

Study concept and design: Diven, Allred, Reed.

Acquisition, analysis, or interpretation of data: Bartenstein, Diven, Reed.

Drafting of the manuscript: Bartenstein, Diven, Allred.

Critical revision of the manuscript for important intellectual content: Bartenstein, Diven, Reed.

Statistical analysis: Bartenstein, Reed.

Administrative, technical, or material support: Reed.

Study supervision: Diven, Reed.

Conflict of Interest Disclosures: None reported.

Previous Presentation: This research was presented at the Texas Dermatological Society 2014 Annual Fall Meeting; October 24-25, 2014; Bastrop, Texas.

1. Boniol M, Autier P, Boyle P, Gandini S. Cutaneous melanoma attributable to sunbed use: systematic review and meta-analysis. *BMJ*. 2012;345:e4757.
2. Wehner MR, Shive ML, Chren MM, Han J, Qureshi AA, Linos E. Indoor tanning and non-melanoma skin cancer: systematic review and meta-analysis. *BMJ*. 2012;345:e5909.
3. Wehner MR, Chren MM, Nameth D, et al. International prevalence of indoor tanning: a systematic review and meta-analysis. *JAMA Dermatol*. 2014;150(4):390-400.
4. Pagoto SL, Lemon SC, Oleski JL, et al. Availability of tanning beds on US college campuses. *JAMA Dermatol*. 2015;151(1):59-63.
5. Texas Department of State Health Services. Tanning Facility Updates. <https://www.dshs.state.tx.us/dmd/tanning.shtm>. Accessed August 16, 2014.
6. Forster JL, Lazovich D, Hickle A, Sorensen G, Demierre MF. Compliance with restrictions on sale of indoor tanning sessions to youth in Minnesota and Massachusetts. *J Am Acad Dermatol*. 2006;55(6):962-967.

OBSERVATION

Parthenium Dermatitis With Deck-Chair Sign

Deck-chair sign is a clinical pattern observed in patients with erythroderma characterized by a selective sparing of skin folds like axillary, inguinal, submammary, and flexures, classically described with papuloerythroderma of Ofuji (PEO).¹ Herein we describe a case of parthenium dermatitis in which deck-chair sign was noted.

Report of a Case | A 75-year-old man from India, a farmer by occupation, presented to the dermatology department for evaluation of generalized itchy red skin eruptions present for 1 month. He had 5-year history of recurrent episodes of pruritic nonexudative erythematous papules on the face, neck, and hands, symptoms worsening in the summer months. He also had a history of regular contact with parthenium during his work. Patient did not have a personal or family history of atopy. There were no constitutional or systemic symptoms. Physical examination revealed widely spread erythematous papules, many of which were coalescing to form plaques. Well-demarcated sparing of abdominal folds, preaxillary folds, and genital areas was noted, producing deck-chair sign (**Figure**). Findings of systemic examination were unremarkable.

Patch testing with 15% parthenium showed a 2+ positive reaction to parthenium. He was treated with topical and systemic steroids on a tapering dose along with sunscreens and emollients. Oral methylprednisolone was prescribed, 32 mg/d, which was tapered by 4 mg every week. The prescription was later switched to azathioprine, 50 mg/d. Strict photoprotective measures were advised along with regular use of sunscreens.

Discussion | *Parthenium hysterophorus* can produce a spectrum of clinical patterns. The dermatitis usually presents as itchy, erythematous, papules and plaques on exposed areas of the body like the face, including upper eyelids, side of neck, the "V" of the upper chest, flexures of the forearms, and cubital and popliteal fossae. Parthenium dermatitis commonly begins as an airborne contact dermatitis pattern. Other patterns include chronic actinic dermatitislike, seborrheic dermatitis, prurigo nodularislike, photosensitive lichenoid eruption, and hands-and-feet dermatitis patterns.² Repeated exacerbations are common due to con-

Figure. A Case of Parthenium Dermatitis



A, Widespread erythematous papules and plaques on the chest and abdomen. B, Sparing of the abdominal and preaxillary skin folds, producing the deck-chair sign.

tinued 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 Ofuji et al¹ 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 pathognomic of PEO, it has also been observed in angioimmunoblastic lymphoma, cutaneous Waldenstrom macroglobulinemia, and acanthosis nigricans.³⁻⁵ In a study of 90 patients with erythroderma, Pal and Haroon⁶ observed deck-chair sign in 5.5% of the cases. This shows that the deck-chair sign is typical but not pathognomic of PEO, and it can be seen in a heterogeneous group of cutaneous disorders, including parthenium dermatitis.

Sathish Pai, MD
Shricharith Shetty, MD
Raghavendra Rao, MD

Author Affiliations: Department of Dermatology, Kasturba Medical College, Manipal University, Manipal, Karnataka, India.

Corresponding Author: Shricharith Shetty, MD, Department of Dermatology, OPD No. 21, Kasturba Medical College Hospital, Manipal, Karnataka, India 576104 (drshricharith@gmail.com).

Published Online: April 29, 2015. doi:10.1001/jamadermatol.2015.0494.

Conflict of Interest Disclosures: None reported.

1. Ofuji S, Furukawa F, Miyachi Y, Ohno S. Papuloerythroderma. *Dermatologica*. 1984;169(3):125-130.

2. Lakshmi C, Srinivas C. Parthenium the terminator: an update. *Indian Dermatol Online J*. 2012;3(2):89-100.

3. Ferran M, Gallardo F, Baena V, Ferrer A, Florensa L, Pujol RM. The 'deck chair sign' in specific cutaneous involvement by angioimmunoblastic T cell lymphoma. *Dermatology*. 2006;213(1):50-52.

4. Autier J, Buffet M, Pinquier L, et al. Cutaneous Waldenstrom's macroglobulinemia with "deck-chair" sign treated with cyclophosphamide. *J Am Acad Dermatol*. 2005;52(2)(suppl 1):45-47.

5. Murao K, Sadamoto Y, Kubo Y, Arase S. Generalized malignant acanthosis nigricans with "deck-chair sign". *Int J Dermatol*. 2013;52(3):377-378.

6. Pal S, Haroon TS. Erythroderma: a clinico-etiological study of 90 cases. *Int J Dermatol*. 1998;37(2):104-107.

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.¹ 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.² However, to our knowledge, Janeway lesions or splinter hemorrhages due to noninfective endomyocarditis, 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/ μ L and 7520/ μ L) despite the absence of other causes of secondary eosinophilia. These findings fit the definition of *hypereosinophilic syndrome* proposed by Simon et al⁴ 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