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PERI-IMPLANT DISEASE AND RISK FACTORS: A NARRATIVE REVIEW

ПЕРИИМПЛАНТНА БОЛЕСТ И РИЗИК ФАКТОРИ: НАРАТИВНА РЕВИЈА

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Abstract

Peri-implant disease is a consequence of the interaction of multiple factors, some of which are known, while others remain unknown. This systematic literature review discusses the various factors associated with peri-implant disease, highlighting their role in its etiopathogenesis. Peri-implant tissue is strongly influenced by various local and systemic factors. Local factors, such as poor oral hygiene, directly and immediately negatively affect peri-implant tissues, while various biomaterials, occlusal loading, and the presence or absence of keratinized mucosa also influence the condition of implants placed in the jaws. The effect of these factors depends on the individual's genotype, epigenetic factors, systemic health, nutrition, and many other contributing factors. Risk factors are numerous, but their impact is not yet fully defined. The advancement of medicine necessitates their continuous research, updating, and improved management strategies. Plaque accumulation and biofilm formation play a significant role in the development of peri-implant disease; however, the influence of other local factors should not be neglected and requires further clarification. Within the peri-implant microenvironment, systemic factors create favorable conditions for the harmful effects of local risk factors. Timely detection of risk factors and their elimination will reduce the risk of peri-implant diseases, and will ensure reliability for precise planning, successful implantation, and a long, stable survival period for the implants. **Keywords:** peri-implantitis, peri-mucositis, risk factors, implant.

Апстракт

Периимплантната болест е последица на заемно дејство на многу фактори, дел од кои се познати, а дел сè уште остануваат непознати.Во овој систематски литературен преглед се дискутира за различните фактори кои се поврзуваат со периимплантната болест, истакнувајќи ја нивната улога во нејзината етиопатогенеза. Периимплантното ткиво е под силно влијание на различни фактори како од локална така и од системска природа. Локалните фактори, како што е лошата орална хигиена,директно и непосредно негативно влијаатврз периимплантните ткива, но различните биоматеријали, оклузалното оптоварување, кератинизираната гингива,истотака,имаат влијание врз состојбата на имплантите поставени во вилиците. Каков ќе биде ефектот од овие влијанија зависи од генотипот на индивидуата од епигенетските фактори, системското здравје, исхраната и многу други дополнителни и помошни фактори. Факторите на ризик се многубројни, но нивното влијание сè уште не е дефинирано во целост. Напредокот на медицината условува нивно постојано истражување, дополнување и надрагдување на нивниот третман. Акумулацијата на плакот и формирањето на биофилмотимаат значајна улога воразвојот на периимплантната болест, но секако дека не треба да се занемари влијанието надругителокални фактори, па дури итреба попрецизно да се разјасни. Во услови на микроимплантна средина, системските фактори создаваат олеснителни услови за штетно влијание на локалните ризик-причинители. Навремена детекција на ризик-факторите и нивна елиминацијаќе го намали ризикот од појава на периимплантни заболувања, а ќе обезбеди сигурност за прецизно планирање, успешно имплантирање и за долг и стабилен период на опстанок на имплантите. **Клучни зборови:** периимплантитис, перимукозитис, ризик-фактори, имплант.

Introduction

Tooth loss is a condition that adversely affects the quality of life of an individual. The loss of teeth primarily impacts self-confidence, significantly impairs the masticatory process and the function of the temporomandibular joint, and may also be associated with systemic conditions, primarily by disrupting the function of the gastrointestinal system.

For completely edentulous patients, available therapeutic solutions include the placement of removable dentures, with or without implants, and fixed implant-supported prostheses[1,2].

However, fixed full-arch implant-supported prostheses attached to implants represent the gold standard for improv-

ing oral health and providing a solution for total edentulism. This procedure achieves the correction of impaired aesthetics and function.

Dental implants are a common choice for addressing partial or total edentulism. Although the use of dental implants is a good choice from an aesthetic and functional perspective, implantation can sometimes result in failure. Therefore, special attention should be paid to possible complications and anatomical limitations, which must always be considered when planning and performing the placement of dental implants[3] (Figure 1.).

Figure 1. *Implant placement - model (drawing) - (taken from a free online source)*[4]

Millions of dental implants are placed each year, but a large number of them develop biological complications such as peri-implant mucositis or peri-implantitis. Adverse effects are generally classified as early and late, where early ones occur before, and late ones occur after the functional loading of the implant[5].

