Review Article

Lymphedema of the Lower Extremities: A Brief Bibliographic Review

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Abstract

The lymphatic system is a vast vascular network responsible for transporting lymphatic fluid, or lymph, which is filtered through lymph nodes distributed along its course. Beyond its circulatory role, this system plays a fundamental part in immune function, contributing to antibody production and facilitating the clearance of cellular debris and microorganisms from the body. Although conservative, non-surgical management remains the cornerstone of treatment, lymphedema is currently considered a chronic and incurable condition. Preventive strategies are essential; however, once diagnosed, management should focus on reducing inflammation, preventing secondary infections, and preserving function. Mechanical therapy continues to represent the gold standard in treatment, particularly compression therapy and specialized physical rehabilitation techniques.

Key words

Lymphedema, lymphatic drainage, edema, compression.

Introduction

Lymph is a protein-rich interstitial fluid whose progressive accumulation leads to the development of lymphedema. This condition encompasses a group of disorders characterized by edema as a significant consequence of impaired lymphatic vascular function, whether involving primary (congenital) or secondary (acquired) lymphatic vessels [1].

The lymphatic circulation is responsible for returning interstitial fluid and proteins to the central venous system. However, when lymphatic stasis occurs, proteins and cellular metabolites accumulate within the extracellular space, leading to water retention and increased interstitial hydrostatic pressure. Secondary to these changes, there is an increase in fibroblasts, keratinocytes, and adipocytes within the affected tissues. Over time, this process results in

increased collagen deposition, excessive connective tissue proliferation, and expansion of subcutaneous and adipose tissue components in the skin.

From a pathophysiological standpoint, the progression of lymphedema closely correlates with histological findings. In early stages, obstruction of otherwise competent lymphatic vessels manifests as soft, pitting edema. As the disease progresses, vascular ectasia and valvular insufficiency promote the persistent accumulation of protein-rich fluid, triggering chronic inflammation, adipose tissue proliferation, and fibrosis. These changes also predispose patients to recurrent episodes of cellulitis [2].

Based on etiology, lymphedema is classified as primary when it results from genetic abnormalities affecting lymphatic development - such as hyperplasia, hypoplasia, or aplasia - and secondary when it arises from mechanical obstruction or acquired injury due to surgery, trauma, malignancy, venous thrombosis, or infection [2].

Although primary lymphedema is relatively rare, with an estimated prevalence of 1.15 per 100,000 individuals under 20 years of age, well-described hereditary forms exist, including Milroy disease and lymphedema–distichiasis syndrome, both inherited in an autosomal dominant pattern. In contrast, secondary lymphedema is the most common form worldwide. In endemic regions, filariasis remains the leading cause; however, in developed countries, iatrogenic etiologies related to oncologic treatment predominate. The incidence following lymphadenectomy varies depending on the anatomical region involved, reaching significant rates after inguinal dissections and axillary procedures for breast cancer [2].

The diagnosis of lymphedema is primarily clinical and requires careful exclusion of differential diagnoses, particularly in early stages when it may be confused with chronic venous insufficiency, lipedema, obesity, cardiac edema, infectious processes, or malignancy. A considerable proportion of patients referred with suspected lymphedema are ultimately found to have an alternative diagnosis. In advanced stages, the clinical presentation becomes characteristic, with progressive edema evolving into non-pitting induration, skin changes such as peaud'orange appearance, heaviness, pain, and recurrent cellulitis. Although clinical assessment remains the diagnostic cornerstone, complementary studies such as duplex ultrasonography are useful for ruling out associated venous pathology and guiding therapeutic decisions [2].

This article aims to provide a structured review of the pathophysiology, diagnostic criteria, and current therapeutic alternatives for the management of lymphedema, ranging from conservative strategies to advances in reconstructive microsurgery, within the context of a condition whose clinical relevance continues to increase.

Methodology

This study follows a qualitative research approach, as data were collected without numerical measurements in order to address the research question. Information was obtained through the review of scientific articles and informational sources, including textbooks and academic websites. The study population included articles documenting the diagnosis, treatment, complications, classification, and clinical manifestations of lymphedema.

The project sample consisted of scientific articles that met previously established inclusion and exclusion criteria. These criteria included publication between 2019 and July 2025, availability in English, Portuguese, or Spanish, and open-access format. The databases used for the literature search were EBSCO, Dialnet, and Google Scholar. Additionally, the review was supplemented with informationfrom *Rutherford's*

Vascular Surgery and Endovascular Therapy textbook.

