Leg Ulcer

thumb|right|Bércový vřed hojící se granulační tkání thumb|right|Bércový vřed povleklý fibrinem **Leg ulcer** (lat. U*lcus Cruris*) is the loss of a skin substance reaching various depths into the subcutaneous tissues. It is a chronic wound with a healing time longer than 6 weeks. It results from severe trophic changes in tissue damaged by a previous disease, most often vascular. It occurs mainly in old age. It is a poly-ethiological disease.[1]

Etiopathogenesis

In most cases, it occurs as a result of severe trophic changes - up to 85% of cases have a venous origin (so-called venous ulceration), 10% of cases have an arterial origin, the remaining 5% have another cause.

External causes: physical, chemical, infectious, artifacts.

Internal causes: venous, arterial, lymphatic, haematogenous, coagulation, metabolic, neuropathic diseases, vasculitis, tumours, mixed disorders. [1]

Venous Ulceration

thumb|right|Bércový vřed žilního původu

Etiopathogenesis

1. **Phlebothrombosis:** After its rejection, partial or complete recanalisation of the affected vein may occur, but mostly with destruction and subsequent insufficiency of the valves in the deep and connecting venous system. The consequences of such macrocirculation disorders are blood stasis and the gradual development of venous hypertension, which has a secondary response in the microcirculation - increased capillary permeability, loss of plasma proteins and the formation of a fibrin pericapillary cuff. These changes, together with rheological disorders, lead to trophic changes in the tissues and to the formation of the so-called post-thrombotic shin ulcer,
    - Ulcerations are deep, extensive, often circular, with irregular, often mined margins, they have a pronounced exudative character, a coated base and are accompanied by extensive edema.
2. **Primary varicosity:** Leg ulcers are a terminal manifestation of chronic venous insufficiency caused by insufficiency of venous valves in the deep venous system, in the connecting veins, or in the superficial venous system, or a combination of all previous disorders.
    - Ulcerations are smaller, have a shallow base and smooth edges;
    - Most often located on the inner side of the distal third of the shin in the place of the so-called Cockett perforators, but also above other places in the shin area.[1]

Arterial ulceration

They are caused by a partial or complete vascular occlusion, the causes of which are most often arteriosclerotic changes. The arterial occlusion first shows black-brown necrosis on the skin in the affected area, and after its detachment, ulcerations with yellow, adherent or greasy coatings appear.

Ulcerations are often circular in shape and localised on the anterior, medial and lateral sides of the lower legs, as well as the insteps, toes and heels. They are quite painful, especially in the horizontal position of the feet. [1]

Examination

- Examination of the venous and arterial system of the lower limbs - sonography (Doppller, Doppler Duplex);
- Examination of the functional fitness of the muscle venous pump (D-PPG);
- In unclear cases - phlebography, isotope phlebography, arteriography;
- Microbiological, ev. mycological examination[1].

Healing

Phase 1: exudative (inflammatory, cleansing)

- Defensive reaction of the organism to various noxae;
- Vasodilation, increased vascular permeability, and chemotactic factors allow the migration of the neutrophils and macrophages to the wound site, where they phagocytose cellular debris, foreign material, and pathogens. At the same time, they secrete inflammatory mediators, cytokines, growth factors, and other chemotactic factors that activate the cells needed for healing. They also release proteolytic enzymes that break down damaged and necrotic tissue. [1].

Phase 2: Proliferative (granulation)

- Creation of new granulation tissue interwoven with blood vessels;
- Cytokines and growth factors stimulate fibroblasts and vascular endothelial cells[1].

Phase 3: Re-epithelialization, remodeling

- Migration, mitotic division and differentiation of cells with the support of growth factors and maturation of collagen fivers;
- The cells of the basal layer migrate to the surface of the skin and at the same time approach by amoeboid movement from the edges of the wound to its centre;
- Eventually, the cells mature, the epidermis is definitively rebuilt with individual layers and functions. In parallel with this process, structural changes occur in the joint and the original wound changes into healed, scar tissue. [1]

In chronic wounds, including shin ulcers, the healing process is disrupted by a number of local and general factors. Therefore, the individual phases are extended, especially the exudative and granulation phases.[1]

Complication

- Contact allergies from the load applied externally
- Erysipelas.

Therapy

- Local and systemic treatment (venopharmaceuticals, vasodilators, analgesics and enzymes preparations);
- Removal of all necrotic, fibrin and bacterial coatings or wound debridement - mechanical, autolytic, chemical, enzymatic;
- Stimulation of granulation and epithelialization - ointment therapy and wet compresses;
- External compression in ulcers of venous and lymphatic etiology - short-stretch bandages[2].

Links

References

1.
2.

Related articles

- Chronic venous insufficiency
- Wound healing