Pityriasis rubra pilaris in association with hepatitis A

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Pityriasis rubra pilaris (PRP) is an idiopathic papulosquamous disease that clinically presents with palmoplantar keratoderma and follicular hyperkeratotic papules that coalesce into scaly erythematous plaques. Although the etiology is unknown, some factors may be playing a role in the etiopathogenesis of PRP, such as genetic factors, internal malignancies, autoimmune diseases, trauma, and infections. A variety of infections are considered to be possible precipitating factors in PRP. In recent years, numerous cases of PRP have been described in association with HIV infection.[1] We described a case demonstrating PRP in association with hepatitis A.

A 5-year-old boy, was admitted to our dermatology department with a 2-month history of a florid erythematous eruption involving the hands, feet, elbows, knees, and face. He had fatigue, jaundice on his eyes, face and urine the week before the exanthem developed, and then he had consulted a pediatrician. Hepatitis A had been diagnosed in our patient, and he was hospitalized for 4 days. One week after his discharge, the first erythematous-scaly follicular eruption occurred on his elbows and knees, then involved other areas. The patient's medical history was unremarkable, and there was no personal or family history of skin disease. Clinical examination revealed wide, confluent erythematous, dense scaling areas and keratotic follicular papules over the neck, chin, and extensor surfaces of the limbs, especially knees and elbows (**Figure 1**). The palms showed erythema, hyperkeratosis, and fissures. Our patient had type III classic juvenile PRP, according to Griffiths' proposed classification. On physical examination, hepatomegaly was determined. Laboratory investigations showed some pathologic parameters: Serum aspartate aminotransferase (AST) 41 U/L (normal 10-37), alanine aminotransferase (ALT) 48 U/L (normal 10-37), hemoglobin 12.8 g/dl (normal 13.6-17), hematocrit 37.6 % (normal 39.5-50), activated partial thromboplastin time (aPTT) 26.3 sec (normal 27.5-36). Anti-Hepatitis A IgM and IgG antibodies were positive. The levels of thyroid hormones were normal. An HIV test, rapid plasma reagin test, and anti-nuclear antibody titer were negative. An ultrasonography showed liver enlargement, with normal structure. Other examinations, particularly alkaline phosphatase, glucose, and bilirubin were normal. An excisional skin biopsy specimen from his left thigh areas showed slight acanthosis, banded alternating orthokeratosis, parakeratosis, plugs of the follicular infundibulum, and a superficial perivascular inflammatory infiltrate. These findings are consistent with PRP. We gave topical low-potency steroid ointment, emollient and keratolytic agent for therapy. Systemic therapy was avoided, due to the hepatic dysfunction.

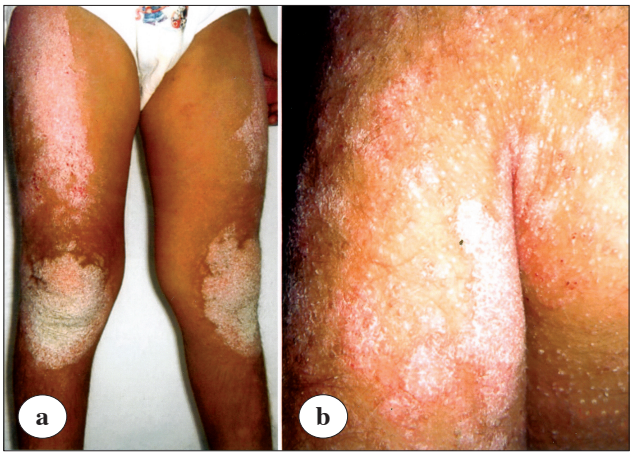

Figure 1 - Keratotic-scaly follicular papules over the extensor surfaces of the **a)** legs and, **b)** shoulder.

Pityriasis rubra pilaris is a rare papulosquamous disease with a typical onset during the first and fifth decades. Griffiths classified PRP into 5 types, based on age and pattern of onset as well as prognosis.[2] Type I is a classical adult onset PRP. Type III is a classical juvenile onset PRP and appears to differ clinically from type I only by onset in childhood.[1] Our case was consistent with type III classic juvenile PRP.

Pityriasis rubra pilaris is an idiopathic disease, which has remained enigmatic, and cases of PRP occur without any preceding event. Many etiologic factors, such as, genetic factors, malignancy, autoimmunity, trauma, and infection are suggested in the etiology of PRP, but the role of these factors are not clearly explained. Pityriasis rubra pilaris has appeared simultaneously with autoimmune diseases, such as, myasthenia gravis, hypothyroidism, and celiac sprue.[1] Pityriasis rubra pilaris has been observed with many infectious diseases. Gross et al,[3] reported that 14 of 173 cases from the English language literature had possible precipitating infection or trauma. Davidson et al,[4] reported that 5 of 57 PRP patients had preceding infection or trauma. All 5 cases were children. Larregue and colleagues,[5] also

described 3 of 4 cases of juvenile PRP occurring after an infection. Some cases of PRP have been described in association with HIV infection since 1990.[1] Cecchi et al,[6] reported a patient who was affected with alopecia universalis, vitiligo, and chronic viral hepatitis C, who subsequently developed an adult-onset PRP and lichen planus. In our opinion, PRP may have been provoked by hepatitis A in this case.

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