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LETTER TO THE EDITOR

Anetodermic lesions following pityriasis rosea and impetigo

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Dear Editor,

Pityriasis rosea (PR) is an acute, exanthematous disease caused by human herpesvirus (HHV)-6 and/or HHV-7. It mainly affects young adults of both genders. The disease begins with a single, erythematous plaque, called herald or mother patch. It is roundish or oval, pink in colour, with slightly elevated borders and slightly depressed center. It usually occurs on the trunk and remains the unique clinical manifestation of the disease for 7 to 10 days, when widespread, clinically similar, although smaller lesions appear. The scalp, face, palms and soles are usually spared. Pruritus is rarely severe.[1,2] Atypical clinical presentations are possible.[3] The duration of the infection ranges from two weeks to three months. The disease is self-healing: therapy is usually unnecessary or symptomatic.

We present a case of anetodermic lesions which appeared in a patient with PR followed by impetigo.

A 32-year-old woman was admitted with a clinical diagnosis of PR. The patient stated that she was in good general health and that she was not in therapy with systemic drugs. She also declared that the rash had appeared one week earlier and was accompanied by severe pruritus.

Dermatological examination revealed numerous erythematous-squamous papules which were widespread on the chest, abdomen, back (with a

Christmas tree arrangement) and upper limbs. They were round or oval, pink to red in colour, 0.4 cm in diameter. The diagnosis of PR was confirmed. No biopsy was performed. A cream containing 8% calamine and hydroxyzine (25 mg/day) were prescribed. The patient returned one week later with several pustules superimposed on previous PR lesions. A diagnosis of impetigo, presumably caused by continuous scratching, was made. Cultures were positive for *Staphylococcus aureus*. The patient was treated with i.m. ceftriaxone (2 g/day for 7 days) and hydroxyzine. The patient was again examined two weeks later: impetigo and itching had almost completely disappeared. However, six weeks later, the patient developed, in the same areas in which previous PR and impetigo lesions had occurred, atrophic lesions. They were round or oval, with a colour ranging from pearly white to pale pink, 0.5 to 1.5 cm in diameter (Figure 1). The consistency was soft: finger pressure allowed herniation of the lesions. No symptom was reported by the patient. General physical examination was normal. All laboratory tests were within normal ranges or negative. A diagnosis of post-PR and impetigo anetoderma was made. However, histopathological examination was consistent with perifollicular anetoderma (Figure 2). We therefore decided to classify this case as “anetodermic lesions following PR and impetigo”. Anetoderma is a rare disease of unknown etiology, characterized by loss of dermal elastic fibers.[4,5] Some varieties of anetoderma are known: familial (with autosomal dominant inheritance pattern), congenital, drug-induced,

primary and secondary anetodermas.[5] Primary anetoderma occurs on healthy-appearing skin (Schweninger-Buzzi anetoderma) or on a previous disease of the skin (Pellizzari-Jadassohn anetoderma).[4,5]

To our knowledge, no cases of anetoderma following PR have been reported in the literature. As previously mentioned, our patient developed impetigo on several lesions of PR. This was the result of scratching caused by severe pruritus. In this patient, it is possible that anetodermic lesions have been the final clinical result of a staphylococcal infection. However, only one case of anetoderma following impetigo has been reported in the literature.[6] Furthermore, very rare cases of anetoderma wich appeared after bacterial infections of the skin have been published: tuberculosis,[7,8] leprosy [7,9-11] and syphilis. [7,8,12-13]

In our patient, the medical history and clinical presentation of the lesions were consistent with the diagnosis of Pellizzari-Jadassohn anetoderma. We excluded familial, congenital and drug-induced anetoderma. The latter was excluded because no cases of anetoderma after the use of calamine, hydroxyzine and ceftriaxone have been reported in the literature. However, histopathological picture of our patient was consistent with the diagnosis of perifollicular elastolysis.[14-22] It is characterized by atrophic, round or oval, white to pink, small or very small, non confluent, asymptomatic lesions, with a central pilosebaceous follicle.[15,16,18] These lesions occur on the neck, shoulders, arms and upper trunk.[15-18] Histopathological picture is

characterized by loss of elastic fibers surrounding hair follicles, without signs of inflammation. Collagen fibers are not involved.[15] A bacterial etiology was suggested, because strains of *Staphylococcus epidermidis* producing elastase were found in the hair follicles of the lesions.[15,17] However, this hypothesis was not confirmed.[16] In some cases, lesions of perifollicular elastolysis were preceded by or associated with acne,[16,18] atopic dermatitis[20] or pseudofolliculitis of Behçet's disease.[21]

In conclusion, we reported a case of anetodermic lesions which appeared in a patient previously affected by PR and impetigo. Clinical manifestations were consistent with the diagnosis of Pellizzari-Jadassohn anetoderma, whereas histopathological picture was typical of perifollicular elastolysis. For these reasons, we decided to classify this case as "anetodermic lesions following PR and impetigo".

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Conflicts of interest: The authors have nothing to declare.

Legends

Figure 1. - (A) and (B) Atrophic lesions, located in the same areas in which previous PR and impetigo lesions had occurred.

Figure 2. - (A) Reduction of elastic fibers in the superficial dermis (haematoxylin and eosin x40). (B) Elastic fibers are absent in the superficial dermis (orcein x40).