CASE REPORT
Indonesia Journal of Biomedical Science (*IJBS*) 2026, Volume 20, Number 1: 12-17
P-ISSN.2085-4773, E-ISSN.2302-2906

Published by
Indonesia Journal of Biomedical Science

Indeterminate leprosy: case report and literature review

Luh Putu Venny Cempaka Sari[1,2], I Gusti Ayu Agung Dwi Karmila[1,2*], Herman Saputra[2,3], Handelia Pinari[1,2], Kevin Jonathan Djuanda[1,2], Ni Putu Wina Widyastuti[1,2], Mario Korwa[1,2]

[1]Dermatology and Venereology Department, Medical Faculty, Universitas Udayana, Denpasar, Bali
[2]Ngoerah Hospital, Denpasar, Bali
[3]Pathological Anatomy Department, Medical Faculty, Universitas Udayana, Denpasar, Bali

*Corresponding to:
I Gusti Ayu Agung Dwi Karmila; Dermatology and Venereology Department, Medical Faculty, Universitas Udayana, Denpasar, Bali
karmila@unud.ac.id
Phone: +62 819-9923-2930

Received: 2025-12-28
Accepted: 2026-02-06
Published: 2026-03-02

ABSTRACT

Background: The World Health Organization (WHO) states that Morbus Hansen (MH) or leprosy is one of seventeen neglected tropical diseases that require special global attention. Individuals infected with leprosy but who have not yet developed a cell-mediated immune response to the organism are classified as indeterminate type. If left untreated, an indeterminate type of leprosy can develop into the tuberculoid or lepromatous type. This case report aims to provide a clinical description of the indeterminate type of leprosy, to increase understanding and awareness of this condition.

Case: A 25-year-old man presented with white patches and numbness on his back for the past year, with a history of being diagnosed with leprosy at the age of 12 and completing leprosy therapy. Physical examination revealed multiple macular patches with decreased sensitivity, without peripheral nerve thickening. Slit skin smear examination found no acid-fast bacteria (AFB). Histopathological examination with Ziehl-Neelsen staining found the epidermis covered by weavy keratin and perivascular lymphohistiocytic patterns, partly following the neurovascular pathway in the superficial part, without finding any AFB. A diagnosis of indeterminate-type leprosy was established with multidrug therapy (MDT) of the paucibacillary (PB) type. Improvement in sensitivity without skin complications was noted after completing treatment.

Conclusion: Early detection and appropriate treatment of leprosy are essential to reduce transmission of *M. leprae* to close family members, especially household contacts. Continuous therapy and regular monitoring are crucial to improve treatment success.

Keywords: Indeterminate leprosy, leprosy, *Mycobacterium leprae*.
Cite this Article: Sari, L.P.V.C., Karmila, I.G.A.A.D., Saputra, H., Pinari, H., Djuanda, K.J., Widyastuti, N.P.W., Korwa, M. 2026. Cytoreductive nephrectomy in pregnant woman with renal cell carcinoma: a case report. *IJBS* 20(1): 12-17. DOI: 10.15562/ijbs.i20v1.659

INTRODUCTION

Hansen's disease, also known as leprosy, is a chronic infection caused by the bacterium *Mycobacterium leprae*.[1,2] These bacteria can infect the mucosal skin tissue and peripheral nerves, causing loss of sensation in the skin with or without dermatological lesions. Clinically, leprosy lesions can be macules or patches of hypopigmentation accompanied by anesthesia, or hypopigmented macules with raised and slightly erythematous edges, or erythematous infiltrates/plaques, or papules and nodules.[2]

The WHO reported data collected from 143 countries in 2016, namely 214,783 new cases of leprosy detected and 171,948 registered cases (prevalence rate of 0.23 per 10,000 population). Of the new cases found, the highest number was in Southeast Asia (115,180), followed by the Americas (26,365), Africa (21,465), and the rest in other regions of the world.[3,4] The prevalence rate of leprosy in Indonesia in 2023 is 0.63 cases per 10,000 population, and the rate of new cases is 5.2 cases per 100,000 population.[5]

Patients with strong cell-mediated immune responses typically exhibit clinical manifestations of few lesions with low or undetectable levels of mycobacteria, and are classified as tuberculoid type. In contrast, patients who are inactive (anergic) to M. leprae display lesions with higher levels of mycobacteria, and are classified as lepromatous type.[6-9] Patients who have not developed a cell-mediated immune response to the organism are classified as the Indeterminate type. If left untreated, Indeterminate leprosy can progress to the tuberculoid or lepromatous type.[7]

Leprosy is one of the infectious diseases that causes very complex problems. These problems are not only medical but also extend to social and economic issues.[10] Leprosy can cause disability during the course of the disease. Delayed early detection and treatment of this disease have been linked to an increased risk of disability in the eyes, hands, and feet.[1]

The following is a report of one case of indeterminate type leprosy in a 25-year-old male. This case report aims to discuss the risk factors, clinical manifestations, diagnosis, and management of Leprosy, particularly the indeterminate type. It is hoped that this case report will increase the understanding of health practitioners so that they can reduce morbidity and

mortality related to the disease and underlying risk factors.

