CASE REPORT

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Case report of a 28 years old woman with lepromatous leprosy mimicking systemic lupus erythematosus

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ABSTRACT

Background: Leprosy has been known as the greatest imitator disease that can mimic a disease condition, including autoimmune disease. This case report aims to demonstrate and remind healthcare providers that leprosy infection may manifest with various autoimmune phenomena reminiscent of classic autoimmune disease, including systemic lupus erythematosus (SLE), and to prevent delayed treatment and further complications.

Case report: A 28-year-old female patient with ulcers all over her body and amputation of her fingers and toes. Based on the positive antinuclear antibodies (ANA) test results, weak positive ANA profile band, anticardiolipin antibodies immunoglobulin M (ACA IgM), and complement C4 test, the patient has been diagnosed with systemic lupus erythematosus (SLE) since eleven years ago. There was no improvement during SLE therapy as the years passed. The patient developed leprosy symptoms such as madarosis, saddle nose, and facies leonine. Slit skin smear for leprosy acid-fast bacilli (AFB) from forehead and chin showed bacterial index +6, skin biopsy examination concluded lepromatous leprosy with bacterial index +5, and IgM anti-PGL-1 serological test examination 3185 u/ml. She was treated with steroids at the hospital and multidrug therapy for multibacillary leprosy (MDT MB) for one year. She had significant improvement after receiving medication.

Conclusion: The diagnosis of leprosy is delayed because of the clinical similarities between the disease and SLE. The clinicians should know leprosy-specific symptoms to avoid inaccurate diagnoses and treatment delays.

Keywords: lepromatous leprosy, leprosy, lupus, systemic lupus erythematosus.
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INTRODUCTION

Leprosy, Morbus Hansen or Hansen's disease, is a chronic infectious granulomatous disease caused by *Mycobacterium leprae* (*M. leprae*). *M. leprae* is an obligatory intracellular bacterium that affects peripheral nerves, skin, and other organs, liver, bones, muscles, and upper respiratory tract. *M. leprae* has a long incubation period, highly variable clinical manifestations, and also can manifest as an autoimmune disease such as systemic lupus erythematosus (SLE) or rheumatoid arthritis.[1–3]

Leprosy reaction occurred because T-cell reactivity to mycobacterial antigens induced random variation in its clinical features, resulting in misdiagnosis and delayed treatment.[2,3]

Indonesia is ranked third in the world, after India and Brazil, with 7.146 new leprosy cases in 2021. Until 2021, 6 provinces and 101 cities have not achieved leprosy elimination. The accuracy in diagnosis and early treatment is required to achieve zero leprosy cases in Indonesia by 2024. We reported a case of leprosy mimicking SLE, with clinical features including multiple face and body ulcers.

CASE DESCRIPTION

A 28-year-old woman from West Jakarta was referred to Sitanala General Hospital Emergency Room, a private hospital in West Jakarta, with chief complaints of many wound ulcers that had spread over her body for two weeks before being admitted to the hospital. Initially, the ulcers were only round and small all over her body, but over two years, the ulcers grew broader, deeper, and covered in pus all over her body. Her fingers and toes turned black and amputated. The patient was denied being diagnosed with leprosy on previous treatment. Instead, the patient has been diagnosed with systemic lupus erythematosus (SLE), an autoimmune disease, and is receiving routine corticosteroid therapy, but there has been no improvement.

The skin lesions began 15 years ago when the patient had numbed white spots on his calves, but she did not seek treatment because she assumed it was only tinea versicolor. After that, over the next 1.5 years, both ears began to feel numb, accompanied by complaints of fatigue, fever, and joint pain. Eleven years ago, the patient went to dr. Cipto Mangunkusumo General Hospital and the ANA test titer was 1/320, fine speckled pattern positive result, ACA IgM 48.7 MPL positive low - medium, ACA IgG negative, anti-double-stranded DNA within normal limits, complement C3 test was normal, and the complement C4 test was positive low (8 mg/dL). Based on laboratory results, the patient was diagnosed with SLE at RSCM hospital and received steroid therapy in

2012. There is no further investigation into the leprosy case because the ANA test and ACA test results were positive. The patient was subsequently routinely controlled at the private hospital in West Jakarta until a year ago. Three years ago, the patient independently conducted an ANA profile band test and obtained a weak positive result for borderline PCNA (+1), concluding that it only increased slightly or could be considered negative for SLE.

