

Leprosy, Type 2 Diabetes, and Peripheral Neuropathy: A Complex Case of Diabetic Foot Ulcer

Zubia Suhail¹, Saima Askari²

ABSTRACT

Background: Peripheral neuropathy is a significant factor in the development of neuropathic foot ulcers, commonly associated with conditions like type 2 diabetes mellitus and leprosy. These ulcers often result from sustained pressure at localized points, compounded by compromised circulation and delayed wound healing. Leprosy, though uncommon, should be considered in the differential diagnosis of neuropathic ulcers, especially in endemic areas.

Case Presentation: An 80-year-old male with a history of type 2 diabetes mellitus, hypertension, ischemic heart disease, and newly diagnosed leprosy presented with a diabetic foot ulcer. The ulcer had been present for 3 months following nail trauma. Clinical examination revealed a BMI of 24.67 kg/m², bilateral ankle-brachial pressure index (ABPI) of 2.5, and a Vibratory Perception Threshold (VPT) of 50. Angiography showed significant atherosclerotic disease, and the patient underwent angioplasty of stenotic segments in the peroneal artery. Despite Charcot foot deformity on radiography, surgery was not required at this early stage.

Management: The patient was treated with a basal-bolus insulin regimen for strict diabetes control, along with broad-spectrum antibiotics (oral clindamycin and ciprofloxacin). Wound debridement and dressing changes were performed daily. After 4 weeks of antibiotic therapy, moxifloxacin continued for 2 weeks. During treatment, the patient developed hypopigmented lesions, leading to a diagnosis of indeterminate leprosy. Multi-drug therapy (rifampicin, dapsone, clofazimine) was initiated. The diabetic foot ulcer healed within 8 weeks with proper treatment and offloading.

Conclusion: This case underscores the importance of considering leprosy in the differential diagnosis of neuropathic foot ulcers, especially in endemic regions. Effective management, including glycemic control, wound care, and multi-drug therapy, is essential to prevent complications and promote healing.

KEYWORDS: Diabetic foot ulcer, Charcot foot, Leprosy, Peripheral neuropathy.

INTRODUCTION

Type 2 diabetes mellitus and leprosy are both established etiological factors for peripheral neuropathy, which predispose individuals to the development of neuropathic foot ulcers. These ulcers often arise due to sustained pressure at localized points, compounded by compromised peripheral circulation, especially in the feet, which are distal

from the heart, resulting in delayed wound healing. Chronic wounds can progress to complications such as osteomyelitis and Charcot foot deformities, further hindering wound recovery.¹ Diagnosing peripheral neuropathy remains complex; however, a comprehensive approach involving detailed clinical evaluation, laboratory investigations, and, when necessary, nerve conduction studies is critical in establishing an accurate diagnosis.²

Address for Correspondence: Dr. Zubia Suhail, MBBS
RMO Foot Clinic,
Baqai Institute of Diabetology & Endocrinology,
Karachi - Pakistan.
E-mail: zubiasuhail@yahoo.com.

Submitted: September 20, 2024 **Revision Received:** October 05, 2024

Accepted for Publication: October 17, 2024

Access this Article Online

URL:
<https://jpes.org.pk/index.php/jpes/article/view/29>

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

How to cite this: Suhail Z, Askari S. Leprosy, Type 2 Diabetes, and Peripheral Neuropathy: A Complex Case of Diabetic Foot Ulcer. JPES. 2024;1(2):87-90.

Leprosy (Hansen's disease) is a curable, tropical, chronic infectious disease caused by the acid-fast bacilli *Mycobacterium leprae* and *Mycobacterium lepromatosis*. It predominantly affects regions in South Asia, Africa, and the Americas, where it remains endemic. The primary mode of transmission is respiratory, via inhalation of aerosols or nasal droplets from individuals with prolonged exposure to the bacteria.³ Clinically, leprosy presents in polar forms, classified into Paucibacillary and Multibacillary categories. According to Ridley and Jopling's classification, leprosy is further subdivided into Tuberculoid, Borderline Tuberculoid, Mid Borderline, Borderline Lepromatous, and Lepromatous forms based on immunological and histological features.⁴

This case report highlights a patient with a known history of type 2 diabetes, newly diagnosed with leprosy, who was presented with a diabetic foot ulcer and radiographic evidence of Charcot foot deformity.

