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Asymptomatic Erythematous and Tan, Shiny, Well-Defined Plaques

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Case Summary

A 56-year-old female presented with a 1-month history of asymptomatic erythematous and tan, shiny, well-defined plaques with peripheral reddish keratotic papules in the bilateral inframammary (Fig. 1) and infrapannicular folds. A waxy texture without induration was noted. The oral and genital mucosa and nails were unaffected. The patient did not have similar involvement elsewhere on the body, including the axilla. She trialed ketoconazole 2% cream without improvement and self-treated with over-the-counter miconazole nitrate powder and petroleum jelly. Medical history was unremarkable. A 4-mm punch biopsy of a representative area on the left lower abdomen was obtained (Fig. 2).

Question 1. What is your diagnosis?

Response Options 1.

A. Lichen sclerosus
B. Granular parakeratosis
C. Candidal intertrigo
D. Inverse psoriasis
E. Morphea

Correct Answer: B

Discussion

Granular parakeratosis is usually seen as grouped red-brown erythematous crusted or keratotic papules and plaques in the axilla, although other intertriginous areas are also possible. This patient did not have the characteristic reddish papules typically seen but rather lightly tan, parchment paper-like texture surrounded by the keratotic reddish papules. This case highlights the varied clinical morphology and involvement of intertriginous sites other than the axilla. On histology, expected features of granular parakeratosis include hyperkeratosis and compact parakeratosis with retained basophilic keratohyalin granules within the stratum corneum.

Granular parakeratosis occurs when the keratinocyte maturation from the stratum granulosum to the stratum corneum is disrupted. Altered maturation may be due to both failed filaggrin processing and keratohyalin granule breakdown. The filaggrin in keratinocyte cytoplasm clumps into basophilic keratohyalin.[1] Malformed cell surface structures and aberrant regulation of components in the cornified envelope can cause retention hyperkeratosis. Another proposed mechanism is a defect in transportation or utilization of filaggrin. As a result, keratin filaments are not properly attached, causing a barrier deficiency. Defective keratinocyte maturation may explain why topical steroids have used for treatment. They act on filaggrin and loricrin production and have a negative regulatory effect on conversion of profilaggrin to filaggrin while increasing proliferation.[2]

The patient's atypical presentation may initially suggest other pathologies, including lichen sclerosus, inverse psoriasis, morphea, or candida intertrigo. Lichen sclerosus is most often

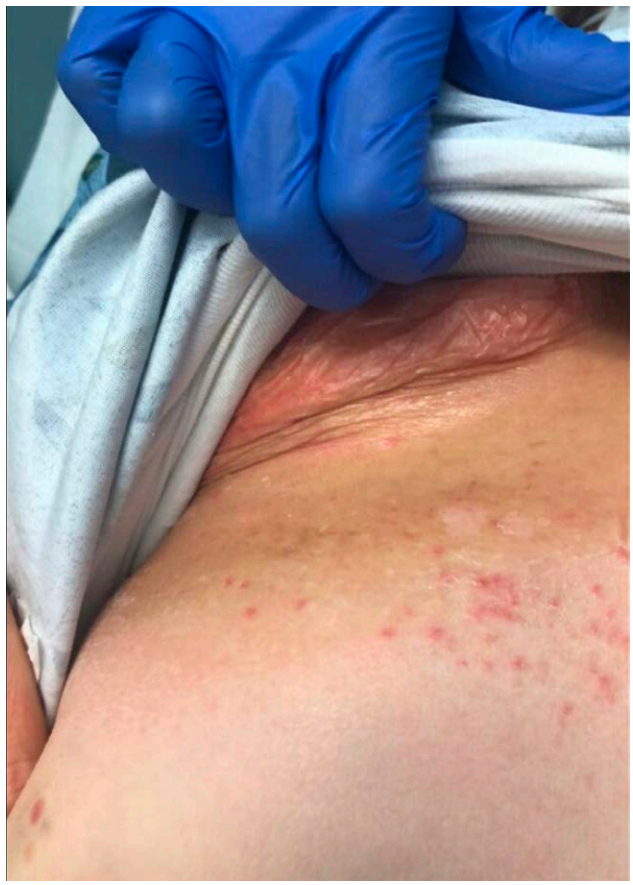

Fig. 1. Erythematous and tan plaques with peripheral keratotic reddish papules in the right inframammary fold.

