Systemic Lupus Erythematosus with Coexistent Psoriasis Vulgaris: a Case Report

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ABSTRACT

We report a 42-year-old female with a 20-year history of systemic lupus erythematosus who subsequently developed psoriasis vulgaris. She has been on chronic, erratic prednisone and hydroxychloroquine intake prior to appearance of psoriatic lesions. Hydroxychloroquine and glucocorticoids are possible triggers of this phenomenon. Both diseases have a shared susceptibility loci and a shared Th17 pathophysiologic pathway. Treatment with methotrexate and selected biologics can target both disease mechanisms.

Key Words: psoriasis, lupus, Th17

Introduction

Systemic lupus erythematosus (SLE) is the prototypic multisystem autoimmune disorder with apparent shift to Th2 immune responses leading to B cell hyperactivity and production of autoantibodies. Psoriasis, on the other hand, is a predominantly Th1 immune response. Increased amounts of Th1 cytokines (IFN-γ and IL-2) are observed. The coexistence of SLE and psoriasisis is considered unusual. Worldwide, there have only been less than 200 published cases of such coexistence, and none locally.

Clinical Case

We have a 42-year-old female with a 20-year history of systemic lupus erythematosus (SLE) and subsequent one year history of psoriasis vulgaris which presented as erythematous, well-defined scaly plaques on the scalp and trunk, along with onycholysis, subungual hyperkeratosis, and oil spots on the nails of hands and feet (Figure 1). She has been on chronic and erratic prednisone intake and hydroxychloroquine prior to appearance of lesions. Histopathologic examination of the trunk lesion revealed parakeratosis with collection of neutrophils (Munros microabscess), hypogranulosis, psoriasiform epidermal hyperplasia with squared-off rete ridges, elongation of the dermal papillae with thin suprapapillary plates, and dilated superficial blood vessels (Figure 2). The findings were consistent with psoriasis. Of note, were the absence of LE changes such as vacuolar interface change, periadnexal infiltrates or increased mucin. Cutaneous lesions of lupus erythematosus were not appreciated. Other laboratory parameters (CBC, urinalysis, chemistry) were normal. ANA was positive; anti-dsDNA was negative.

The patient was seen by Rheumatology Section. Considering her SLE is inactive and her psoriasis is stable, prednisone was slowly tapered: 7.5 mg OD for 1 month, 5.0 mg OD for 1 month, 2.5 mg OD for 1 month until discontinuation. Monthly laboratory examinations (CBC, creatinine, and urinalysis) are required to monitor the status of nephritis. Hydroxychloroquine was maintained at a dose of 200 mg during this time. Tapering of both drugs is usually not done simultaneously as we run the risk of pushing the patient to a nephritic flare. Tapering of hydroxychloquine was eventually started. The goal was to manage her with methotrexate. No additional topical steroids were given. Emollients applied ad libitum was advised.

Discussion

The prevalence of SLE is 4.3 to 45.3 per 100,000.[1] in the Asia-Pacific and only approximately 1.1% of lupus patients had coexisting psoriasis.[2] Worldwide, there have only been less than 200 published cases of such coexistence and none in the Philippines.

Psoriasis and SLE belong to what is called an immunologic continuum proposed by McDermott and McGonagle (Figure 3). At one end of this spectrum are the autoinflammatory disorders and at the other end are the autoimmune disorders. SLE is the prototypical autoimmune disorder. Psoriasis is classified as a mixed pattern disorder with both autoinflammatory (early disease and flares) and