Some of the causes are known, while others still need to be investigated.6 However, the onset and progression of peri-implant disease are the result of the interaction of many factors (known and unknown) in each individual, which can combine in different ways[7].

Peri-implant diseases are pathological inflammatory processes resulting from a disrupted microbiome that affects the integrity of soft and hard structures[8]. Peri-implant mucositis precedes peri-implantitis, in which the primary inflammatory process of the soft tissues is followed by destructive processes affecting the alveolar bone. Peri-implantitis can progress rapidly and negatively impact osseointegration[9]. Scientific evidence confirms that certain systemic factors, such as diabetes mellitus, cardiovascular diseases, obesity, and alcohol consumption, among others, play an important role in modulating bone physiology and wound healing[10].

Therefore, these conditions may be significant risk indicators or factors for the prevalence and incidence of peri-implant diseases[11]. Local factors should by no means be overlooked as possible etiological factors. These include the material from which implants are made, typically titanium[12], oral hygiene[13], and the influence of the peri-implant microbiome on the implant surface[14].

Based on these data, the aim of this paper was to identify the risk factors and explain their influence on the occurrence of peri-implant diseases.

Method

This systematic literature review discusses the various factors associated with peri-implant disease, highlighting their role in its etiopathogenesis. The relevant articles cited in this study were retrieved from the MEDLINE/PubMed database, published in English, with no time frame restrictions.

This article discusses various factors associated with peri-implant disease, highlighting their role by presenting their respective advantages and disadvantages. The search strategy for relevant articles included studies retrieved from the MEDLINE/PubMed database, published in English, with no time restrictions. This narrative review encompasses and cites original research, narrative reviews, and systematic literature reviews. The keywords used to conduct the research were “peri-implantitis” and “risk factors.” A total of 484 works, including abstracts and full-text articles, were identified in the PubMed database using these keywords.

The first selection, i.e., potentially suitable articles, was made based on the criterion of being fully published in the database (full text), with a title corresponding to the stated objective, which included a total of 151 articles and excluded 333 abstracts. After the initial selection, articles presenting case reports, short communications, or articles with predominantly preventive content were excluded from this selection. According to this criterion, 84 studies were discarded. All articles that met these specified criteria were characterized as potentially relevant studies and suitable for further analysis.

Full texts were retrieved without contacting or obtaining approval from the study authors.

For greater precision in the selection, inclusion and exclusion criteria were established to guide the process.

Inclusion criteria:

- Study design: original research, narrative reviews, systematic reviews
- Population: patients with peri-implantitis and peri-mucositis
- Factors: systemic and local risk factors
- Language: English

Exclusion criteria:

- Study design: case reports, short communications, and brief reports
- Abstracts
- Articles in languages other than English

Within the limitations of this narrative review, the findings are based on the literature available according to the criteria and are discussed narratively without conducting a systematic analysis.

Etiology and Risk Factors in Peri-Implant Diseases

Peri-implant tissue is strongly influenced by various factors of both local and systemic nature. Local factors, primarily poor oral hygiene, directly and immediately negatively affect peri-implant tissues, while genetic and epigenetic factors, systemic health, and nutrition influence the health of each individual and their susceptibility to various diseases, including peri-implant diseases.

Systemic Factors

The factors influencing the success of implant therapy are numerous and heterogeneous. The most common systemic diseases considered to affect the survival of implants in the jaws are diabetes mellitus, cardiovascular diseases, Crohn's disease, hypothyroidism, osteoporosis, chemotherapy, and radiotherapy in the treatment of head and neck malignancies. All of these listed risk factors belong to the group of factors representing a relative contraindication for implantation. Although many of them have been studied for a long time, there is still no consensus or definitive guidelines on this issue. For clinicians whose field of practice is implantology, the rule of an individual approach to each patient applies. Specifically, the therapist is obligated to adapt the decision for implant placement, as well as the treatment plan, according to the assessment made regarding the risk factor. It is considered that in most situations, the degree of control of the systemic disease is more important than the disease itself.

However, in implantology, there are also absolute contraindications for implant therapy. These include intravenous bisphosphonate therapy, myocardial infarction, cerebrovascular insult (stroke), placement of artificial heart valves or transplants, active malignancy treatment, acute high-grade malignancy condition, risk of hemorrhage, immunosuppression, psychiatric conditions, and substance abuse.

