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Papular Urticaria

Adam S. Stibich, MD, Newark, New Jersey

Robert A. Schwartz, MD, MPH, Newark, New Jersey

Papular urticaria is a common and often distressing childhood disorder manifested by chronic or recurrent papules caused by a hypersensitivity reaction to the bites of mosquitoes, fleas, bedbugs, and other insects. Individual papules may surround a wheal and often have a central punctum. The histopathology of papular urticaria consists of mild subepidermal edema, extravasation of erythrocytes, interstitial eosinophils, and exocytosis of lymphocytes. Papular urticaria may represent a clinical challenge, particularly during spring and summer months.

Arthropods have important clinical implications for the clinician. Their bites and stings may induce allergic reactions, ranging from little more than annoying to life threatening. Many arthropod products are capable of inciting allergic responses in sensitized individuals. One such reaction is papular urticaria or lichen urticatus. Papular urticaria was originally described in 1813 by Bateman.¹

Clinical Manifestations

Papular urticaria (Figure) is a common childhood disease that is manifested by a chronic or recurrent papular eruption caused by a sensitivity reaction to the bites of mosquitoes, fleas, bedbugs, and other insects.^{2,3} Although cases have been described in infants as young as 2 weeks, it is seen primarily in children between 2 and 7 years of age, particularly in those with a history of atopic dermatitis. The disorder usually appears in late spring and summer. Papules may occur on any part of the body but tend to be grouped in clusters on exposed areas, particularly the extensor surfaces of the extremities.^{4,5} These papules may appear to a lesser extent on the face and neck, trunk, thighs, and buttocks and generally spare the genital, perianal, and axillary regions.^{2,4} However, location depends on the arthropod involved.^{6,7}



Papular urticaria on the legs.

Individual lesions are seen as 3- to 10-mm, firm urticated papules, often with a central punctum. They may be excoriated, lichenified, or secondarily infected with crust formation.^{4,8,9} The lesions recur in crops and all stages of development; regression may be noted. Most lesions persist for 2 to 10 days and, after resolution, may result in temporary postinflammatory erythema or pigmentation. If exposure to the parasite is allowed to continue, the attacks may persist for an average of 3 to 4 years, perennially or recurring seasonally; occasionally they may persist into adolescence or adulthood.^{2,10,11}

Pathology

Histologic features of typical papular urticaria can be classified into 4 variants: lymphocytic, eosinophilic, neutrophilic, and mixed cellular.¹² A localized perivascular infiltrate with lymphocytes, histiocytes, eosinophils, and mast cells in the upper dermis; variable edema between collagen fibers; and a light scattering of eosinophils and mast cells away from vessels in the upper and mid dermis are

Drs. Stibich and Schwartz are from Dermatology, New Jersey Medical School, Newark.

Reprints: Robert A. Schwartz, MD, MPH, Dermatology, New Jersey Medical School, 185 S Orange Ave, Newark, NJ 07103-2714.

evident.¹³ Spongiosis with exocytosis and vesicle formation is present in the epidermis, overlying the most marked and superficial perivascular infiltrate. In older excoriated papules, the histologic changes are usually modified by the effects of scratching. In addition, the development of epidermal necrosis, crusting, and a dermal infiltrate with neutrophils and more abundant lymphocytes makes histologic diagnosis more difficult. Whenever possible, a biopsy should be performed on newly formed lesions that are not excoriated.^{4,14,15}

Immunohistochemistry results reveal abundant T lymphocytes (CD45RO, CD3) and macrophages (CD68). B lymphocytes (CD20) and dendritic antigen-presenting cells (S100) are absent. In a study of 30 patients with papular urticaria, direct immunofluorescence staining for deposition of IgA, IgG, IgM, C3, and fibrin was negative in 100% of the patients studied.¹² In 3 patients studied by Heng et al,¹⁶ immunofluorescence tests in fresh lesions of papular urticaria had various amounts of particularly granular deposits of C1q, C3, and IgM in dermal blood vessel walls.

Pathogenesis and Etiology

Papular urticaria may be a result of a Type I hypersensitivity reaction in response to a hematogenously disseminated antigen deposited by an arthropod bite in a sensitized patient.¹² Patients with the condition must be previously sensitized to parasitic antigens. This presumably explains why papular urticaria rarely occurs in neonates. Most infants are not sufficiently exposed to biting insects to develop hypersensitivity. Experiments have shown that with repeated exposure to antigen, hyposensitization takes place, and the child "outgrows" the condition.¹⁷ The adolescent then responds to an insect bite in the way most adults do: a transient wheal develops, but no persistent papule forms.

Differential Diagnosis

The histopathologic features of papular urticaria are not specific and thus make for a broad differential diagnosis. Some conditions with characteristics similar to those of papular urticaria include papular forms of atopic dermatitis, allergic contact dermatitis, drug-induced reaction, id reaction, miliaria rubra, papulovesicular polymorphous light eruption, papular acrodermatitis of childhood (Gianotti-Crosti syndrome),^{18,19} linear IgA bullous dermatosis, and pityriasis lichenoides et varioliformis acuta.^{12,20-21} Similarly, papular urticaria and insect bites are identical histopathologically.² Some distinguishing features in an arthropod bite are a necrotizing nidus

and flame figures, which may be present. Intra-epidermal vesicles (large centrally and smaller peripherally) are not uncommon.¹²

Treatment

The most effective treatment for papular urticaria is identification and removal of its cause. In some instances, this may be difficult if not impossible, and patients should be treated symptomatically while the source of the rash is sought. We advocate mild topical steroids and systemic antihistamines for control of pruritus. Secondary infection warrants the use of topical or oral antibiotics. Disinfecting all pets and fumigating the home may produce a dramatic cure.²²⁻²⁵ Insect repellent should be applied to the skin when outdoors.²⁶

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