CASE

A 25-year-old male patient, Javanese, Indonesian citizen, came to the Dermatology and Venereology Outpatient Clinic complaining of white spots on his back accompanied by numbness for 1 year. The white spots first appeared on his back and then appeared on his chest and remained (neither enlarging nor shrinking). There were no complaints of pain, itching, numbness in the hands and feet, or eyebrow hair loss.

The patient had a history of leprosy at the age of 12 and had completed multidrug therapy for multibacillary-type (MDT MB) children in 2011. At that time, the patient reported white and red patches on the body accompanied by numbness and loss of sensation in the hands and feet. After completing 12 months of treatment, the patient reported that the skin lesions had improved and there was no history of red patches or painful lumps on the skin. The patient denied any history of other systemic diseases, drug allergies, or food allergies. The patient's uncle and cousin have a history of leprosy. The patient also stated that the patient's family lives in the same house as the patient's uncle. The patient denied any history of other systemic diseases, drug allergies, or food allergies in the family. Physical examination and general status were found to be within normal limits. Dermatological status revealed multiple hypopigmented macules and patches in the anterior and posterior thoracoabdominal regions, with well-defined borders, some round and some geographic in shape, measuring 0.8–1.6 cm and 0.6 x 1.2 cm – 1.5 x 2 cm, were found to be discretely scattered, with a localized distribution (**Figure 1**). Sensitivity testing revealed decreased sensation of touch, pain, and temperature in the hypopigmented patches on the back. Nerve examination revealed no thickening or enlargement. Sensory testing with monofilament revealed normal results in the radial nerve, ulnar nerve, median nerve, common peroneal nerve, and bilateral posterior tibial nerve. Voluntary muscle testing (VMT) revealed motor strength of 5 in the radial nerve, ulnar nerve, median nerve, and bilateral posterior tibial nerve.