Diabetes mellitus

Diabetes is typically preceded by a prediabetic condition, which is defined as moderate hyperglycemia that does not reach the threshold for a diabetic state[15]. However, diabetes and prediabetes are very common metabolic disorders that cause hyperglycemia, leading to microangiopathies and macroangiopathies[16]. It has been confirmed that they are associated with periodontal disease[17]. delayed wound healing[18], and disorders of bone metabolism[19] Diabetes mellitus, as well as prediabetic conditions, represent a common and growing health problem with extensive harmful effects on the entire organism. In the oral cavity of patients with diabetes, periodontal disease is more advanced, and the destructive processes are more pronounced[20].

Under such circumstances, osteoblast activity is reduced, while osteoclasts progressively multiply and enhance resorption processes. Collagen production is also decreased[21].

Unlike uncontrolled diabetes, in moderately controlled diabetes, there is an insignificant difference in implant failure rates. The duration of diabetes can influence the failure of implant therapy. This status is explained by the likely existence of microvascular complications that affect the wound healing process. The literature confirms that the success rate of implant therapy in patients ranges from 85.5% to 100%[22,23].

Diabetes is often a disease present in the population over 65 years of age[24]. where the need for implants is also most common[24]. For a long time, it was considered that this disease is a relative contraindication[25,26]. However, the general consensus in professional circles is that implant placement is permissible in patients with well-regulated blood glucose levels. If glycemia is not regulated, implantation should be avoided[27].

It has been proven that implant integration can be achieved independently of glucose levels, but an important benchmark to strive for is an HbA1c value below 8%. Clinical experiences confirm that an additional factor influencing implant failure is the combination of two risk factors: diabetes and bone loss, with approximately 1 mm more bone loss observed in diabetic patients[26].

The greatest risk of implant loss in diabetics exists within the first 5 years after implantation; failure in the subsequent five-year period is less common.

Cardiovascular Diseases

It is known that cardiovascular diseases (CVD) have an impact on the development of peri-implantitis. They compromise blood flow, reducing the supply of oxygen and nutrients to the tissues. These processes negatively affect the healing process[26], osseointegration, and alveolar bone resorption processes[28].

The resulting tissue hypoxia leads to reduced fibroblast activity, decreased collagen synthesis, impaired

macrophage function, and increased capillary pressure, creating conditions conducive to the development of infection.

Some studies[29,30] have shown that patients with CVD and previously diagnosed periodontal disease have a significantly increased risk of mortality, while the association of CVD with peri-implantitis has been confirmed by a correlation between CVD risk markers. Specifically, high levels of triglycerides and uric acid are not only biochemical markers of these diseases but have also been proven to be risk parameters for peri-implantitis[31]. Further investigation of the correlation between CVD and peri-implant inflammation could encourage clinicians to take relevant measures before the intervention to improve implant success rates and alleviate patient discomfort. Moreover, this correlation could further support the link between local inflammation and systemic diseases and promote research in this direction.

A definitive recommendation is that implant therapy in patients with cardiovascular diseases should be carefully assessed and monitored, because evidence suggests that cardiovascular diseases can affect the level of marginal bone around implants, and an unfavorable status in this regard is a possible risk factor for peri-implant diseases.

Crohn's Disease

Crohn's disease is an autoimmune disorder of an idiopathic nature, characterized by granulomatous changes affecting the mucosa of the large intestine. The changes, which manifest in the form of erosions or ulcerations, are present on the intestinal wall and are the cause of gastrointestinal discomfort[32].

In the oral cavity, this disease manifests with lesions similar to those in the lower digestive tract, resembling recurrent aphthous ulcers (smaller and more numerous). The mucosa is cobblestoned and folded, while the gingiva is of a hyperplastic type.

The stance of clinicians regarding diagnosed Morbus Crohn is to perform only urgent interventions during the active phase of the disease. Since treatment is carried out using immunosuppressants or corticosteroids, healing time is impaired, and the predisposition to infection is increased[33]. Based on its course and mechanism of action, it is believed that Crohn's disease has a negative impact on successful implant osseointegration[34].

Thyroid Gland Disorders

Disorders of thyroid gland function affect bone metabolism. Thyroxine (T4) and, to a lesser extent, triiodothyronine (T3) regulate the healing process. In hypothyroidism, the maturation and activity of bone cells are reduced, therefore bone formation is diminished, which can be a reason for implant failure.

