

Case Report

Systemic Lupus Erythematosus and Psoriasis: A Case Report

Jaouad Yousfi^{1*}, Soukaina Oumlil¹, Laila Benjilali¹, Lamiaa Essaadouni¹¹Department of Internal Medicine, University Hospital of Mohammed VI, Marrakesh, Morocco

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Abstract: Psoriasis is an autoimmune chronic inflammatory skin disease. Systemic lupus erythematosus (SLE) is a connective tissue disorder with variable clinical features ranging from mild joint and skin involvement to life-threatening renal, hematologic, or central nervous system involvement. Despite their high frequency in our population, their coexistence is uncommon, reported only in few case reports, raising hypotheses about shared pathogenetic mechanisms. We report a 43-year-old female with psoriasis accompanied by SLE.

Keywords: Psoriasis, skin disease, Systemic lupus erythematosus (SLE).

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INTRODUCTION

Psoriasis is a chronic-recurrent inflammatory disease which affect 1-3% of general populations [1-3]. It is a progressive erythematous-squamous dermatitis of unknown etiology. Psoriasis can be aggressive by developing arthropathy, erythrodermic or pustulosis forms. The etiopathogenesis of psoriasis is not completely understood. Genetic, immunologic and environmental factors have been suggested [4]. Systemic lupus erythematosus (SLE) is an autoimmune disease with a broad spectrum of manifestations, from localized cutaneous lesions (acute, subacute, and chronic) with a benign course, to a rapidly progressive and fatal systemic illness [3, 5]. The prevalence of SLE has been estimated from 14.6 to 122 cases per 100,000 [6, 7].

Although SLE has been described in association with other autoimmune diseases such as rheumatoid arthritis, scleroderma, mixed connective tissue disease, autoimmune thyroid diseases and pernicious anemia [8], its coexistence with psoriasis is very rare [9, 10]. This association is very challenging due to the similarity of the clinical features (cutaneous and articular), but also due to the difficulty of its management, since medications used to treat one condition can exacerbate or even trigger the symptoms of the other [1-4, 6, 10, 11].

CASE PRESENTATION

A 43-year-old woman, with a history of a well-controlled type 2 diabetes mellitus for 13 years on premixed insulin and glimepiride at a dose of 3 mg / day, and hypothyroidism treated by thyroid hormone replacement therapy (Levothyroxine 100 µg /day), presented to the hospital with erythematous-squamous plaques localized over the scalp and extensor surfaces of the elbows and knees. The diagnosis of psoriasis was made based on typical clinical findings and a skin biopsy.

The patient received methotrexate, and achieved complete remission after six months. One year after the diagnosis of psoriasis, the patient presented with drug-resistant chronic headache, alopecia, photosensitivity, malar rash and polyarthralgia involving knees, wrists and elbows.

The results of laboratory tests included an erythrocyte sedimentation rate of 60 mm in the first hour, C- reactive protein of 70g/dl.

Serum anti-nuclear antibodies were positive with a titer of 1/640 and a homogeneous pattern. The titer of anti-dsDNA was high at 40 IU/ml.

Kidney function was normal and proteinuria was 0.1 g /24 h. A brain MRI showed micro nodular white matter lesions.

The patient also presented with chest tightness and dyspnea (NYHA class II) due to interstitial lung disease.

Infection workup, for lung involvement, was negative (Genexpert and cyto-bacteriological examination of the sputum). The electrocardiogram and the echocardiogram were normal.

The diagnosis of SLE with cutaneous, articular, neurologic and pulmonary involvement was established, and the patient was started on low-dose corticosteroid therapy. She also received cyclophosphamide for the neurologic and pulmonary involvement. The cumulative dose received was 12 g.

After 5 years of follow-up, complete remission was achieved; SLE remained clinically quiescent, and no exacerbation of psoriatic skin lesions was observed.

DISCUSSION

The coexistence of psoriasis with SLE has been reported in the literature [4, 6, 12]. A study reported psoriasis in 0.6% of 520 patients with SLE [13]. Other series respectively collected ten cases over 25 years [6]; five cases over ten years [4] and three cases over seven years [14]. Zalla and Muller identified 42 cases of SLE among 9420 psoriasis patients in a 10-year retrospective study [4]. The prevalence rate of psoriasis coexisting with SLE is around 1.1%, slightly higher in women, due to the higher prevalence of SLE in this population [15].

The pathogenesis of this association is not fully understood, as the underlying autoimmune mechanism is still unknown. Dysregulation of the immune response and T cell activation by superantigens appear to be the common pathogenetic mechanism of these two pathologies [2, 12], as does mutations in the human leukocyte antigen (HLA) gene [14]. However, psoriasis and SLE can appear independently in the same patient, without necessarily having a causal relationship between them [1].

These patients seem to have an increased risk for photosensitivity and anti-SSA antibodies [4, 14]. According to Kullick *et al.*, [14], the presence of anti-SSA antibodies would be a serological marker suggestive of the SLE – Psoriasis association. However, they are not always present, and some authors reported the coexistence of the two conditions in the absence of anti-SSA antibodies [10, 13, 16]. Additionally, anti-SSA antibodies may be positive in some patients with psoriasis without such an association [17]. Sulphasalazine and phototherapy (Psoralen and ultraviolet-A (UV-A) light) for psoriasis has been linked to SLE development in isolated cases [13, 14, 18, 19]. Thus, Kullick *et al.*, recommend testing for anti-SSA antibodies in psoriasis patients before starting

phototherapy. Their positivity would contraindicate the treatment with UV-A rays [14].

The coexistence of SLE and psoriasis was reported without contributing factors [20], notably drug intake [4, 6]. In our case, the patient developed SLE after psoriasis although she didn't receive phototherapy and tested negative for anti-SSA antibodies.

This coexistence is a therapeutic challenge since antimalarial drugs (chloroquine and hydroxychloroquine), which are fundamental in SLE treatment, may induce or exacerbate psoriatic skin lesions [13, 21]. This drug would be responsible for severe forms of psoriasis; erythrodermic and pustular [22]. It could lead to its development in patients with a family history of psoriasis, or exacerbate pre-existing psoriatic lesions; but they would not induce the disease. The aggravating role of antimalarial drugs is reported in more than 18% of cases; they would have a nonspecific role in the stimulation of epidermal proliferation [23]. However, extension of the psoriatic lesions is only noted in 6.5 to 18% of cases with psoriatic arthritis treated by antimalarial drugs [24, 25].

In addition, prolonged corticosteroid therapy would lead to the development of generalized pustular and erythrodermic forms [26].

Methotrexate seems to be the drug of choice in SLE associated with extensive psoriasis worsened by antimalarial drugs and/or by the reduction of corticosteroids [27]. It should be introduced at a low weekly dose as soon as the corticosteroid tapering is initiated. Its effectiveness is indisputable since complete remissions are noted in 60% of cases [28]. The side effects of methotrexate are dominated by hepatotoxicity and hematotoxicity warranting regular clinical and laboratory monitoring [27].

CONCLUSION

Psoriasis and systemic lupus erythematosus are relatively common diseases in the population but their association is rare. Their coexistence poses a diagnostic and therapeutic challenge to the clinician. Therapeutic choices need careful consideration to avoid the exacerbation of any of these diseases.

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