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Application of Platelet-Rich Fibrin in Soft Tissue Management of Oral Implantation

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Abstract: Platelet-rich fibrin (PRF) possesses the functions of regulating the local microenvironment, promoting soft tissue repair and maintaining tissue stability by virtue of its three-dimensional fibrin network structure and the sustained release of active ingredients, laying a favorable application foundation in the soft tissue management of oral implantation. This article reviews the structural characteristics and biological properties of PRF, as well as its applications in wound closure of implant sites, soft tissue augmentation and keratinized gingiva reconstruction.

Keywords: Platelet-rich fibrin; Oral implantation; Soft tissue management

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1. Introduction

The long-term success of oral implant rehabilitation relies not only on the osseointegration of implants, but is associated with the peri-implant soft tissue conditions [1]. Stable and sufficient soft tissue can form an effective biological barrier, reduce the risk of bacterial invasion, and play a vital role in maintaining the gingival margin morphology and prosthetic aesthetics. In clinical practice, however, affected by periodontitis destruction, prolonged edentulous time, absorption of hard and soft tissues after tooth extraction, and individual differences, the implant site is often complicated by insufficient soft tissue volume, inadequate width of keratinized gingiva, and difficulty in wound closure of the surgical area, all of which tend to impair the stability and long-term efficacy of implant rehabilitation. For the management of deficient soft tissue conditions in implant areas, conventional therapeutic methods at present mainly include connective tissue grafting, free gingival grafting and local flap techniques. Nevertheless, such surgical approaches are accompanied by drawbacks such as relatively large surgical trauma, high operational sensitivity and limited patient acceptance. Platelet-rich fibrin (PRF), first proposed by Dohan et al. in 2006, is a platelet concentrate prepared by centrifugation of autologous venous blood without the addition of anticoagulants or exogenous activators [2]. Based on a three-dimensional fibrin network, it contains platelets, leukocytes and a variety of growth factors. It can form a bio-scaffold with a certain supporting effect locally and release active ingredients sustainably, thereby participating in the process of wound healing and tissue regeneration [3]. For this reason, PRF has been gradually applied in implant-related soft tissue management and achieved favorable clinical outcomes in wound healing and improvement of local soft tissue conditions. Therefore, based on the

biological mechanism of PRF in implant soft tissue repair, this article reviews its applications in wound management of implant sites, soft tissue augmentation and keratinized gingiva reconstruction.

2. Biological basis of PRF in implant soft tissue regeneration

2.1. Structural characteristics and bioactive components of PRF

PRF is a second-generation platelet concentrate obtained by centrifugation of autologous venous blood, and its formation process simulates the physiological coagulation reaction of the human body. Without the addition of anticoagulants, blood is divided into three layers after centrifugation: the upper layer is platelet-poor plasma, the middle layer is PRF gel, and the lower layer is the red blood cell layer. Among them, PRF is located in the middle area and presents a milky white gel state. This structure derives from the three-dimensional network formed by the gradual polymerization of fibrin, in which platelets and various cellular components are stably embedded, constructing a biological scaffold with certain elasticity and mechanical supporting capacity. The fibrin network can form a relatively stable spatial structure in tissue defects and closely adhere to the surrounding tissues. It provides mechanical support for local tissues in the early healing stage, and meanwhile offers attachment sites for cell adhesion and tissue ingrowth. Zhang Wentao et al. [4] observed in the repair of severe alveolar ridge defects in the maxillary anterior region that the application of PRF achieved favorable soft tissue coverage and enhanced wound stability. Its three-dimensional fibrin network supports cell migration and tissue ingrowth, as well as sustains the continuous release of active ingredients. Accordingly, the symmetry of the gingival margin contour and the fullness of the gingival papillae are improved, the risk of “black triangle” formation is relatively reduced, and the local tissue morphology is maintained more stably. In terms of histological configuration, the most prominent feature of PRF is its three-dimensional fibrin network structure. Composed of cross-linked fibrin fibers with uniformly distributed internal pores, this structure can effectively capture and store platelets, leukocytes and a variety of cytokines [5]. This structure endows PRF with good plasticity, allowing it to be clinically compressed into membrane materials or directly applied to wound filling in the form of gel, while providing a suitable microenvironment for cell adhesion, migration and proliferation. Compared with traditional platelet concentrates, PRF requires no exogenous coagulation activators, and its fibrous structure is more similar to that of natural blood clots, which is conducive to maintaining the continuity and stability of the tissue repair process.