A retrospective study was conducted on 27 patients with hypothyroidism. The results obtained were compared with 29 patients as a control group. The overall success rate after more than 1 year was 95% and 97%, respectively[35] These findings are consistent with results obtained in other studies[35], leading to the main conclusion that well-controlled hypothyroidism does not affect the final success rate of implants.

Osteoporosis

Osteoporosis is a systemic disease manifested by bone degradation and reduced bone density. Bone loss in men and women is 0.5% annually. Postmenopausal women have an increased risk of developing the disease, as a negative calcium balance is registered, which is twice as low during menopause. Most clinicians agree that osteoporosis is not a contraindication for implant placement, unless the implant candidates are on parenteral bisphosphonate therapy.

There is a consensus that dental implant placement is safe for patients who have been receiving oral bisphosphonate therapy for less than three years without other systemic diseases. However, if the therapy is shorter than three years but the patient is also on corticosteroids, or the oral bisphosphonate therapy is longer than three years, the therapy should be discontinued three months prior to the procedure. Therapy can be resumed three months after implant placement, i.e., once bone healing has been achieved[36].

In a study by Cho[37] involving postmenopausal women with osteoporosis, a cumulative success rate of 100% was registered. Resonance frequency analysis at 12 months showed a mean implant stability test value of 71.4±5.52, indicating excellent osseointegration. The average bone loss around the implant was 0.54±0.35mm. During the study, no implant defects were registered, and satisfactory results were recorded for bleeding on probing and peri-implant pocket depth. Changes in bone mineral density (BMD) and bone turnover markers (BTMs) were minimal.

Radiotherapy and Chemotherapy

In the treatment of oncological cases, radiotherapy, as well as chemotherapy, besides destroying malignant cells, non-selectively damages healthy cellular structures as well. All of this therapy results in numerous oral complications, among which osteoradionecrosis is worth mentioning. It occurs in 8.2% of treated patients[32]. If surgical intervention is necessary, maximum selectivity of the procedure is required, with a recommendation to avoid interventions. If they need to be performed for justified or necessary reasons, they should be carried out atraumatically and under antibiotic protection[38].

Studies on humans and animals show a 12 times greater risk of implant failure in patients undergoing oncology therapy.

Radiotherapy destroys malignant tumor masses through ionizing radiation. These processes release free radicals that damage cellular DNA, disrupt replication, and cause the death of mitotic cells. Radiotherapy primarily destroys cells that are integral parts of tissues with high mitotic activity. Bone structures are most sensitive; under the influence of radiation therapy, osteoblasts lose their function, thereby disrupting bone remodeling. In contrast to strong osteoblastic sensitivity, osteoclasts survive more safely, intensify their activity, and lead to resorptive, destructive, and osteolytic processes[32].

The outcome of radiotherapy is strongly influenced by the interval (time) between radiation treatment and implant placement, as well as the total radiation dose administered. All patients who received a high radiation dose (65 Gy or more) have a low rate of implant rehabilitation success. Furthermore, osseointegration is exceptionally poor in cases of reduced healing capacity of the implant bed[39].

On one hand, most implantologists agree that implant placement in this category of patients is best performed 12-18 months after the completion of therapy. On the other hand, it is true that as the time since radiation increases, due to progressive endarteritis processes, hypovascularization occurs, making the bone increasingly less suitable for dental implant placement.

There is limited data on the effect of chemotherapy on implant success. Retrospective studies have shown a negative impact of chemotherapy on implant success, but a higher survival rate was registered if chemotherapy was administered immediately before or one month after implant placement[40].

The length of the implant placed is also responsible for unsuccessful implant therapy. Thus, if the implant is short, the incidence of failure is more frequent, especially in irradiated bone. Therefore, it is advisable to use implants with greater length, a rougher surface, and a screw-shaped implant design.

Genetic Predisposition

Starting from the fact that not all people with the same risk factors develop peri-implant disease, it is believed that genetic predisposition may explain the development of this condition[41]. Biomarkers in peri-implantology of a biochemical and genetic nature are sophisticated tools capable of precisely identifying factors involved in the pathology of peri-implant disease, having the ability to compensate for the limitations of other routine diagnostic methods. Genetic biomarkers, which are constant, are suitable for risk assessment and can be measured before the onset of the disease; on the other hand, biochemical markers are the biomarkers of choice for monitoring the onset, intensity, and activity of the disease[42]. The most widespread genetic polymorphisms are single nucleotide polymorphisms (SNPs), which are variations in the DNA sequence affecting more than 1% of the population.

