Dermacase

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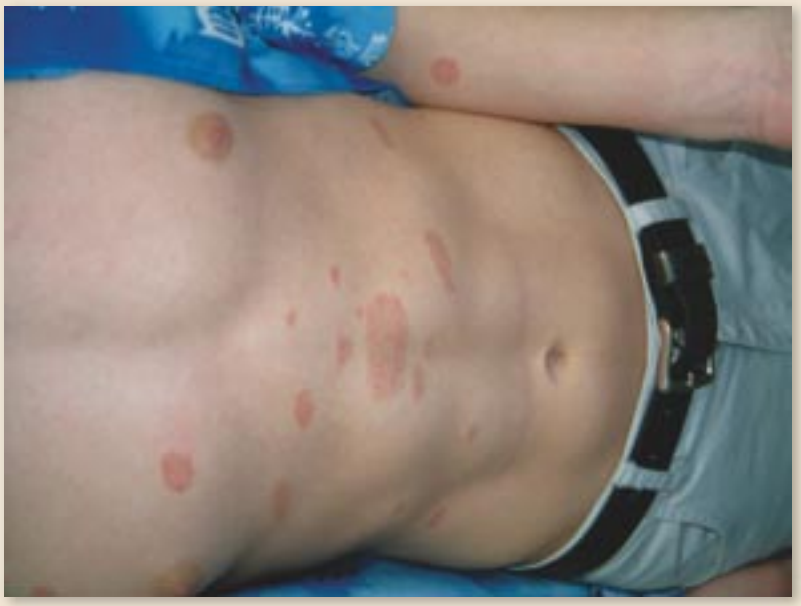

CAN YOU IDENTIFY THIS CONDITION?

An 18-year-old healthy man presents with oval erythematous papulosquamous pruritic lesions on his trunk. Most of the lesions have flesh-coloured centres and slightly elevated scaly borders. The rash started with a single lesion on his abdomen and progressed over the next 2 weeks despite treatment with ciclopirox olamine (Loprox®).

The most likely diagnosis is:

1. Psoriasis
2. Pityriasis rosea
3. Lichen planus
4. Secondary syphilis

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2. Pityriasis rosea

Pityriasis rosea (PR) is a common, acute, self-limited papulosquamous condition that most often affects those between 10 and 35 years old.[1,2] It is thought to occur more often in women; there is no racial predilection; and it occurs throughout all seasons with increased incidence in spring and fall.

Etiology

The etiology of PR is not fully understood. Some studies suggest it is of viral origin. Its clinical presentation supports this view: cases often cluster within families, close contacts, and immunocompromised people. A single episode appears to give lifelong immunity to 97% of patients. As it does with mononucleosis, treatment with ampicillin has been noted to increase the distribution of secondary eruption. Recent studies have shown human herpesvirus 7 (HHV-7) viral DNA in the cutaneous lesions, lymphocytes, and plasma of patients with PR. Because HHV-7 is common in healthy people, however, more studies need to be done to establish a true causal link.

Several immunologic studies have also suggested a viral etiology for PR. Higher levels of CD_4 T lymphocytes, Langerhans cells, and anti–immunoglobulin M to keratinocytes in people with PR support this hypothesis. An autoimmune process has been also proposed; many drugs, including captopril, metronidazole, ketotifen, clonidine, gold, isotretinoin, D-penicillamine, barbiturates, and levamisole, cause eruptions resembling those of PR. Bacille Calmette-Guérin and diphtheria toxoid are also known to cause PR-like eruptions. Drug-associated PR can have a prolonged course and leave hypopigmented or hyperpigmented areas on the skin.

Presentation

In most cases, PR has a characteristic clinical presentation. The first cutaneous manifestation, a "herald patch," is an oval pink macule or plaque with central clearing and peripheral scale pointing inward, usually on the trunk or extremities. This macule enlarges rapidly over a few days to up to 10 cm in diameter. Most studies indicate that a herald patch is found in more than 50% of cases.

Headache, gastrointestinal symptoms, fever, malaise, and arthralgias precede cutaneous manifestations in up to 5% of PR cases. Three to 14 days after the initial herald patch appears, crops of lesions erupt in a more generalized fashion over the trunk, neck, abdomen, and proximal extremities. These lesions often resemble the herald patch and are usually smaller, bilateral, symmetrical macules, papules, and plaques with their long axes parallel to lines of cleavage. This arrangement results in a characteristic "Christmas tree" distribution. The eruption usually lasts 3 to 12 weeks, but can last up to 5 months. A prolonged course is especially common in PR-like drug eruptions. Pruritus is a common complaint and is severe in about 25% of cases.

Although most cases present as outlined above, some variation in presentation, location, and morphology has been reported. There might be several herald patches or no herald patch at all. The extent of eruption can vary from numerous to very few lesions. Children are more prone to developing generalized eruptions with acral involvement or an inverse variant of PR.[3] In an inverse variant, lesions are concentrated on the face and distal extremities, and there are none or very few on the trunk.

Occasionally, PR is localized to one area of the body or involves the oral mucosa. Oral involvement is usually characterized by asymptomatic punctate hemorrhages; ulcerations; and erythematous macules, plaques, vesicles, and bullae; and is more common in children, black patients, and patients with generalized eruptions. Black patients tend to develop a more widespread papular eruption with facial and scalp involvement

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and generalized lymphadenopathy. Lesions might appear darker than the surrounding skin and have no inner collarette of scale. Hypopigmentation is common in black patients after PR resolves.

The morphologic forms of PR include papular, vesicular, pustular, purpuric (hemorrhagic), and urticarial, depending on the appearance of individual lesions. Diagnosis can be difficult with atypical presentations; skin biopsy might be required. Histologically, PR presents as superficial perivascular dermatitis with epidermal and dermal changes. Focal parakeratosis, hyperplasia, and focal spongiosis are seen in the epidermis. In the dermis, extravasated red blood cells with a perivascular infiltrate of lymphocytes, histiocytes, eosinophils, and monocytes often appear.

Differential diagnosis

Differential diagnosis includes psoriasis, secondary syphilis, erythema multiforme, lichen planus, PR-like drug eruptions, and tinea corporis infection. There are no laboratory investigations to diagnose PR.

Treatment

Pityriasis rosea is a self-limited disease, so patient education and reassurance are highly recommended. Moisturizers and nonprescription lotions with calamine and menthol can be used to treat symptoms of pruritus. Topical corticosteroid creams and oral antihistamine medications are indicated for severe cases of pruritus. Those with intractable pruritus or vesicular PR variant and dark-skinned people with widespread PR might benefit from a short course of systemic corticosteroids, such as oral prednisone (0.5 to 1 mg/kg daily for 7 days). Ultraviolet B phototherapy can decrease the severity of PR but not change its course. Oral erythromycin in divided doses for 14 days was shown to decrease the severity of PR in one double-blind, placebo-controlled clinical study[4] and might prove beneficial for severe cases.

References

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4. Sharma PK. Erythromycin in pityriasis rosea: a double-blind, placebo-controlled clinical trial. *J Am Acad Dermatol* 2000;42(2 Pt 1):241-4.