

Papular Pityriasis Rosea

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GOAL

To describe a case of papular pityriasis rosea (PR)

OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Outline the varying clinical presentations of PR.
2. Discuss the theoretical etiologies of PR.
3. Describe therapy for PR.

CME Test on page 48.

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Pityriasis rosea (PR) is a seasonal papulosquamous disorder that can be easily confused with a wide variety of similar appearing cutaneous disorders. This is particularly evident in its atypical papular form. We present a case report of atypical papular PR, along with a discussion of clinical presentation, histologic criteria, proposed etiology,

and treatment options. Papular PR is atypical, presenting in a minority of patients, and may pose a diagnostic challenge. Being familiar with these atypical characteristics will facilitate accurate and timely diagnosis.

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The views expressed are those of the authors and are not to be construed as official or as reflecting those of the US Air Force Medical Service or the US Department of Defense. This work is in the public domain. The authors report no conflicts of interest. Reprints: Lt Col Michael R. Murchland, MC, FS, USAF, Department of Dermatology, Wilford Hall Medical Center, 2200 Berquist Dr, Suite 1, Lackland Air Force Base, San Antonio, TX 78236-5300 (e-mail: michael.murchland@tomdw.whmc.af.mil).

Pityriasis rosea (PR) is an acute, self-limited, inflammatory disorder that primarily affects children and young adults. It is characterized by a distinctive papulosquamous skin eruption classically distributed on the trunk and proximal extremities. PR has been described in the medical literature for more than 200 years, though the dermatosis was first called *pityriasis rosea*, literally "rose-colored scale," by the esteemed French physician Camille Gilbert in 1860.¹ A condition seen throughout the world, PR may occur in patients of all ages; however, approximately 75% of cases occur between the ages of 10 and 35 years.² While there exists a slight female predominance, no racial predisposition has been noted. The disease is more



Figure 1. Papulosquamous truncal dermatitis with herald patch.

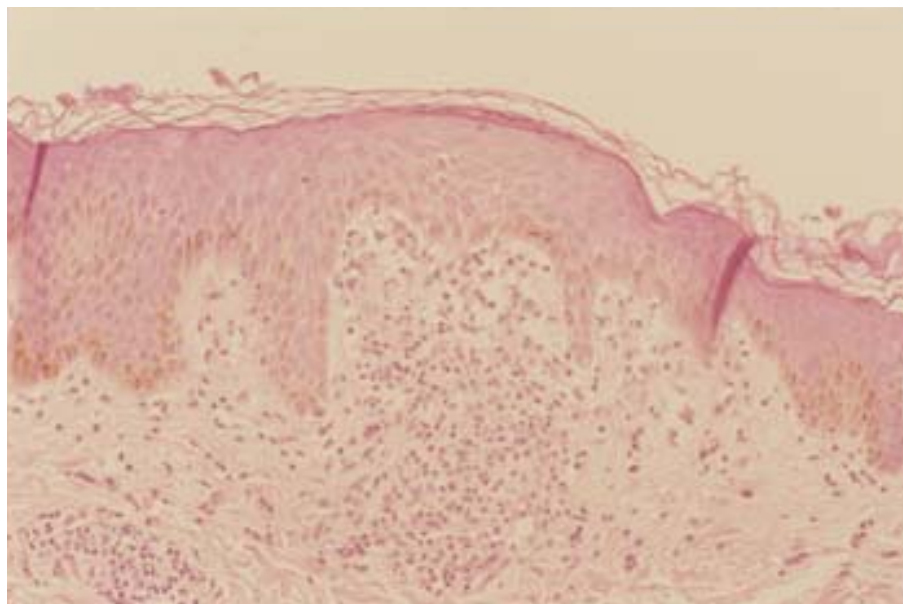


Figure 2. Focal spongiosis, parakeratosis, and extravasated red blood cells (H&E, original magnification $\times 200$).

common in the fall and winter and during the spring months in temperate climates.

Case Report

A 36-year-old African American man presented to the dermatology clinic with a 1-week history of a pruritic papulosquamous rash all over his back, chest, and neck (Figure 1). Antihistamines provided only minimal relief. He reported no antecedent illness or recent use of medication. Results of a potassium hydroxide (KOH) preparation were negative for hyphae. A biopsy of the

skin revealed focal spongiosis, parakeratosis, and extravasation of red blood cells (Figure 2).

Clinical Presentation

The clinical findings of PR are remarkably consistent in most patients. The initial manifestation is the herald patch (a solitary, erythematous, oval plaque that gradually enlarges for 1 to 2 days, ranges from 2 to 6 cm in diameter, and is found most commonly on the anterior aspect of the chest). The reported prevalence of the herald patch varies widely from series to series but typically occurs in



Figure 3. Grouped 2- and 3-mm scaly papules on the back.

50% to 90% of cases.³ A generalized eruption consisting of crops of salmon-colored, 5- to 10-mm ovoid macules and papules follows the appearance of the herald patch, normally within 7 to 14 days. The characteristic lesions are covered with a delicate collarette of scale at their margins and arranged with their longest axis parallel to the lines of cleavage, forming the so-called Christmas-tree pattern on the trunk and proximal aspect of the extremities. Usually, the hands and feet are spared. The eruption classically persists for 2 to 12 weeks, though prolonged cases of up to 5 months have been reported.¹