SNPs that have been the subject of multiple studies in the context of peri-implantitis are related to inflammatory interleukin genes and proteins involved in bone metabolism. These genes, when mutated, can cause an abnormal inflammatory response and/or reduced peri-implant osseointegration, resulting in the development of this disease. In patients who are at higher genetic risk for peri-implantitis, these inflammatory molecules may serve as potential biomarkers[43].

An association with implant loss, present peri-implantitis, or peri-implant marginal bone loss has been demonstrated for certain genetic polymorphisms. No association has been proven for IL-2, IL-6, TNF-α, or TGF-β1 genetic polymorphisms, while an association has been registered for IL-1A and IL-1B genetic polymorphisms. An association exists in the Iranian population with OPG, RANKL, IL-17, but there is no clear association regarding biological complications.

Literature based on clinically followed cases emphasizes that functional polymorphisms of IL-1α (C-889T), IL-1β (C+3954T, C-511T), and the composite IL-1 genotype can be used as predictive markers for peri-implant disease, while the TNFα G-308A polymorphism is not associated with peri-implant disease[43], table 1.

Table 1 shows the systemic and local risk factors in patients with peri-implantitis and peri-mucositis cited in this article.

Local Factors

Oral hygiene

Oral hygiene has a strong influence on both the periodontal tissue of a healthy tooth (periodontium) and the surrounding peri-implant tissue. It has been proven that increased levels of dental plaque promote inflammatory and destructive processes around the implant[44].

In lesions with peri-implantitis and periodontopathy, gram-negative anaerobic bacteria have been identified compared to healthy periodontal and peri-implant tissues. However, greater microbial diversity has been found in peri-implant tissues[45]. Furthermore, in peri-implantitis, there is predominantly an infiltrate rich in inflammatory cells, B-lymphocytes, and plasma cells. Unlike the periodontal tissues of a healthy tooth, the implant lacks cementum and the periodontium (the protective and func-

Table 1. Systemic and local risk factors in patients with peri-implant mucositis and peri-implantitis

	Peri-implant lesions	Authors
Systemic risk factors		
Diabetes mellitus	✓	Oates TW[23]
Cardiovascular disorders	✓	Ustaoğlu G[30]
Crohn's disease	✓	Alsaadi G[33]
Bisphosphonate therapy	✓	Rotim Ž[35]
Osteoporosis	✓	Cho JM[37]
Radiotherapy	✓	Cekić-Arambašin[31]
Chemotherapy	✓	Gabrić Pandurić D[39]
Genetic predisposition	✓	Chang Z[42]
Local factors		
Oral Hygiene	✓	Matsuda S[44]
Dysbiosis	✓	Cui Z[46], Roccuzzo A[47], Chen T[48], Gasimi B[49], Goncalves LF[50], Belibasakis GN[51], Sumida S[52], Lindhe J[53]
Periodontosis	✓	Matsuda S[44], Cui Z[46], Chen T[48], Gasimi B[49]
Smoking	✓	Maló PS[57],Reis INRD[58]
Keratinized mucosa	✓	Shaya F[59]
Occlusal loading		Vigolo P[60], Rossi F[61] Blanes RJ[62], Lindquist LW63, Heitz-Mayfield LJ[64]
Titanium (Ti)		Franchi M[67], Schwarz F[68], Berglundh T[69]

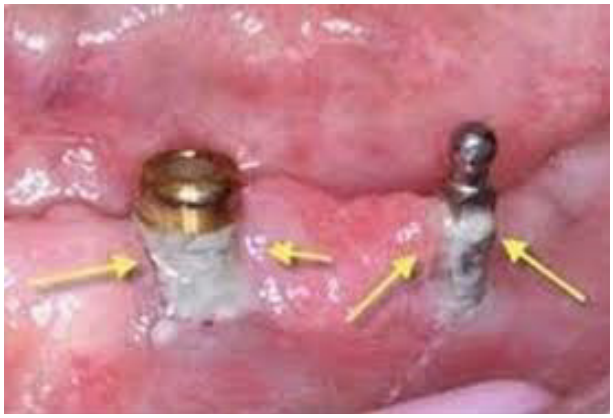

Figure 2. *Plaque accumulation on the implant surfaces (Taken from Roccuzzo et al.47)*

tional tissue layer) overlying the bone, which makes the peri-implant tissue less resistant to the action of dental plaque. This characteristic histological and morphological structural composition represents an easily surmountable barrier to the altered oral microbiome, i.e., dental plaque. There are arguments confirming that, histologically, peri-implant lesions are twice as large and have more blood vessels and inflammatory infiltrate in the connective tissue compared to periodontal tissues[46] (Figure 2.).

