Purpura Morphology, Setting May Indicate Cause

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CHICAGO — The morphology and distribution of purpura can help to narrow a differential diagnosis, Warren W. Piette, M.D., said at the American Academy of Dermatology’s Academy 2005 meeting.

Purpura is the result of one of three hemorhagic mechanisms: simple, inflammatory, and occlusive. Purpura distribution further focuses the diagnosis, particularly for inflammatory hemorrhage.

“I’d like to resurrect a portion of our skills [of visual diagnosis] … and meld them with the new technologies,” said Dr. Piette, a dermatologist at the John H. Stroger, Jr., Hospital of Cook County in Chicago.

Simple hemorrhages at sites of minor trauma are among the most common causes of purpura. These lesions tend to be nonpalpable, ecchymotic, and nonblanching, with a linear or geometric morphology. Underlying conditions include problems with platelet function, the coagulation cascade, or poor dermal support typically resulting from chronic corticosteroid use or sun damage.

Petechial purpura, measuring 4 mm or less, also results from simple hemorrhage. These are macular and nonblanchable, and typically are associated with a platelet count of 10,000/mm³ to 20,000/mm³.

Inflammatory hemorrhage, in contrast, is associated with palpable purpura that is more problematic to diagnose. Such lesions are typically erythematous and may or may not be vasculitic. Conditions resulting in inflammatory hemorrhage include leukocytoclastic or necrotizing vasculitis, although a few nonvasculitic conditions can be associated with palpable purpura and include chronic pigmented purpura, pityriasis lichenoides et varioliformis acuta (PLEVA), lymphomatoid papulosis, and erythema multiforme.

Vasculitic purpura can be differentiated, based on morphology, as purpura triggered by immune complexes and purpura resulting from pauci-immune mechanisms, he said. Immune complex vasculitis usually has a largely dependent distribution, and the pauci-immune variety has a random distribution.

In vasculitic purpura, it’s important to determine the variety of immune complex vasculitis, because IgA-associated vasculitis is more likely than IgG- or IgM-predominant vasculitis to persist, recur, ulcerate, and be associated with systemic disease, he said. Three varieties of renal disease—two of which can lead to renal failure—are associated with IgA vasculitis.

There are eight major categories of occlusive or ischemic hemorrhages and up to 50 possible differential diagnoses associated with them. The most important of the major categories are the cutaneous diseases. In three identical clinical scenarios with variable morphology, “the patient’s telling us, ‘don’t look at my lab [values], look at my skin,’” he said.

The first patient’s lesions were petechial size, but were palpable and partially blanchable. Palpable purpura with blanching negates the diagnosis of simple petechiae, and implicates inflammatory hemorrhage. The patient proved to have septic vasculitis associated with meningococcal disease. The diagnosis was confirmed with findings on biopsy of leukocytoclastic vasculitis and spotty fibrin deposition.

The second patient had simple hemorrhage with ecchymotic-sized purpura. The differential diagnosis needed to consider a range of platelet problems, including disseminated intravascular coagulation and multiple cascade coagulation abnormalities. In this case, purpura was the result of a bleeding disorder from a clotting factor abnormality or fibrinogen consumption below critical levels.

The last patient had noninflammatory retiform purpura as a result of occlusion. Laboratory or histologic confirmation can take hours to days in such patients, he said, while the diagnosis can be strongly suggested by the morphology of the lesion and the clinical setting.

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