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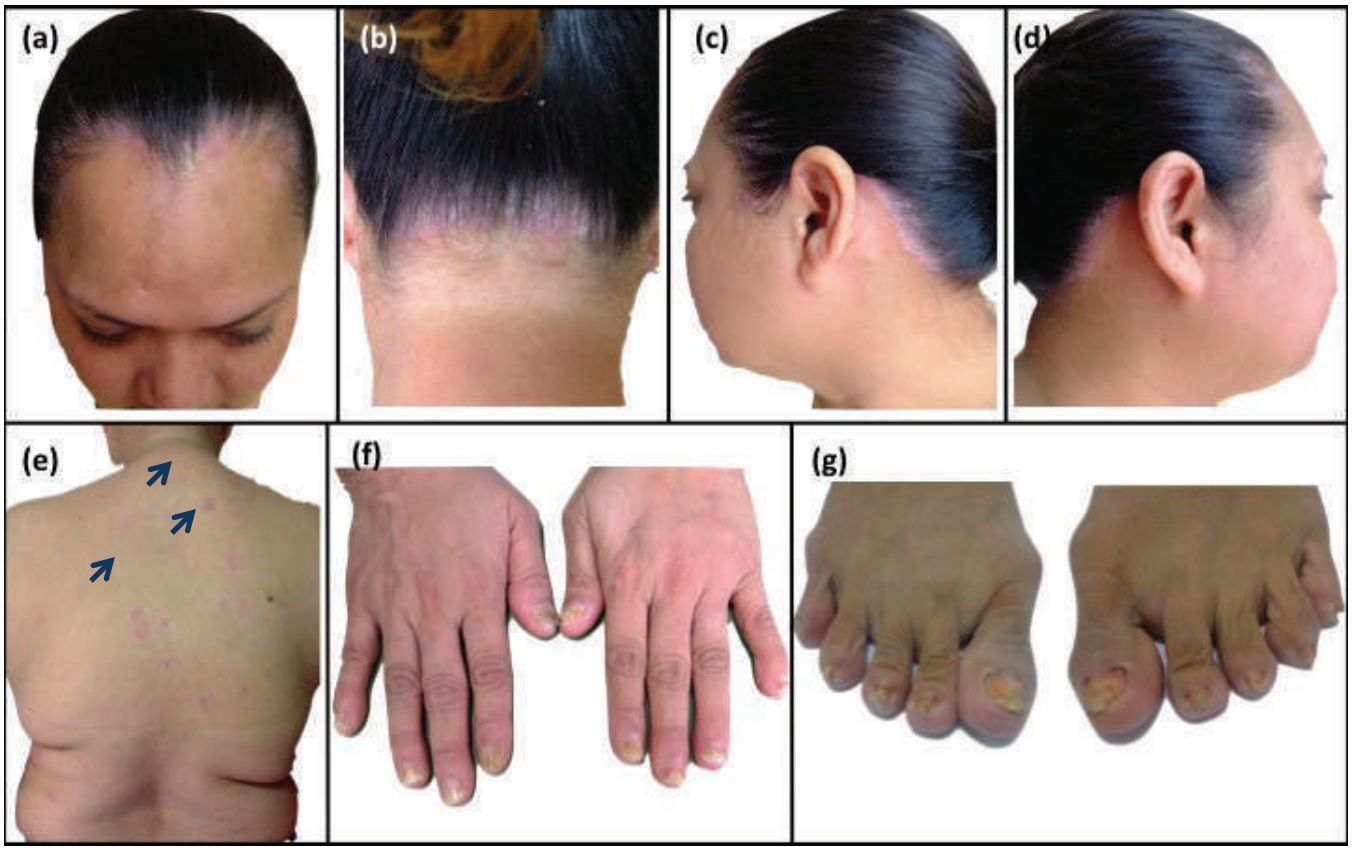

Figure 1. Cutaneous findings of psoriasis patient upon consultation. A-D. Erythematous well-defined scaly plaques on the frontal, occipital and parietal areas. E. Erythematous well-defined scaly plaques on the back where biopsy was done. F-G. Onycholysis, subungual hyperkeratosis and oil spots noted on the nails of the hands and feet.

