CASE REPORT

Staged Surgical Management of Diabetic Foot Infections in Advanced Lower-Limb Lymphedema: A Case Series

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Abstract

We report a case series from the Department of General Surgery, Sree Balaji Medical College and Hospital, describing severe lower-limb lymphedema complicated by diabetic foot infections following minor trauma, and highlighting a staged management strategy combining urgent infection control with definitive lymphedema-directed surgery. Three patients with long-standing grade 3 lymphedema and type 2 diabetes mellitus (T2DM) presented with acute painful swelling of the foot associated with systemic symptoms, consistent with soft-tissue infection on a background of chronic lymphatic obstruction. Case 1 was a 39-year-old woman with a medial foot abscess who underwent prompt surgical debridement with tissue sent for culture and sensitivity, followed by culture-directed antibiotics, compression-based edema control, and negative pressure wound therapy (VAC) to promote granulation and wound contraction. Case 2 was a 45-year-old man with filarial lymphedema and an infected lateral foot ulcer; after initial wound control and optimization of glycaemic status and local wound care, he underwent a second-stage procedure at 20 days consisting of lymphedema debulking and split-thickness skin grafting (6 × 5 cm, harvested from the thigh, meshed, stapled), with drain placement, layered closure, and compression/immobilization dressing. Case 3 was a 45-year-old man with Doppler-confirmed preserved arterial flow and no deep venous thrombosis who underwent incision and drainage/debridement with culture-guided antibiotics and adjunct VAC therapy, alongside continued compression, elevation, and offloading. This series emphasizes that in patients with advanced lymphedema and T2DM, early source control and culture-directed antimicrobials, combined with edema control and NPWT, can prepare the wound bed, while selected cases may benefit from staged definitive debulking with split-thickness grafting to facilitate durable wound closure and reduce recurrence risk.

Keywords: Lymphedema, Type 2 diabetes mellitus, Diabetic foot infection, Debridement, Negative pressure wound therapy, Debulking surgery

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Introduction

Lower-limb lymphedema is a chronic, progressive disorder of lymphatic transport characterized by persistent interstitial fluid accumulation with secondary inflammation, adipose deposition, and fibrosis, ultimately leading to skin thickening and architectural distortion.(1) In advanced disease (International Society of Lymphology stage III), the limb may become markedly enlarged with fibrotic changes, hyperkeratosis and papillomatosis, and the risk of recurrent infection increases because lymphatic dysfunction impairs local immune surveillance and tissue homeostasis.(2, 3) Chronic lymphedema is a recognized risk factor for recurrent cellulitis/lymphangitis, and repeated episodes can further damage lymphatics, perpetuating a cycle of infection, edema, and poor healing. In tropical and subtropical settings, lymphatic filariasis remains an important cause of secondary lymphedema and can progress to disabling elephantiasis with long-term morbidity.(4)

Type 2 diabetes mellitus (T2DM) compounds this risk profile by increasing susceptibility to soft-tissue infection through hyperglycaemia-associated immune dysfunction, microvascular compromise, and neuropathy-related trauma, and it is a major driver of infected foot wounds requiring hospitalization and surgery.(5) IWGDF/IDSA Guidelines emphasize that management of diabetic foot infection hinges on early recognition, metabolic optimization, assessment for deeper infection, prompt surgical source control when indicated (incision and drainage/debridement), and use of appropriately obtained tissue/pus cultures to guide

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antibiotic therapy.(6) When T2DM coexists with long-standing lymphedema, wounds may be particularly recalcitrant because chronic edema and fibrosis impair oxygen diffusion and antibiotic penetration and increase mechanical stress on the skin barrier, thereby delaying granulation and epithelialization.(7) First-line lymphedema treatment remains conservative—compression-based therapy, skin care, and limb elevation—while surgical options are considered for selected patients, especially those with advanced, fibroadipose-predominant disease or recurrent infection despite optimal conservative therapy. Surgical strategies include physiological procedures (lymphaticovenular anastomosis and vascularized lymph node transfer) and excisional approaches (debulking/Charles-type procedures and liposuction), chosen according to stage and tissue characteristics. In complex infected foot wounds, negative pressure wound therapy (NPWT) is commonly used as an adjunct after debridement to promote granulation and manage exudate.(8, 9) Against this background, we report a case series from the Department of General Surgery, Sree Balaji Medical College And Hospital, describing severe lower-limb lymphedema complicated by diabetic foot infections following minor trauma, and illustrating a staged approach combining urgent infection control with definitive lymphedema-directed surgery (including debulking with split-thickness skin grafting) to facilitate durable wound closure and reduce recurrence risk.