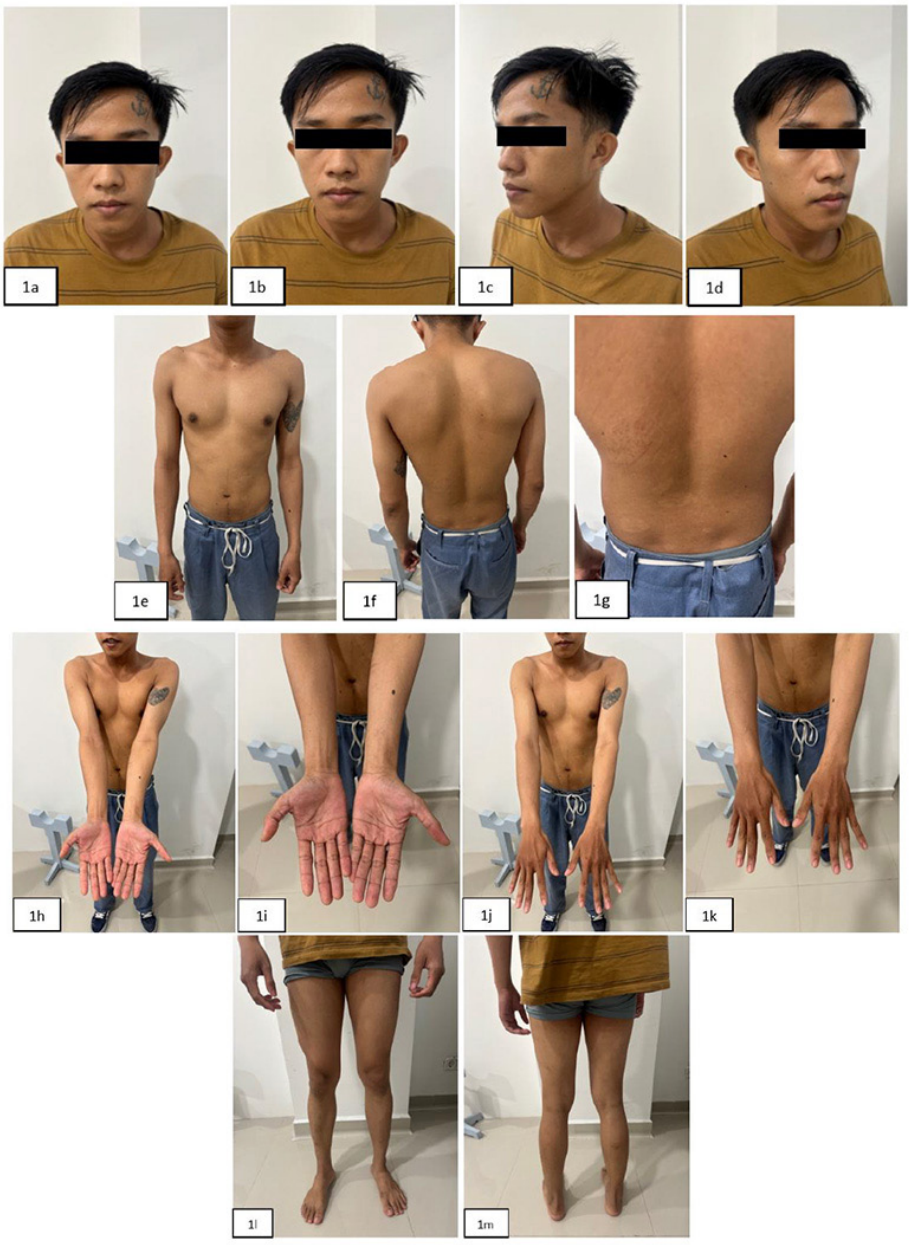

Figure 1. Clinical presentation during the initial visit (**a-m**) anterior and posterior thoracoabdominal region, showing multiple hypopigmented macular patches.

Skin slit smear examination revealed no acid-fast bacilli (AFB) in the right ear lobe, left ear lobe, or skin lesions. The complete blood count, liver function, random blood glucose, and renal function results were within normal limits. Histopathological examination of hypopigmentation patches in the posterior thoracoabdominal region. Hematoxylin-Eosin staining revealed skin tissue consisting of the epidermis, dermis, and its adnexa, and subcutaneous fat. The epidermis was covered by weavy keratin. In the superficial layer, a perivascular lymphohistiocytic pattern was observed, partly following the neurovascular pathways. No granuloma pattern was observed in this specimen. The morphological findings showed a superficial perivascular lymphohistiocytic pattern partly following the neurovascular pathways without a granuloma. In the Ziehl-Neelsen (ZN) histochemical stain, no acid-fast bacilli were found. The morphological findings are consistent with indeterminate-type leprosy (**Figure 2**).

Based on the medical history, physical examination, and supporting tests, the patient was diagnosed with indeterminate-type leprosy. The treatment given was multidrug therapy for paucibacillary-type leprosy (MDT-PB), consisting of rifampicin 600 mg once a month and dapsone 100 mg every 24 hours intraorally, with a treatment plan for 6 months, neurotropic vitamins B_1, B_6, and B_{12} 1 tablet every 24 hours intraorally, and 10% urea cream every 12 hours topically on dry skin. Patients were given explanations and education about the course of the disease, leprosy reactions, current treatment including its benefits and side effects, the 6-month MDT-PB treatment plan, disability and disability prevention (check, protect, and care), and contact tracing. After 6 courses of MDT-PB treatment, there was improvement in the sensitivity of the lesions, and no leprosy reactions were found. Repeat slit skin smear examination showed no acid-fast bacilli (AFB).

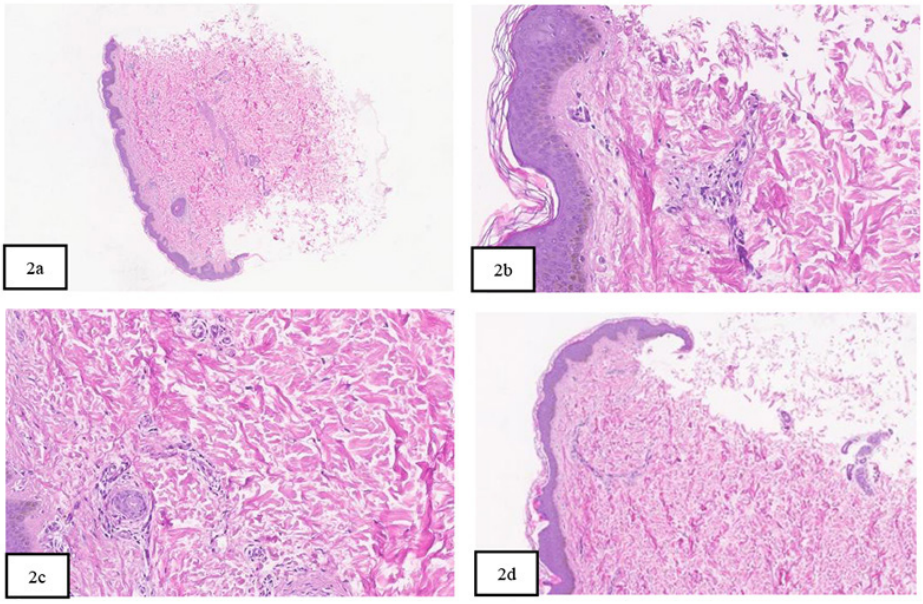

Figure 2. Histopathological examination of hypopigmentation lesions (**a-d**). The epidermis is covered by weavy keratin. In the superficial layer, there is a perivascular lymphohistiocytic pattern, partly following the neurovascular pathway.

