OA Dr. Elisabeth LAHNSTEINER
FÄ für Allgemeinmedizin und Familienmedizin

Wound bed conditioning with autologous wound patch in the extremities

Neues aus der Forschung & Innovationen – JATROS 06/2022
OA Dr med.univ. Elisabeth Lahnsteiner

Epidemiologically, there are significant interdisciplinary challenges in the treatment of acute and chronic wounds of various etiologies due to the increase in sports and leisure accidents as well as changes in demographics. Understanding the causal relationships of clinical presentations, risk factors, and differential diagnoses, as well as the processes of wound healing disorders, is crucial for achieving long-term therapeutic success and patient satisfaction through restored mobility and quality of life.

Wound healing disorders can have traumatic, patient-related, or iatrogenic causes or a combination of these factors. The uniqueness of disturbed wound healing for traumatology and orthopedics lies in the potential involvement of bone. The primary focus of treatment must be to prevent secondary damage, such as chronic osteitis/osteomyelitis or functional impairment, through appropriate measures.

Acute traumatic or prolonged wound healing disorders such as wound edge necrosis, wound dehiscence, and hematomas require prompt emergency revision with uncompromising debridement and comprehensive necrectomy and sequestrectomy.

Strategies for definitive soft tissue repair range from simple wound closure using secondary sutures to split-thickness skin grafts to local or free flap transfer, with these measures also applicable to the management of chronic wound healing disorders.

Causes of disturbed wound healing

The physiological healing process of post-traumatic or chronic postoperative wounds is often disrupted by numerous factors. Reduced blood flow or vascular injuries cause local painful hypoxemia and cell death. Necrosis, exudate, hematomas, phlegmonous soft tissue infections, suture material intolerance predispose to wound dehiscence or fistulation. Osteomyelitis and osteolysis are common accompanying phenomena.

Lack of immobilization and pressure relief on the operated limb as well as inadequate orthopedic footwear adapted to the changed anatomical conditions are also significant disrupting factors in the reparative process. The comorbidity of accompanying systemic diseases such as metabolic disorders, circulatory disorders, immunodeficiency, and limited mobility requires a multidisciplinary treatment strategy.

Biology of wound healing

The wound healing process is a complex molecular and biochemical process controlled by the interaction of cytokines, proteases, growth factors, and chemotactic processes.

The multifaceted processes such as chemotaxis and phagocytosis, connective tissue proliferation, collagen degradation and remodeling, angiogenesis, and re-epithelialization are finely coordinated.

The course of wound healing can be divided into coordinated and interacting processes.

In hemostasis, after initial vasoconstriction, vasodilation with reactive hyperemia follows. Increased vascular permeability leads to the leakage of blood plasma into the interstitium, clinically resulting in local edema. This reactive process is controlled by prostaglandins from tissue defects, histamines from mast cells, and vasoactive amines (e.g., serotonin) from activated platelets.

The platelets within the blood clot are not only responsible for hemostasis but also release numerous wound healing mediators such as EGF, PDGF, IGF-1, and TGF-1.

In the inflammatory phase, chemotactic signals mediate the process of leukocyte diapedesis, facilitating the passage of leukocytes from blood vessels into the wound area. Neutrophilic granulocytes play a crucial role in wound cleansing. They phagocytose and eliminate bacteria, foreign material, and devitalized tissue, thus preventing wound infection. Neutrophils synthesize and release inflammatory mediators such as TNF-alpha and IL-1, activating fibroblasts and epithelial cells. They produce and store large amounts of aggressive proteases and free oxygen molecules, which they use to digest phagocytosed material. Upon their death, these toxins enter the wound area and can contribute to a prolonged inflammatory process under increased cellular load. Ultimately, they are excreted or phagocytosed by macrophages along with exudate, debris, and toxins.

Macrophages play a key role in wound repair by mediating the transition from the inflammatory phase to proliferation. They produce numerous cytokines and growth

factors, e.g., TNF-alpha, PDGF, VEGF, TGF-alpha, TGF-ß, IL-1, IL-6, IFG, and FGF. These factors lead to the ordered recruitment and proliferation of fibroblasts and endothelial cells and the formation of high-quality granulation tissue. Depletion of monocytes and tissue macrophages disrupts the physiological wound healing process, resulting in inadequate autologous wound debridement and delayed fibroblast proliferation. Macrophages regulate and stimulate the complex processes of fibroplasia and angiogenesis.

In the proliferative phase, the provisional matrix plays a crucial functional role. It serves as an important reservoir for growth factors and cytokines, anchoring and inert guiding structure during cell division, migration, and differentiation. Matrix metalloproteases enzymatically regulate the process of cell migration over and through the ECM. If the critical balance of MMP and TIMP in the catabolic and anabolic remodeling process is disturbed, excessive MMP degradation can cause the breakdown of growth factors and their receptors, resulting in a prolonged healing process.

