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Leprosy Trophic Ulcer Management

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ABSTRACT

Background: Leprosy is a chronic granulomatous infection caused by *Mycobacterium leprae*, also a polymorphic disease with a wide range of neurocutaneous manifestations. Unfortunately, delayed diagnosis and treatment are still an issue in endemic poor resource settings and in non-endemic countries due to global migration. Ulcer is not a common feature in leprosy patients, except during reactional states, Lucio's phenomenon (LP), or secondary to neuropathies. **Objective:** To study the treatment options to manage leprosy trophic ulcer. **Method:** The Google-scholar, Science-Direct database, and ResearchGate from 2010 until September 2023 were searched using the keywords "leprosy ulcer", "leprosy trophic ulcer", "plantar leprosy ulcer", and "leprosy ulcer management." All available cohort studies, case-series, case-reports, and expert reviews were included with an emphasis on leprosy trophic ulcer management. **Results**: The trophic ulcer evolves initially from a trauma/deep fissure/callosity or tenderness over pressure-bearing areas of palms and soles. Anesthesia of the foot is the central factor in the pathogenesis of plantar ulcer. Sufficient rest for a simple ulcer is essential for ulcer healing. Topical treatment to promote wound healing includes phenytoin, zinc oxide, platelet-rich plasma (PRP), platelet-rich fibrin (PRF), and hydrocolloid dressing. Various surgical methods are available for management of plantar ulcers, including skin grafts and different types of flaps. In primary care settings, early detection, patient education, and self-care practices are essential in preventing severe ulcerations and recurrence. A clinical sign that can be suspected is the stage of threatened ulcer, called the pre-ulcerative with aseptic inflammation, usually occurring under a joint or a bony prominence of the foot and characterized by edematous lesions. Initial management in primary care including wound care and protection, limiting mobility, and reducing heavy loads on the wound area, especially in the lower extremities. **Conclusion:** Leprosy trophic ulcer management includes sufficient rest, topical treatment to promote wound healing, and various surgical methods.

Keywords: *leprosy; trophic ulcer; wound healing*

BACKGROUND

Leprosy is a chronic granulomatous infection caused by *Mycobacterium leprae*, also a polymorphic disease with a wide range of neurocutaneous manifestations, which usually appear after a long period of incubation (3–7 years). Clinical suspicion after careful examination of skin and neural alterations is of utmost importance for the diagnosis of leprosy. Unfortunately, delayed diagnosis and treatment are still an issue in endemic poor resource settings and in non-endemic countries due to global migration. Ulcer is not a common feature in leprosy patients, except during reactional states, Lucio's phenomenon (LP), or secondary to neuropathies[1]. The trophic ulcer evolves initially from a trauma/deep fissure/callosity or there may be tenderness over pressure-bearing areas of palms and soles. The area breaks down into a frank painless ulcer discharging serosanguineous fluid[2]. Delayed diagnosis or misdiagnosis contributes to advanced disease and irreversible disabilities[1].

RESEARCH METHODS

The Google-scholar, Science-Direct database, and ResearchGate from 2010 until September 2023 were searched using the keywords "leprosy ulcer", "leprosy trophic ulcer", "plantar leprosy ulcer", and "leprosy ulcer management." All available retrospective and prospective studies, case-series, case-reports, and expert reviews were included with an emphasis on leprosy trophic ulcer management. The observations were analysed to find the procedures and efficacy of leprosy trophic ulcer management.

RESULTS

Pathogenesis of Leprosy Trophic Ulcer

Anesthesia of the foot is the central factor in the pathogenesis of plantar ulcers. Plantar anesthesia, unprotected walking, poor quality of scar formation resulting from previous ulceration, excessive load on the scar, and persisting foci of infection are some of the main factors for the recurrence of plantar ulcers. The pathogenesis of plantar trophic ulcers has been extensively investigated. Five mechanisms that produce damage to insensitive tissues include: (1) continuous pressure, causing necrosis due to lack of blood supply; (2) concentrated high pressure, causing cutting/