DISCUSSION

Leprosy is a disease that attacks the skin and then develops into a secondary stage, causing peripheral neuropathy with the potential for long-term disability and stigma. This disease can cause physical disability if not treated properly, especially in productive age groups, which can interfere with functional status.[1,9]

Based on epidemiological studies, leprosy is commonly found in developing countries as a result of these countries' limited ability to provide adequate services in the areas of health, education, and social and economic welfare to their communities.[10,11] The global distribution of new cases of Leprosy in 2020 shows that the top five countries are, in order, India, Brazil, Indonesia, the Democratic Republic of Congo, and Bangladesh. The highest rates of detection of new cases of leprosy were also reported by countries in Africa and Southeast Asia. Of the 127 countries that reported data in 2020, India, Brazil, and Indonesia reported the highest number of new cases (>10,000 cases).[9] In Indonesia itself, the reported prevalence rate of leprosy in 2023 was 0.63 cases per 10,000 population, and the rate of new cases discovered was 5.2 cases per 100,000 population.[5] In terms of gender, men are more likely to develop leprosy than women. The average male-to-female ratio globally is reported to be 2:1. This condition can be caused by various factors, including differences in behavior and lifestyle. Men are associated with more outdoor activities, making them more susceptible to *M. leprae* infection, while women are more accustomed to taking care of themselves and maintaining their health. Men pay less attention to their own health and have difficulty accessing public health services.[12,13]

Regarding age, leprosy can occur in various age groups, but it is reported to be dominant in populations over 14 years of age.[14] In this case, the patient was a 25-year-old male. The patient is of Javanese ethnicity (Jember) and an Indonesian citizen. According to the literature, the patient falls into the category of groups with reported cases of leprosy, with males and those over 14 years of age reported as having a higher incidence of leprosy. In addition, Indonesia is also considered one of the countries with endemic leprosy.

M. leprae bacteria are obligate intracellular and aerobic. *M. leprae* are rod-shaped, measuring 1-8 µm x 0.5 µm, acid-fast (AFB) and alcohol-resistant, and are gram-positive bacteria.[1,15] Another causative agent is *Mycobacterium lepromatosis* (*M. lepromatosis*), which was described in 2008 in Mexico in a patient with fatal Virchowian disseminated leprosy, and was found to have a different deoxyribonucleic acid sequence from *M. leprae*.[16] However, despite the considerable genetic differences between these two species, their clinical manifestations and treatments are similar, and it is only possible to distinguish between them by performing molecular testing.[17] *M. leprae* reproduces more slowly than *M. lepromatosis*, with a generation time of approximately 12 to 13 days.11 This indicates that *M. leprae* has a long incubation period, which can range from 2 to 10 years after infection, depending on the form of the disease.[18]

M. leprae shows tropism for Schwann cells, keratinocytes, and macrophages, as well as a tendency to spread more efficiently in cooler areas of the body, such as nerves near the skin surface, the skin itself, and the membranes of the upper respiratory tract.[1] Humans are the primary carriers of *M. leprae* infection. The most common route of transmission of leprosy is through contact with droplets expelled from the upper respiratory tract of patients with multibacillary leprosy (MB). In addition, transmission can also occur through skin contact or vertical transmission.[7]

Genetic risk factors have been reported to be associated with leprosy. In general, genetic factors influence the transmission and clinical course of leprosy.[19] Single-nucleotide polymorphism (SNP) association studies indicate that the low lymphotoxin-α (LTA)-producing allele is the primary genetic risk factor for early-onset leprosy.[20] SNP or other genes associated with disease and/or reaction development in leprosy are the vitamin D receptor (VDR), tumor necrosis factor (TNF)-α, interleukin (IL)-10, interferon (IFN)-γ, human leukocyte antigen (HLA) genes, and toll-like receptor (TLR)-1.[21] Associative studies have identified polymorphic risk factors in the promoter region shared by two genes, namely parkinson (PARK)-2, which encodes an E3-ubiquitin ligase called Parkin, and the parkin coregulated gene (PACRG).[22] A study also showed that genetic variants of nucleotide-binding oligomerization domain-containing protein (NOD)-2 are associated with susceptibility to leprosy and the development of leprosy reactions (type I and type II).[23] In this case, there is a possibility of a genetic factor. This is because the patient's uncle and cousin have a history of leprosy.

