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Superficial Epithelioma with Sebaceous Differentiation Presented as a Yellow Plaque

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Dear Editor:

Superficial epithelioma with sebaceous differentiation (SESD) is a distinct, rare, benign neoplasm first described by Rothko et al.¹ in 1980. In the literature, SESD has also been termed reticulated acanthoma with sebaceous differentiation², seborrheic keratosis with sebaceous differentiation, and acanthomatous superficial sebaceous hamartoma³.

A 54-year-old Korean woman presented with an asymptomatic yellow plaque, with well-defined borders on her

right anterior hairline; the plaque had initially appeared 13 years prior (Fig. 1). She had no underlying disease ex-



Fig. 1. A well-defined yellow plaque (arrow) was evident on the right side of the anterior hairline.

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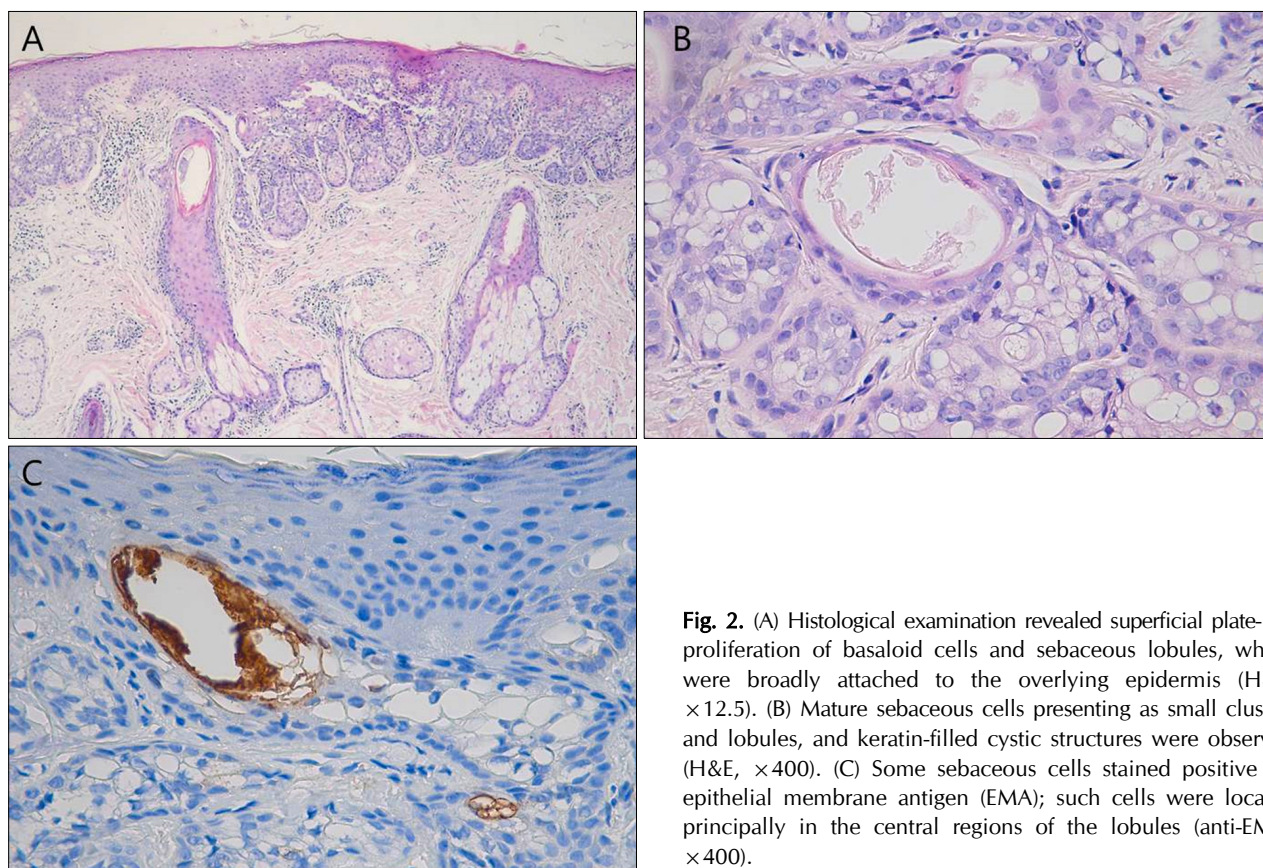


