

Department of Dermatology, Radboud University
Nijmegen Medical Center, Nijmegen, The
Netherlands

Funding sources: None.

Conflicts of interest: None declared.

Correspondence to: H. Jorn Bovenschen, MD, PhD,
Department of Dermatology, Radboud University
Nijmegen Medical Center, P.O. Box 9101,
NL- 6500 HB Nijmegen, The Netherlands

E-mail: j.bovenschen@derma.umcn.nl

REFERENCES

1. Garnier G. Vulvite érythémateuse circonscrite bénigne a type erythroplastique. Bull Soc Fr Dermatol Siphiligr 1954;61: 102-4.
2. Ee HL, Yosipovitch G, Chan R, Ong BH. Resolution of vulvitis circumscripta plasmacellularis with topical imiquimod: two case reports. Br J Dermatol 2003;149:638-41.
3. Frega A, Rech F, French D. Imiquimod treatment of vulvitis circumscripta plasmacellularis. Int J Gynaecol Obstet 2006;95:161-2.
4. Bhaumik J. Vulvitis circumscripta plasmacellularis: success with a modified treatment regimen using imiquimod. J Obstet Gynaecol 2006;26:72.
5. Novak N, Yu CF, Bieber T, Allam JP. Toll-like receptor 7 agonists and the skin. Drug News Perspect 2008;21:158-65.

doi:10.1016/j.jaad.2009.08.018

Dermal hypersensitivity reaction as a prodrome to Hodgkin lymphoma

To the Editor: The definition of dermal hypersensitivity reaction (DHR) as a histopathologic diagnosis with a specific clinical correlate is poorly established. Clinical entities often associated with this histologic pattern vary widely: urticaria, drug reactions, arthropod bite reactions (papular urticaria), spongiotic (eczematous) dermatitis, reactions associated with systemic malignancy, pruritic urticarial papules and plaques of pregnancy, bullous pemphigoid, and scabies.¹ Callen et al² have previously reported the association between adult onset intractable eczema and noncutaneous lymphoma. We present a case whereby recalcitrant pruritic urticarial dermatitis, a histopathologic finding of DHR, and review of systems prompted screening for malignancy beyond age appropriate recommendations which resulted in the diagnosis of Hodgkin lymphoma (HL).

A 71-year-old man presented with a 2- to 3-month history of a progressively worsening generalized urticarial dermatitis, primarily erythematous papules and wheals with scant overlapping scaling and xerosis, which was not controlled with systemic steroids. He was noted to be dermatographic and his trunk and extremities were involved. Crusted excoriations overlapped these areas (Fig 1). His

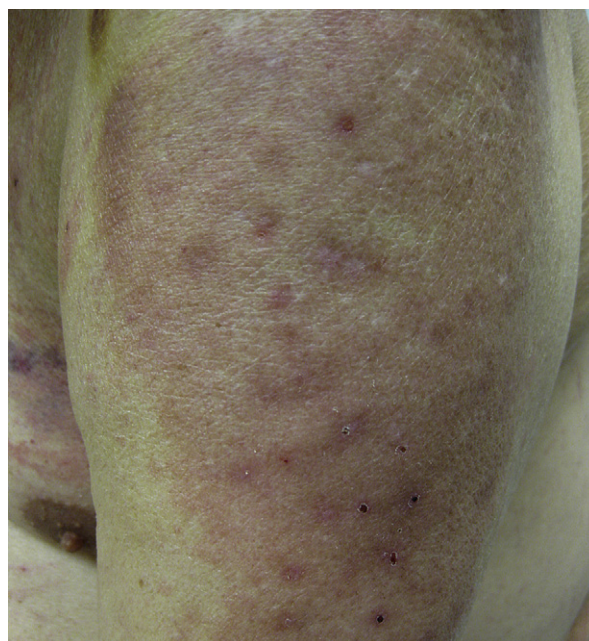


Fig 1. An urticarial dermatitic process with excoriation on the upper left arm.

medications on presentation were unchanged, and his only complaint was increasing lower back pain, which he attributed to his spinal stenosis.

A complete chemistry panel and blood cell count were within normal limits. A recent chest radiograph revealed no masses and no lymphadenopathy. He was prescribed emollients, combination antihistamines, and clobetasol ointment. Hematoxylin–eosin (Fig 2) staining and direct immunofluorescence (DIF) studies were performed on biopsy specimens; mild spongiosis, a perivascular lymphocytic infiltrate, edema of the dermis, and numerous eosinophils were identified, periodic acid–Schiff staining was negative for fungal organisms, and DIF studies were entirely negative. Overall, the pattern was consistent with a DHR.