Population migration from rural areas to cities contributes to an increase in cases of leprosy, where high population density can affect poor living conditions and hygiene. High density and poor hygiene can affect indoor air quality, thereby increasing the risk of transmission of leprosy, especially for people living together in one house. High intensity and short physical distance between people with leprosy increase the risk of leprosy.[24,25] In one systematic review study, it was reported that a history of close and prolonged contact, especially with multibacillary patients, males, and individuals over 60 years of age, is are factors that support the transmission of leprosy.[15] In addition, an individual's immunological status greatly influences the transmission and clinical manifestation of leprosy, which is mediated by the host's cellular immune system. Leprosy has many similarities with tuberculosis, in that tissue damage is not caused solely by the bacterial agent, but also by the individual's immune response to the *M. leprae* bacteria.[24] In addition to internal factors, there are several external environmental factors that can influence the occurrence of leprosy infection. High humidity is a condition favored by M. leprae. Although the bacteria can survive for 9 days in dry nasal secretions, M. leprae can survive for up to 46 days in moist soil..[24] In this case, there was a history of prolonged contact with a patient with leprosy. The patient said that his uncle and cousin had a history of leprosy and that the patient's family lived in the same house as the patient's uncle. Although the patient no longer lives in the same house, he said that his family visited him during the Eid al-Fitr holiday a year ago.

The two routes by which *M. leprae* leaves the human body that are often described are the skin and the nasal mucosa.[1] M. leprae is commonly found in the superficial keratin layer of the skin of lepromatous leprosy patients, indicating that the organism can be shed along with sebaceous secretions. The number of bacilli from nasal mucosal lesions in lepromatous cases ranges from 10,000 to 10,000,000. Nasal secretions from lepromatous patients can produce as many as 10,000,000 live organisms per day.[21] *M. leprae* then enters other human bodies through direct contact with the skin or nasal mucosa originating from droplets.[15] After infection occurs, clinical symptoms in the peripheral nerves or skin will appear in varying degrees. The shortest reported incubation period for leprosy is only a few weeks. This report is based on the very rare incidence of leprosy in neonates. The maximum incubation period reported is 30 years or more. It is generally agreed that the average incubation period for leprosy is between 3 and 10 years.[21] The clinical symptoms that arise also vary, depending on the cellular immunity of the patient. If the cellular immunity is competent, the clinical picture will be tuberculoid, whereas if the cellular immunity is impaired, the clinical picture will be lepromatous.[7]

The clinical manifestations of leprosy can generally take the form of macules or patches of hypopigmentation with anesthesia, or hypopigmented macules accompanied by raised and slightly erythematous edges, or erythematous infiltrates/plaques, or papules and nodules.[2] There are various classification systems used in cases of leprosy, but the most commonly used are the World Health Organization (WHO) classification and the Ridley and Jopling classification.[2] Based on the WHO classification system, leprosy is categorized into two types, paucibacillary (PB) and multibacillary (MB). The PB-type is characterized by fewer than or equal to 5 lesions, a negative bacterial index, and involvement of one nerve. Meanwhile, the MB-type is defined as leprosy lesions on the skin that number more than or equal to 6 lesions, a positive bacterial index, or involvement of two or more nerves.[1,6] The Ridley and Jopling classification system classifies leprosy into tuberculoid leprosy (TT); borderline tuberculoid (BT); borderline (BB); borderline lepromatous (BL) and lepromatous leprosy (LL).[6,7,14] The New Indian Classification in 1981 classified leprosy into several types, indeterminate (I), tuberculoid (T), borderline (B), lepromatous (L), and polyneuritis (P). Specifically, the indeterminate-type generally occurs in patients who have not developed a cell-mediated immune response to *M. leprae*. The first skin lesions that appear in the indeterminate-type are small to medium-sized hypopigmented patches, mostly located on the thighs and face, but can also be found on other parts of the body, with blurred edges and accompanied by complaints of loss of sensation. In addition, hair growth and nerve function are not involved, so there are no common manifestations such as madarosis or peripheral nerve thickening.[8,21] In this case, the patient complained of white patches on the chest and back that appeared several years ago. The white patches first appeared on the back and then on the chest. The white patches were said to be persistent, neither enlarging nor shrinking. The patient reported numbness in the white patches on the back, without itching or pain in the affected areas.

