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Resolution of inverse psoriasis after treatment with levodopa for Parkinson's disease

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Abstract

Inverse psoriasis is characterized by the development of erythematous shiny plaques at intertriginous areas of the body. It has a prevalence of 2% worldwide. The usefulness of levodopa in psoriasis was discovered in 1970 but nowadays it is not a standard therapy for this condition. A 74-year-old woman was diagnosed with Parkinson's disease subsequent to the development of extensive inverse psoriasis. The skin lesions were resistant to classical topical and systemic medications. Treatment with levodopa was initiated in order to treat her neurological problem and progressive remission of the skin lesions was noted. We highlight the role of dopamine in the pathophysiology of this dermatosis.

KEYWORDS

intertriginous psoriasis, inverse psoriasis, levodopa, Parkinson's disease

1 | INTRODUCTION

Psoriasis is a chronic recurrent inflammatory skin disease characterized by the development of scaly well-defined erythematous plaques. It has a prevalence of 2% worldwide and the most common clinical presentation is psoriasis vulgaris (Limaye, 2015). Inverse or intertriginous psoriasis affects 3–7% of patients with psoriasis (Omland & Gniadecki, 2015). Erythematous, shiny non-scaly plaques appear at intertriginous areas of the body (Omland & Gniadecki, 2015). The relationship between dopamine and psoriasis was described in the 70s and good therapeutic responses of psoriasis with the use of this drug were reported.

We describe the case of a patient with inverse psoriasis refractory to standard treatment who was subsequently diagnosed with Parkinson's disease. The cutaneous manifestations remitted after treatment with levodopa.

2 | CLINICAL CASE

A 74-year-old woman was attended at the Dermatology Unit due to itchy skin lesions of 1-month duration extensively affecting the skin folds and dorso-lumbar area. Physical examination showed shiny erythematous well-demarcated plaques affecting predominantly the intertriginous areas of the body (Figure 1).

A clinical diagnosis of inverse psoriasis was done and the patient was treated with topical drugs including corticosteroids, calciprotriol, antibiotics, and anti-fungals without improvement of skin lesions. Then she received acitretin 0.5 mg/kg/day for 3 months showing no response. Afterwards treatment with cyclosporine 3 mg/kg/day was administered for 3 months also with a lack of response. Finally, given the therapeutic failure to the previous treatments, the patient was admitted to the Dermatology Department. During her admission we carried out a skin biopsy that showed parakeratosis, acanthosis, and epidermal hyperplasia compatible with psoriasis. The analytical studies including blood count, complete general biochemistry with hepatic and renal profile, protein electrophoresis, complement, serologies for hepatitis B, hepatitis C, and human immunodeficiency virus were negative or normal. The autoimmunity study including antinuclear antibodies was also negative. Thus we confirmed the diagnosis of intertriginous psoriasis. During her admission we also noted facial hypomimia and rigidity in her body movements. We ruled out concomitant medication with potential anti-dopaminergic effects and consulted the Neurology Unit. The diagnosis of idiopathic Parkinson's disease was confirmed in the neurological study. Consequently, treatment with levodopa–carvidopa at increasing doses up to a dose of 25/250 mg every 8 hr was

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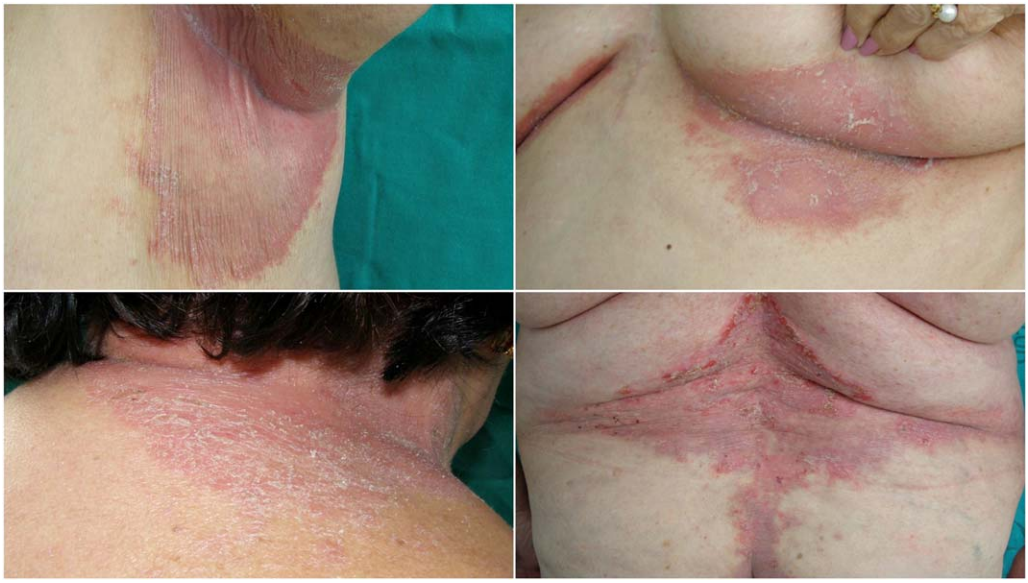

FIGURE 1 Erythematous well demarcated plaques localized at intertriginous areas of the skin

given to our patient. After a few days on this medication both Parkinson symptoms and psoriatic lesions gradually improved. The patient achieved a complete remission after three weeks and remained asymptomatic for more than a year. Then she had a recurrence of the inverse psoriasis related to the abandonment of levodopa on her own account. Again the re-establishment of the dopaminergic treatment was followed by the remission of the skin symptoms. Nowadays she remains asymptomatic after another year of treatment (Figure 2).