Etiology

Two main types of lymphedema have been described. **Primary lymphedema** is an uncommon hereditary condition resulting from congenital malformations of the lymphatic system, also referred to as congenital lymphatic dysplasia. **Secondary lymphedema**, in contrast, is caused by mechanical disruption of normal lymphatic flow. It is most commonly associated with inflammatory and infectious processes, surgical procedures, radiation-induced scar tissue, compression of lymph nodes by a growing tumor, or lymph node removal during surgery [3].

Incidence

Incidence rates in secondary lymphedema vary depending on the anatomical site involved. It has been reported that approximately 8–56% of patients undergoing treatment for breast cancer with axillary lymph node dissection develop lymphedema within the first two postoperative years.

Similarly, patients treated for vaginal, ovarian, endometrial, cervical, prostate, or colorectal cancer have a significant risk of developing lymphedema affecting the abdominal region, genital area, or lower extremities. It is also important to highlight that individuals treated for head and neck cancers may develop lymphatic involvement in less visible areas, such as the pharyngeal region [3].

Pathophysiology

Clinically, lymphedema can be defined as a condition in which interstitial volume increases by more than 20% for a period exceeding six months in an irreversible manner. This sustained overload leads to elevated intracapillary pressure within lymphatic vessels, progressive dilation of their lumens, and eventual structural damage to the valves or the development of vascular ectasia [3].

The persistent accumulation of lymphatic fluid promotes fibroblast proliferation and collagen deposition, resulting in fibrosis and further obstruction of lymphatic flow. In addition, interstitial fluid buildup combined with impaired immune response generates a pro-inflammatory state, creating a favorable environment for recurrent infections of the skin and soft tissues [2].

Clinical manifestations

Key Clinical History Clues [1]

- A positive family history of lower limb swelling should raise suspicion of hereditary (primary) lymphedema.
- The onset of painless edema in an adolescent without an identifiable secondary cause should suggest primary or idiopathic lymphedema.
- A history of chronic diarrhea and weight loss may indicate mesenteric lymphangiectasia.
- The presence of milky fluid within cutaneous vesicles suggests chylous reflux.
- Recent travel to tropical countries accompanied by inflammatory symptoms should prompt suspicion of filariasis.

Main signs and symptoms

Edema

Based on the underlying pathophysiology, the accumulation of lymphatic fluid generates increased interstitial pressure that initially manifests as pitting edema, which may improve with limb elevation [4].

Edema typically begins distally in the feet, leading to the characteristic “square toes” appearance. A positive Stemmer sign - defined by the inability to pinch and lift the skin at the base of the second toe - is a classic clinical finding.

Dorsal forefoot swelling may produce the so-called “buffalo hump” appearance. The surrounding tissue may progressively acquire a

firm or "woody" texture due to fibrosis and tissue induration [4].

Skin Changes

In early stages, the skin may appear pinkish-red with mildly increased local warmth due to enhanced capillary blood flow. Over time, however, the skin thickens and develops hyperkeratotic areas, lichenification, and a characteristic peaud'orange (orange peel) appearance [5].

Although ulcerations are less common than in chronic venous insufficiency, they may occur. Unlike venous stasis disease, skin hydration and elasticity are generally better preserved, and severe ischemic compromise is uncommon. Some patients develop warty lesions or small vesicles with clear lymphatic drainage (lymphorrhea), while others may present with chylous reflux, characterized by milky discharge (chylorrhea).

A subset of patients exhibit yellow discoloration of the nails, known as yellow nail syndrome. While not pathognomonic for lymphedema, it is frequently associated with impaired lymphatic drainage, reduced nail growth, and increased nail fragility.

Pain

Patients typically report a sensation of heaviness or mild discomfort in the affected limb. Severe pain is uncommon and should prompt evaluation for complications such as infection or neuropathic pain related to scar tissue or prior radiation therapy [6].

Clinical staging [7]

Clinically, lymphedema can be classified into the following stages:

- **Stage 0 (Latent or Subclinical Stage):** Accumulation of lymphatic fluid and perilymphatic fibrosis without visible edema.
- **Stage I:** Reversible edema that pits on pressure and significantly improves or resolves with limb elevation or compression therapy. No evident fibrosis.
- **Stage II – Early:** Irreversible edema that does not fully resolve with elevation. Pitting may still be present (positive Godet sign).
- **Stage II – Late:** Moderate to severe fibrosis with edema that may be pitting or non-pitting.
- **Stage III:** Irreversible edema with recurrent inflammatory episodes, marked fibrosis, and sclerosis of the skin and subcutaneous tissue. The skin becomes hard or "woody," with trophic changes such as papules or lymph-filled vesicles. Lymphostatic elephantiasis may develop at this stage.

