Adaptation between periodontal factors and restorative effects: a narrative review

Qingmeng Li*

Oral medicine, Air Force Medical University, Xi'an, 710032, China

Abstract: The restorative effects of implants, full crowns, and removable dentures are closely related to periodontal factors. Subgingival periodontal pathogens, excessive immune responses, and unfavorable anatomical factors can lead to periodontal tissue inflammation, causing alveolar bone resorption, which is detrimental to the function of restorations. They even lead to the occurrence of peri-implantitis, dental stomatitis, gingival atrophy, etc. Additionally, the connection method between the implant and the abutment, the margin length of fixed prostheses, and the roughness of complete dentures can also impact periodontal status by affecting biological width and plaque attachment. Regular plaque removal, control of systemic diseases, and improvement of prosthetic performance can enhance the adaptation of restorations to periodontal tissues, thereby improving restorative effects. This article aims to explore the interactions between periodontal factors and restorative effects to provide a scientific basis and guidance for clinical treatment.

1 Introduction

Implants, full crowns, and complete dentures, as the most common fixed and removable prostheses, greatly meet the aesthetic needs of patients with dental and tooth loss. However, the effectiveness of these restorations not only depends on their design and materials but also closely relates to the patient's periodontal health. Various factors, including subgingival periodontal pathogens, individual immune responses, and gingival biotypes, can cause inflammation of periodontal tissues, leading to alveolar bone resorption and thereby affecting the stability and functional performance of restorations. Additionally, poorly designed restorations can adversely impact periodontal status, exacerbating conditions such as peri-implantitis and periodontitis. For instance, excessively long crown margins can disrupt biological width, and overly rough implant materials can increase plaque accumulation, both of which can accelerate alveolar bone resorption. Therefore, understanding the compatibility between periodontal factors and restorations is crucial for enhancing the long-term retention of prostheses in the oral cavity and maintaining aesthetics.

2 Periodontal factors influence the repair effect

The long-term retention rate of prostheses in the oral cavity is closely related to periodontal factors after dental prosthetic restoration. Accumulation of dental plaque on the prosthesis can lead to periodontitis and peri-implantitis, subsequently affecting the restoration outcome. Moreover, periodontal pathogens release virulence factors that influence the oral immune microenvironment, resulting in immune-related periodontal damage. Additionally, inherent periodontal anatomical factors significantly impact the effectiveness of the restoration.

2.1 Oral Microbial Factors

The oral cavity is an environment where multiple microorganisms coexist. Studies have shown that dental plaque on teeth and prostheses is the initiating factor for periodontal disease after restoration [1]. When plaque accumulates excessively and the microbial homeostasis is disrupted, the number of Gram-negative anaerobic bacteria increases, which is associated with chronic periodontitis and peri-implantitis [2]. Among these anaerobic bacteria, the "red complex"—comprising Porphyromonas gingivalis, Treponema denticola, and Tannerella forsythia—exhibits the highest pathogenic potential. These bacteria express various virulence factors that directly damage periodontal tissues, disrupt the host defense system, invade gingival epithelial cells, and promote destructive immune responses in the host. For instance, the major virulence factor of Porphyromonas gingivalis, gingipain (gingival protease), can degrade collagen fibers in the gingiva, leading to inflammatory responses in periodontal tissues [3]. Therefore, if dental plaque around the prosthesis and adjacent teeth is not promptly removed after restoration, it can lead to gingival swelling, periodontitis, and peri-implantitis, subsequently causing periodontal tissue destruction, manifested as loosening of abutment teeth or implants, gingival recession, and severely affecting functionality and aesthetics.

* Corresponding author: 2801950756@qq.com

2.2 Host Immune Microenvironment

The immune response is a double-edged sword; it can kill bacteria on one hand but also attack body tissues, causing immune imbalance. The substantial accumulation of plaque around the prosthesis can induce inflammation, leading to the infiltration of inflammatory cells, primarily B lymphocytes and T lymphocytes, into the surrounding soft tissues of the prosthesis, resulting in clinical manifestations such as congestion and edema. Macrophages play dual roles in promoting and inhibiting inflammation, while neutrophils are key cells in both innate and adaptive immunity and are crucial drivers of acute inflammation. Additionally, there is an upregulation of cytokine expression levels around the prosthesis. Pro-inflammatory cytokines such as IL-1β and TNF-α can induce the activation of osteoclasts and promote alveolar bone resorption by increasing RANKL expression and reducing OPG expression in osteoblasts [4]. Further extensive infiltration of inflammatory cells can cause tissue destruction and bone loss, leading to alveolar bone resorption and even implant loosening and loss [5].