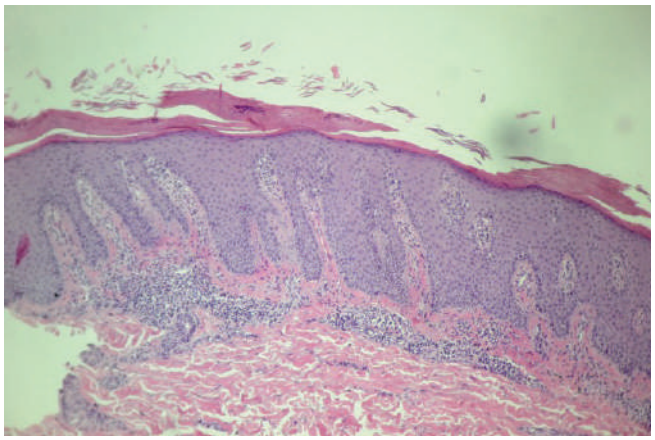

Figure 2. Histopathology of the lesion on the back, H&E stain, 10x magnification showing hyperkeratosis, hypogranulosis, psoriasiform epidermal hyperplasia and thinning of the dermal papilla.

autoimmune (plaque psoriasis) characteristics.[3] T lymphocytes in psoriatic lesions are polarized as T helper (Th) 1 and T cytotoxic subsets. Increased amounts of Th1 cytokines (interferon-gamma and interleukin 2) are observed. SLE is characterized by the apparent shift of Th 2 immune responses leading to the production of autoantibodies.[4] The shift is further supported by the findings of excessive Th 2 cytokines, notably interleukin (IL) 6 and IL-10.[4]

No established trigger for psoriasis in an SLE patient exists in literature as of this writing. The theories remain the same as with the general population. Several case reports have shown that steroid withdrawal causes flare of psoriasis, conversion to pustular forms and even induction of pustular psoriasis de novo.[5,6,7] Antimalarials have also been described to trigger the appearance of psoriasis and converted preexisting psoriasis to pustular and even erythrodermic forms.[8]

However, the development of both psoriasis and SLE in a patient is not impossible. Li and colleagues[9] identified two common susceptibility loci (rs8016947 and rs4649203) shared by SLE and psoriasis in the Chinese Han population using genome-wide association studies in multiple hospitals in China. The upregulation of the Th 17 immune pathway is a mechanism also shared by both diseases.[10] IL 17, produced by Th17 cells, is well known to play a major role in maintaining chronic inflammation in psoriasis.[9]