Case Series

Case 1: A 39-year-old woman with a 3-year history of T2DM and a 20-year history of chronic right lower-limb lymphedema presented with pain and swelling of the right foot for one week following trivial trauma, associated with fever, burning micturition, and difficulty in walking. On examination, she had grade 3 lymphedema of the right lower limb with a localized abscess over the medial aspect of the right foot. In view of an acute soft-tissue infection in a patient with diabetes and longstanding lymphedema (both of which are known to predispose to severe infection and delayed wound healing), she was evaluated with routine inflammatory and metabolic work-up and assessed for concurrent urinary tract infection, and glycaemic control and limb elevation were instituted as part of standard care. She underwent surgical wound debridement with freshening of wound edges, removal of sloughed tissue, and collection of tissue for culture and sensitivity; meticulous haemostasis was achieved followed by thorough wound irrigation and application of a sterile compression dressing with a crepe bandage. The intraoperative and postoperative periods were uneventful. Empirical antimicrobial therapy was initiated and subsequently tailored according to culture and sensitivity results, and negative pressure wound therapy (VAC therapy) was applied to promote granulation tissue formation and facilitate wound contraction, along with ongoing compression-based lymphedema care and optimisation of diabetes management.

Case 2: A 45-year-old man with an 8-year history of T2DM and a 15-year history of chronic right lower-limb lymphedema secondary to filariasis presented with pain and swelling of the right foot for one month following minor trauma, accompanied by fever and difficulty in walking. Clinical examination revealed grade 3 lymphedema of the right lower limb with an ulcer over the lateral aspect of the right foot, clinically consistent with an infected traumatic/diabetic foot wound occurring on the background of chronic lymphatic obstruction (a setting known to predispose to recurrent soft-tissue infections, poor local tissue oxygenation, and delayed healing). The patient was managed with standard measures including optimization of glycaemic control, limb elevation, meticulous wound care, and empiric antibiotics that were subsequently tailored based on wound/tissue culture and sensitivity, with assessment for deep infection as indicated. After initial surgical wound control, and once the wound bed was healthy, the patient underwent a second-stage procedure 20 days after the initial surgery consisting of combined lymphedema debulking and split-thickness skin grafting: excess lymphedematous tissue from the lower right leg was excised (debulking) to reduce diseased fibrofatty load and facilitate durable wound closure, haemostasis was secured, and the wound was closed in layers using Vicryl with placement of a surgical drain; the skin was approximated using Ethilon. A 6 × 5 cm split-thickness graft was harvested from the left thigh, meshed, applied to the exposed recipient area, and secured with staples. A sterile compression dressing was applied using Bactigras (paraffin gauze dressing) and a POP (plaster-of-Paris) support/immobilization dressing.

Case 3: A 45-year-old man with a 10-year history of T2DM and a 15-year history of chronic lymphedema presented with pain and swelling of the right foot for one week following trivial trauma, associated with fever, burning micturition, and difficulty in walking. On examination, he had grade 3 lymphedema of the right lower limb, and a localized abscess was noted over the medial aspect of the left foot. Baseline blood investigations were within normal limits, and a bilateral lower-limb venous Doppler study demonstrated diffuse subcutaneous edema extending from the mid-calf to the foot on the right side, with triphasic flow in the dorsalis pedis and posterior tibial arteries, no evidence of deep venous thrombosis, and no saphenofemoral or saphenopopliteal junction incompetence. In view of an acute soft-tissue infection in a patient with diabetes and longstanding lymphedema (a high-risk combination for rapid progression of infection and impaired wound healing), he was managed with limb elevation, strict glycaemic optimisation, analgesia, and initiation of empirical broad-spectrum antibiotics with subsequent tailoring based on pus/tissue culture and sensitivity, and evaluation for urinary tract infection given the dysuria. After obtaining anaesthesia fitness, he underwent surgical wound debridement/incision and drainage of the abscess with removal of sloughed tissue, freshening of wound margins, and collection of tissue for microbiological analysis; haemostasis was achieved

followed by copious wound irrigation and application of a sterile compression dressing with crepe bandaging to support edema control. Once healthy granulation tissue was established, negative pressure wound therapy was used to promote granulation and facilitate wound contraction, alongside continued compression-based lymphedema care and offloading to aid ambulation and prevent recurrence.