crushing by mechanical violence; (3) heat/cold, causing burning or frost bite; (4) repetitive mechanical stress of moderate degree, causing inflammation and autolysis; and (5) pressure on the infected tissue, resulting in spread of infection. Ten percent of the ulcers arise from perceived or unperceived neglected injuries resulting in infection and tissue damage, and 5% arise from neglect of deep cracks in the dry, anhidrotic, and hyperkeratotic skin of the sole or from infection underneath[3]. The pathogenesis of plantar ulcers is unique and includes three stages. The pre-ulcerative stage of aseptic inflammation is characterized by increased stress exerted over a period that gives rise to a traumatic (aseptic) inflammation in the subcutaneous layer of the sole, which is most vulnerable to mechanical stress. This usually occurs under a joint or a bony prominence just distal to the head of a metatarsal. The stage of concealed ulcer is the stage of the necrotic blister. The inflamed site undergoes necrosis due to the stress of continued walking, with the subcutaneous tissue undergoing necrosis. The liquefied tissue mixed with blood is forced to the surface by continued walking to present as a blister. With the formation of the necrotic blister, the destruction of the subcutaneous tissue is complete. The stage of overt or open ulcer is the stage when the skin overlying the blister breaks open and the necrotic area becomes exteriorized[3].

Leprosy Trophic Ulcer Management

Sufficient rest for simple ulcer (non-infected, not deeper than dermis) is essential for ulcer healing. Rest the wounds to prevent progression. This is especially important for plantar blisters/ulcers. This can be achieved by walking with the help of crutch or canes[4].

The mechanism by which phenytoin accelerates wound healing is unknown. Clinical, animal, and *in vitro* studies suggest that phenytoin may be involved in the healing process at several levels including stimulating fibroblast proliferation, enhancing the formation of granulation tissue, decreasing collagenase activity (by reducing its production or secretion or both), promoting deposition of collagen and other connective tissue components, decreasing bacterial contamination, and decreasing wound exudate. Biopsies of phenytoin treated open wounds show neovascularization, collagenization, and decreased polymorphonuclear and eosinophil cell infiltration[5]. Sehgal et al used a 100-mg quantity of phenytoin sodium (10 tablets of 10 mg each) crushed to a fine powder using a mortar and pestle, then mixed with 10 g of zinc oxide (ZnO) paste. The quantity of ZnO in the paste was 24.0–26.0%. After 4 weeks of treatment with topical phenytoin sodium zinc oxide paste, it showed the appearance of uneven, red granulation tissue progressing to healing (Fig. 1). Sehgal et al found that phenytoin sodium zinc oxide paste was found to be an efficacious, cost-effective, and well-tolerated alternative therapy. ZnO paste may decrease bacterial infections and assist in the promotion of epithelialization[6].

