Surgical Intervention for Lymphedema

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KEYWORDS

• Lymphedema • Surgery • Lymph node transfer • Axillary reverse mapping
• LYMPHA • Lymphovenous anastomosis • Vascularized lymph node transfer
• Liposuction

KEY POINTS

- Lymphedema is a chronic, progressive disease with no curative treatment.
- Surgical treatment options are effective at managing early and late stage lymphedema.
- Standardized methods for quantifying lymphedema, universal reporting standards, and an increased amount of high-quality evidence are necessary to advance understanding and management of lymphedema.

INTRODUCTION

Lymphedema is a chronic, progressive disease that affects approximately 140 to 200 million people worldwide.[1,2] There is no curative treatment and palliation is challenging. The incidence is difficult to quantify as early stage lymphedema is often underreported until it necessitates intervention. The etiology includes congenital malformations (primary) and direct injury to the lymphatic channels (secondary). Oncologic treatment for solid tumors is the leading cause of secondary lymphedema in the developed world. In the upper extremity, it is most often associated with breast cancer treatment. Patients with breast cancer who have undergone axillary lymph node dissection and/or radiotherapy are a particularly susceptible group, with reported lymphedema rates as high as 65% to 70%.[3,4] Other causes of secondary lymphedema include trauma, neoplastic obstruction, or inflammatory destruction of the lymphatics. Obesity-induced lymphedema occurs in super obese patients with body mass indexes of greater than 50 to 60 kg/m^2 stemming from overwhelmed or damaged lymphatics secondary to increased adipose tissue and fibrosis.[5,6]

Lymphedema can manifest as mild to severe arm swelling, pain, dysfunction, disfigurement, lipodermatosclerosis, skin ulceration, cellulitis, and rarely lymphangiosarcoma. Treatment of lymphedema includes both nonsurgical and surgical strategies.

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Nonsurgical management involves meticulous skin care, limb elevation, lifelong external compression therapy (both static and pneumatic), and physical therapy with manual lymph drainage and massage to minimize symptoms. Surgical options have been reserved for failure of conservative management historically, but recent data suggest early intervention with surgical techniques may reduce incidence of symptom progression.[7–9] Preventative surgical techniques have been described to reduce the initial disruption of the lymphatics and maintain function. Microsurgical techniques, including lymphaticovenous anastomosis (LVA), vascularized lymph node transfer (VLNT), and lymphaticolymphatic bypass aim to restore the underlying physiologic impairment. Additional surgical interventions such as liposuction and surgical excision remove affected tissues to effectively decrease the drainage load. The successful selection of surgical therapy depends on the stage of lymphedema with LVA and VLNT more suitable for fluid-predominant disease and suction-assisted protein lipectomy (SAPL) for solid disease. Open debulking and reductive procedures are used for management of late-stage solid lymphedematous disease.

ANATOMY AND PATHOPHYSIOLOGY

Lymphedema is an abnormal accumulation of protein-rich interstitial fluid within the interstitial space. It can occur anywhere in the body, most commonly in the lower extremity, followed by the upper extremity and genitalia. Disruptions in the interstitial pressures lead to an imbalance between the arterial capillary inflow, an increased demand for lymphatic outflow, and the decreased capacity of the lymphatic circulation.[10–12]

Secondary lymphedema occurs because of surgical, inflammatory, neoplastic, or traumatic destruction of the dermal lymphatics and their outflow tracts. During early stage lymphedema, compensatory mechanisms including lymphatic regeneration make up for the initial insult. At later stages, the lymphatic capillaries become overwhelmed and damaged leading to fibrosis, thickened basement membranes, and loss of permeability of the lymphatic capillaries.[11] This breakdown allows protein to leak into the interstitial tissues, which increases the tissue colloid osmotic pressure. Water then accumulates in the interstitial space. The edematous tissues signal increased numbers of fibroblasts, adipocytes, keratinocytes, and inflammatory cells. These cell types cause increased collagen deposition, adipose accumulation, chronic inflammation, and fibrosis of the skin and subcutaneous tissues.[11,13] Clinical manifestations include nonpitting edema with overlying skin changes. Stasis of the protein-rich fluid makes the subcutaneous tissues prone to recurrent bacterial and fungal infections, which ultimately leads to progressive damage of the lymphatics.[14]

The enlarged and edematous limb can subsequently cause debilitating and chronic pain, decreased quality of life (QoL), psychosocial issues, increased infection risk, higher medical costs, and loss in productive days for those afflicted with the disease.[15,16] Although the incidence, onset, and progression of lymphedema differ greatly among patients, there are several associated risk factors that have been identified. These risk factors include obesity (body mass index $\geq$30 kg/m^2), number of nodes resected during oncologic surgery, radiation therapy, high rates of paclitaxel use, infection, and underlying genetic makeup.[16,17]

CLINICAL PRESENTATION

Patients who have undergone breast cancer treatment with surgery, radiation, and/or chemotherapy have a lifetime risk of lymphedema occurrence[17,18] and should be monitored with a low threshold of suspicion. Most patients become symptomatic within 8 months of surgery and 75% will present in the first 3 years.[17]