2.3 Anatomical Factors

Biological width determines the stability of implants and the marginal position of full crown restorations. The biological width of natural teeth, which is the constant distance of approximately 2mm from the base of the gingival sulcus to the crest of the alveolar bone, includes 1mm of epithelial attachment and 1mm of connective tissue attachment [6]. The biological width of implants is typically around 3-4mm, including the gingival sulcus epithelium, junctional epithelium, and the connective tissue below the implant-bone interface. Its biological significance lies in the necessity to maintain sufficient soft tissue thickness and bone wall thickness during implantation; otherwise, to preserve the biological width, bone resorption and implant instability may occur. If the crown margin is too long during full crown restoration, disrupting the biological width, it can lead to gingival recession and gingivitis. Additionally, the morphology of the gingiva affects the outcome of full crown restorations. Patients with a thick gingival phenotype generally have a greater biological width and better aesthetic outcomes compared to those with a thin gingival phenotype, which, however, is more favorable for maintaining oral hygiene and health post-restoration [7]. Furthermore, the crown-to-root ratio, root anatomical structure, and periodontal ligament area all influence the prognosis of the restoration [8].

The influence of various periodontal factors on the restoration effect is summarized in the figure 1.

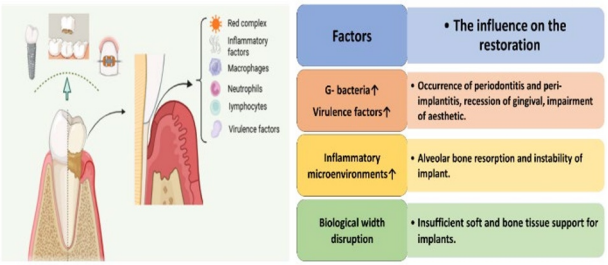

Figure 1. The Increase of Gram-negative Bacteria, Infiltration of Inflammatory Cells, and Factors Such as Biological Width Can Lead to Alveolar Bone Resorption, Adversely Affecting Restoration.

3 The Impact of Adverse Periodontal Factors on Different Types of Restorations

3.1 Impact on Implants

Peri-implantitis is considered an inflammatory disease closely related to oral microorganisms, characterized by inflammation of the peri-implant mucosa and progressive loss of supporting bone [9]. Increased plaque accumulation, soft tissue inflammation, and marginal bone loss can reduce the biological width (i.e., less than 2 mm), increasing the risk of peri-implantitis. Therefore, procedures such as keratinized gingiva augmentation can improve the health and stability of the tissues around implants [10]. Additionally, the design features of implants, including the nature of the connection between the implant and the abutment, as well as the surface characteristics of the abutment and implant, can also affect the maintenance of peri-implant soft tissue integrity. For example, compared to externally connected implants, conical connections are less prone to peri-implant diseases. This is because bacteria are less likely to invade the implant/abutment interface in conical connection implants, ensuring better sealing and less bone loss [11].

3.2 Impact on Fixed Prostheses

The compatibility between periodontal factors and the restoration affects the aesthetic outcome and the inflammatory response around the restoration. The position of the preparation margin, the shape of the restoration, and the design and cleanliness of the bridge influence the gingival tissue's response to the restoration [6]. On the one hand, if the crown margin does not fit well with the tooth structure, it can lead to the dissolution of the adhesive, causing gingivitis, microleakage, marginal discoloration, and secondary caries [12]. On the other hand, any roughness and irregular edges in the structure of fixed prostheses can lead to microbial attachment, worsening oral hygiene, and leading to gingivitis and periodontitis. Additionally, the size of the periodontal ligament area is closely related to periodontal support and can be used clinically to assess whether a tooth can serve as a good abutment. When the occlusal force on the periodontal ligament is too great, or there is inflammation

in the periodontal tissues, it can cause alveolar bone resorption and destruction, manifesting as alveolar ridge resorption [13].