Fig. 2. (A) Histological examination revealed superficial plate-like proliferation of basaloid cells and sebaceous lobules, which were broadly attached to the overlying epidermis (H&E, $\times 12.5$). (B) Mature sebaceous cells presenting as small clusters and lobules, and keratin-filled cystic structures were observed (H&E, $\times 400$). (C) Some sebaceous cells stained positive for epithelial membrane antigen (EMA); such cells were located principally in the central regions of the lobules (anti-EMA, $\times 400$).

cept asthma and no family history of similar skin lesions. The initial clinical impression was a xanthoma and we performed a biopsy. Histological evaluation revealed superficial plate-like proliferation of sebaceous lobules that were attached, at multiple points, to the overlying epidermis (Fig. 2A). Many basaloid cells were evident between the sebaceous lobules of the superficial dermis. No cytological atypia was evident. Keratin-filled cystic structures and focal ductal differentiation were observed within the tumor mass (Fig. 2B). The tumor cells were positive for epithelial membrane antigen (EMA) (Fig. 2C) and pan cytokeratin, but negative for carcinoembryonic antigen (CEA). Immunohistochemical staining of nuclei for the DNA mismatch repair proteins MSH2 and MLH1 was positive.

SESD is a rare histologically distinct neoplasm. The clinical appearance varies; SESD may present as papules, nodules, and/or plaques that vary in hue (erythematous, tan-to-brown, hyperkeratotic, or pearly)⁴. Biopsy is essential to diagnose SESD. Our current case was clinically distinctive, presenting with yellow plaque simulating a xanthoma. SESD appearing as yellow papules or plaque has been but very rarely described.

Histopathologically, SESD exhibits superficial plate-like

proliferation of basaloid cells that are broadly attached to the overlying epidermis. Additional features include keratin-filled cysts, a well-formed ductal structure, and sebocytes of varying degrees of maturity presenting as single cells, cell clusters, and lobules³. The zones of sebaceous differentiation are located primarily at the lower poles of the lesions. Histological differential diagnoses include several other epithelial tumors exhibiting differentiation toward sebaceous cells, including nevus sebaceus, sebaceous hyperplasia, sebaceous adenoma, sebaceous epithelioma, and sebaceous carcinoma⁴. However, SESD can be easily differentiated by the superficial plate-like architectural pattern. The predominance of mature sebaceous lobules may explain the yellow hue of the lesion in our patient.

The histogenesis of SESD remains unclear. However, the sebaceous differentiation pattern suggests that the tumor originates from the pilosebaceous unit. The absence of stainable CEA within the proliferative epithelium argues against an eccrine derivation. In an immunohistochemical study of keratinocyte differentiation markers, the tumor showed homogenous staining for K14 but negative staining for K10, involucrin, or filaggrin in the basaloid tumor cells⁵. In addition, the secretory gland epithelium markers,

K7 and EMA, were detected in nests of sebocytes and ducts. Accordingly, the authors suggested that SESD tumor cells differentiated toward sebofollicular epithelium rather than toward interfollicular epidermis⁵. Several reports have found that SESD and Muir-Torre syndrome (MTS) are not associated¹⁻³. The fact that the nuclei of lesional cells stained uniformly for MSH-2 and MLH1, and the clinical history, support the lack of association of SESD and MTS in our present case.

We thus report a case of SESD in whom the lesion presented clinically as a yellow plaque. The histological features were typical of SESD, with a predominance of mature sebaceous lobules.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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