

LETTER TO THE EDITOR

Isolated Epidermolytic Acanthoma in a Renal Transplant Recipient

Ji-Hye Yang, M.D., Jae-Kyung Kim, M.D., Chong-Hyun Won, M.D., Sung-Eun Chang, M.D., Mi-Woo Lee, M.D., Jee-Ho Choi, M.D., Kee-Chan Moon, M.D.

Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Dear Editor:

Epidermolytic acanthoma (EA) was first reported in 1970 when six patients with solitary tumors clinically resembled warts were observed. Histologically these tumors showed epidermolytic hyperkeratosis (EH) without features of other epidermal tumors¹. The exact etiology of EA remains unknown, but immunosuppression is thought to be associated with EA although case report is lacking.

A 64-year-old man presented with a solitary papule on his scrotum. The lesion first appeared 7 years earlier and had gradually grown over time, but had not triggered symptoms such as itching or pain. This patient had no history of similar skin lesions. He had received a kidney transplant 7 years previously, and noted the scrotal lesion occurred immediately after starting an immunosuppressive regimen, consisted of tacrolimus (1 mg/day), azathioprine (50 mg/day), and deflazacort (6 mg/day). In addition, this patient also had type 2 diabetes and hypertension, both of which were controlled by medication. There was no family history of skin disease.

Physical examination showed a solitary, brownish, verrucous, and keratotic papule, 1 cm in diameter, on the left side of the scrotum (Fig. 1). Histologic examination of a shave biopsy specimen of the lesion showed orthokeratotic hyperkeratosis, papillomatosis, and acan-

thosis. Hypergranulosis was also noted, and the cells of the granular and upper spinous layers contained numerous, large, clear, perinuclear space with basophilic keratohyaline granules and eosinophilic materials (Fig. 2). As a result of these clinical and histological findings, the patient was diagnosed with isolated EA. The scrotal lesion resolved after ablative CO₂ laser treatment, with no evidence of recurrence during a 1-year follow-up period. EA is a solitary tumor that histologically shows EH without features of other epidermal tumors. EA is considered to be an acquired benign tumor, typically occurs in middle-aged adults. The lesions are characteristically brownish papules less than 1 cm in diameter with a verrucous surface. EA can be classified into solitary and disseminated subtypes, called isolated epidermolytic acanthoma (IEA) and disseminated epidermolytic acanthoma (DEA), respectively. Isolated lesions may occur anywhere on the body, but the disseminated subtype usually occurs on the back and the genitoscrotal area.



Fig. 1. A solitary, brown papule, measured 1 cm in diameter, with a verrucous surface on the scrotum.

Received May 31, 2010, Revised July 14, 2010, Accepted for publication July 14, 2010

Corresponding author: Mi-Woo Lee, M.D., Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, 388-1 Pungnap-dong, Songpa-gu, Seoul 138-736, Korea. Tel: 82-2-3010-3460, Fax: 82-2-486-7831, E-mail: miumiu@amc.seoul.kr

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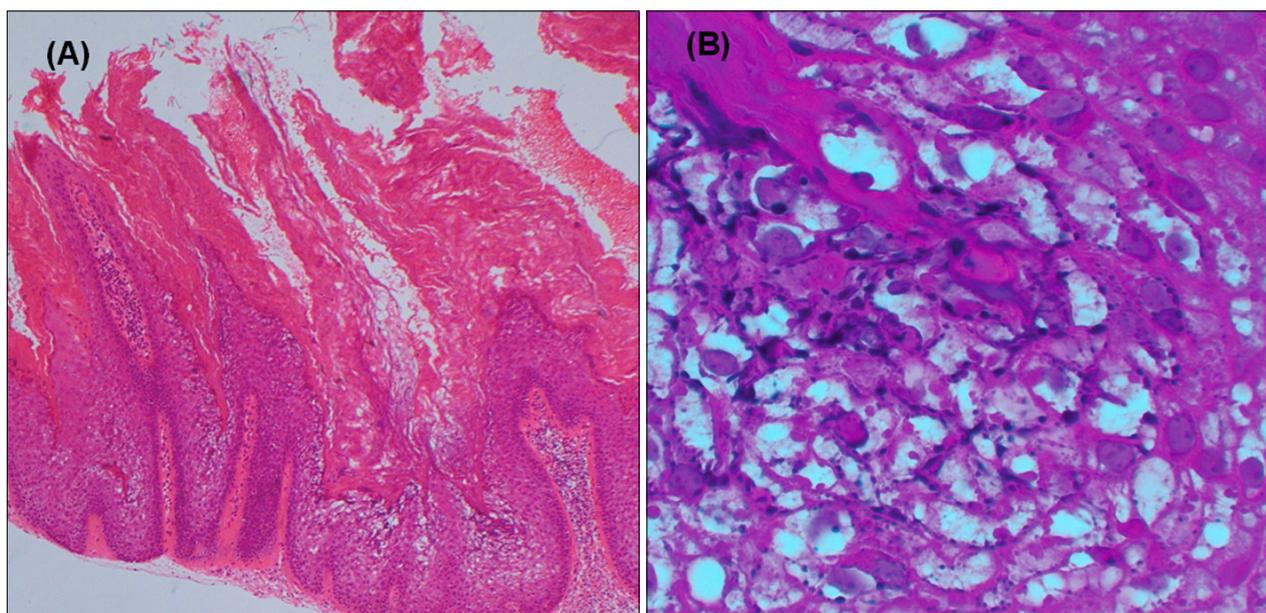


Fig. 2. Histologic examination of the scrotal lesion showing epidermolytic hyperkeratosis: vacuolar degeneration, increased keratohyaline-like bodies, hypergranulosis and compact hyperkeratosis (A: H&E, $\times 40$, B: H&E, $\times 400$).

EA is characterized histologically by EH, which consists of random-sized clear spaces around the nuclei in the stratum spinosum and granulosum, reticulated, lightly-staining material forming indistinct cellular boundaries, a markedly thickened granular zone containing an increased number of small and large, irregularly shaped basophilic keratohyaline-like bodies, and compact hyperkeratosis². EH may be congenital or acquired. Congenital EH includes bullous congenital ichthyosiform erythroderma, systemized epidermal nevus, and hereditary palmoplantar keratosis, whereas acquired EH include IEA and DEA.

The etiology of EA remains unknown and the differences between IEA and DEA in the pathogenesis are also unclear. Mutations in the keratin 1 and 10 genes have been observed in IEA³. EA may also be induced by exogenous factors, such as viruses or trauma. Human papillomavirus (HPV) was thought to be a cause of EA, because of the wart-like appearance, but these lesions have not been found to contain HPV DNA⁴. Immunosuppression has been known to be associated with the pathogenesis of DEA. Immunosuppression could inhibit immune surveillance, preventing abnormal keratinocyte clones from being recognized or removed. Alternatively, immunosuppression may allow latent abnormal keratinocyte clones to proliferate. To date, DEA with disseminated superficial porokeratosis and verruca vulgaris has been observed in a kidney transplant patient⁵, and DEA has also

been observed after PUVA⁶. Although our patient had a solitary lesion and the possibility of coincidental occurrence of IEA may exist, this case suggests that IEA could be associated with immunosuppression, because the skin lesion had appeared immediately after commencing immunosuppressive treatment. Further case reports and studies are needed to elucidate the relationship between immunosuppression and EA.

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