3.3 Impact on Removable Dentures

Denture stomatitis is a common infectious disease in wearers of removable dentures, characterized by erythema or tissue edema in the denture-bearing area. Numerous studies have shown that Candida albicans, as the primary microbial driver of denture stomatitis, mainly infects host cells through its secreted invasive enzymes, adhesins, and hyphal formation as virulence factors [14]. The higher the surface energy and roughness of the denture material, the easier it is for pathogenic microorganisms to adhere. Therefore, smooth or highly polished denture surfaces not only extend the lifespan of dentures and enhance aesthetics but also help maintain oral hygiene and reduce plaque adhesion. Additionally, patients with dry mouth are also prone to denture stomatitis because their saliva lacks innate immune-related molecules and cannot flush the oral cavity, leading to Candida adhesion and colonization in the mouth [15].

4 Controlling periodontal pathogenic factors and improving repair effectiveness

4.1 Controlling Dental Plaque

Plaque accumulation is the initiating factor for periodontal disease around restorations in the oral cavity. To control dental plaque, regular removal and maintaining oral hygiene are essential. After crown restoration, in addition to paying attention to personal cleaning methods such as brushing and flossing the restoration margins, regular visits to the dentist for manual or ultrasonic scaling can reduce the number and proportion of periodontal pathogens, thereby reducing the incidence of secondary caries and periodontal disease [16]. However, when performing ultrasonic scaling on porcelain crowns and implants, metal tips should not be used to avoid damaging the porcelain or scratching the implant surface; instead, carbon fiber tips or resin tips can be chosen. If severe periodontal disease occurs around the full crown or implant, subgingival scaling and periodontal surgery can be performed to remove plaque and promote periodontal tissue regeneration [17]. Surgical treatments include flap surgery and guided bone regeneration [18].

4.2 Improving the performance of restorations

Improving the performance of restorations is crucial for better repair outcomes. Firstly, poor fit between the crown neck and shoulder, overly long crown margins, and excessive subgingival shoulder width can lead to plaque accumulation at the crown margins, often causing gingival hyperplasia, swelling, and bleeding, resulting in gingivitis and secondary caries. Secondly, prolonged retention of adhesives in the gingival sulcus or on the tissue surface of the bridge can cause gingivitis and even affect the stability of the retainer [19]. Therefore, improving the marginal fit between the restoration and tooth structure and promptly removing excess adhesive are very important. Additionally, maintaining a good axial contour of the restoration is vital; excessive or insufficient convexity can lead to food impaction or direct impact on the gingiva, causing inflammatory reactions and gingival recession [20]. Furthermore, enhancing the smoothness of the restoration surface can reduce plaque adhesion, thereby reducing inflammation. Titanium-based implants, as the most widely used implants, may trigger the release of titanium dissolution products into the surrounding tissues when there is corrosion on the implant surface, improper implant-abutment interface positioning, or excessive adhesive, stimulating inflammation and foreign body reactions in the peri-implant tissues and affecting the development of peri-implantitis [21].

4.3 Correcting Bad Habits and Controlling Systemic Diseases

Numerous studies have shown that smoking and diabetes are identified risk factors for peri-implantitis, making it crucial to correct unhealthy habits [22]. Although the definitive relationship between smoking and peri-implantitis has not been established, smoking may increase the incidence of bone loss, enhance susceptibility to peri-implantitis, impair wound healing, and lead to implant loosening. Some studies indicate that smokers, compared to non-smokers, generally exhibit significantly higher levels of pro-inflammatory cytokines, probing depth, suppuration, bleeding, and plaque scores at implant sites, poorer oral hygiene, and lower compliance with supportive periodontal therapy. Controlling systemic diseases is also critical. Studies have shown a positive correlation between type 2 diabetes and peri-implantitis. In the oral environment, hyperglycemia can trigger inflammation, oxidative stress, apoptosis, and decreased immune regulation ability [23]. Diabetic patients experience prolonged postoperative osseointegration and soft tissue healing periods, increasing the risk of complications such as wound infection and poor osseointegration.

Figure 2 summarizes ways of controlling periodontal pathogenic factors and improving repair effectiveness.

