Coexistence of Psoriatic Arthritis and Systemic Lupus Erythematosus

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Psoriasis is a common skin disease that affects 1–3% of the general population. Systemic lupus erythematosus is much less common than psoriasis. Its overall prevalence in the United States ranges between 14.6 and 50.8 cases/100,000 adults. Although SLE has been described in association with other autoimmune diseases such as rheumatoid arthritis, scleroderma, mixed connective tissue disease, autoimmune thyroid diseases, pernicious anemia and others [1], the coexistence of these two diseases is very rare [2-4]. A PubMed search of the English-language medical literature showed no previously published cases of psoriatic arthritis associated with SLE. We report the first two cases of patients suffering from both psoriatic arthritis and SLE.

Patient Description

Patient 1

A 52 year old white woman was diagnosed in 1980 as suffering from plaque-type psoriasis, based on typical clinical findings and a skin biopsy. She was treated with various topical glucocorticoid preparations and annual climatotherapy (bathing in Dead Sea water, mud therapy and exposure to sun rays) in the Dead Sea area. In 1990 she developed asymmetric polyarthritis for the first time, which affected several proximal interphalangeal joints and the wrists, and typical dactylitis of the second, third and fourth right toes. She also had small effusions in both knees. Serology tests for rheumatoid factor and antinuclear antibodies were negative. The serum levels of C3 and C4 complement factors were normal. The patient was treated with nonsteroidal anti-inflammatory drugs and was advised to bring forward her annual trip to the Dead Sea region for climatotherapy, a treatment that she had undergone every year for the previous 10 years.

The patient was forced to discontinue treatment at the Dead Sea when she developed a low grade fever, general weakness, and a diffuse erythematosus rash that first appeared on her neck and subsequently spread over her body with associated loss of hair. Her typical psoriatic skin lesions remained unchanged. She was admitted to the dermatology department. Positive findings on admission included temperature 37.3°C, marked hair loss, an acute maculopapular rash associated with typical plaque-like psoriatic lesions, arthritis in a few PIP joints, both wrists, knees and ankles, and dactylitis of the second, third and fourth right toes. The results of laboratory tests included a sedimentation rate of 100 mm in the first hour, a low serum hemoglobin concentration of 9.7 g/dl, and normal white blood cell and thrombocyte counts. Serum ANA was positive at a dilution of 1:640 and the titer of anti-dsDNA was high at > 200 IU/ml (normal < 20). Serum C3 and C4 complement components were low at 31 mg/dl and 4 mg/dl, respectively. Serology tests for RF, anti-Ro, anti-La, and VDRL were negative.

The patient also developed marked proteinuria with a 24 hour protein secretion of 2.1 g and active urine sediment. She refused kidney biopsy and was treated with high dose prednisone (1 mg/kg body weight). The response was good, with disappearance of the rash and gradual reduction of the proteinuria. Her C3, C4 and anti-dsDNA tests returned to normal values, while the ANA remained positive.

Since then the patient has received oral methotrexate therapy (12.5 mg/week) and low dose prednisone (5 mg/day). The psoriatic skin lesions continue to fluctuate.

Patient 2

A 40 year old Bedouin man presented to the clinic with a 4 week history of a painful right foot. At the age of 21 he was diagnosed with SLE on the basis of prolonged fever, symmetric polyarthritis of the hands and feet, membrano-proliferative glomerulonephritis, high titer of anti-dsDNA antibodies > 200 IU/ml, and low levels of C3 < 40 mg/dl and C4 < 10 mg/dl complement components. He was treated with oral cyclophosphamide and corticosteroids with a very good response. Cyclophosphamide therapy was discontinued after 18 months. His disease remained clinically quiescent, but the patient was lost to follow-up.

In 1995, 18 years after the diagnosis of SLE, the patient was admitted to the hospital because of anemia, symmetric arthritis of the metacarpal phalangeal and PIP joints, severe headache and low grade fever. On admission the serum hemoglobin concentration was 11.0 g/dl, white blood cell count 6050/mm^3, and platelet count 120,000/mm^3. A lumbar puncture revealed 38 white blood cells/mm^3 and 65% neutrophils. The cerebrospinal fluid protein and glucose levels were 196 mg/dl (normal 40–70) and 25 mg/dl (normal 20–45), respectively. A direct Gram stain as well as fungal and Ziehl-Nelson stains were all negative. Blood and CSF cultures

SLE = systemic lupus eythematosus

PIP = proximal interphalangeal
ANA = antinuclear antibodies
RF = rheumatoid factor

CSF = cerebrospinal fluid

were sterile. The ANA was positive (1:640) and the anti dsDNA titer was > 200 IU/ml. The patient was treated as active SLE with corticosteroids and hydroxychloroquine and had a good response. He was followed in the outpatient clinic and his disease remained clinically quiescent.