In terms of composition, PRF is rich in cellular components, such as platelets and leukocytes. The α-granules of platelets store a variety of growth factors, including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), and insulin-like growth factor (IGF). As essential biological regulatory molecules, these growth factors bind to cell surface receptors and participate in the regulation of cell proliferation, chemotaxis, differentiation, and extracellular matrix synthesis. Released in a relatively coordinated manner at different stages, they exert synergistic effects on the proliferation, adhesion and mineralization of osteoblasts [6]. Meanwhile, leukocytes contained in PRF can secrete multiple inflammation-related factors and participate in the regulation of local inflammatory responses and immune processes, thereby maintaining a microenvironment conducive to tissue repair. In addition, PRF exerts regulatory effects on various cell types, including osteoblasts, periodontal ligament cells, gingival fibroblasts, oral epithelial cells and mesenchymal stem cells, which provides the biological basis for bone tissue repair and soft tissue healing [7]. Notably, the fibrin network formed by PRF possesses a slow degradation property. The encapsulated growth factors are released sustainably rather than in an abrupt burst within a short period. This maintains stable biological activity signals in the early stage of wound healing and continuously regulates cellular behaviors and tissue reconstruction, which serves as a crucial feature distinguishing PRF from traditional platelet concentrates.

2.2. Mechanism of PRF-mediated soft tissue healing

At the cellular behavioral level, PRF exerts a promotive effect on gingival fibroblasts. A variety of growth factors released by PRF enhance cell proliferative activity and accelerate cell migration, enabling cells at the wound margin to

rapidly accumulate toward the defective area. Meanwhile, the capacity of fibroblasts to secrete collagen is improved with increased deposition of type I collagen, which gradually restores the tension and structural stability of soft tissues and lays the foundation for soft tissue reconstruction. In terms of matrix remodeling, the fibrin scaffold provided by PRF presents a spatial structure similar to that of natural blood clots and serves as a support for cell adhesion and spreading. With the gradual degradation of the scaffold, the local extracellular matrix is continuously replaced by newly formed collagen, transforming the wound from a loose clot structure into dense connective tissue, which manifests as enhanced tissue continuity and improved quality of wound closure. Angiogenesis is also significantly regulated. Vascular-related growth factors sustainably released from PRF accelerate the formation and uniform distribution of new capillaries in the wound area. The establishment of microcirculation shortens the duration of ischemia, facilitates the delivery of oxygen and nutrients, and thereby creates favorable conditions for epithelial coverage and connective tissue maturation. Wu Jing et al. [8] also pointed out that PRF is rich in growth factors such as PDGF, VEGF and TGF-β, which can promote the proliferation of vascular endothelial cells and the secretion of pro-angiogenic factors, participate in extracellular matrix synthesis, and exert positive effects on tissue repair and regeneration. In the regulation of inflammatory responses, leukocytes enriched in PRF and various cytokines released by them jointly participate in the local immune response, enabling the wound to form a relatively ordered inflammatory state at the early stage. Studies have shown that during PRF-assisted treatment, the levels of inflammatory factors such as IL-1β, IL-8 and TNF-α are significantly decreased, indicating that PRF can modulate the release of inflammatory mediators in the early stage of injury and reduce the degree of local inflammatory tissue damage [9]. The release rhythm of inflammatory mediators is precisely regulated, which not only ensures the necessary initiation of inflammation after injury, but also avoids tissue destruction caused by excessive persistent inflammatory response, so as to alleviate local exudation and cellular injury.