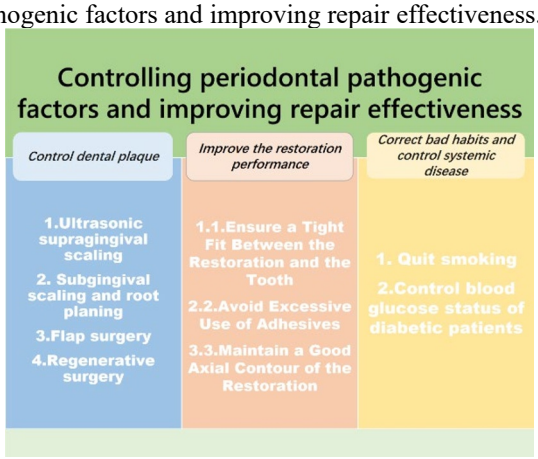

Figure 2. Methods of controlling periodontal pathogenic factors and improving repair effectiveness

5 Conclusion

In summary, periodontal factors are closely related to the effectiveness of restorations. Oral microbial factors, the host immune microenvironment, and anatomical factors determine the success of restorations by affecting the health of periodontal tissues. For example, the presence of the red complex and the expression of virulence factors can lead to gingival swelling, periodontitis, and peri-implantitis, destroying periodontal tissues and causing restoration failure. Additionally, during the restoration of implants, fixed prostheses, and removable dentures, disruption of biological width and excessive plaque accumulation can adversely affect periodontal health. Therefore, controlling dental plaque through ultrasonic or manual methods, improving the performance of restorations, managing systemic diseases, and correcting bad habits can reduce the incidence of periodontal diseases and improve restoration outcomes.