CASE HISTORY

An 80-year-old man with a history of type 2 diabetes mellitus, hypertension, smoking, and ischemic heart disease (IHD), residing in Hyderabad, presented to the Foot Clinic at the Baqai Institute of Diabetology and Endocrinology in Karachi with a right foot ulcer that had been present for last 3 months, following nail trauma. He was vitally stable, with a BMI of 24.67 kg/m², bilateral ankle-brachial pressure index (ABPI) of 2.5, and a Vibratory Perception Threshold (VPT) of 50. Biochemical tests are shown in Table-I.

Considering increased ABI and history of IHD, angiography was done, and it showed significant atherosclerotic disease in visualized vessels, more marked in the calf vessels and angioplasty of multilevel stenotic segments of the peroneal artery was done with partial improvement.

In terms of treating the patient effectively, a basal bolus insulin regimen was started for strict control of diabetes along with broad-spectrum antibiotic therapy which includes oral clindamycin 300 mg every 6 hours for gram-positive coverage and oral ciprofloxacin 500 mg every 12 hours for gram-negative coverage. This regimen was continued for 4 weeks, followed by 2 weeks of moxifloxacin. In addition to the antibiotics, he received wound debridement and dressing changes. Cleaning of the foot ulcer with the help of 0.9% normal saline and debridement was done. Although the patient had Charcot foot changes as visible on the x-ray (Fig 1), he wasn't treated with any surgical procedure as these changes were not deforming the foot structure and were still in the mild stages of the disease. However, complete bed rest along with proper offloading with the help of footwear was advised and a total duration of 8 weeks was taken for proper healing of the foot ulcer.

While undergoing treatment for the diabetic foot ulcer, the patient developed bilateral pedal swelling, which was initially thought to be due to prolonged bed rest (to avoid additional pressure or strain on the ulcer) or his previous history of ischemic heart disease. After

ruling out underlying heart failure, he was advised to elevate his legs, which provided some relief. However, after some time, small hypopigmented lesions appeared on his arms, legs, back, and ears. Physical examination revealed tactile hypoesthesia with no superficial nerve thickening or pain. The lesions had poorly defined borders and displayed a dry, wrinkled appearance. A dermatological consultation was promptly arranged for further evaluation and a punch biopsy of the hypopigmented lesions was sent which showed a granulomatous – inflammatory – infiltrate – consisting of scattered histiocytes lymphocytes. A diagnosis of indeterminate leprosy was made and multi-drug therapy was started which includes rifampicin, 600mg once a month; dapsone, 100mg daily; and clofazimine, 300mg once a month and 50 mg daily for 6 months. This multi drug therapy is aimed at preventing resistance to *M. leprae* by single-drug treatment and to prevent relapse.

The diabetic foot ulcer has healed with appropriate treatment, and the patient's swelling has shown significant improvement following the prescribed interventions. The patient is advised to continue the multidrug therapy for leprosy, as per the standard regimen, to prevent relapse and resistance. Ongoing management, including glycemic control and regular monitoring of foot care, is essential to ensure continued recovery and prevent further complications.

DISCUSSION

This case report highlights the management complexities of an 80-year-old male with type 2 diabetes mellitus, hypertension, ischemic heart disease, and a right foot ulcer due to trauma from a nail injury. His presentation is not uncommon in diabetic patients, where foot ulcers are a significant complication. However, this case stands out because of the development of hypopigmented lesions during the



Fig.1: X-ray of left foot AP and lateral view showing mild osteomyelitic changes from 2nd to 5th metatarsal head.

Clinical Investigations:

Table-I: Clinical investigations performed in the outpatient department.

