%, was given twice daily at the weekends to enhance the therapeutic effect in one of them.[4,5]

The diagnosis of folliculocentric LSA is challenging and should be considered in the differential diagnoses of atrophic and folliculocentric lesions. The present case illustrates the clinical, dermoscopic, and histopathologic characteristics in the rare variant. A thorough evaluation helps to identify the folliculocentric component and leads to the final diagnosis.

ARTICLE INFORMATION

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