References

1. Mombelli A. Maintenance therapy for teeth and implants. Periodontol 2000. 2019 Feb;79(1):190-199. doi: 10.1111/prd.12255. PMID: 30892766.
2. Daubert DM, Weinstein BF. Biofilm as a risk factor in implant treatment. Periodontol 2000. 2019 Oct;81(1):29-40. doi: 10.1111/prd.12280. PMID: 31407437.
3. Mysak J, Podzimek S, Sommerova P, Lyuya-Mi Y, Bartova J, Janatova T, Prochazkova J, Duskova J. Porphyromonas gingivalis: major periodontopathic pathogen overview. J Immunol Res. 2014; 2014:476068. doi: 10.1155/2014/476068Epub 2014 Mar 25. PMID: 24741603; PMCID: PMC3984870.
4. Baseri M, Radmand F, Hamedi R, Yousefi M, Kafil HS. Immunological Aspects of Dental Implant Rejection. Biomed Res Int. 2020 Dec 9; 2020: 7279509. doi: 10.1155/2020/7279509. PMID: 33376734; PMCID: PMC7744195.
5. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Periodontol. 2018 Jun;89 Suppl 1: S267-S290. doi: 10.1002/JPER.16-0350. PMID: 29926957.
6. Abduo J, Lyons KM. Interdisciplinary interface between fixed prosthodontics and periodontics. Periodontol 2000. 2017 Jun;74(1):40-62. doi: 10.1111/prd.12189. PMID: 28429481.
7. GAI Yun, LIU Zhiming, ZENG Shen. Effect of gingival biotype on total crown repair and gingival crevicular fluid inflammatory factors. Shanghai Journal of Stomatology. doi: 10.19439/j.sjos.2023.06.019.
8. Ivanovski S, Lee R. Comparison of peri-implant and periodontal marginal soft tissues in health and disease. Periodontol 2000. 2018 Feb;76(1):116-130. doi: 10.1111/prd.12150. Epub 2017 Nov 30. PMID: 29193334.
9. Schwarz F, Ramanauskaite A. It is all about peri-implant tissue health. Periodontol 2000. 2022 Feb;88(1):9-12. doi: 10.1111/prd.12407. PMID: 35103327.
10. Thoma DS, Naenni N, Figuero E, Hämmerle CHF, Schwarz F, Jung RE, Sanz-Sánchez I. Effects of soft tissue augmentation procedures on peri-implant health or disease: A systematic review and meta-analysis. Clin Oral Implants Res. 2018 Mar;29 Suppl 15:32-49. doi: 10.1111/clr.13114. PMID: 29498129.
11. Laleman I, Lambert F. Implant connection and abutment selection as a predisposing and/or precipitating factor for peri-implant diseases: A review. Clin Implant Dent Relat Res. 2023 Aug;25(4):723-733. doi: 10.1111/cid.13185. Epub 2023 Feb 24. Erratum in: Clin Implant Dent Relat Res. 2023 Oct;25(5):984. doi: 10.1111/cid.13267. PMID: 36825512.
12. Srimaneepong V, Heboyan A, Zafar MS, Khurshid Z, Marya A, Fernandes GVO, Rokaya D. Fixed Prosthetic Restorations and Periodontal Health: A Narrative Review. J Funct Biomater. 2022 Feb 1;13(1):15. doi: 10.3390/jfb13010015. PMID: 35225978; PMCID: PMC8883934.
13. Analysis of Gingivitis after Repair of Fixed Prosthodontics/JIAO Xue-feng, DENG Xin-yu, WEN Yue- jin, et al.//Medical Innovation of China, 2014. 11(34):067-070
14. McReynolds DE, Moorthy A, Moneley JO, Jabra-Rizk MA, Sultan AS. Denture stomatitis-An interdisciplinary clinical review. J Prosthodont. 2023 Aug;32(7):560-570. doi: 10.1111/jopr.13687. Epub 2023 Apr 18. PMID: 36988151
15. Sterzenbach T, Helbig R, Hannig C, Hannig M. Bioadhesion in the oral cavity and approaches for biofilm management by surface modifications. Clin Oral Investig. 2020 Dec;24(12):4237-4260. doi: 10.1007/s00784-020-03646-1. Epub 2020 Oct 27. PMID: 33111157; PMCID: PMC7666681.
16. Zhang Y, Wang X, Li H, Ni C, Du Z, Yan F. Human oral microbiota and its modulation for oral health. Biomed Pharmacother. 2018 Mar; 99:883-893. doi: 10.1016/j.biopha.2018.01.146. Epub 2018 Feb 20. PMID: 29710488.
17. Digel I, Kern I, Geenen EM, Akimbekov N. Dental Plaque Removal by Ultrasonic Toothbrushes. Dent J (Basel). 2020 Mar 23;8(1):28. doi: 10.3390/dj8010028. PMID: 32210213; PMCID: PMC7175112.
18. Schwarz F, Jepsen S, Obreja K, Galarraga-Vinueza ME, Ramanauskaite A. Surgical therapy of peri-implantitis. Periodontol 2000. 2022 Feb;88(1):145-181. doi: 10.1111/prd.12417. PMID: 35103328.
19. de Avila ED, van Oirschot BA, van den Beucken JJJP. Biomaterial-based possibilities for managing peri-implantitis. J Periodontal Res. 2020 Apr;55(2):165-173. doi: 10.1111/jre.12707. Epub 2019 Oct 22. PMID: 31638267; PMCID: PMC7154698.
20. SUN Yuyan, MENG Weiyan. Research Progress on Predisposing Factors of Peri-implantitis[J]. Journal of Oral Science Research, 2024, 40(5): 389-392.

21. Chen L, Tong Z, Luo H, Qu Y, Gu X, Si M. Titanium particles in peri-implantitis: distribution, pathogenesis and prospects. Int J Oral Sci. 2023 Nov 23;15(1):49. doi: 10.1038/s41368-023-00256-x. PMID: 37996420; PMCID: PMC10667540.
22. Dreyer H, Grischke J, Tiede C, Eberhard J, Schweitzer A, Toikkanen SE, Glöckner S, Krause G, Stiesch M. Epidemiology and risk factors of peri-implantitis: A systematic review. J Periodontal Res. 2018 Oct;53(5):657-681. doi: 10.1111/jre.12562. Epub 2018 Jun 7. PMID: 29882313.
23. Liccardo D, Cannavo A, Spagnuolo G, Ferrara N, Cittadini A, Rengo C, Rengo G. Periodontal Disease: A Risk Factor for Diabetes and Cardiovascular Disease. Int J Mol Sci. 2019 Mar 20;20(6):1414. doi: 10.3390/ijms20061414. PMID: 30897827; PMCID: PMC6470716.