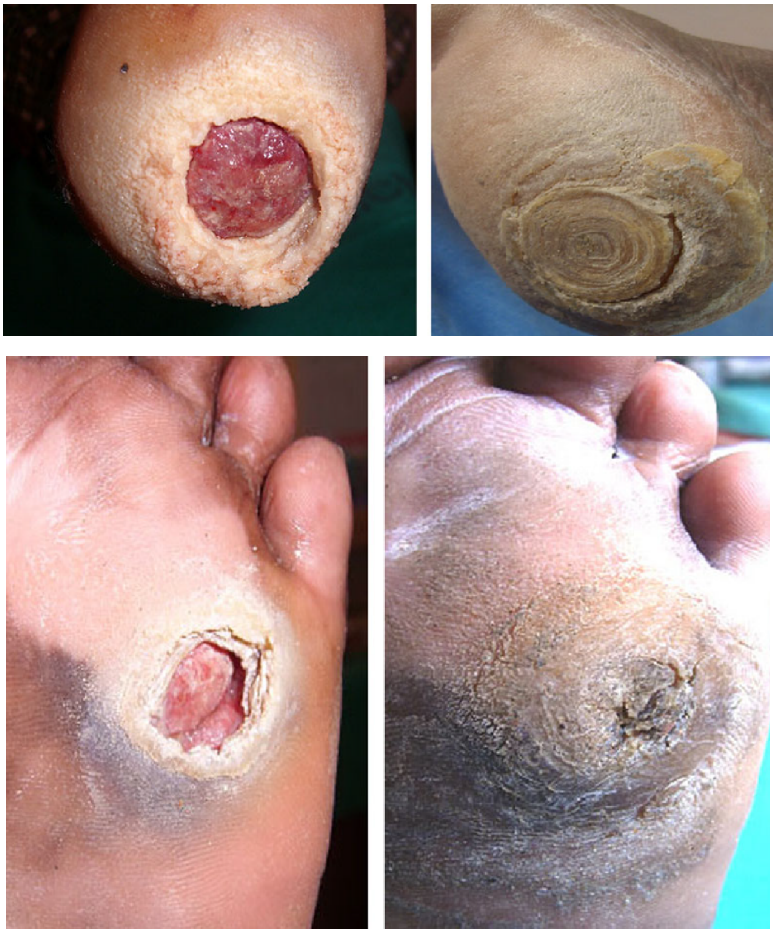

Figure 1. Before (a,c) and after (b,d) 4 weeks of treatment with topical phenytoin sodium zinc oxide paste, showing the appearance of red, vascularized, uneven granulation tissue progressing to healing.

Recent studies have reported the effectiveness of platelet-rich plasma (PRP) in the management of chronic ulcers. Platelet-rich plasma contains growth factors to increase the proliferation of mesenchymal stem cells, osteoblasts, fibroblasts, endothelial cells, and epidermal cells, thereby stimulating tissue regeneration, new blood vessel vascularization, and re-epithelialization, which are beneficial in the process of wound healing. Suryawati et al report a non-healing trophic ulcer in a leprosy patient treated with Treated with a combination of injection (0,1 ml subcutaneous injection with 1 cm distance from previous injection) and topical PRP once/week after wound cleansing and debridement[7]. Anandan et al studied around 50 leprosy patients with trophic ulcer treated with wound debridement followed by PRP topically applied once a week for 6 weeks. Approximately 46 patients (92%) showed complete healing and ulcer healing mean time was 4,38 weeks[8]. Saha et al compared two groups, which group A was treated with total contact casting and PRP injected intra and perilesionally and topically applied once every 2 week for 8 weeks, group B was treated with total contact casting only once every 2 week for 8 weeks. The study found that ulcer surface area reduction in group A was 91.10 ± 9.65% and group B was 79.77 ± 17.91%[8]. In an analytic cohort study by Sari et al, they reported 20 leprosy patients treated with 2 times PRP gel topical application on plantar ulcer. There were significant differences in size of lesions ($p = <0.05$), the number of macrophages ($p = <0.001$), number of capillaries ($p = 0.003$), and the amount of granulation ($p = 0.032$)[9]. Tissue growth factors, platelet derived growth factor (PDGF) and epidermal growth factor (EGF) are reported to be useful in treating neuropathic ulcers by expediting wound healing and tissue regeneration. Besides PRP, other platelet-derived autologous products, platelets rich fibrin (PRF) is a rich source of these growth factors and they have been previously shown to be effective in treating non-healing, chronic leg ulcers, including neuropathic ulcers. Nagaraju et al studied 9 non-healing trophic ulcers more than 6 weeks treated with PRF weekly application. The mean percentage improvement in the area was 93.52%, and volume was 97.74% at the end of the second sitting. All ulcers closed by a maximum of five sittings. No adverse events were observed[10]. Ghatge et al found that PRF was safe, cost-effective, easy to prepare, and an effective modality in the management of trophic ulcer in leprosy[11]. Vinay et al studied 10 patients with non-healing trophic ulcers more than 12 weeks treated with PRF weekly application. Eight patients showed complete healing after 2-4 PRF dressing sessions, and no adverse events were observed[12].

