

Multibacillary leprosy with positive serology misdiagnosed as systemic lupus erythematosus

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Accepted 24 June 2022

DESCRIPTION

A woman in her 30s with no medical or family history was diagnosed with systemic lupus erythematosus (SLE) 3 months ago when she presented with alopecia, arthritis, photosensitivity, malar rash and diffuse maculopapular skin rash on her upper and lower extremities. Work-up revealed positive serology for SLE (positive antinuclear antibody (ANA) and antidouble-stranded DNA (anti-dsDNA) antibodies). Her complete blood count, urine analysis and urinary protein-creatinine ratio were within normal limits. She was started on hydroxychloroquine and prednisone 60 mg/day. Weeks later, she presented with worsening of her skin rash that extended to involve her whole body, tingling and numbness of her hands and feet, which was not present at the time of the initial presentation. On clinical examination, she had diffuse hair thinning and arthritis involving her wrists, metacarpophalangeal joints and knees. Her face showed diffuse erythema with nodular lesions on the cheeks and chin. Her trunk and extremities also showed diffuse erythematous nodular skin lesions (figure 1), and she had diminished sensation in all extremities. Based on her serology and physical findings, she fulfilled the Systemic Lupus International Collaboration Clinics classification criteria for SLE; however, nodular skin lesions were not typical of SLE or worsened on immunosuppressive therapy. Therefore, she was referred to the dermatology department for assessment. A skin biopsy was obtained, and pathology revealed diffuse infiltration of the dermis with foamy histiocytes, and Ziehl-Neelsen stain demonstrated acid-fast bacilli within the macrophages (figure 2). The diagnosis of multibacillary leprosy was confirmed with a skin biopsy, and the rheumatological manifestations



Figure 1 Erythematous nodular skin lesions involving the face, trunk and extremities.

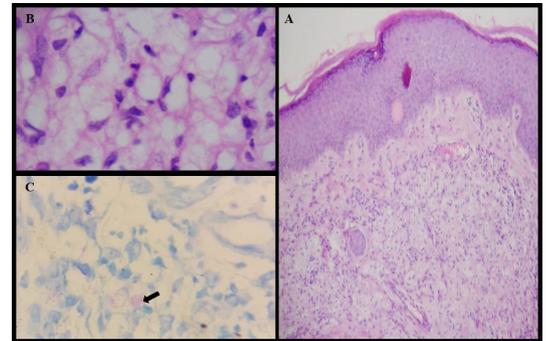


Figure 2 Skin biopsy: (A) demonstrating diffuse infiltrate of foamy histiocytes in the dermis separated from the epidermis by a green zone (H&E stain at 100× magnification), (B) dermis showing foamy histiocytes (H&E at 400× magnification) and (C) Ziehl-Neelsen stain at 400× magnification demonstrating acid-fast bacilli within macrophages, some of which are clumped (globi) (arrow).

and positive serology were attributed to leprosy. Hydroxychloroquine was discontinued, and prednisone was tapered over a period of 6 months. Antimycobacterium therapy with rifampin, dapsone and clofazimine was given for a year. During her follow-up, she had significant improvement in her skin nodules and arthritis.

Leprosy classically presents with neurological and skin manifestations followed by musculoskeletal involvement as Charcot's arthropathy and symmetrical polyarthritis.¹ Other reported manifestations include fever, vasculitis, epididymitis, glomerulonephritis, pericarditis and pleurisy.² Less common, yet clinically diverse presentations of leprosy include haemolytic anaemia, alopecia, photosensitivity, generalised lymphadenopathy, glomerulonephritis, serositis and Raynaud's phenomenon. Leprosy also includes a wide spectrum of laboratory manifestations because of the various immunological responses; a variety of auto-antibodies can be detected in leprosy patients such as ANA, anti-dsDNA, antimitochondrial antibodies, rheumatoid factor, antineutrophil cytoplasmic and antiphospholipid antibodies. It has been hypothesised that ANA presenting in leprosy patients results from weak cross-reactivity with complexed nucleic acids and nucleoproteins exposed after cell destruction in chronic inflammation.³ Other pathogenic mechanisms include molecular mimicry, and some studies reported shared idiotypes among antibodies derived from patients with leprosy and SLE.⁴

Previously published case reports of leprosy mimicking SLE, where the patients fulfilled



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To cite: Shoela S, Eshak N, Beltagy A, et al. *BMJ Case Rep* 2022;**15**:e251497. doi:10.1136/bcr-2022-251497

classification criteria for SLE, however, worsened on immunosuppressive treatment, the diagnosis of leprosy was eventually proven through skin biopsies, and patients clinically improved on the multidrug therapy for leprosy.^{2,3} Conversely, there are also reports of leprosy infection triggering lupus flare, in the form of arthritis, skin rash, lupus nephritis, pulmonary haemorrhage and autoimmune haemolytic anaemia.⁵

Learning points

- ▶ Chronic infections as leprosy, tuberculosis, HIV can induce positive antinuclear antibody, which is not necessarily an indication of autoimmune disease.
- ▶ Leprosy should be in the differential diagnosis in patients presenting with rash and neurological manifestations, whether sensory or motor, especially in endemic areas or travellers from endemic countries.
- ▶ Delayed diagnosis of leprosy may lead to a patient suffering irreversible structural damage.
- ▶ Immunosuppressive therapy has a role in the management of rheumatological manifestations of leprosy, but it must be given in conjunction with the standard multidrug therapy for leprosy.

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Contributors SS, NE, AB took care of the patient while being admitted to the hospital. MA wrote the manuscript. NE and MA revised the final manuscript. All the authors approved the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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