The physical examination at Sitanala Hospital revealed madarosis, saddle nose, facies leonine, ulcers covered in crusts in the malar area, multiple ulcers in the body region and upper and lower extremities, amputation of fingers II, III, IV, V bilateral, and amputation of toes II, III, IV, V bilateral (Figure 1). Based on laboratory evaluation found anemia (Hb 10.4 g/dl), hypoalbuminemia (2.2 g/dl), normal kidney function, results of blood aerobic resistance culture found *Staphylococcus aureus*, slit skin smear for leprosy results was BI:6, MI: 0. The skin biopsy results indicated Morbus Hansen with lepromatous leprosy type (LL-type MH) with a bacterial index of 5+ (Figure 2) and the results of IgM anti-phenolic glycolipid (PGL)-1 serological test examination is 3185 u/ml. The diagnosis of LL-type MH can be established following further consultation with internal medicine specialists, and it was determined that SLE could be ruled out. During her hospitalization, the patient received intravenous injections of methylprednisolone 125 mg every 6 hours, tapering off intravenously, omeprazole 40 milligrams every 12 hours intravenously, meropenem 1 gram every 8 hours intravenously, metronidazole 500 milligrams intravenously, albumin 1 flash every 24 hours intravenous drip.

The patient's condition improved after receiving treatment at dr. Sitanala General Hospital received MDT MB therapy from the public health center in West Jakarta for two weeks. The patient's body temperature and appetite improved, she could engage in mild activities, and the ulcers began to heal (Figure 3).

