Lymphatic Ulcer of Lower Limb

15

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15.1 Introduction

Lymphedema of the limbs is characterized by obstruction of limb collecting lymphatics by inflammation or mechanical obstruction after trauma or oncological surgery and irradiation. Lack of natural tissue fluid drainage pathways is followed by its accumulation in the interstitial space leading to expansion of tissues called clinical edema. The stagnant tissue edema fluid contains proteins, recirculating immune cells, and cellular debris. Moreover, bacteria penetrating the sole of the skin cannot be evacuated due to lack of outflow pathways to the regional lymph nodes. Among the complications of lymphedema, the most frequent are dermatolymphangioadenitis (DLA), skin fibrosis, deposition of fat tissue in the subcutis, and skin ulcers.

15.2 Pathology

Ulcer development is one of the late complications of chronic lymphedema. Although not frequent, it is a very serious condition of advanced stages [1] (Figs. 15.1 and 15.2). Ulcers are usually formed in the lower parts of the calf or on the dorsum of the foot. Their location is not limited to the internal aspect of the calf as it is the case with venous ulcers. Various locations are dependent on where skin injuries mostly take place. Shoe abrasions are common. Moreover, the large hanging dependent swollen skin fragments often touch the surface, and the epidermis is damaged followed by lymph leakage. Subsequently, the denuded skin surface is colonized by skin flora. Oozing of lymph precludes covering of the surface by edge keratinocytes. In addition, microbes present in the lymphedematous tissues and

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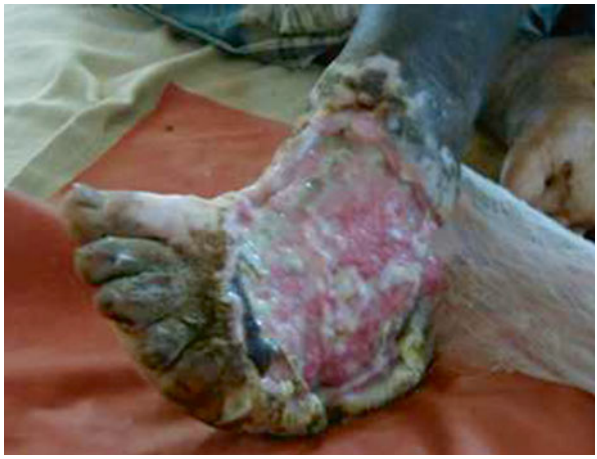

Fig. 15.1 Large foot ulcer in lymphedema of the lower limb stage IV. This ulcer requires debridement and long-term topical therapy concurrently with procedures for decrease of edema of the whole limb

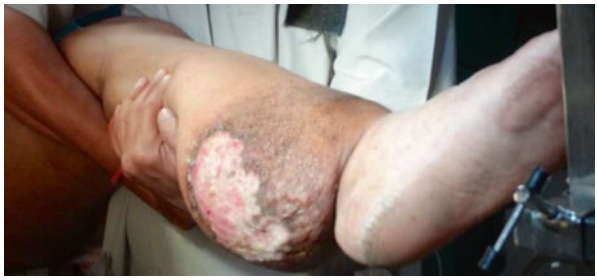

Fig. 15.2 Lymphedema with ulcer

lymph enhance the local host immune reaction [2, 3]. Infiltrating granulocytes phagocytize bacteria and granulation tissue debris and release enzymes preventing the covering of the ulcer surface by keratinocytes. Lymphedema skin ulcers are shallow with smooth edges in contrast to venous ulcers. However, in advanced stage of lymphedema and elephantiasis, necrosis of tissue may occur and tendons and fascia can be seen in the ulcer bottom.

What is specific for obstructive lymphedema is presence of large accumulated mass of bacteria in the swollen tissues deprived of lymphatic outflow [3]. This is predisposed to local inflammation and subsequently ulcer formation. Moreover, patients with obstructive lymphedema suffer from recurrent attacks of dermato-lymphangioadenitis (DLA) caused by bacteria present in a dormant state in the stagnant tissue fluid and lymph [2, 4, 5]. This microflora is, together with microbes colonizing from the environment, responsible for non-healing of ulcers and poor healing after the debulking surgery. Interestingly, the detected bacteria are in vitro sensitive to most antibiotics but not penicillin; however, penicillin is clinically most effective [2]. This means that there are other non-defined non-culturable bacterial strains responding to penicillin, which may be responsible for non-healing.