The clinical manifestations of leprosy are not limited to the skin, but can also involve the nerves.[1] The most common neurological manifestation of leprosy is peripheral nerve damage accompanying skin lesions, especially in the skin nerve fibers and nerve trunks. The pattern and distribution of nerve damage are

influenced by the number of bacteria infiltrating the nerves, as well as the patient's immunological response to the infected nerves. Based on this, the clinical manifestations of peripheral nerve damage can be classified into sensory disorders, motor disorders, and autonomic disorders.[7,9] Leprosy generally involves the superficial nervous system, such as the auricularis magnus, medianus, ulnaris, sural, posterior tibialis, and superficial peroneal nerves.[10] In this case, sensitivity examination revealed a decrease in touch, pain, and temperature sensation in the hypopigmented patches on the back. Nerve examination did not reveal any thickening or enlargement. Sensory examination with monofilament and motor examination with VMT were within normal limits in the radial nerve, ulnar nerve, median nerve, and bilateral posterior tibial nerve.

A diagnosis of leprosy can be made if the following criteria are met: (1) hypopigmented or erythematous skin lesions or red patches of skin with loss of sensation; (2) thickened or enlarged peripheral nerves with loss of sensation and/or weakness of the muscles innervated by those nerves; and (3) skin smears showing acid-fast or bacilli observed in skin smears/biopsies. The presence of all three criteria indicates a diagnostic accuracy of 95%.[7,26] Various tests can be useful in confirming the diagnosis of leprosy. These tests include measuring antibody titers against *Mycobacterium* antigens with rapid lateral flow devices; cell-mediated cytokine release assays (such as IFN-γ detection, similar to commercial complete blood tests used to detect infection by *M. tuberculosis*); amplification of *Mycobacterium* DNA with polymerase chain reactions (PCR); or the use of metabolomics to detect molecular features specific to *M. leprae* infection in blood or urine.[1] Histopathological examination with skin biopsy can be performed to confirm the diagnosis of leprosy. Skin biopsies should include the dermis layer and, if possible, extend to the subcutaneous layer of the lesion. Hematoxylin-eosin staining should be supplemented with the Fite-Faraco staining method or another method to detect acid-fast bacilli.[7]

However, there are differences in establishing a diagnosis of indeterminate-type leprosy. Specifically, in indeterminate leprosy, the diagnosis is confirmed by biopsy showing perineurovascular infiltration. Acid-fast bacilli are not commonly found on histopathological examination.[9,27] Other histopathological findings include focal, perivascular, perianaxial, and perineural lymphohistiocytic inflammatory infiltrates. In the lepromin test, indeterminate-type leprosy will show a negative result, so it is considered PB type for treatment purposes.[27] In this case, a slit skin smear examination showed no acid-fast bacilli (AFB) in the right ear lobe, left ear lobe, or skin lesions. Histopathological examination with Hematoxylin-Eosin staining showed superficial perivascular lymphohistiocytosis partially following the neurovascular pathway without granuloma. Ziehl Neelsen (ZN) histochemical staining did not reveal acid-fast bacilli. The morphological findings were consistent with indeterminate-type leprosy.

The management of leprosy includes pharmacological and non-pharmacological treatments.10 Pharmacological therapy includes MDT recommended by the WHO to treat leprosy based on the PB and MB-types. For treatment purposes, indeterminate-type leprosy is considered PB-type based on lepromin test results.[27] There are three MDT options that are currently available: for PB-type (MDT-PB) and MB type (MDT-MB). The adult MDT-PB package for single lesions includes rifampicin 600 mg plus ofloxacin 400 mg, and minocycline 100 mg (ROM) single dose. The adult MDT-PB package consists of rifampicin 600 mg and dapsone 100 mg taken once a month under supervision and dapson 100 mg once a day taken independently. Six packages are given, to be completed within 6-9 months. In general, MDT containing rifampicin has a bactericidal mechanism of action, while other anti-leprosy drugs are bacteriostatic.[6,22] In this case, the patient was given multidrug therapy for paucibacillary-type (MDT PB) package and completed the 6-month treatment plan.

The clinical course of leprosy is associated with the development of disabilities, particularly in the eyes, hands, and feet, affecting both soft tissue and bone, which leads to disability and deformity. Leprosy also causes social stigma.[1,8-12] However, in indeterminate-type leprosy, it has been reported that approximately 75% of lesions heal spontaneously. Additionally, the prognosis for this type of leprosy is very good, and leprosy reactions have never been reported.[8] In this case, the *Mycobacterium* species was not examined through PCR testing. However, this case was traced through histopathological examination until the completion of treatment.