Classification

This vascular disorder is broadly divided into two main categories: primary and secondary lymphedema. Primary lymphedema can be further classified according to genetic background and age of onset.

Primary lymphedema

Primary lymphedema shows a higher prevalence in females compared to males, with an approximate ratio of 3:1. Although it is hereditary in origin, it encompasses a heterogeneous group of disorders and is therefore subdivided into different classifications [1].

1. Classification by age of onset

- **Congenital:** May occur sporadically or follow an autosomal dominant inheritance pattern. It is strongly associated with intrauterine lymphatic dysfunction and chromosomal abnormalities such as Turner syndrome, trisomy 21, and Klinefelter syndrome. Although involvement may be limited to a single limb, edema can also affect the face and genital region.
- **Praecox (Early-Onset):** The most common form of primary lymphedema, accounting for approximately 94% of cases. It demonstrates a marked female predominance (10:1 ratio) and typically

presents between 1 and 35 years of age. Edema is usually unilateral and confined to the lower limb in most patients. The role of estrogenic hormones in its pathogenesis remains under investigation.

- **Tarda (Late-Onset):** The least common subtype, appearing after 35 years of age and representing approximately 10% of patients with primary lymphedema.

2. Classification by morphology [8]

Primary lymphedema may also be classified according to lymphatic vascular morphology:

- **Aplasia:** Absence of identifiable lymphatic collecting vessels, typically seen in congenital and early-onset forms.
- **Numerical hypoplasia:** Reduced number and diameter of lymphatic vessels below normal values.
- **Hyperplasia:** Increased number of lymphatic vessels with associated tortuosity and dilation; this pattern is more frequently observed in males.

3. Classification by Anatomical Involvement

- **Distal obstruction:** Patients typically present with mild, bilateral swelling. Prognosis is generally favorable, as most cases demonstrate limited progression during the first year of disease and follow an indolent course.
- **Proximal obstruction:** Accounts for more than 50% of cases. It involves proximal lymphatic vessels or lymph nodes, often demonstrating intranodal fibrosis on imaging or histological evaluation. Although swelling may be unilateral, it is usually classified as severe. Due to proximal involvement, disease progression tends to be extensive, and surgical intervention is often considered the most effective therapeutic approach in selected patients.

Secondary lymphedema [1]

Secondary, or acquired, lymphedema represents the most common form of lymphatic vascular dysfunction and results from damage to previously normal lymphatic vessels. The most common causes worldwide include malignancy and filariasis.

- **Cancer:**Breast cancer treatment is most frequently associated with acquired lymphatic insufficiency, particularly affecting the upper extremities following axillary lymph node dissection or radiation therapy.
- **Filariasis:**A parasitic infection caused primarily by *Wuchereriabancrofti*,*Brugiamalayi*, and *Brugiatimori*. It is the leading cause of secondary lymphedema in tropical and developing countries and is transmitted by mosquitoes.

The pathophysiological process involves inflammation and perilymphatic fibrosis, as well as sclerosis of lymph nodes caused by adult worms. Reactive hyperplasia and dilation of collecting channels lead to valvular damage and vessel wall injury, ultimately resulting in lymphatic obstruction. Filarial lymphedema may rapidly progress to disabling elephantiasis.

Other less frequent causes of secondary lymphedema include pregnancy, vascular trauma (such as burns or extensive wounds), bacterial and fungal infections, snake or insect bites, contact dermatitis, and rheumatoid arthritis, among others.

Diagnosis

The diagnosis of lymphedema is primarily clinical and should include a thorough medical history, with particular attention to prior cancer, radiotherapy, surgery, or infections. Physical examination typically reveals asymmetry between the affected limb and the contralateral extremity. A circumference difference greater than 2 cm is generally considered diagnostic. Accurate and consistent measurement of limb circumference is essential for monitoring disease progression [9].

For many years, lymphoscintigraphy has been considered the gold standard for diagnosing lymphedema. This technique allows visualization

of major lymphatic vessels and assessment of collateral circulation. It also helps predict response to conservative treatment with complete decongestive therapy. The procedure involves subdermal injection of a non-ionic radiotracer into the interdigital spaces, followed by tracking of lymphatic flow using a gamma camera [10].

Magnetic resonance lymphangiography (MR lymphangiography) provides detailed anatomical visualization of lymphatic vessels and surrounding structures.Indocyanine green (ICG) lymphography, performed through subdermal interdigital injection, enables real-time visualization of the lymphatic system using near-infrared fluorescence imaging, allowing identification of patent lymphatic vessels and mapping of lymphatic pathways [11].