Modern wound dressing was also studied in leprosy trophic ulcer management. Hydrocolloid was a hydrogel mixed with synthetic rubber and sticky materials, excellent exudate absorption properties, and suitable for a severe exudative wound[13]. Cikutra et al reported a case of trophic ulcer in a 61-year-old woman with lepromatous leprosy, treated with hydrocolloid application to the ulcer every three days. The improvement of trophic ulcer in this case was seen on the 28th day as the ulcer has become shallower and smaller. On the 70th day, the ulcer healed into an eutrophic cicatrix. Based on this case report, the use of hydrocolloid is an effective choice of treatment when used on the right type of wound and with routine monitoring. (Fig. 2)[14] Prakoeswa et al did an experimental study with patient with chronic ulcer leprosy topically apply the drug every 3 days until the ulcer closed for a maximum of 8 weeks, comparing human amniotic membrane – mesenchymal stem cell – conditioned medium **(**HAMMSC-CM), HAMMSC-CM with vitamin C, and HAMMSC-SC with vitamin E. Every group showed good clinical outcomes with no complications or side effects[15].

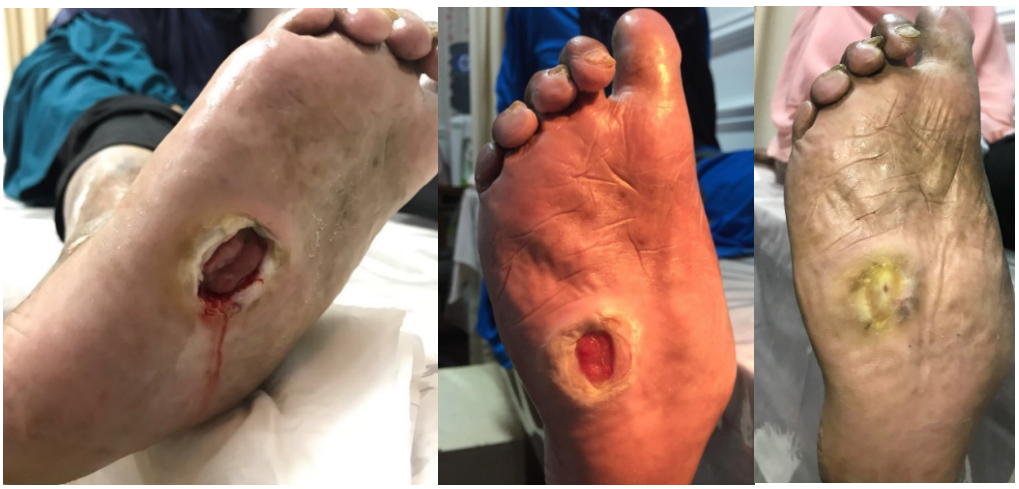

Figure 2. Wound dressing with hydrocolloid dressing, replaced every 3 days, resulted in healed ulcer after 28 days (b) and 70 days (c), eutrophic scar was observed

Various surgical methods are available for management of plantar ulcers, including skin grafts and different types of flaps. In a study by Gahalaut et al, a total of 40 leprosy patients were managed using different types of local superficial flaps (advancement, rotation, transposition and first toe web flap). Majority of ulcers healed within 4 weeks and patients were discharged within 6–8 weeks postoperatively. Ulcers recurred in only 25% of all the ulcers operated upon and we observed that management of plantar ulcers by using appropriate local superficial plantar

flaps is a viable option and worth trying in view of low incidence of recurrence. Other case report by Yuwantana and Wirohadidjojo, suction blister epidermal grafting (SBEG) was done on a right plantar ulcer for 16 months in a young female leprosy patient. On 60th day, complete closure of the wound was observed (Fig. 3)[16].