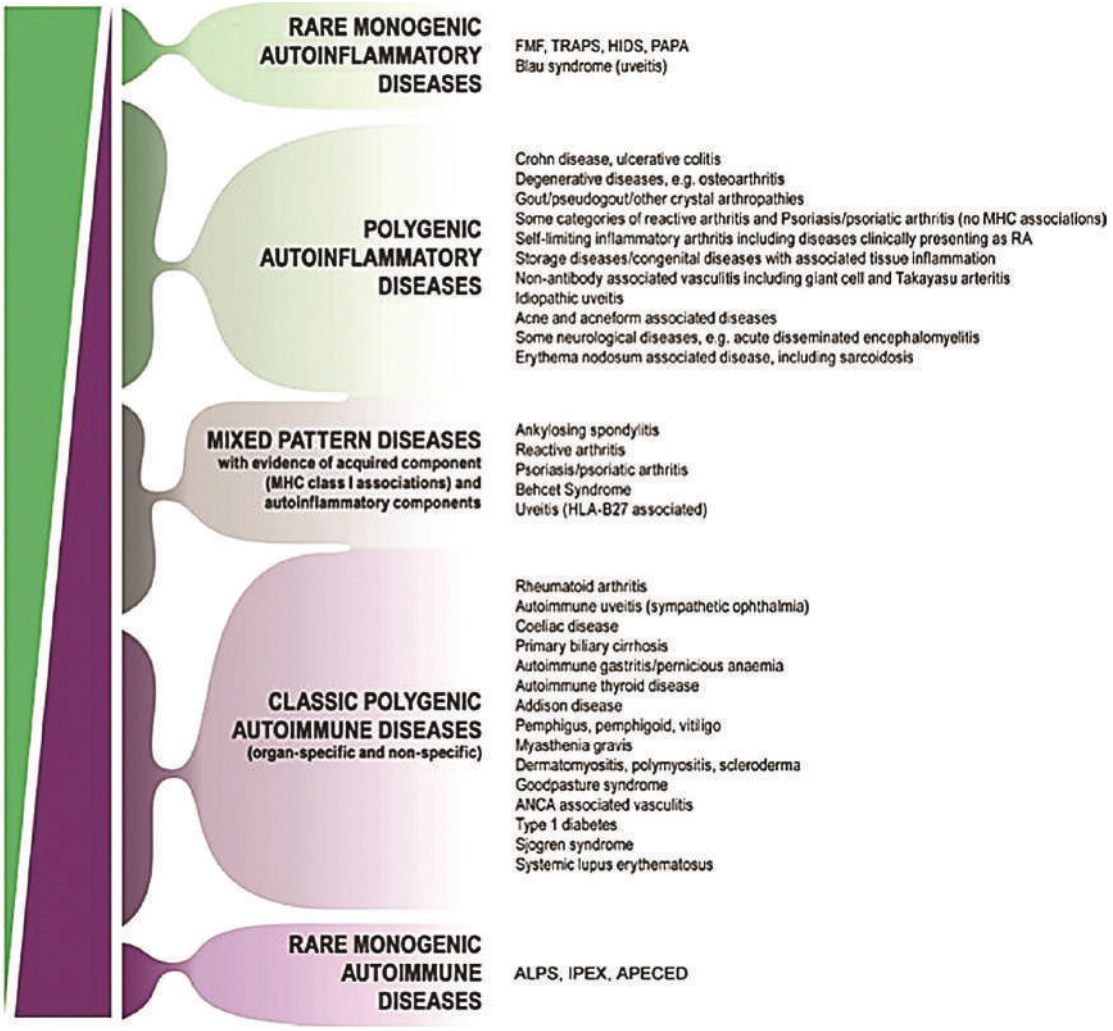

Figure 3. The immunologic continuum proposed by McDermott and McGonagle depicting autoinflammatory, autoimmune and mixed patter disorders. Lifted from McGonagle D, McDermott MF. A Proposed Classification of the Immunological Diseases. PLoS Med. 2006 Aug; 3(8):e297.

The proliferation of Th 17 cells is stimulated by IL 23.[11] Both IL 17 and IL 23 are elevated in SLE patients although their roles are still unknown.[12,13]

Perhaps the most important implication of the coexistence of these diseases lies in the management. The cornerstone of management for patients with SLE without major organ involvement include glucocorticoids, antimalarials, NSAIDs, and, in refractory cases, immunosuppressive agents.[14] In a subset with lupus nephritis like our patient, management includes the use of immunosuppressants. Phototherapy is avoided as it may have cutaneous effects. Exposure to sunlight is a well-known environmental factor in the induction and exacerbation of both cutaneous and systemic lupus erythematosus. UV light, especially UVB, is an important trigger in many patients with SLE.[4] In mild psoriasis, topical steroids are usually employed for control. For moderate to severe cases, phototherapy is recommended. Drugs such as methotrexate, cyclosporine, acitretin, sulfasalazine, mycophenolate mofetil, and biologicals are also included in the treatment. Steroids and antimalarials are generally not recommended because of the risk inducing rebound phenomenon and conversion to pustular psoriasis.

The goal is to achieve the lowest possible pharmacologic intervention that would possibly target both diseases. One viable option is methotrexate. For SLE without major organ involvement, methotrexate may be given for immuno-suppression. Concurrently, it is a first-line systemic therapy for psoriasis as it is highly efficacious for severe disease and all clinical variants of psoriasis. Targeted immune modulators or biologic therapies have become increasingly utilized in autoinflammatory and autoimmune diseases TNF-α inhibitors, ustekinumab, and abetacept have been shown to have promising effects on patients with both diseases.[10]

Statement of Authorship
All authors have approved the final version submitted.

Author Disclosure
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