Discussion

Chronic lower-limb lymphedema and T2DM represent a high-risk clinical overlap in which minor skin trauma can precipitate disproportionate soft-tissue infection, delayed healing, and recurrence. Lymphedema is characterized by impaired lymphatic transport with progressive interstitial fluid accumulation, chronic inflammation, adipose/fibrotic tissue deposition, and skin barrier changes; advanced disease is clinically marked by non-pitting edema, thickening, papillomatosis/hyperkeratosis, and increased susceptibility to bacterial entry.(3) In addition, lymphedema is associated with impaired local immune surveillance and recurrent cellulitis/erysipelas, which can become frequent and severe once skin integrity is compromised.(10) Lipsky et al. (2012) and Senneville et al. (2023) added that diabetes magnifies infection risk through a combination of neutrophil dysfunction, microvascular disease, and (in many patients) peripheral neuropathy and biomechanical stress, culminating in delayed wound healing and higher rates of deep infection and amputation when infected foot wounds are not rapidly controlled.(6, 11)

Across all three cases, a common pathway was evident: trivial/minor trauma preceded local infection in an edematous limb with long-standing lymphatic obstruction, and systemic symptoms (fever) raised concern for rapidly progressive soft-tissue infection. The acute management priority in such presentations is early source control—incision and drainage for abscess and prompt debridement of devitalized tissue—combined with systemic antibiotic therapy and optimization of metabolic factors that impair healing. Current diabetic foot infection guidance emphasizes that diagnosis is primarily clinical,(6) but treatment outcomes depend on timely debridement/drainage when purulence, abscess, or necrosis is present, with reassessment for deeper infection (including osteomyelitis). Case 1 highlighted a short symptom duration (one week) with a localized medial foot abscess on a background of grade 3 lymphedema and T2DM. Surgical debridement with freshening of wound margins and removal of slough was aligned with IWGDF/IDSA Guidelines, where debridement reduces bacterial burden, removes nonviable tissue, and converts a chronic/infected wound environment into one more conducive to granulation and re-epithelialization.(6, 12) The collection of tissue for culture and sensitivity is particularly important because guideline-based care recommends obtaining a post-debridement specimen (preferably tissue rather than superficial swab) to guide definitive therapy, while empiric antibiotics should be adjusted once microbiology results are available. The case also incorporated limb elevation and compression-based care as adjuncts to reduce edema and improve local tissue mechanics; compression forms a core component of complex decongestive therapy (CDT) for lymphedema, alongside skin care, exercise, and (when feasible) manual lymphatic drainage; in corroboration with Jahan et al. (2025) and Zhang et al. (2022).(13, 14)

Negative pressure wound therapy (NPWT/VAC) was used after initial infection control to promote granulation and wound contraction. IWGDF 2023 update support NPWT primarily in selected contexts—most consistently for post-surgical wounds rather than uncomplicated non-surgical ulcers—reflecting that the benefit signal is stronger when used after operative debridement/amputation or to manage complex wound beds.(15) Systematic reviews and meta-analyses including Chen et al. (2021) and Hasan et al. (2015) have similarly reported faster wound size reduction/granulation and potential improvements in healing outcomes in appropriately selected diabetic foot wounds, though certainty varies with study design and wound type.(16, 17) In the present series, NPWT was appropriately positioned as an adjunct after source control and antimicrobial therapy, rather than a substitute for debridement or antibiotics.