15.3 Role of the Lymphatic System in Ulcer Healing

Wound healing should not be considered as a process limited to the damaged tissues. It is always accompanied by an intensive response of the regional and, in advanced stages, the systemic lymphatic (immune) system (Fig. 15.3). Penetration of microorganisms through the epidermis and cellular changes caused by tissue injury are almost immediately recognized in the local lymphatic system irrespective of the topography of tissue. Blood immune cells and plasma humoral factors extravasate by the process of chemotaxis and increased capillary permeability. The migrating immune cells incorporate the microbial antigens as well as self-antigens from the apoptotic disintegrated tissue parenchymal cells and migrate via initial and collecting lymphatics to the regional lymph nodes. There, the elimination of antigens and raising of antigen-specific lymphocytes take place.

Intensive transport of microbial and self-antigens along lymphatic to lymph nodes and cellular reaction in the lymphoid tissue result in the formation of antigen-specific cohorts of cytotoxic lymphocytes. It remains so far unknown whether these cells migrate back from the bloodstream to the ulcer and, if so, whether they participate in the healing process. The effect of homing lymphocytes may be pro- and anti-inflammatory as well as lymphatic pro- and antiangiogenic. Lymph nodes are sites for quick reaction to bacteria resulting in their elimination. They may also be sites for raising tolerance to own antigens from wound cellular debris. Maybe in delayed wound healing, the low level tolerance is not sufficient to overcome an

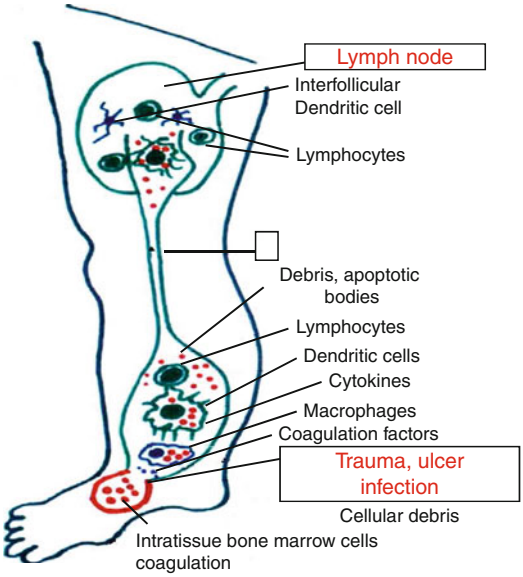

Fig. 15.3 Schematic presentation of how pathological events in the limb evoke reaction of the lymphatic system: lymphatics and nodes

excessive mass of self-antigens. It may then be suggested that in the non-healing ulcers, the aggressive lymph node-derived cells may prevent healing by attacking own granulation cells. Around 20 % of long-lasting venous ulcers are complicated by systemic allergic reactions. An open question remains whether there is a closed functional loop "wound-regional lymph node-blood circulation-wound" and what may be the tasks of the lymphocytes and precursors of dendritic cells circulating in this loop. The hypothetical loop is "wound-afferent lymphatics-lymph node-efferent lymphatics-blood-wound": antigens are transported from the wound via afferent lymphatics to the lymph node; once there, a processing of the antigen takes place followed by proliferation of antigen-specific lymphocytes; these cells are released and transported along efferent lymphatic via the thoracic duct to the blood circulation; some of them are trapped in the liver, gut, bone marrow, and spleen and inform local lymphoid tissue about penetration of the body by microbes and release of own cellular debris; these antigen-specific cells are further extracted from blood at the wound site; there, they participate in the healing and reconstruction processes and, however, may also attack own granulation cells in the autoimmune process. Debris help additional colonization by bacteria. This may explain delayed wound healing and systemic allergic reaction seen in some 20 % patients with long-lasting ulcers. Scintigraphy may help in the localization of the pathology (Fig. 15.4).

15.4 Treatment of Lymphatic Ulcers

Treatment is directed at (a) decrease of lymphedematous limb volume and (b) local procedures for healing.

15.4.1 Conservative Procedures in Lymphedema

Conservative therapy includes intermittent pneumatic compression, bandaging or stocking, and penicillin prophylaxis against recurrent attacks of DLA.