Given all these specific characteristics around the implant, it is quite logical that poor oral hygiene has a detrimental effect on implant survival. If plaque deposits are not removed through maintaining oral hygiene, they easily cause gingival inflammation, rapidly infiltrate the deeper structures, i.e., the bone, and cause destruction and

resorption, which affects implant stability, with the possibility of complete loss.

Dysbiosis

When the microbial homeostasis in the periodontium is disrupted for any reason, pathogens within the biofilm trigger a series of immune reactions in the host organism that can cause periodontopathy in natural teeth. This disruption of microbial homeostasis is also characteristic of peri-implant disease. Elucidating the complex role of the microbial community, as well as the immune mechanisms in oral health and disease, is crucial for the development of improved preventive, diagnostic, and therapeutic strategies[46].

Although many mechanisms are known, many questions remain about how changes in bacterial populations contribute to the development and progression of these conditions.

Microbiological similarity between periodontal and peri-implant tissues has been demonstrated; however, differences have been registered, with a richer and more diverse microbiological finding in tissues diagnosed with peri-implantitis and peri-implant mucositis[47].

In the oral cavity, a microbiological equilibrium exists, where microorganisms are in a symbiotic relationship with the host, maintaining optimal oral health. When the physiological oral microecological balance is disrupted, pathogenic microbes proliferate freely, causing inflammation and tissue damage[48,49]. In periodontal pockets, bacteria such as Treponema, Porphyromonas gingivalis, Fusobacterium, and Prevotella are numerous, and the count of bacteria such as Fusobacterium and Selenomonas increases with the deepening of periodontal pockets[50] (Figure 3.).

In the same individual, identical genotypes of P. gingivalis have been demonstrated in both periodontopathic and peri-implantitis sites. The possibility of bacterial transmission is supported by research that identified the presence of P. gingivalis, T. forsythia, Aggregatibacter actinomycetemcomitans, and Prevotella intermedia in saliva and on the tongue[52,53].

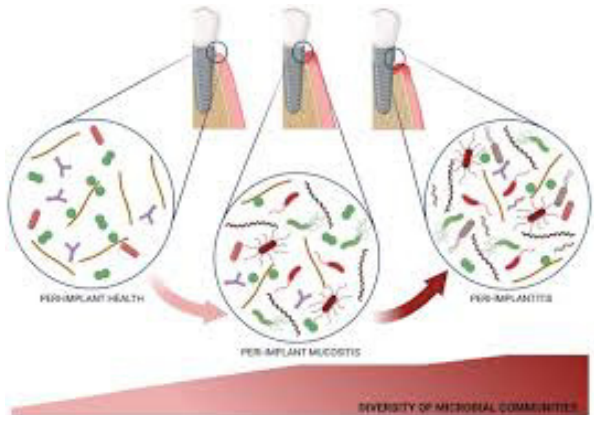

Figure 3. *Differences in the bacterial microbiome around peri-implant tissue: healthy, peri-implant mucositis, peri-implantitis (taken from Belibasakis et al.[51])*

Smoking

Smoking is one of the significant risk factors, belonging partially to the group of systemic factors due to its harmful effects on all organs and tissues, but also partially as a local factor due to its direct effect on all tissues and organs in the oral cavity. Numerous studies confirm that smoking is associated with the onset and progression of periodontal tissue inflammation[54-58]. Cigarette smoking impairs neutrophil and phagocyte chemotaxis, disrupts humoral immunity, alters serum immunoglobulin levels (IgG and IgE), increases the production of reactive oxygen species (ROS), and promotes the release of proteolytic enzymes, collagenases, and elastases, which directly contribute to the destruction of periodontal tissues.54 Research has shown that smoking-induced subgingival dysbiosis occurs before the clinical symptoms of periodontal disease[55], meaning these preclinical findings are precursors to periodontitis.