Case 2 illustrated a longer symptomatic course (one month) with an ulcer on the lateral aspect of the right foot in a patient with filarial lymphedema. In endemic regions, lymphatic filariasis is a major cause of secondary lymphedema;(18) chronic lymphatic damage predisposes to recurrent “acute attacks” of dermato-lymphangio-adenitis/cellulitis and progressive disability, and morbidity management emphasizes meticulous skin care, hygiene, prompt treatment of entry lesions, and compression where feasible.(19) The staged approach—initial wound control followed by definitive volume-reducing surgery—was clinically logical because persistent stage 3 lymphedema can perpetuate maceration, mechanical stress, and recurrent infection, thereby preventing durable closure even after the acute infection improves.(3) Carroll & Singhal (2024) and Chen et al. (2025) added that excisional (debulking) procedures are generally reserved for advanced lymphedema with severe fibroadipose overgrowth and skin changes when conservative measures fail or when recurrent infections and nonhealing wounds threaten limb salvage; they aim to reduce the diseased tissue load, improve function, and facilitate long-term compression.(20, 21) The combined debulking with split-thickness skin grafting (SSG) performed 20 days after the initial procedure addressed two barriers simultaneously: (1) a complex wound requiring coverage and (2) a hostile lymphedematous milieu that impairs perfusion and healing. Layered closure with absorbable sutures, drain placement to reduce seroma/lymphorrhea, and secure graft fixation with staples followed by non-adherent dressing and immobilization are consistent with standard reconstructive principles for graft take in lower-limb wounds, particularly where edema and shear forces threaten adherence.(20)

Case 3 reinforced the importance of vascular and venous evaluation when infection occurs in a chronically swollen limb. The venous Doppler findings—diffuse subcutaneous edema without deep venous thrombosis, no saphenofemoral/saphenopopliteal junction incompetence, and triphasic distal arterial flow—were reassuring for adequate macrovascular inflow and reduced the likelihood that venous reflux or acute venous occlusion was the primary driver of swelling. Assessment for peripheral arterial disease is essential in diabetic foot care because ischemia worsens infection outcomes and can mandate revascularization to achieve healing; bedside waveform/vascular testing forms part of standard evaluation.(22) The management pathway in Case 3—antibiotics with culture-directed tailoring, incision/drainage and debridement, compression support, and NPWT once granulation was established—again reflects the principle that infection control must precede advanced wound technologies or reconstructive steps. A notable feature in Cases 1 and 3 was concomitant dysuria/burning micturition, raising the possibility of urinary tract infection as a systemic inflammatory trigger or coexisting infection. In diabetic patients, intercurrent infections can destabilize glycemic control and worsen wound outcomes;(23) therefore, evaluating and treating concurrent infections while tightening glucose control is a pragmatic component of perioperative and wound management, even though the cornerstone of limb salvage remains local source control and appropriate antibiotics.

Long-term, prevention of recurrence is inseparable from chronic lymphedema care. Mihalache et al. (2025) emphasize ongoing compression (garments/bandaging), skin care to prevent fissures and fungal intertrigo, exercise/weight management, and patient education to reduce cellulitis episodes; in filarial lymphedema, morbidity management programs similarly prioritize hygiene, skin care, and prompt treatment of entry lesions.(24) In patients with diabetes, the same period must include structured foot care (daily inspection, protective footwear, callus care), offloading when wounds are present, and optimization of vascular risk factors—measures that reduce re-ulceration and subsequent infection.

Conclusion

In this case series, patients with long-standing grade 3 lower-limb lymphedema and T2DM developed severe foot soft-tissue infections following minor trauma, highlighting the synergistic risk of rapid infection progression and delayed wound healing. Early source control with incision and drainage/debridement, culture-directed antimicrobial therapy, strict glycaemic optimisation, edema control with elevation and compression, and adjunctive negative-pressure wound therapy enabled effective infection control and wound bed preparation. In selected advanced cases where persistent lymphedema impeded durable healing, staged definitive lymphedema surgery with debulking and concurrent split-thickness skin grafting provided a practical limb-salvage strategy by reducing diseased tissue load and facilitating stable wound closure. Overall, an integrated multidisciplinary approach that addresses both acute infection and the underlying lymphedema is essential to improve healing, reduce recurrence, and preserve function in diabetic patients with advanced lymphedema.

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