In 1998 a laparoscopic cholecystectomy was performed due to acute cholecystitis and cholelithiasis. After surgery he developed a classical psoriatic-like lesion of the umbilical area near the laparoscope insertion scar.

In 2003 he presented with painful toes on his right foot. Physical examination revealed dactylitis of his fourth and fifth right toes, arthritis in the second MCP, PIP and distal interphalangeal joints of his left hand and arthritis of the third PIP joint of the right hand. No other physical findings consistent with active SLE were found. The ANA was positive at 1:40 and the anti dsDNA titer was 40 IU/ml. The C3 and C4 levels were markedly low. The urine sediment was normal. He was diagnosed as suffering from psoriatic arthritis and improved following 2 months therapy with methotrexate.

Comment

Zalla and Muller [5] identified 42 cases of SLE among 9420 psoriasis patients in a 10 year retrospective study. They calculated the prevalence rate of psoriasis coexisting with SLE or other photosensitive disorders in their patient population as 0.69% of patients with psoriasis and 1.1% of patients with SLE. Female psoriasis patients developed SLE more often than males. In most reported cases psoriasis preceded the onset of SLE, but both diseases can appear concurrently and SLE can precede psoriasis.

The most common type of psoriasis that was associated with lupus was plaque-like and the majority of patients developed SLE, while the minority developed discoid lupus erythematosus only. In those cases in which psoriasis preceded SLE, as in our first patient described here, the trigger for the onset of SLE could have been the exposure to ultraviolet light in the Dead Sea area, which may have induced a severe photosensitivity reaction. PUVA (psoralen-ultraviolet A) therapy and sulphasalazine, which are used to treat psoriasis and psoriatic arthritis, may also induce SLE, but these were not given to our patient.

Antinuclear antibodies may appear in anti-tumor necrosis factor-treated patients. There have been a few reports of a lupus-like syndrome that resolves with cessation of therapy. Our first patient who suffered from psoriatic arthritis did not receive anti-TNF therapy.

However, SLE and psoriasis also have some common precipitating factors, such as infection, drugs and trauma. Our second patient presents a classic case of psoriasis and psoriatic arthritis precipitated by trauma (laparoscopic surgery). There are no specific serologic markers for this situation, but anti-Ro/SSA antibodies have been reported to occur in increased frequency in such cases.

We do not know for certain why the coexistence of these two disorders is so rare. It is unlikely that the existence of one (SLE or psoriatic arthritis) protects against the development of the other. It is possible that when a patient with SLE or psoriasis develops arthritis, it is mistakenly related to the underlying disease and an additional diagnosis is not sought.

In summary, the coexistence of SLE and psoriatic arthritis is very rare despite a few triggering factors that have the potential to precipitate each of these diseases. This coexistence probably represents a chance association.

MCP = metacarpal phalangeal

TNF = tumor necrosis factor

References

1. Lorber M, Gershwin ME, Shoenfeld Y. The coexistence of systemic lupus erythematosus with other autoimmune diseases: the kaleidoscope of autoimmunity. *Semin Arthritis Rheum* 1994;24:105–13.
2. Lynch WS, Roenigk HH,Jr. Lupus erythematosus and psoriasis vulgaris. *Cutis* 1978;21:511–16, 23–5.
3. Millns JL, Muller SA. The coexistence of psoriasis and lupus erythematosus. An analysis of 27 cases. *Arch Dermatol* 1980;116:658–63.
4. Hays SB, Camisa C, Luzar MJ. The coexistence of systemic lupus erythematosus and psoriasis. *J Am Acad Dermatol* 1984;10:619–22.
5. Zalla MJ, Muller SA. The coexistence of psoriasis with lupus erythematosus and other photosensitive disorders. *Acta Derm Venereol Suppl (Stockh)* 1996;195:1–15.

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