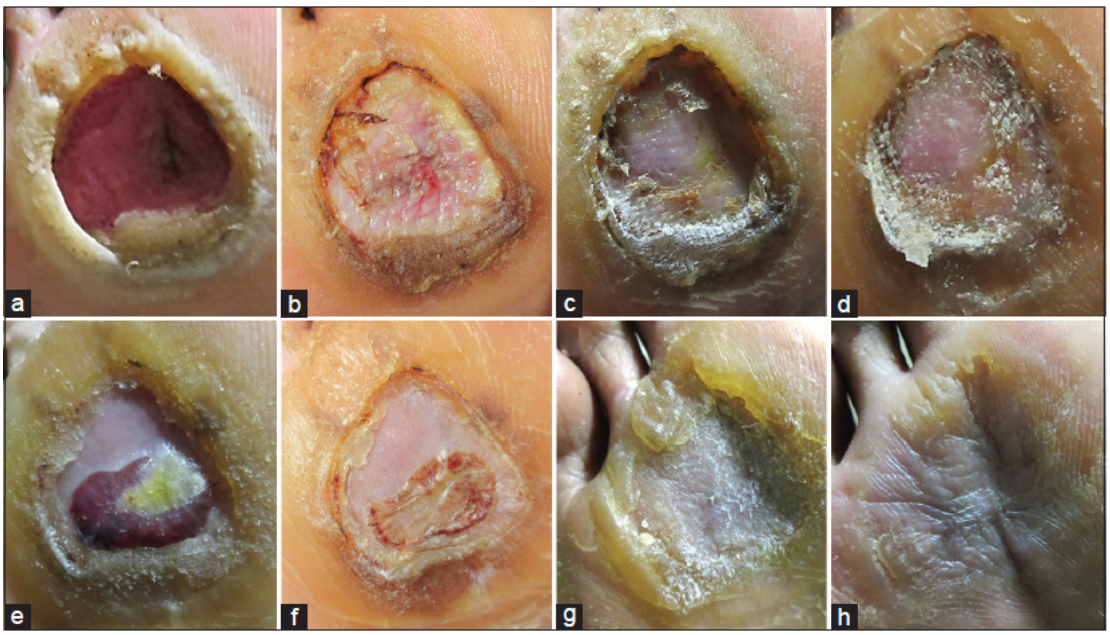

Figure 3. Clinical progress of the recipient site. Clinical progress of the wound: (a) preoperative, (b) day 7, (c) day 10, (d) day 14, (e) day 21, (f) day 35, (g) day 49, and (h) day 63.

Leprosy Trophic Ulcer Management in Primary Health Care

Management of leprosy trophic ulcers in primary healthcare (PHC) centres, known as *Puskesmas* in Indonesia, involves early identification and intervention to prevent the progression of these ulcers. Early signs of trophic ulcers, such as redness, swelling, or tenderness in pressure areas, especially on the feet, should prompt healthcare workers to educate patients about self-care practices. Patients are advised to avoid walking barefoot to reduce pressure on the ulcer-prone areas, and they are encouraged to frequently inspect their feet for unnoticed injuries. This preventative approach is crucial in preventing the development of severe ulcerations[17].

Wound care in PHC settings is primarily focused on simple, cost-effective, and accessible methods. For instance, the use of saline dressings and zinc oxide paste has been found effective in keeping the ulcer site clean and promoting healing. Regular debridement of necrotic tissue, if present, is essential to reduce infection risk. Additionally, health workers may apply hydrocolloid dressings to maintain a moist wound environment, which aids in faster healing. They may also use readily available, low-cost materials such as gauze impregnated with phenytoin, which has shown promising results in promoting wound granulation and epithelialization[17-18].

Immobilization techniques are often employed in primary care to support healing by reducing pressure on the ulcer site. PHC staff may recommend using crutches or canes to offload pressure from affected feet. Simple modifications, such as padding footwear or using orthotic devices, can help minimize stress on the ulcers, promoting faster healing and reducing the likelihood of recurrence. In severe cases, plaster casts or other immobilizing devices may be applied to protect the affected area from mechanical pressure and further injury[18]. Education and self-care training are crucial components of managing leprosy trophic ulcers in primary healthcare. Patients are taught to perform daily foot inspections, practice appropriate hygiene, and avoid factors that could lead to injury. Family members are often involved in this education process to provide additional support. By empowering patients and their families with knowledge and skills, PHC can help prevent the recurrence of trophic ulcers, reduce the burden on healthcare services, and improve patients' quality of life[17-18].

CONCLUSIONS

Leprosy trophic ulcer management includes sufficient rest, topical treatment to promote wound healing in leprosy trophic ulcer, and various surgical methods.

Acknowledgments

None.

Ethical Approval

The research received ethical clearance from the Prof. dr. I.G.N.G. Ngoerah Hospital. The approval date is May 28th, 2020, with number 1119/UN14.2.2.VII.14/LT/2020.

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Conflicts Of Interest

There are no conflictsof interest.

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