Angiogenesis and neovascularization are also induced by growth factors such as b-FGF, TGF-ß, and VEGF. The local wound environment, including hypoxia, acidic pH, and high lactate levels, stimulates vascular formation. Adequate oxygen supply and nutrient enrichment for reparative tissue processes are restored. Granulation tissue primarily consists structurally and histologically of a dense capillary network, proliferating fibroblasts, tissue macrophages, surrounded by a matrix of collagen, glycosaminoglycans (hyaluronic acid), and glycoproteins.

In the remodeling phase and re-epithelialization, the migration and proliferation of keratinocytes are stimulated by the "free edge effect" and local release of growth factors EGF, TGF-alpha, TGFß, and KFG, as well as the expression of WF receptors, initiating the formation of an intact basal lamina with cornified epidermis. Mechanical stability is critically influenced by the geometric shape of the wound, surgical methodology, and wound etiology.

Biology of chronic wound healing

In chronic wounds, the physiological healing process is disrupted in the inflammatory or proliferative phase.

Chronic wounds typically exhibit excessive cellular inflammatory activity. The exudate of chronic wounds is rich in pro-inflammatory cytokines such as TNF-alpha and IL-1 beta and increased concentrations of free oxygen radicals. Macrophages are inadequately activated, leading to suppression of healing-promoting cytokines and

growth factors release. The phenomena of premature cell aging in the chronic wound milieu, increased activity of pro-inflammatory cytokines, oxidative stress, or an overload of bacterial toxins are suspected causes of disrupted tissue regeneration. The accumulation of senescent fibroblasts with increased production of proteolytic enzymes exacerbates the chronic healing process.

The role of platelets in the healing process

Platelets play a key role in the repair mechanism of soft tissue defects in both acute and chronic wounds. They provide essential growth factors such as FGF, PDGF, TGF-ß, EGF, VEGF, IGF, which are essential for the migration, differentiation, and proliferation of stem cells. Additionally, platelets stimulate fibro
A combination of standardized phased wound bed conditioning in therapeutic combination with Platelet Rich Plasma (PRP) and PRF (Platelet Rich Fibrin) wound patch stimulates the formation of high-quality granulation tissue while simultaneously reducing inflammatory exudation in traumatic and chronic wounds in the extremities. The indication and correct timing of application of an Autologous Wound Patch require careful clinical wound diagnosis by a physician and surgical soft tissue management. Platelet-rich plasma thus represents a promising new therapeutic adjunct in the treatment of wounds of various etiologies due to its biological mode of action.

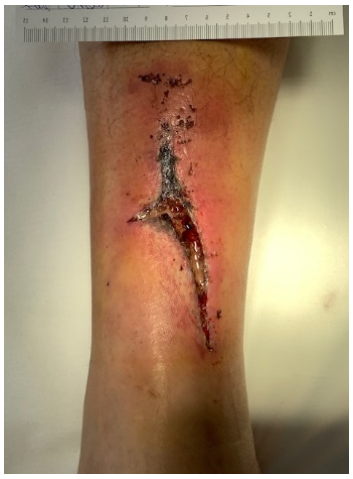
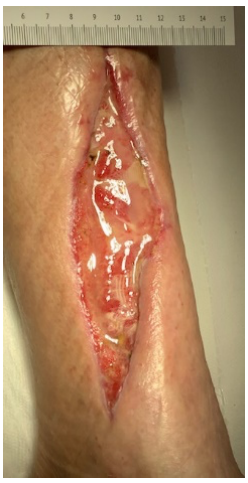
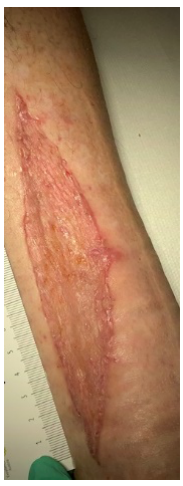

Abb 1: Vlc cruris sin permagna infecta, Kompartementsyndrom
Abb 2: Autolog. Wundpach
Abb 3 : Meshgraft TX + biolog. Wundverband mit PRF

Author:
WUND.ORDINATION
WUND.CHIRURGIE
OA Dr med.univ.Elisabeth Lahnsteiner
Leitung Ärzteteam Wundheilung, WIen
Ehem. Leitung Wundambulanz Orthopädisches Spital Speising, Wien

1190 Heiligenstädterstraße 46-48 /Ebene 4/ Top 3
Handy: 0676 72 69 680
E-Mail: info@wundordination.at
Homepage: www.wundordination.at