Urea	43 mg/dl	10-50 mg/dl
LDL-Cholesterol	140 mg/dl	Without Known Coronary Artery Disease - <= 130 mg/dl With Known Coronary Artery Disease - <= 100 mg/dl Normal (for diagnosis) - < 5.7 %
Hemoglobin A1c (HbA1c)	7.20%	Pre-Diabetes (for diagnosis) - 5.7 - 6.4 % Likely Diabetes (for diagnosis) - >= 6.5 %
Hemoglobin (Hb)	10.8 g/dl	14.7-16.3 g/dl
Hematocrit (HCT /PCV)	35%	41.9-48.7 %
Red Blood Cell (R.B.C)	5.59 million/ul	4.5-6.5 million/ul
Mean Corpuscular Volume (MCV)	62 fl	76-96 fl
Mean Corpuscular Hemoglobin (MCH)	19 Pg	26-32 Pg
Mean Corpuscular Hemoglobin Concentration (MCHC)	31 g/dl	32-36 g/dl
Red blood cell distribution width (RDW)	14.10%	< 15.0 %
T.L.C	11.3 x 1000/ul	4.0-11.0 x 1000/ul
Neutrophils Count	86%	40-75 %
Lymphocytes Count	7%	20-45 %
Eosinophils Count	1%	1-6 %
Monocytes Count	6%	2-8 %
Basophil Count	%	<= 1 %
Platelets Count	271 x 1000/ul	150-400 x 1000/ul
Band Cells	%	0-4 %
Creatinine (Serum)	0.78 mg/dl	0.9-1.3 mg/dl
Estimated glomerular filtration rate	77 mL/min/1.73 m2	
Sodium	134 meq/l	136-145 meq/l
Potassium	4.45 meq/l	3.8-5.2 meq/l
Chloride	105 meq/l	96-107 meq/l
Bicarbonate	26 meq/l	22-29 meq/l

treatment of diabetic foot ulcers, raising the possibility of leprosy, an uncommon but important consideration in certain regions like Pakistan.

Foot Ulcers in Diabetic Patients: Diabetic foot ulcers are well-documented in the literature as a common complication of type 2 diabetes mellitus and standard management of diabetic foot ulcers includes antibiotic therapy, wound debridement, and offloading.⁵ Similarly, our patient received empirical antibiotic treatment and required frequent wound care and debridement, underscoring the importance of maintaining a clean wound bed to facilitate healing.

Neuropathy and Ulcer Progression: The pathophysiology behind diabetic foot ulcers often includes neuropathy, which was a key factor in the patient's ulcer progression in our case and neuropathy, especially in elderly diabetic patients, often goes unnoticed due to a lack of pain sensation.⁶ Moreover, neuropathy in diabetic patients leads to delayed recognition of foot injuries, which further complicates wound healing.⁷ These findings align with our case, where the patient's foot ulcer progressed as a result of sensory neuropathy.

Coexistence of Leprosy and Diabetes: Leprosy is a chronic inflammatory disease and remains a public health problem in many developing countries. The global leprosy strategy 2021-2030 calls for accelerating action to reach the goal of ZERO LEPROSY. According to WHO guidance on interruption of transmission and elimination of leprosy disease 2023 there is an explicit 3-phase pathway which includes interruption of transmission, elimination of leprosy disease and post-elimination surveillance.⁸ The possibility of leprosy complicating a diabetic foot ulcer is not frequently discussed in the context of routine clinical care as it is a relatively uncommon but important consideration in endemic regions. A case report described a patient with both diabetes and leprosy who developed foot ulcers and Charcot neuroarthropathy,⁹ like the current case. Their management also involved multidrug therapy for leprosy (rifampicin, dapsone, and clofazimine) in conjunction with wound care. In our case, early diagnosis of leprosy was crucial for appropriate treatment to avoid progression and prevent complications.

Management of Charcot Foot: Charcot foot, another complicated factor in our patient, is a recognized complication in diabetic patients with neuropathy. There are several stages of Charcot neuroarthropathic foot ulcer determined with the help of different imaging modalities that are helpful in planning treatment. In severe cases Charcot foot can be treated surgically with the help of osteotomy, surgical debridement and arthroplasty. The management of Charcot neuroarthropathy involved therapeutic strategies such as offloading and the use of specialized footwear, which were implemented in our case to mitigate further joint degeneration and prevent additional structural damage.¹⁰ Unlike severe cases that require surgical intervention,¹¹ of our patient's Charcot changes were mild, and surgery was not needed, demonstrating the importance of conservative management in the early stages.

