Lepromatous leprosy mimicking systemic lupus erythematosus

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Abstract

We describe a case of a 36-year-old woman who presented with bilateral symmetrical polyarthritis of the hands, malar rash, and discoid rash over three months. She was initially diagnosed and treated as systemic lupus erythematosus but later proven to have lepromatous leprosy. The patient showed a remarkable improvement after the administration of steroids. Due to typical features of a connective tissue disorder, a positive serological marker, photosensitivity, typical age of onset and initial response to treatment led to the confusion and delay in diagnosis of lepromatous leprosy. Further, this case emphasizes the importance of obtaining a detailed contact history and taking a skin biopsy even in a typical connective tissue lesion. Our case illustrates an unusual presentation of lepromatous leprosy mimicking systemic lupus erythematosus.

Key words: systemic lupus erythematosus, leprosy, vasculitis

Introduction

Leprosy, also known as Hansen disease, is a chronic infectious disease caused by *Mycobacterium leprae*. Although leprosy primarily involves skin and the peripheral nervous system, it gives rise to a wide range of clinical and serological manifestations. The clinical manifestations of leprosy include erythema nodosum, arthritis, fever, skin erythema, vasculitis,

epididymitis, glomerulonephritis, pericarditis, and pleurisy.^{1,2} Leprosy may also manifest with a variety of autoimmune phenomena resembling autoimmune diseases, such as systemic lupus erythematosus (SLE) or rheumatoid arthritis. Therefore, leprosy may mimic a variety of disease conditions.

Case description

A 36-year-old woman presented with low grade fever, bilateral symmetrical polyarthritis of hands with morning stiffness for three months. She also experienced photosensitivity. She had no significant past medical history. On examination, she had tender and swollen proximal interphalangeal joints (PIP) on both hands (Figure 1). There was evidence of subcutaneous indurations on both arms resembling scleroderma, which suggested the possibility of mixed connective tissue or overlap syndrome. A malar rash and a discoid rash behind the ears were observed. In addition, there was a desquamation rash over both soles (Figure 2). She also had mild to moderate splenomegaly.

A working diagnosis of SLE/overlap syndrome was made. Her blood picture revealed a erythrocyte sedimentation rate of 120 mm/1st hour, haemoglobin of 9.6 g/dL, with normal white cells and platelets. The blood picture revealed anaemia of chronic disorder. Rheumatoid factor was negative, but the anti-nuclear antibody (ANA) test was positive (1:160). Other serological markers, double stranded DNA (dsDNA), U1 small nuclear ribonucleoprotein, anti-centromere antibodies, and antitopoisomerase antibodies (Scl70) were negative.

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Figure 1. Swollen PIP joints.

Figure 2. Desquamation rash.

Her complement levels (C_3 , C_4) were normal. An ultrasound scan of her abdomen showed moderate splenomegaly. A skin biopsy from her upper arm was sent for histology. She was symptomatic with painful arthritis and a desquamation rash over both soles. She fulfilled the criteria for diagnosis of SLE and was started on aceclofenac sodium 100 mg twice a day, hydroxy-chloroquine 200 mg daily and prednisolone 30 mg daily. Her symptoms subsided over two weeks and the prednisolone dose was reduced to 10 mg daily in one month. However, a painless vasculitic rash appeared on both of her palms in one month. The skin biopsy had moderate to large number of acid-fast organisms within the foamy histiocytes keeping with borderline lepromatous leprosy (Figure 3,4). She was referred to the dermatology clinic for the commencement of multidrug therapy. She had good response to this therapy.

Figure 3. Skin biopsy showing a collection of foamy histiocytes in the dermis. Haematoxylin and eosin stain at x10.

Figure 4. Wade fite stain at 20X showing histiocytes containing multiple acid-fast bacilli.

Discussion

SLE is an autoimmune disorder characterized by antibodies to nuclear and cytoplasmic antigens, multi-system inflammation, protean clinical manifestations and a relapsing and remitting course. More than 90% of cases of SLE occur in women, frequently starting during childbearing age. The Systemic Lupus International Collaboration Clinics (SLICC) criteria for SLE classification require fulfillment of at least four criteria with at least one clinical criterion and one immunologic criterion or lupus nephritis as the sole clinical criterion in the presence of ANA or anti-dsDNA antibodies.³

Leprosy, a chronic granulomatous infection caused by *Mycobacterium leprae*, classically presents with cutaneous and neurological manifestations. Musculoskeletal involvement of leprosy is the third most common manifestation but is often underdiagnosed and underreported. The wrists, proximal interphalangeal joints, metacarpophalangeal joints, elbows, ankles, and knees can be affected. Arthritis due to leprosy can be divided into four types: Charcot joints, septic arthritis, acute polyarthritis of lepra reaction and chronic arthritis resembling rheumatoid arthritis.⁴ Acute polyarthritis of lepra reaction is acute in onset and affects the small joints of the hands and feet, with the arthritis settling down within a few weeks.⁴ Chronic arthritis manifests as symmetrical polyarthritis clinically identical to rheumatoid arthritis.^{4,5} Skin lesions related to leprosy can have varied and complex manifestations including macules, papules, nodules, psoriasis-like lesions, annular erythema, and pigmentation.⁶ At times, articular involvement may be the sole presenting manifestation without cutaneous lesions.

Autoantibodies can be detected in some cases of leprosy, which may result in the condition being easily mistaken for SLE.^{7,8} It has been reported that leprosy patients can be positive for RF, in addition to detection of other autoantibodies, including ANA, anti-Sjogren's syndrome related antibodies, and anti-dsDNA. The presence of autoantibodies in leprosy sera might be associated with the release of autoantigens due to tissue injury or the molecular mimicry of the pathogens that induce cross-reactivity.⁹ Our patient had positive ANA (1:160), features suggestive of chronic cutaneous lupus (discoid lupus), acute cutaneous lupus (malar rash), synovitis involving small joints of both hands and more than thirty minutes of morning stiffness. The diagnosis of SLE was made as above criteria were fulfilled in our patient. She initially showed signs of improvement with treatment of SLE.

However, the following features can be of help to differentiate leprosy from SLE: leprosy is more common among men, it often causes loss of eyebrows instead of alopecia and the associated peripheral nerve damage primarily occurs during the early stages of the disease. Reportedly, 55% of leprosy patients present with some degree of peripheral nerve damage at diagnosis,¹⁰ whereas only 3.9% of SLE patients present with such lesions.¹¹ A skin biopsy is useful when the definitive diagnosis is difficult. Wade fite staining might reveal the presence of acid-fast bacilli, while a positive lupus band test indicates SLE.

Conclusion

Our case illustrates an unusual presentation of lepromatous leprosy that was initially diagnosed as SLE. Recognition of rheumatic manifestations in leprosy is important as it may mimic SLE. Specifically, leprosy must be considered as a differential diagnosis in patients with unusual rheumatic manifestations and persistent skin lesions in an endemic area. The early differentiation between the two diseases is of utmost importance to institute appropriate treatment and reduce patient morbidity and mortality. It is necessary to increase the awareness of clinicians for early diagnosis of leprosy when the presentations are unusual.

Conflicts of Interest

None of the authors have financial or academic conflicts of interests.

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Case report

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