

growth of the tissues supplied.⁴ This may explain the appearance of granulomatous rosacea, a potentially immunologically mediated condition, in the location of prior nerve-related infection.^{1,5}

In the case presented, 1 or more of the proposed mechanisms may have contributed to the development of an isotopic response. Further investigation of the immunologic, vascular, and neurologic changes after viral infection may clarify the link between primary herpes virus infection and the appearance of secondary dermatoses.

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Funding sources: None.

Conflicts of interest: None declared.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the US Navy, US Army, or the Department of Defense.

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<http://dx.doi.org/10.1016/j.jaad.2014.09.040>

Large epidermal cleft formation in verrucous-keratotic malignant melanoma of the heel

To the Editor: Verrucous-keratotic malignant melanoma is an uncommon clinical variant of malignant melanoma that can be confused with benign lesions. We report a verrucous-keratotic melanoma on the heel that we had difficulty diagnosing accurately at the first examination.

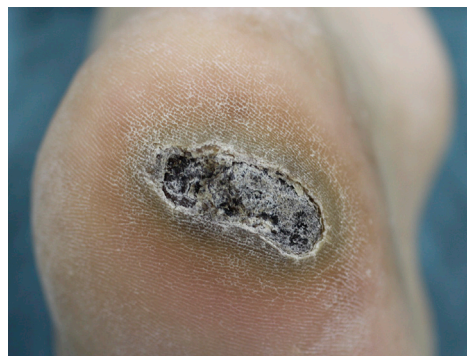


Fig 1. Verrucous-keratotic malignant melanoma. A verrucous hyperkeratotic plaque on the left heel, mimicking a plantar wart.

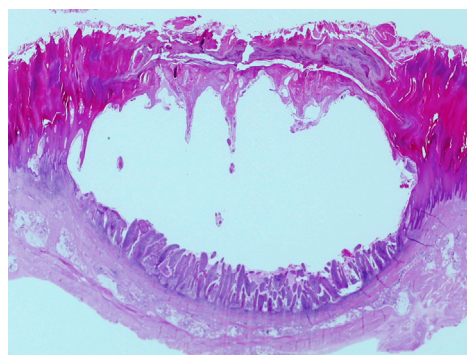


Fig 2. Verrucous-keratotic malignant melanoma. An extremely large cleft is seen at the center of the tumor.

A 65-year-old man came to our department with a pigmented hyperkeratotic lesion on his right heel. The blackish lesion had appeared a half-year before his first visit, and it had gradually enlarged.

Physical examination revealed a black, slightly elevated plaque 3.0 × 1.0 cm in size (Fig 1). The surface was ragged and covered with thick scales. Histopathologic examination of sample from shave biopsy revealed atypical melanocytes scattered throughout the horny layers. We totally excised the lesion with a wide margin. A histologic examination of the resected tumor showed prominent epidermal hyperplasia and elongated dermal papillae. Dissociation between melanoma cells in the basal layer and the suprabasal epidermis had caused a large cleft to form in the epidermis (Fig 2). Numerous atypical melanocytes were observed lining up in a row on the basement membrane.

We evaluated it as Clark level II, and sentinel lymph node biopsy was carried out. As a result, no metastasis was recognized in the sentinel lymph nodes.

Although verrucous-keratotic malignant melanoma is not included in the widely accepted

classification of Clark et al,¹ this case suggests that we should keep verrucous-keratotic malignant melanoma in mind as an important differential diagnosis for verrucous, hyperkeratotic lesions. In this case, the subtle perilesional clinical appearance of dark infiltration at the ridges might have been a clue to the diagnosis of melanoma.

The intercellular adhesion between melanoma cells is much weaker than that between normal keratinocytes, because melanocytes or melanoma cells do not have desmosomes. In addition, membranous expression of E-cadherin, which is responsible for melanocyte-keratinocyte adhesion, is sometimes decreased in melanoma progression.² Thinning of the epidermis with attenuation of the basal and suprabasal layers, described as “consumption of the epidermis,” is frequently seen in areas of direct contact between the epidermis and melanoma cell nests.³ Consumption of the epidermis occasionally results in the formation of a cleft separating the thin epidermis and melanoma cell nests.³ Braun-Falco et al⁴ reported that the clefts are a reliable diagnostic criterion for malignant melanoma. It has been suggested that the greater the Breslow depth, the greater the consumption of the epidermis in a melanoma lesion.⁵ However, the Breslow depth of the current lesion did not appear to be very great, although we were unable to measure it accurately because of the large size of the cleft. The presence of the verrucous architecture may be associated with the presence of epidermal consumption. In the current case, melanoma cells completely replaced the basal layer keratinocytes, forming only a single-layer lining on the basement membrane. This characteristic infiltration pattern of melanoma cells with fragile cell adhesion might have led to the large cleft with features mimicking the tombstone appearance of pemphigus vulgaris, although in the current lesion, the tombstones were melanoma cells instead of the basal keratinocyte tombstones of pemphigus vulgaris lesions.

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Funding sources: None.

Conflicts of interest: None declared.

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<http://dx.doi.org/10.1016/j.jaad.2014.09.044>

Acral localized acquired cutis laxa: Report of a case associated with inflammatory arthritis

To the Editor: Acral localized acquired cutis laxa (ALACL) is a distinct variant of acquired cutis laxa characterized by loose redundant skin primarily confined to the distal extremities. It is often associated with signs or symptoms of a preceding inflammatory dermatosis, such as urticaria, erythema, swelling, or pruritus. Disease associations include multiple myeloma, idiopathic urticaria, Raynaud phenomenon, and rheumatoid arthritis. We report a case associated with inflammatory arthritis in childhood.

A 41-year-old woman with a history of polyarticular juvenile idiopathic arthritis diagnosed at age 3 presented with asymptomatic loose, pendulous skin confined to her volar finger pads and toes. She noted the onset of loose skin at age 6 and described ill-defined erythema, warmth, and pruritus of her hands that preceded the development of the skin changes. The degree of skin laxity remained stable since childhood. There was no history of urticaria. Family history was relevant for a paternal grandfather with rheumatoid arthritis.

On examination, there was loose redundant skin on the finger pads bilaterally that gave them a flat rounded appearance (Fig 1). The volar finger pads were soft on palpation and the skin remained depressed when pressure was applied, giving them an appearance that has been described as “chewing gum-like.”¹ The plantar surface of the toes had similar skin changes, although less striking. Facial appearance was normal and did not show advanced aging.