On follow-up after 4 weeks, he was noted to have lack of improvement and a new complaint of night sweats, and increased doses of antihistamines and the addition of a leukotriene inhibitor were instituted as a steroid-sparing alternative. Additional studies, including thyroid stimulating hormone, antithyroglobulin and antithyroperoxidase antibodies, tryptase, angiotensin sensitivity test, alanine aminotransferase, alkaline phosphatase, and serum protein electrophoresis with immunofixation were normal. A computed tomographic scan was also obtained in response to the patient's recalcitrant dermatitis and new review of systems, which revealed a soft tissue pelvic mass and lymphadenopathy. Lymph node biopsies were performed, revealing the presence of Reed–Sternberg cells, which are pathognomonic for HL. He underwent

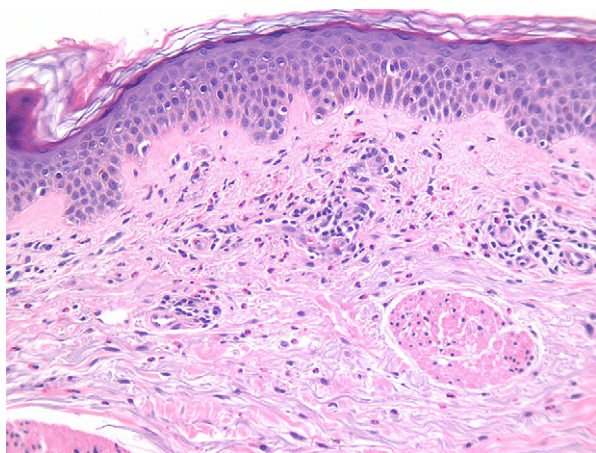


Fig 2. A skin punch biopsy specimen revealed mild spongiosis with a perivascular lymphocytic infiltrate, dermal edema, and numerous eosinophils. (Hematoxylin–eosin stain; original magnification: $\times 40$.)

chemotherapy and within 1 month (2 cycles) was noted to have complete resolution of his skin lesions.

Pruritus is a common nonspecific symptom in HL, often preceding the diagnosis by 5 years. Here we present a case in which pruritus and a treatment resistant urticarial dermatitis were the most salient presenting features of HL and histopathologically correlated to DHR. Though the clinical presentation of urticaria has been described as a nonspecific manifestation of HL,³ perhaps it is a specific sign when seen in combination with DHR and HL.¹

Nonspecific cutaneous findings of HL can be seen in up to 50% of patients with HL,⁴ and they include eczema,² hyperpigmentation, prurigo, ichthyosis, vasculitis, bullous eruptions, erythema nodosum, and generalized hyperhidrosis.^{5,6} In contrast, the frequency of specific, cutaneous HL is very low, ranging from 0.5%⁶ to 7.5%,⁷ and includes bullae, painless nodules that often ulcerate, papules, papulosquamous eruptions, and ulcers. Histologically, cutaneous HL reveals atypical cells, which are often positive for CD30 and CD15, and the presence of Reed–Sternberg cells. Our patient's histopathology lacked cutaneous Reed–Sternberg cells or any lymphocytic features indicating malignancy.

Although dermatologic findings rarely justify the use of computed tomographic scans as screening options for malignancies, the combination of failed conventional therapy, the unique histologic finding of DHR, nightsweats, and the clinical presentation of recalcitrant urticarial dermatitis prompted a higher clinical suspicion of malignancy. Expanding our differential for cutaneous findings of HL, both nonspecific and specific, can contribute to early detection. Our case demonstrates the need for increased

tenacity in pursuing a diagnosis, even in the face of rather mundane, nonspecific findings.

John Vu, MD, PhD, Jonban Ho, MD, and Joseph C. English III, MD

University of Pittsburgh Department of Dermatology, Pittsburgh, Pennsylvania

Funding sources: None.

Conflicts of interest: None declared.

Correspondence to: Joseph C. English III, MD, Associate Professor of Dermatology, Clinical Vice Chairman of Quality and Innovation, Director of Teledermatology, University of Pittsburgh Department of Dermatology, 200 Lothrop St, Ste 3825 Presby South Tower, Pittsburgh, PA 15044.

E-mail: englishjc@upmc.edu

REFERENCES

1. Fung MA. The clinical and histopathologic spectrum of "dermal hypersensitivity reactions," a nonspecific histologic diagnosis that is not very useful in clinical practice, and the concept of a "dermal hypersensitivity reaction pattern". *J Am Acad Dermatol* 2002;47:898-907.
2. Callen JP, Bernardi DM, Clark RA, Weber DA. Adult-onset recalcitrant eczema: a marker of noncutaneous lymphoma or leukemia. *J Am Acad Dermatol* 2000;43(2 pt 1):207-10.
3. Cavalli F. Rare syndromes in Hodgkin's disease. *Ann Oncol* 1998;9(suppl 5):109-13.
4. Szur L, Harrison CV, Levene GM, Samman PD. Primary cutaneous Hodgkin's disease. *Lancet* 1970;1:1016-20.
5. Rubenstein M, Duvic M. Cutaneous manifestations of Hodgkin's disease. *Int J Dermatol* 2006;45:251-6.
6. Smith JL Jr, Butler JJ. Skin involvement in Hodgkin's disease. *Cancer* 1980;45:354-61.
7. Benninghoff DL, Medina A, Alexander LL, Camiel MR. The mode of spread of Hodgkin's disease to the skin. *Cancer* 1970;26:1135-40.

doi:10.1016/j.jaad.2010.01.057

Imatinib mesylate–induced pseudoporphyria

To the Editor: A 63-year-old white woman with a history of chronic myelogenous leukemia (CML) was referred to our clinic for evaluation of a 19-month history of worsening blistering and skin fragility of her extremities (Fig 1). A previous biopsy specimen revealed a paucinflamatory subepidermal split consistent with porphyria cutanea tarda (PCT) and negative direct immunofluorescence (DIF) with normal serum and urine porphyrin studies. Our biopsy of a characteristic lesion was consistent with PCT, with a negative DIF and repeat porphyrin studies that again were normal. Discontinuation of her thrice weekly naproxen and daily estrogen supplement for