

Clinical Image

A Sudden-Onset, Diffuse, Purpuric Rash in a Teenager

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CLINICAL IMAGE

A fifteen-year-old male presented with a purpuric rash of one-week duration. The rash began on his abdomen and spread to his extremities within two days. The patient received a course of prednisone; however, the rash progressed. He denied any recent medication changes, vaccinations, or arthropod exposure. Physical exam was significant for 3-5 mm monomorphic non-blanching purpuric papules on his abdomen, groin, and buttocks, with fewer lesions on his trunk and extremities (Figures 1, 2). A biopsy of a lesion was taken from the right hip. Histopathology demonstrated a superficial lymphocytic perivascular infiltrate, erythrocyte extravasation in the superficial dermis, and parakeratosis over foci of spongiosis. These findings were consistent with purpuric pityriasis rosea (PR).

Pityriasis rosea (PR) is a self-limiting exanthem that usually presents between the ages of 10 to 35 and has a female predominance. It begins with a solitary papule or plaque, referred to as a “herald” patch, with an erythematous or psoriasiform appearance. The herald patch either grows in circumference or fades, while smaller lesions develop within one to three weeks after the herald patch’s appearance. These smaller lesions follow Langer’s skin lines, resulting in a “Christmas tree” distribution. The rash may be pruritic. The lesions commonly resolve within 6 weeks [1]. Several studies have demonstrated an association between the onset of PR and systemic reactivation of human herpesvirus-6 (HHV-6) and HHV-7 in some patients [2]. In others, PR has also been associated with recent upper respiratory tract infections and drugs, including barbituates, captopril, and isotretinoin [3]. PR is typically treated symptomatically, although acyclovir and erythromycin have both been used to shorten the course of the disease [1].

The histopathologic characteristics of PR typically include focal parakeratosis and spongiosis (Figure 3) [1]. Lymphocytic exocytosis, erythrocyte extravasation, acanthosis, and a thinned granular layer may also be present. The extravasated erythrocytes in purpuric PR can be found between rete ridges without associated capillaritis or vasculitis [3].

Purpuric PR is a rare variant of the disease. Other forms of atypical PR include vesicular, hemorrhagic, urticarial, and papular forms [4]. This variant has been found in both pediatric and adult

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populations. The purpuric lesions may present in tandem with more typical patches, or as the primary morphology [3,4]. The course and prognosis of the disease is otherwise similar to classic PR [3].

The lesions of purpuric PR may mimic Henoch-Schönlein (HSP) or other IgA vasculidities, pityriasis lichenoides, pigmented purpuric dermatoses, pityriasis lichenoides et varioliformis acuta (PLEVA), and Kaposi’s sarcoma [5]. The diagnosis thus depends on the presence of associated symptoms or diseases, as well as



Figure 1 Purpuric papules on the patient’s elbows and forearms one week after the onset of the rash.



Figure 2 Purpuric papules on the patient’s chest and abdomen one week after the onset of the rash.

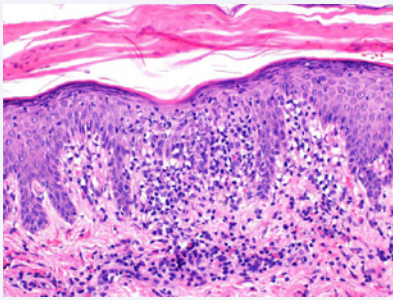


Figure 3 20x hematoxylin and eosin stain of a cross-section of the dermis in this teenager. There is a superficial lymphocytic perivascular infiltrate with extravasation of red blood cells.

the histopathology of the lesions. PR can be distinguished from PLEVA by its sudden-onset of monomorphic lesions and limited duration.

REFERENCES

1. Drago F, Broccolo F, Rebora A. Pityriasis rosea: an update with a critical appraisal of its possible herpesviral etiology. *J Am Acad Dermatol.* 2009; 61: 303-318.
2. Watanabe T, Kawamura T, Jacob SE, Aquilino EA, Orenstein JM, Black JB, et al. Pityriasis rosea is associated with systemic active infection with both human herpesvirus-7 and human herpesvirus-6. *J Invest Dermatol.* 2002; 119: 793-797.
3. Bhalla N, Tambe S, Zawar V, Joshi R, Jerajani H. Localized purpuric lesions in a case of classical pityriasis rosea. *Indian J Dermatol Venereol Leprol.* 2014; 80: 551-553.
4. Chuh A, Zawar V, Lee A. Atypical presentations of pityriasis rosea: case presentations. *J Eur Acad Dermatol Venereol.* 2005;19:120-126.
5. Ermertcan AT, Ozgüven A, Ertan P, Bilaç C, Temiz P. Childhood Pityriasis rosea inversa without Herald Patch Mimicking Cutaneous Mastocytosis. *Iran J Pediatr.* 2010; 20: 237-241.

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