Constituents in tobacco, such as nicotine and benzo[a]pyrene, exhibit direct antimicrobial effects upon contact with oral microbial communities, leading to non-specific bactericidal effects that reduce the physiological microbiome and immune cell populations, creating opportunities for pathogenic bacteria to proliferate[56].

Knowing the biological proximity of these two tissues, and referring to the easy bacterial transmission, the impact of smoking on peri-implant tissues is justified.

Nicotine causes vasoconstriction and impaired epithelialization of lesions, which is why it has long been considered a potential risk factor for implant longevity. Nicotine affects osseointegration, increases soft tissue inflammation, and influences bone loss around the implant. Its impact on marginal bone loss around implants is well documented[57,58]. It has been proven that heavy smokers have twice the risk of implant failure compared to non-smokers or moderate smokers.

Keratinized Mucosa

The lack of keratinized mucosa may be associated with poor soft tissue condition, presence of peri-implant mucositis and peri-implantitis. It is believed that an adequate width of keratinized mucosa is related to plaque accumulation, inflammation, recession, and attachment loss, but bone loss has not been confirmed.

It has been demonstrated that a wider band of keratinized gingiva may be beneficial for patients in the treat-

ment of peri-implantitis compared to situations with a reduced width of keratinized mucosa. With wider keratinized gingiva, reduced probing depth, absence of inflammation, and improved bone stability have been registered. Combined correction of hard and soft tissue defects around lesions caused by peri-implantitis may facilitate treatment success and help maintain peri-implant stability[59].

However, it is considered that the ultimate outcome for implant survival depends on the need to meet certain criteria concerning the keratinized mucosa for the implant to be successfully placed and to achieve stable osseointegration without risk.

Occlusal loading

Under physiological circumstances, no association has been found between occlusal loading, malpositioned implants, as causes of bone resorption around the implant[60,61]. However, some experiences suggest that poorly fabricated superstructures and improperly positioned implants may cause peri-implant tissue resorption[62]. But if the tissue is affected by peri-implantitis and occlusal loading exists, bone resorption is considered very likely[63].

On the other hand, implants with very rough surfaces, i.e., surfaces coated with sprayed titanium plasma or hydroxyapatite, carry a certain risk. Manipulation during implant placement has a serious effect on bone resorption, especially if the implant is placed deeply into the bone and cemented with cement whose excess cannot be removed. Cement remnants can cause infection around the implant[64]. However, this risk is avoided by screw-retaining the abutment to the intraosseous part of the implant, a common practice in modern implantology.

Titanium as a Risk Factor

As an implant material, titanium began to be used just under 100 years ago in America, and the first titanium-based implant-supported prostheses were reported in 197765. Due to their long lifespan, corrosion resistance, good biocompatibility, and low Young's modulus, titanium and its alloys have been widely used for dental implants[66].

Currently, titanium implants are the most widely used commercial dental implants. However, factors such as friction between the implant and bone surface, wear caused by biomechanical loading, and the biological effect of fretting corrosion inevitably lead to the release of titanium particles into the surrounding tissues[67].

There is evidence of an association between titanium particles and peri-implant disease, but its role as a risk indicator for peri-implantitis remains to be further clarified and determined[68,69]. The role of titanium particles in the progression of peri-implantitis may be explained by delving deeper into the analysis of risk factors affecting the release of titanium particles around the implant and the dispersion of these particles, as well as their potential role in the initiation and progression of peri-implant disease.

The findings from which the conclusions in this article were drawn, relating to the influence of risk factors on peri-implant disease, are based on the analysis of the results obtained from the reviewed literature.

Conclusion

Risk factors are numerous, but their exact impact has not yet been definitively proven. They are still being studied, refined, and further elaborated. The development of technological and research methodologies will undoubtedly lead to new insights regarding these factors and new approaches to therapy and prevention.

Plaque accumulation and biofilm formation have been demonstrated to play a significant role in the onset and progression of peri-implant disease, while the role of prosthetic factors such as occlusal loading is a very likely cause requiring further investigation. All other systemic factors create a favorable environment for the easier expansion of local risk factors.

Undoubtedly, risk reduction is of great importance for precise planning, successful implantation, and long-term stability of implant survival in the jaws.

Reference

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