Atypical disease occurs in 10% to 15% of patients and may pose a diagnostic challenge.⁴ Our patient presented with papular PR, a rare form of the disorder that is more common in young children, pregnant women, and Afro-Caribbeans (Figure 3).⁵ Scabies, lichen planus, and a lichenoid drug reaction may have similar appearances and occasionally are confused with papular PR. Patients with this morphologic type more likely exhibit features of inverse PR, another unusual variant that is characterized by involvement of usually spared areas, such as the face, axilla, and groin. Inverse papular PR may mimic papular acrodermatitis of childhood (Gianotti-Crosti syndrome). The most common atypical manifestation is the absence of a herald patch.⁴ Urticaria, vesicles, bullae, lichenoid lesions, nonpalpable purpura, erythema multiforme-like lesions, gigantic plaques, and exfoliative dermatitis all have been reported.⁶

Although PR is recognized easily in its classic presentation, difficult cases are encountered sometimes. Early in the course of the disease, the herald

patch may be mistaken for tinea corporis. A KOH preparation of skin scrapings is utilized to make this distinction. Nummular eczema and nummular asteatosis also may be considered. When the generalized eruption of PR occurs, cutaneous processes, such as secondary syphilis, tinea versicolor, psoriasis or parapsoriasis guttata, and seborrheic dermatitis, should be included in the differential diagnosis. The lesions of erythema dyschromicum perstans, irritated pityriasis alba, and pityriasis lichenoides chronica, which can follow the lines of skin cleavage on the trunk, also must be differentiated from PR. Rare cases of purpuric PR may be confused with Kaposi sarcoma, vasculitis, Henoch-Schönlein purpura, or various causes of thrombocytopenia.¹ In vesicular PR, the possibility of varicella must be considered.

A number of medications have been associated with a PR-like dermatosis. These include captopril, isotretinoin, metronidazole, D-penicillamine, levamisole, gold, bismuth, arsenic, barbiturates, and clonidine.⁶ Vaccines, such as bacillus Calmette-Guérin and diphtheria toxoid, also have been reported to cause similar eruptions.¹ The lesions found in drug-induced PR may exhibit classical features; however, they tend to be recalcitrant to therapy and have a prolonged course. In addition, they are usually less numerous, larger in diameter, and more commonly lead to marked hyperpigmentation than the typical lesions. Frequently, the Christmas-tree distribution is absent.⁶

Diagnosis

Because the diagnosis of PR usually is made by the clinical appearance of the eruption, a biopsy of the

skin is rarely necessary. Histopathologic findings are relatively nonspecific and represent those of a subacute or chronic dermatitis. Typically, an undulating epidermis is seen with focal parakeratosis, as well as a diminished granular layer and spongiosis. Small spongiotic vesicles, sometimes mimicking Pautrier microabscesses because of the collection of lymphocytes within them, are a characteristic feature.⁷ The papillary dermis shows some edema and occasionally homogenization of collagen, along with a mild-to-moderate lymphohistiocytic perivascular infiltrate. Older lesions may exhibit a relative increase in the number of eosinophils in this infiltrate. The herald patch demonstrates the standard features of a secondary lesion of the disease. Dyskeratotic cells in the epidermis and extravasated erythrocytes in the dermal papillae may help distinguish atypical PR from other dermatoses.⁸

Etiology

The precise etiology of PR is unknown. The most widely accepted theories regarding its pathogenesis point to an infectious—particularly viral—cause. Many observations support this view, including the seasonal variation in occurrence; spontaneous regression of lesions; occasional presence of prodromal symptoms; relative rarity of recurrence; and increased prevalence in immunocompromised patients, such as pregnant women and recipients of bone marrow transplants.⁹ PR also occurs in small epidemics in fraternity houses, Turkish baths, and military establishments.¹⁰ Moreover, the viral hypothesis is supported by reports of an association between PR and preceding upper respiratory tract infections.¹¹

A number of viruses, including picornaviruses and *Parvovirus*, have been explored historically as causative agents of PR, without success. The most convincing evidence for a specific viral etiology was reported in 1997 by investigators who used polymerase chain reaction analysis to detect human herpesvirus-7 (HHV-7) DNA in the plasma, peripheral blood mononuclear cells, and skin of a series of patients with PR.¹² Based on their findings, the authors suggested the possibility that PR may be a clinical manifestation of HHV-7 reactivation. Subsequent studies, however, cast doubt on these findings. One such study reported no difference in the prevalence of HHV-7 (or HHV-6) in mononuclear cells between patients with PR and those with other skin disorders,¹³ while another demonstrated lower levels of HHV-7 DNA in PR lesions than morphologically normal skin.¹⁴ Like their predecessors, these findings have come under scrutiny.¹⁵ Clearly, more work needs to be done to

make any definitive statements regarding the etiology, infectious or otherwise, of PR.

Comment

The imperfect understanding of PR is reflected by the numerous modes of therapy used in the past. These have consisted of injections with arsenic and bismuth, staphylococcal toxoid, streptococcal and typhoid vaccines, milk, and PR scales dissolved in saline solution.⁶ Because the lesions disappear spontaneously within a limited time frame, treatment usually is aimed at relieving pruritus, which may be severe in approximately 25% of patients.³ Therapy includes emollients, corticosteroid creams, antihistamines, and, rarely, for severe forms, systemic steroids or dapsone. UVB radiation, administered in 5 consecutive daily erythrogenic exposures, has been reported to decrease pruritus and hasten the involution of cutaneous lesions.¹⁶ Another study showed that UVB phototherapy decreased the severity of disease but did not alter itching or course of the disease.¹⁷ Like all potential treatments, it is most efficacious if it is initiated early. Furthermore, in a recent study of 90 subjects, oral erythromycin was found to be effective in treating PR, with a complete response (the disappearance of all lesions without the development of new lesions within 2 weeks of the start of treatment) noted in 73% of patients.¹⁸ While these findings further complicate the debate regarding the infectious etiology of PR, they also add an intriguing option to the therapeutic arsenal. Our patient was treated with erythromycin and antihistamines, and rapid improvement of his symptoms was noted within one week.

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