CONCLUSION

This case report highlights the significant association between peripheral neuropathy, potentially due to diabetes or leprosy, in the development of diabetic foot ulcers. Despite leprosy being uncommon, it is an important consideration in neuropathic foot ulcers in endemic areas.

Recommendations:

- Clinicians should consider leprosy as a potential cause of peripheral neuropathy in patients with diabetes, especially in endemic areas.
- Early detection through clinical examination and diagnostic tests is essential for timely intervention.
- Self-awareness programs and foot care education should be prioritized to improve patient outcomes and reduce the risk of ulcer progression.
- Comprehensive management, including proper glycemic control, multidrug therapy for leprosy, and the use of offloading devices, should be implemented to prevent complications and promote healing.

Grant Support & Financial Disclosures: None.

REFERENCES

1. Faramarzi MR, Farid GA, Babamiri B, Lotfy H, Goli R, Faraji N, et al. Management of an infected wound complicated by osteomyelitis secondary to neuropathy caused by previous leprosy; successful treatment with gauze ribbon, multivitamins, and maggot therapy. *Clinical Infection in Practice*. 2024 Jul 1;23:100370.
2. Yusuf S. Case Report: Delayed Diagnosis of Leprosy-Related Neuropathic Ulcer, Insights from a Case of Delay to Diagnose across Four Clinical Settings. *F1000Research*. 2024 Oct 11;13:1211.
3. Bhukhan A, Dunn C, Nathoo R. Case Report of Leprosy in Central Florida, USA. 2022. *Emerging Infectious Diseases*. 2023;29(8):1698-1700. doi:10.3201/eid2908.220367.
4. Jariyakulwong N, Julianon N, Saengboonmee C. Lepromatous leprosy with a suspected 30-year incubation period: A case report of a practically eradicated area. *Journal of Taibah University Medical Sciences*. 2022 Aug 1;17(4):602-5.
5. Bus SA, Armstrong DG, Gooday C, Jarl G, Caravaggi C, Viswanathan V, Lazzarini PA, International Working Group on the Diabetic Foot (IWGDF). Guidelines on offloading foot ulcers in persons with diabetes (IWGDF 2019 update). *Diabetes/metabolism research and reviews*. 2020 Mar;36:e3274.
6. Pop-Busui R, Ang L, Boulton AJ, Feldman EL, Marcus RL, Mizokami-Stout K, Singleton JR, Ziegler D. Diagnosis & treatment of painful diabetic peripheral neuropathy. *American Diabetes Association*, 2022: 1-32.
7. Sharma S, Rastogi A. Genetic Signature for the Causation of Charcot Neuro-osteoarthropathy of Foot in Diabetes: A Systematic Review. *The International Journal of Lower Extremity Wounds*. 2024 May 6:15347346241252549.
8. Jariyakulwong N, Julianon N, Saengboonmee C. Lepromatous leprosy with a suspected 30-year incubation period: A case report of a practically eradicated area. *Journal of Taibah University Medical Sciences*. 2022 Aug 1;17(4):602-5. (<https://www.sciencedirect.com/science/article/pii/S1658361222000245>)
9. Hsu JH, Wu YH, Hsiao PF. Histoid leprosy complicated with Charcot neuroarthropathy: A case report. *Dermatologica Sinica*. 2021 Jul 1;39(3):137-8.

AUTHORS:

1. Dr. Zubia Suhail, MBBS
RMO Foot Clinic,
Baqai Institute of Diabetology & Endocrinology,
Karachi - Pakistan.
2. Dr. Saima Askari, MBBS, FCPS Medicine & Endocrinology,
Consultant Endocrinologist & Assistant Professor,
Baqai Institute of Diabetology & Endocrinology,
Baqai Medical University, Karachi - Pakistan.