15.4.2 Surgical Procedures in Lymphedema (Figs. 15.5 and 15.6)

Surgical revision of the groin and inguinal lymph nodes is the first pre-debulking step. Enlarged lymph node may be anastomosed to the great saphenous vein according to the technique described by the author [6, 7]. In case the lymph nodes were found fibrotic but afferent lymphatic still patent, the latter should be implanted into the saphenous vein. In case of total fibrosis of nodes and afferent vessels, the inguinal fossa should be cleansed without ligation of any afferent vessels to allow fluid to leak to the wound [8]. Leakage stops within days.

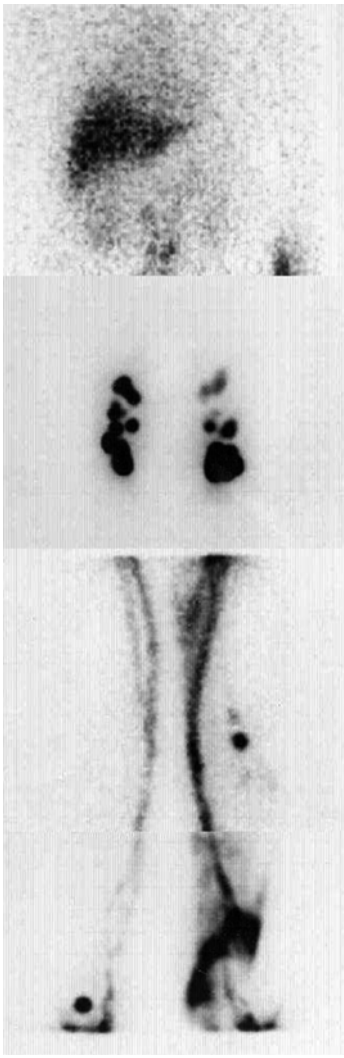

Fig. 15.4 Lymphoscintigram of the left lower limb with calf ulcer. Dissemination of tracer at and around the ulcer, dilated afferent lymphatic collectors, and enlarged lymph nodes

15.4.3 Treatment of Ulcer

The ulcer should be treated as any other ulcer by debridement, topical antiseptics, and nanosilver dressings. Results are often not satisfactory. Then debulking of the fragment of ulcerated tissue should be done. Systemic antibiotics should be given perioperatively and for as along as wound healing is not completed. Thereafter, long-term penicillin (Penidure) should be given in a dose of 1,200,000 u., i.m.,

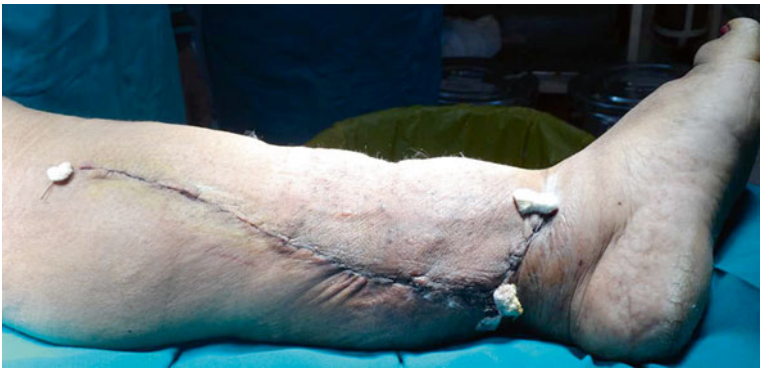

Fig. 15.5 Debulking of tissues with ulcer immediately after surgery

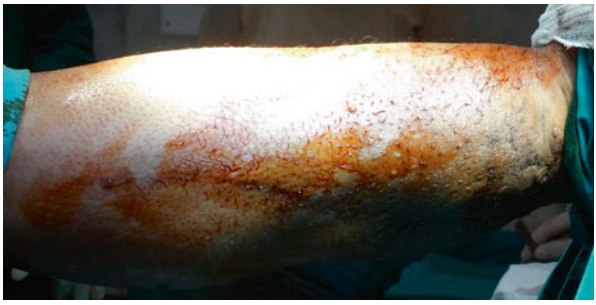

Fig. 15.6 Three months after debulking, just prepared for debulking on the lateral side

every 3 weeks for 1 year or longer, to prevent recurrence of ulceration and DLA attacks. Postoperative compression with elastic bandages and stockings is mandatory. When operative wounds are healed up, intermittent pneumatic compression is highly recommended every day for 1 h at 80–120 mmHg pressures, followed by immediate wrapping of the limb to prevent edema fluid reaccumulation [9, 10].

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