3. Application efficacy of PRF in soft tissue management of oral implantation

3.1. Wound closure of implant site and socket preservation after tooth extraction

After the formation of an extraction wound, the wound surface is mostly covered by a blood clot. With a relatively loose structure, the clot is susceptible to displacement or rupture due to the oral environment and mechanical stimuli, resulting in unstable wound margin adaptation and delayed healing. When PRF is placed into the extraction socket or covered over the wound surface, it forms a dense local fibrin network that closely adheres to the wound. It provides early mechanical support to the wound margin, reduces marginal collapse, and enables more continuous epithelial migration toward the wound center. Deng Xuelian et al. [10] indicated that the molecular components of PRF resemble those of natural blood clots. It acts as a biological scaffold to provide a microenvironment for cell proliferation and differentiation, improves local healing by promoting angiogenesis and tissue repair, and thereby facilitates wound stability and continuous tissue regeneration. As healing progresses, the fibrin matrix formed by PRF is gradually replaced by newly regenerated tissue, allowing a smoother transition from the initial clot state to dense connective tissue. Meanwhile, wound contraction is accelerated, and the incidence of wound dehiscence and bleeding is reduced, maintaining the continuity of local tissues. Clinically, this is manifested as shortened wound healing time and improved healing quality. Wang Fang et al. [11] confirmed that the application of PRF at extraction sites markedly shortens wound healing time. The growth factors contained in PRF promote the repair of traumatic regions, favor local tissue recovery and structural maintenance, and optimize the overall wound healing process. Previous studies have also found that PRF application alleviates postoperative swelling and pain, which is associated with improved local microcirculation and regulated inflammatory responses. Pu Lili et al. [12] supported this viewpoint, reporting that the use of PRF after mandibular impacted third molar extraction significantly reduces postoperative pain and swelling, achieves better soft tissue healing, and lowers the incidence of complications compared with conventional treatment.

3.2. Soft tissue augmentation and keratinized gingiva reconstruction

Tooth loss is often accompanied by alveolar ridge resorption, which leads to reduced soft tissue volume and a thinner gingival biotype. This weakens the resistance of peri-implant tissues to mechanical stimulation and dental plaque, further increasing the risk of marginal bone resorption and soft tissue recession. Relevant studies have indicated that insufficient peri-implant soft tissue thickness reduces the tissue buffering capacity against external stimuli, impairs anti-inflammatory and reparative potential, and renders tissues more vulnerable to plaque aggression, thereby raising the likelihood of peri-implant tissue destruction [13]. Under such compromised soft tissue conditions, the dependence on regenerative materials increases. When PRF is placed around implants or in the submucosal space, it can form stable adhesion with the recipient tissues. Its fibrin network provides early spatial support and is gradually replaced by newly synthesized collagen during healing, resulting in increased local soft tissue thickness. Wang Huichao et al. [14] further confirmed that PRF helps improve peri-implant soft tissue attachment and local stability. Two months after treatment, partial or complete gingival coverage was achieved at the implant neck, accompanied by increased width of keratinized gingiva. There is a significant reduction in probing depth and sulcus bleeding index compared with baseline. PRF also exerts favorable effects on keratinized gingiva reconstruction. In the second-stage implant surgery or soft tissue contouring procedure, PRF can be used as a covering material or combined with flap elevation to expand the area of keratinized tissue and reduce surgical site contraction and wound margin retraction. Tang Qiong et al. [15] compared the clinical outcomes of extraction sockets filled with fresh blood clot versus PRF gel. The results showed that the PRF group presented a more prominent increase in keratinized gingiva width, improved gingival soft tissue thickness, and relatively less wound shrinkage during postoperative follow-up, verifying that PRF possesses superior advantages in maintaining soft tissue volume and promoting keratinized gingiva formation.

4. Summary

PRF has demonstrated favorable clinical efficacy in the soft tissue management of oral implantation. Owing to its fibrin network structure and bioactive components, PRF presents considerable application value in wound closure, soft tissue augmentation, and keratinized gingiva reconstruction. Its clinical performance is closely correlated with centrifugation parameters, material form selection and specific application methods. Differences in operational conditions lead to variations in the structural characteristics and the release pattern of bioactive ingredients of PRF. Further standardization of preparation parameters and application protocols is still required to improve the stability of its clinical application.

Disclosure statement

The author declares no conflict of interest.

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