Doppler ultrasonography may also be used; however, its primary role is to exclude venous pathologies in patients presenting with significant limb edema, such as post-thrombotic syndrome, deep or superficial venous thrombosis, or chronic venous insufficiency [12].

Conservative and surgical treatment

Conservative treatment

There is currently no definitive cure for lymphedema. Conservative measures include weight reduction strategies in obese patients and supervised physical activity. Obesity promotes a persistent pro-inflammatory state, as adipose tissue deposits contribute to lymphatic vessel damage. Reducing systemic inflammation is therefore essential. Additionally, spontaneous muscle contractions during physical activity facilitate lymphatic flow and provide clinical benefit [2].

Recommended exercises include [13]**:**

- **Standing exercises:** Heel and toe walking; walking with weight-bearing on the medial and lateral borders of the foot; alternating knee elevation during ambulation.
- **Seated exercises:** Flexion and extension movements of knees, ankles, and toes.
- **Supine exercises:** Cycling motions with alternating directions; scissor movements; lower limb extensions.

The most effective conservative treatment is Complete Decongestive Therapy (CDT). This approach combines manual lymphatic drainage, compression bandaging or garments, and lymphomyokinetic exercises to promote adequate lymphatic flow and drainage [14].

CDT is based on four fundamental pillars [2]:

1. Compression therapy
2. Manual lymphatic drainage
3. Therapeutic exercise
4. Skin care

It consists of two phases [2]:

- **Phase I (Intensive/Reduction Phase):** Aims to reduce limb volume and optimize skin care. Typically performed five times per week by a trained lymphedema therapist for 4–8 weeks.
- **Phase II (Maintenance Phase):** Begins immediately after Phase I to preserve achieved results. Requires lifelong daily self-management by the patient or caregiver.

Although highly effective, CDT requires substantial time commitment from both patient and therapist, and practitioners must be specifically trained in manual lymphatic drainage techniques [13].

Contraindications to CDT include: Deep vein thrombosis, renal, hepatic, or pulmonary insufficiency, severe peripheral arterial disease, aortic aneurysm, acute infections, extensive skin ulcers, pregnancy, recent surgery, uncontrolled hypertension, and active malignancy [13].

Components of conservative therapy [13]

Manual Lymphatic Drainage (MLD): Developed in 1936, MLD consists of gentle skin pressure to stretch superficial lymphatic vessels, enhancing their contractility and facilitating

lymph flow to relieve congestion and reduce subcutaneous fibrosis.

Treatment begins by stimulating adjacent non-affected areas to prepare them to receive redirected lymphatic flow.

Compression Bandaging: The primary technique during Phase I of CDT. It includes a tubular liner, padding (foam, polyester, or cotton), finger bandaging, and multiple layers of short-stretch bandages. These create a pumping effect through low resting pressure and high working pressure during muscle contraction.

Bandages are typically worn 24 hours per day, except during bathing or massage. During maintenance, compression garments are worn during the day and bandages at night.

Compression Garments: Daily and consistent use is essential to maintain limb volume reduction. Garments must be properly fitted, graduated, and made of low-elasticity, bidirectional fabric to ensure higher distal pressure and maintain unidirectional lymphatic flow.

Recommended pressure ranges from 20–50 mmHg, and garments should be replaced every 3–6 months.Contraindications include arterial insufficiency, acute heart failure, extreme limb deformity, deep skin folds, extensive ulcers, severe peripheral neuropathy, and lymphorrhea.

Non-Elastic Compression Devices: Commercial adjustable devices using Velcro-fastened, non-elastic support bands around the limb. These are useful for patients unable to independently perform complex bandaging techniques.

Intermittent Pneumatic Compression (Pressotherapy) [15]

In selected cases, intermittent pneumatic compression may be added. This method uses controlled air pressure to simulate massage, promoting relaxation and sufficient compression to activate the lymphatic system, enhance fluid and toxin elimination, and improve vascular conditions such as varicose veins and lymphedema.

Devices typically apply pressures between 30–60 mmHg to avoid vascular injury [13].

Contraindications include: [13]

- Deep vein thrombosis
- Advanced venous or arterial obstruction
- Active infections
- Severe cutaneous fibrosis
- Uncontrolled hypertension
- Aortic aneurysm
- Severe peripheral arterial disease
- Cardiac decompensation
- Hepatic, renal, or respiratory insufficiency
- Pregnancy

Psychological Support

Psychological support is essential in comprehensive management. Due to significant physical changes, patients may experience depression, social withdrawal, and reduced quality of life. Addressing aesthetic and functional concerns, encouraging social integration, and reinforcing long-term therapeutic adherence are crucial components of care [16].