3 | DISCUSSION

The skin and the central nervous system share a common origin through the ectoderm. Dermatological conditions such as bullous pemphigoid or seborrheic dermatitis are associated with neurological disease. A relationship between domapine and the pathophysiology of psoriasis has been known since the 70s. In 1970 Andre Barbeau described the cutaneous and neurological improvement in patients

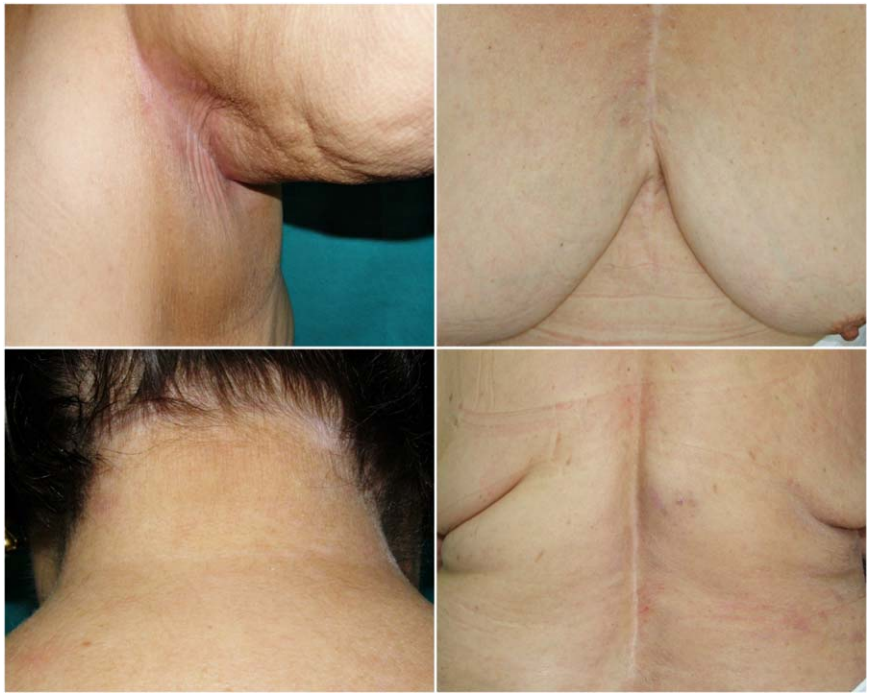

FIGURE 2 Physical examination of the patient after one year of treatment with levodopa, showing resolution of inverse psoriasis

with psoriasis in addition to neurological Parkinson symptoms after treatment with levodopa alone or in combination with a peripheral decarboxylase inhibitor (Devoitille, De la Brassine, Dethier, & Couteaux-Dumont, 1973). Later, in 1973 Lewis carried out a more thorough study with 19 patients treated with levodopa in which 14 of them showed an improvement in both conditions. Skin lesions located on the trunk and limbs responded better to treatment, however the improvement demonstrated at the scalp and nails was slower or even non-existent (Lewis, 1973; Paver, Doyle, Krivan, & Kossard, 1974). Three years later Francois Savery conducted a double-blind crossover study in order to compare the effect of levodopa versus placebo in 35 patients with plaque psoriasis but without associated neurological symptoms. He demonstrated that levodopa increases the concentration of cyclic adenosine monophosphate (AMPc) after 3–6 months of treatment in a good proportion of patients. Furthermore, these results were correlated with an improvement in the dermatological symptoms. Patients who did not show a clinical improvement did not increase their levels of AMPc, so there is a significant relationship between these two aspects (Savery, Karassik, & Gast, 1976). Therefore in 1973 it was postulated that levodopa may be processed locally into dopamine by the enzyme dopa-decarboxylase. Dopamine would stimulate the different enzymes that produce an increase in cyclic AMP, which can reduce or decrease the speed of cell division in the epidermis. It should be noted that the period of time between the start of treatment and the clinical improvement is variable between the different researches. In the study conducted by Barbeau, the psoriatic lesions improved in 16 days approximately. In Lewis's study, the improvement was estimated after a minimum period of 4–6 weeks and in Savery's after 3 months of treatment. In other posterior small studies this period was even greater. In the case of our patient the improvement began after 3 weeks, and the relapse of the skin lesions had a causal relationship with therapeutic non-compliance.

In conclusion, we described a clinical case that may demonstrate a relationship between dopamine and psoriasis. Awareness by doctors of this fact may allow a better diagnosis and management of both conditions. Treatment with levodopa can be an effective treatment in patients with refractory psoriasis associated with Parkinson's disease (Kalb et al., 2009).

CONFLICT OF INTEREST

The authors have no conflict of interest to report.

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