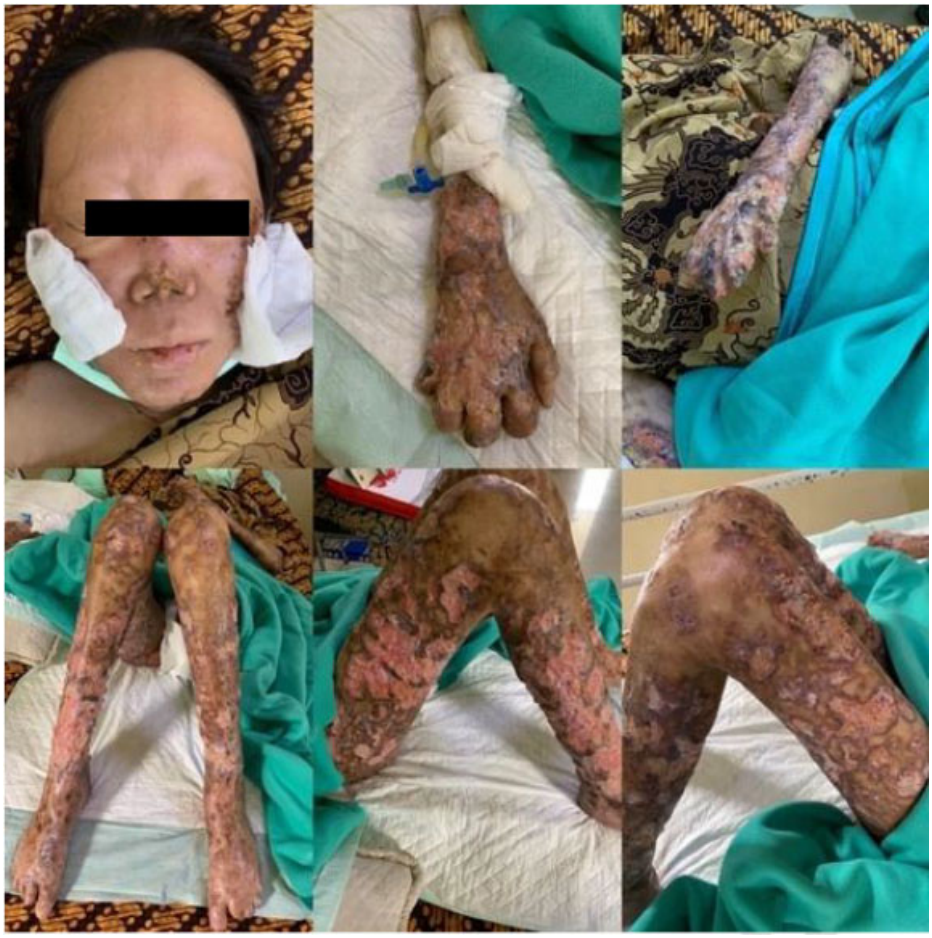

Figure 1. The physical examination of the patient at the initial assessment. Madarosis, saddle nose, and multiple ulcers with deformity on both arms and legs were seen.

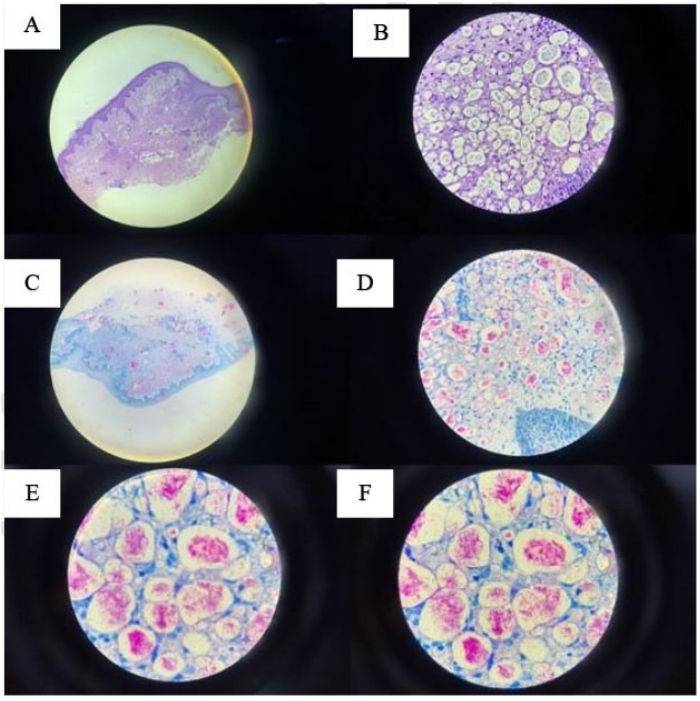

Figure 2. Skin biopsy results from dr. Sitanala General Hospital showed granulation with positive AFB (A-F).

DISCUSSION

M. leprae is transmitted through droplets from the nose and mouth or close contact

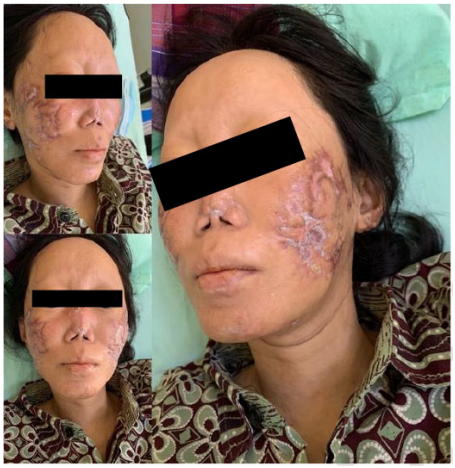

Figure 3. Post-therapy, after two weeks, showed improvement in lesions.

over months with someone with untreated leprosy. The long incubation period of *M. leprae* bacteria, and the variety of clinical manifestations complicate the diagnosis.[4–6]

Systemic lupus erythematosus (SLE) is a chronic inflammatory autoimmune disease with a wide range of clinical manifestations that primarily affects women of reproductive age and is associated with a high mortality rate. The accumulation of autoantibodies and immune complexes characterizes this disease, which causes inflammation and tissue damage. SLE is defined when a patient manifests a ten-point score, with the entry criterion of the antinuclear antibodies (ANA) test at a titer of 1/80 using The American College of Rheumatology (ACR) classification. This criterion also has been used in daily practice.