Pharmacological management

There is insufficient evidence to support routine pharmacological therapy for lymphedema. Diuretics or sympathetic blockade may be used in early stages in selected patients, particularly those with concomitant heart failure.Flavonoids and coumarin derivatives, such as diosmin, may provide benefit by enhancing lymphatic trunk contractility and reducing capillary permeability and stasis; however, evidence remains limited and further studies are required [16].

Surgical treatment

1. **Vascularized Lymph Node Transfer (VLNT):** This procedure is indicated for patients with a deficient or absent lymphatic system, particularly in secondary lymphedema. It involves

transferring a free flap containing functional lymph nodes to the affected extremity, followed by microvascular anastomoses to restore lymphatic drainage. The transferred lymph nodes act as biological "sponges," absorbing lymphatic fluid and facilitating its passage into the venous system through natural lymphatic–venous connections within the flap [2].

2. **Ablative or Reductive Surgical Management:** This approach aims to remove excess skin and subcutaneous tissue without directly addressing the underlying pathophysiology of lymphedema. It is indicated in advanced stages, particularly late Stage II and Stage III disease characterized by non-pitting edema. The procedure generally combines liposuction with surgical excision and has demonstrated favorable outcomes in patients with advanced lymphedema who are not candidates for physiologic microsurgical interventions [17].

Complications

Among the most frequent complications are the following:

Cutaneous Infections

- Infections play a dual role in lymphedema, as they may act both as a precipitating factor and as a complication of untreated disease. Because lymph is a protein-rich fluid, it provides an optimal medium for bacterial and fungal proliferation. Lymphedematous tissue also serves as a portal of entry for pathogens, contributing to recurrent infections. It is estimated that 20–30% of patients with lymphedema develop cutaneous infections [1].
- **Cellulitis:**A common complication characterized by erythema, warmth, pain, skin irritation, and inflammation. It may be accompanied by peaud'orange changes and represents one of the most significant infectious complications of lymphedema [2].
- **Malignancy:**Lymphangiosarcoma is a rare but severe malignant tumor that may arise in the setting of chronic lymphedema of any etiology. It has been reported in patients with longstanding massive edema, post-mastectomy lymphedema, primary lymphedema, or filarial lymphedema. Clinically, it presents as reddish-blue or purple macular or papular skin lesions, sometimes progressing to subcutaneous nodules. Although typically a late complication, it is potentially life-threatening [18, 19].

Conclusions

In conclusion, lymphedema is a chronic and progressive disorder with significant functional, aesthetic, and psychological impact. Its management requires a comprehensive understanding of its pathophysiology, clinical manifestations, classification, and therapeutic options. This review highlights the importance of early recognition of cardinal signs, progressive skin changes, and stage-based evolution, enabling timely diagnosis, which remains primarily clinical and may be supported by imaging studies when anatomical confirmation or differential diagnosis is required.

Accurate classification into primary or secondary forms - along with further subclassification by age of onset, morphology, and anatomical involvement - not only enhances etiopathogenic understanding but also directly influences therapeutic decision-making and prognosis. Secondary lymphedema associated with malignancy and filariasis continues to represent a significant public health challenge, while primary forms demand a high index of clinical suspicion, especially in younger populations and in the presence of a positive family history.

From a therapeutic perspective, although no definitive cure currently exists, Complete

Decongestive Therapy remains the cornerstone of conservative management, demonstrating effectiveness in reducing limb volume and preventing complications when consistently and appropriately implemented. Compression therapy, manual lymphatic drainage, therapeutic exercise, and meticulous skin care constitute a structured approach that requires patient adherence, ongoing education, and close follow-up.

Surgical alternatives, both physiologic and reductive, represent valuable options in selected cases, particularly in advanced stages or in patients with severe lymphatic compromise. When properly indicated, these interventions can significantly improve symptoms and quality of life.

Ultimately, lymphedema management should be multidisciplinary and holistic, incorporating psychological support and patient education due to the substantial emotional and social burden associated with the disease. Early detection, prompt intervention, and individualized treatment strategies are essential to prevent progression to irreversible stages. Strengthening healthcare professional training and promoting further epidemiological research will contribute to optimizing prevention, diagnostic, and therapeutic strategies, thereby improving prognosis and quality of life for affected individuals.

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