CONCLUSION

Indeterminate-type of leprosy is an early form of leprosy that is not yet stable, characterized by pale white patches (hypopigmentation) or redness on numb skin, often appearing in people with compromised immunity, and can heal spontaneously or develop into another type of leprosy depending on the body's immune response. Although it often heals on its own, early diagnosis and treatment with MDT-PB for 6 months is considered better to prevent this type of leprosy from progressing to tuberculoid or lepromatous leprosy.

CONFLICT OF INTEREST

There is no conflict of interest on the part of the author regarding the publication of this case report.

ETHICS IN PUBLICATION

The patient has received information and signed an informed consent form to use the patient's medical information and clinical photographs for educational and scientific publication purposes.

FUNDING

There was no funding sponsorship for the care, examination, and treatment of patients, nor for the publication of this case report.

AUTHORS CONTRIBUTION

The LPVCS author is responsible for patient examination, care, and

treatment, as well as literature review and manuscript preparation. The IGAADK author is responsible for publication correspondence. The HS author is responsible for supervising histopathological examination. The authors of the HP, KJD, NPWW, and MK are responsible for patient care.

GENERATIVE AI DISCLOSURE

There is no usage of artificial intelligence (AI) in the manuscript preparation.

REFERENCES

1. Salgado CG, de Brito AC, Salgado UI, Spencer JS. Leprosy. In: Kang S, Amagi M, Bruckmer AL, Enk AH, Margolis DJ, McMichael AJ, Orringer JS. Fitzpatrick's Dermatology, 9th ed. McGraw-Hill Education. 2019:2892–924.
2. Alinda MD, Geani S, Agusni RI, Kusumaputra BH, Reza NR, Prakoeswa CRS, Listiawan MY. Diagnosis and Management of Leprosy. Periodical of Dermatology and Venereology. 2020;32(2):149-157. DOI: 10.20473/bikk.V32.2.2020.149-157
3. World Health Organization. Guidelines for the Diagnosis, Treatment, and Prevention of Leprosy. New Delhi: WHO. 2018:1-87.
4. Kementerian Kesehatan Republik Indonesia. Pedoman Nasional Pelayanan Kedokteran Tata Laksana Kusta. Jakarta: Kementerian Kesehatan Republik Indonesia. 2020:4–8. (Bahasa)
5. Kementerian Kesehatan Republik Indonesia. Profil Kesehatan Indonesia Tahun 2023. Jakarta: Kementerian Kesehatan Republik Indonesia. 2024:186–92. (Bahasa)
6. Riyaz N, Sehgal VN. Leprosy: Trophic Skin Ulcers. Skinmed. 2017;15(1):45–51.
7. Maymone MBC, Laughter M, Venkatesh S, Dacso MM, Rao PN, Stryjewska BM, Hugh J, Dellavalle RP. Dunnick CA. Leprosy: Clinical aspects and diagnostic techniques. J Am Acad Dermatol. 2020;83(1):1–14. DOI: 10.1016/j.jaad.2019.12.080
8. Alrehaili J. Leprosy Classification, Clinical Features, Epidemiology, and Host Immunological Responses: Failure of Eradication in 2023. Cureus. 2023;15(9):1–11. DOI: 10.7759/cureus.44767
9. Chen KH, Lin CY, Su SB, Chen KT. Leprosy: A Review of Epidemiology, Clinical Diagnosis, and Management. J Trop Med. 2022;2022(7):1–13. DOI: 10.1155/2022/8652062
10. Gracino ME, dos Santos BM, Garbelin MV, Nishi V, Silva D, Kondo RN, Mariano RD. Leprosy: A systematic review. International Journal of Dermatological Science. 2020;2(1):8-21. DOI: 10.33545/26649772.2020.v2.i1a.10
11. World Health Organization. Global Leprosy Strategy 2016-2020: Accelerating towards a Leprosy-Free World. Monitoring and Evaluation Guide [Internet]. World Health Organization. 2016 [Cited on November, 24th 2025]. Available on: https://apps.who.int/iris/handle/10665/208824