Clinicians should consider leprosy while making a differential diagnosis of SLE that has less typical clinical symptoms, especially if it is in an area where leprosy is endemic. The patient may complain of symptoms that are not specific for leprosy, such as malar rash, arthritis, photosensitivity, polyneuropathy, muscle weakness, generalized lymphadenopathy, hepatosplenomegaly, glomerulonephritis, and the presence of autoantibodies as occurs in patients with SLE. Although the clinical picture can resemble SLE, leprosy has characteristic symptoms that can help clinicians identify this disease, such as numb skin lesions, nerve numbness, and nerve pain in the nerves. This skin lesion can affect the peripheral nerves of the skin, especially the posterior tibial, ulnar, median, and peroneus common nerves. The similarity of skin lesions in leprosy and skin lesions in SLE makes it challenging to establish a diagnosis.[1,6]

This case report found only clinical criteria such as oral ulcers, discoid lupus erythematosus, and positive low C4. The authors relied on laboratory findings from ten years ago for the immunological criteria. Due to limited resources, laboratory tests re-examination could not be performed, and the ACR criteria cannot be used for this patient. Positive results from tests such as rheumatoid factor, ANA test, anti-syndrome, Sjogren's, and anti-double stranded DNA have been reported in cases of leprosy.[3,7]

Based on the findings of the anamnesis, physical examination, slit skin smear examination, laboratory examination, IgM anti-phenolic glycolipid (PGL)-1serological test examination, and histopathological examination, the diagnosis of Morbus Hansen with Lepromatous Leprosy type (MH LL) is achievable. The MDT-MB therapy can be administered for one year and evaluated monthly. The IgM anti-PGL-1 serological test examination in leprosy patients, according to Srihartati E *et al.* (2010), is instrumental in determining the diagnosis and evaluating the treatment of leprosy.[8]

In this case report, the patient had wound ulcers that spread over her body. One of the common complications in leprosy with peripheral nerve damage is acute or chronic ulcers based on the pathogenesis and consequences. However, the approach must be multidisciplinary in patients with chronic ulcers that are already ongoing. Other than the standardized leprosy chemotherapy, novel treatments such as applying stem cells (SC) could be an option. Based on a study conducted by Alinda *et al.* (2021), stem cell treatment such as adipocyte-derived mesenchymal stem cells-conditioned medium (ADMSC-CM) and amniotic membrane mesenchymal stem cells-conditioned medium (AMSC-CM), are potential therapeutic agents in the management of chronic ulcer in leprosy.[9]

The key learning from this case is that colleagues should always consider leprosy as a differential diagnosis in SLE because leprosy could induce clinical manifestations that mimic autoimmune diseases and other diseases.

CONCLUSION

Leprosy is a disease with various clinical manifestations (the greatest imitator disease) that can mimic an autoimmune disease, making the diagnosis and initial treatment challenging for medical practitioners, in this case, reporting the clinical features of leprosy mimicking SLE since ten years ago. The clinical features of leprosy, which can closely mimic many other diseases, should concern clinicians with a differential diagnosis of leprosy. Hence, the diagnosis and initial treatment are not too late, and the chain of transmission can be broken as quickly as possible.

CONFLICT OF INTEREST

None.

PATIENT'S CONSENT

The patient approved clinical documentation and publication.

AUTHORS CONTRIBUTION

All authors have contributed from patient examination, treatment, follow-up medication, reference finding, manuscript preparation, and publication.

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