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Prior case report oral presentation without questions by J. Alex Miles, MD, at UNC-Duke Conference on February 23, 2021.

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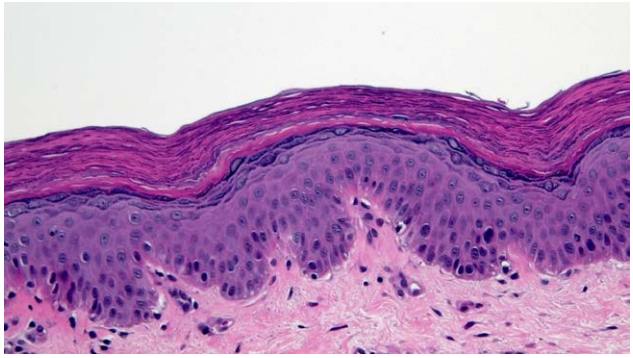

Fig. 2. H&E-stained punch biopsy from a representative lesion on the left abdomen, 200× magnification.

found in the genital area with wrinkled, ivory, shiny papules or plaques, and epidermal atrophy. It is thought to be an autoimmune disease of the skin leading to a loss of self-tolerance to one or more proteins, such as ECM-1. On histology, lichen sclerosus features compact hyperkeratosis in the stratum corneum, vacuolar interface changes, and band-like sub-epidermal hyalinization overlying a lymphocytic infiltrate of the upper dermis.

Another considered differential is inverse psoriasis, which can have the distribution seen in this patient. However, it often includes bright red, well-demarcated plaques that lack scale due to the moist nature of the folds. Epidermal hyperplasia, attributed to IL-17A and IL17F secretion, and elongation of rete ridges with acanthosis are common histology findings for psoriasis.[3]

Morphea can present with asymptomatic plaques that are tan centrally with peripheral erythema; however, induration is typically palpated within the plaques. Additionally, histology would have revealed spongiosis, foci of parakeratosis, epidermal acanthosis, and a superficial neutrophilic infiltrate. These changes are attributed to an elevation of T-lymphocytes producing IL-4 and TGF-beta, which increase the expression of extracellular matrix proteins and inhibit matrix degradation.

Finally, Candidal intertrigo may be considered, as the distribution in intertriginous sites is similar. However, a bright red coloration, not the tan, waxy nature of these plaques, would be expected; and mats of yeast intermingled with pseudohyphae would be seen on histology.

Question 2. What histopathological features would you most likely find on biopsy?

Response Options 2.

A. Spongiosis, foci of parakeratosis, epidermal acanthosis, and a superficial neutrophilic infiltrate, PAS-positive hyphal forms in the stratum corneum
B. Compact hyperkeratosis in the stratum corneum, vacuolar interface changes, band-like sub-epidermal hyalinization overlying a lymphocytic infiltrate of the upper dermis
C. Epidermal hyperplasia and elongation of rete ridges with acanthosis, confluent parakeratosis, and diminution of the granular layer
D. A thickened parakeratotic stratum corneum with retention of keratohyalin granules
E. Mats of yeasts intermingled with pseudohyphae

Correct Answer: D

Question 3. What is the proposed mechanism to explain the histopathology findings?

Response Options 3.

A. Defect in conversion of profilaggrin to filaggrin
B. Antibodies to extracellular matrix 1 (ECM-1)
C. Activation of T-lymphocytes resulting in production of IL-4 and TGF-beta
D. Secretion of IL-17A and IL-17F by Th17 cells resulting in proliferation of keratinocytes
E. Opportunistic growth and ability to transition between yeast and hyphal forms

Correct Answer: A

Conflicts of Interest

None.

References

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