12. Martoreli Júnior JF, Ramos ACV, Alves JD, Crispim J de A, Alves LS, Berra TZ, Barbosa TP, da Costa FBP, Alves YM, dos Santos MS, Gomes D, Yamamura M, Pinto IC, Fuentealba-Torres MA, Nues C, Pieri FM, Arcoverde MAM, dos Santos FL, Arcêncio RA. Inequality of gender, age and disabilities due to leprosy and trends in a hyperendemic metropolis: Evidence from an eleven-year time series study in Central-West Brazil. PLoS Negl Trop Dis. 2021;15(11):e0009941. DOI: 10.1371/journal.pntd.0009941
13. Liu YY, Yu MW, Ning Y, Wang H. A study on gender differences in newly detected leprosy cases in Sichuan, China, 2000-2015. Int J Dermatol. Desember 2018;57(12):1492–9. DOI: 10.1111/ijd.14148
14. Prakoeswa CRS, Lubis RS, Anum Q, Argentina F, Menaldi SL, Gunawan H, Yuniati R, Mulianto NR, Siswati AS, Widasmara D, Rusyati LMM, Mamuaja EH, Muchtar V, Agusni RI, Kusumaputra BH, Alinda MD, Listiawan MY. Epidemiology of Leprosy in Indonesia: a Retrospective Study. Periodical of Dermatology and Venereology. 30 Maret 2022;34(1):29–35. DOI: 10.20473/bikk.V34.1.2022.29-35
15. Bratschi MW, Steinmann P, Wickenden A, Gillis TP. Current knowledge on Mycobacterium leprae transmission: a systematic literature review. Lepr Rev. 2015;86(2):142–55.
16. Sharma R, Singh P, McCoy RC, Lenz SM, Donovan K, Ochoa MT, Estrada-Garcia I, Silva-Miranda M, Cruz FJS, Balagon MF, Stryjewska B, Scollard DM, Pena MT, Lahiri R, Williams DL, Truman RW, Adams LB. Isolation of Mycobacterium lepromatosis and Development of Molecular Diagnostic Assays to Distinguish Mycobacterium leprae and M. lepromatosis. Clinical Infectious Diseases. 2020;71(8):262–9. DOI: 10.1093/cid/ciz1121
17. Avanzi C, Singh P, Truman RW, Suffys PN. Molecular epidemiology of leprosy: An update. Infection, Genetics and Evolution. 2020;86(12):1–11. DOI: 10.1016/j.meegid.2020.104581
18. Reibel F, Cambau E, Aubry A. Update on the epidemiology, diagnosis, and treatment of leprosy. Med Mal Infect. 2015;45(9):383–93. DOI: 10.1016/j.medmal.2015.09.002
19. Alter A, Alcaïs A, Abel L, Schurr E. Leprosy as a genetic model for susceptibility to common infectious diseases. Hum Genet. 2008;123(3):227–35. DOI: 10.1007/s00439-008-0474-z
20. Alcaïs A, Alter A, Antoni G, Orlova M, Van Thuc N, Singh M, Vanderborght PR, Kotach K, Mira MT, Vu HT, Ngyuen TH, Nguyen NB, Moraes M, Mehra N, Schurr E, Albel L. Stepwise replication identifies a low-producing lymphotoxin-α allele as a major risk factor for early-onset leprosy. Nat Genet. 2007;39(4):517–22. DOI: 10.1038/ng2000
21. Bhat RM, Prakash C. Leprosy: an overview of pathophysiology. Interdiscip Perspect Infect Dis. 2012;12(1):1–13. DOI: 10.1155/2012/181089
22. Mira MT, Alcaïs A, Nguyen VT, Moraes MO, Di Flumeri C, Vu HT, Mai CP, Nguyen TH, Nguyen NB, Pham XK, Sarno EN, Alter A, Montpetit A, Moraes ME, Moraes JR, Doré C, Gallant CJ, Lepage P, Verner A, Van De Vosse E, Hudson TJ, Abel L, Schurr E. Susceptibility to leprosy is associated with PARK2 and PACRG. Nature. 2004;427(6975):636-40. DOI: 10.1038/nature02326
23. Berrington WR, Macdonald M, Khadge S, Sapkota BR, Janer M, Hagge DA, Kaplan G, Hawn TR. Common polymorphisms in the NOD2 gene region are associated with leprosy and its reactive states. J Infect Dis. 2010;201(9):1422-35. DOI: 10.1086/651559
24. Joshi P. Epidemiology of Leprosy. In: Kumar B, Kar HK, editors. IAL Textbook of Leprosy, 2nd ed. New Delhi: Jaypee Brothers Medical Publishers. 2017:33–50.
25. Leano HA de M, Araújo KM da FA, Bueno I de C, Niitsuma ENA, Lana FCF. Socioeconomic factors related to leprosy: an integrative literature review. Rev Bras Enferm. 2019;72:1405–15. DOI: 10.1590/0034-7167-2017-0651
26. Alemu Belachew W, Naafs B. Leprosy: diagnosis, treatment and follow-up. Journal of the European Academy of Dermatology and Venereology. 2019;33(7):1205–13. DOI: 10.1111/jdv.15569
27. Gomes AP, Miguel PSB, Martins e Mafra FB, De Moura ACL, Braga LM. Leprosy: A clinical review. Brazilian Journal of Health and Biomedical Sciences. 2022;20(2):151–60. DOI: 10.12957/bjhbs.2021.63968