continued exposure and seasonal variation. In untreated cases, it may gradually spread and eventually progress to erythroderma over variable periods of time. Patch testing helps to determine the cause of the contact dermatitis.

In this patient, we noted characteristic sparing of major skin folds of the abdomen and axillary regions, producing the deck-chair sign. This is a classic sign for PEO, which was first reported by Ofujii et al in 1984 to describe 4 cases of papulo-erythroderma with flat-topped papules that became generalized erythrodermic plaques with characteristic sparing of skin folds and flexures. Although deck-chair sign is considered pathognomonic of PEO, it has also been observed in angioimmunoblastic lymphoma, cutaneous Waldenstrom macroglobulinemia, and acanthosis nigricans. In a study of 90 patients with erythroderma, Pal and Haroon observed deck-chair sign in 5.5% of the cases. This shows that the deck-chair sign is typical but not pathognomonic of PEO, and it can be seen in a heterogeneous group of cutaneous disorders, including parthenium dermatitis.

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Janeway Lesions and Splinter Hemorrhages in a Patient With Eosinophilic Endomyocarditis

Janeway lesions and splinter hemorrhages are a cutaneous sign of infective endocarditis (IE). Janeway lesions are nontender, erythematous or violaceous maculae on the palms and/or soles that are also found in several noninfective diseases, such as systemic lupus erythematosus and myxoma. Splinter hemorrhages on multiple nails without any obvious trauma are indicative of systemic causes: not only IE, but antiphospholipid syndrome, vasculitis, and treatment with systemic tyrosine kinase inhibitors or hemodialysis. However, to our knowledge, Janeway lesions or splinter hemorrhages due to noninfective endocarditis, such as eosinophilic endomyocarditis, have not been reported.

Herein, we present a case of both Janeway lesions and splinter hemorrhages that arose in eosinophilic endomyocarditis and faded away as the underlying disease improved.

Report of a Case | A man in his 30s was referred to our department for asymptomatic skin lesions of the fingernails and toes that arose approximately 1 month after diagnosis with multiple cerebral infarctions and cardiac failure. His consciousness and vital signs were normal. Nontender, small, and erythematous maculae on the toes, suggestive of Janeway lesions, were observed (Figure 1A). Linear reddish-brown streaks were noted on the distal portions of the nail plates of all fingers, which were consistent with splinter hemorrhages (Figure 1B). There was no history of direct trauma to the fingertips or toes.

A skin biopsy specimen obtained from the macular lesion on the right fifth toe showed thrombi or thromboemboli within the blood vessels in the dermis (Figure 2) without any abscess formation, which is seen in Janeway lesions in IE.3 Blood tests on 2 occasions in the course of 2 weeks revealed eosinophilia (eosinophil counts, 15 340/μL and 7520/μL) despite the absence of other causes of secondary eosinophilia. These findings fit the definition of hypereosinophilic syndrome proposed by Simon et al in 2010: eosinophilia found on more than 1 occasion and exclusion of secondary eosinophilia for the diagnosis.

Electrocardiograms, which initially showed a small negative T-wave in leads II, III, and aVF that became flat in subsequent days, suggested cardiomyopathy. Findings of diagnostic imaging studies with contrast-enhanced computed tomography, cardiac magnetic resonance imaging, and transthoracic echocardiography suggested thrombi in the right ventricle, subendocardial late and poor gadolinium enhancement, and noncompaction of the left ventricle with the hypertrophied wall, all of which are consistent with eosinophilic myocarditis rather than IE.5 A diagnosis of IE was excluded because the patient had no sign of infection (ie, no fever, negative blood cultures, and no vegetation on cardiac valves).

Based on these detailed clinical evaluations, we diagnosed the patient as having Janeway lesions and splinter hemorrhages associated with eosinophilic endomyocarditis secondary to hypereosinophilic syndrome, also known as Loeffler endomyocarditis. With systemic steroids and thrombolytic therapy, the patient’s general condition, eosinophilia, and abnormal findings on imaging studies were mostly improved 3 months later. As he recovered from the underlying disease, the Janeway lesions and the splinter hemorrhages eventually completely disappeared (Figure 1C and D).

Discussion | To our knowledge, this is the first reported case of Janeway lesions or splinter hemorrhages in eosinophilic endomyocarditis. Both cutaneous manifestations are results of distal cutaneous vascular insufficiency following multiple embolisms, which can
occur from any source. We speculate that the source of the embolisms in the present case was intraventricular thrombus. The present case has reminded us that noninfective endocarditis should be included in the differential diagnosis of Janeway lesions and splinter hemorrhages. In addition, these cutaneous signs may indicate how well the treatment for the underlying disease works.

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