Review Article

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Dentistry Section

Bleeding on Probing around Dental Implants: A Narrative Review

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ABSTRACT

Peri-implant bleeding is an important early sign of inflammation around dental implants and serves as a diagnostic indicator of peri-implant mucositis and peri-implantitis, both of which can lead to implant failure if, not treated promptly. The present review aims to provide insights into Bleeding on Probing (BOP) around dental implants. Data collection for this review involved gathering and analysing relevant literature to synthesise findings, identify patterns, and evaluate key results across studies. Histological studies reveal that peri-implant bleeding is associated with the infiltration of inflammatory cells such as neutrophils, macrophages, and lymphocytes, which is comparable to periodontal disease. This inflammation disrupts the peri-implant epithelium, increases vascularisation, and destroys connective tissue, all of which indicate early tissue damage around the implant. Inflammation also affects Peri-implant Crevicular Fluid (PICF), leading to increased levels of pro-inflammatory cytokines such as Interleukin (IL) 1β and Tumour Necrosis Factor (TNF)-α, as well as Matrix Metalloproteinases (MMPs) that contribute to tissue destruction. These molecular alterations make PICF an effective diagnostic tool for determining the severity of peri-implant disease. Clinically, BOP around implants is a commonly used metric to assess peri-implant health, although its prognostic value varies due to structural differences between peri-implant tissues and those surrounding natural teeth. However, significant or prolonged bleeding indicates poor healing, infection, or inadequate tissue management, all of which can impede osseointegration-the process by which the implant integrates with the bone. While peri-implant bleeding is a natural component of the healing process under controlled conditions, persistent bleeding may signal complications. Therefore, peri-implant bleeding should be monitored regularly to facilitate early detection and management, thereby preventing peri-implant disorders and ensuring the long-term success of dental implants.

Keywords: Osseointegration, Peri-implant crevicular fluid, Periodontal disease

INTRODUCTION

Dental implants have become a widely accepted solution for replacing missing teeth due to their durability and functionality. BOP is a well-established diagnostic indicator in clinical periodontology, first demonstrated by Mühlemann HR and Son S in 1971 [1], Lenox J and Kopczyk R in 1973 [2], Greenstein G et al., in 1981 [3], and Weinberg and Hassan in 2012 [4]. BOP has also been shown to have a strong negative predictive value for the development of future disease [5]. Patients under maintenance treatment with a full-mouth BOP score<20% were reported to have a lower risk of progressive attachment loss [6]. Therefore, BOP is one of the parameters incorporated into various periodontal risk assessment techniques.

REVIEW OF LITERATURE

Peri-implant diseases are categorised as peri-implant mucositis or peri-implantitis. Peri-implant mucositis is defined as soft-tissue inflammation around a functional dental implant with BOP, whereas peri-implantitis is differentiated by the loss of supporting marginal bone in addition to normal bone remodeling [7]. Previous research has found that periodontal diseases, including peri-implantitis, contribute significantly to implant failure, which is often measured by the plaque index and bleeding on soft-tissue probing around implants. Consequently, the long-term success of dental implants depends on the health of the surrounding periodontium. Maintaining peri-implant tissue health is therefore crucial for implant longevity. If, peri-implant diseases are not detected and managed appropriately, they may lead to implant loss [8].

The BOP around dental implants is a crucial parameter for diagnosing inflammation in the peri-implant mucosa. It is recognised as a clinical measure to distinguish between peri-implant health and disease [9], consistently serving as a diagnostic indicator for both peri-implant mucositis and peri-implantitis. Differences in the bacterial composition associated with BOP around dental implants compared to natural teeth suggest that peri-implant BOP has prognostic value, with its presence or absence correlating with the deterioration or stability of peri-implant conditions over time. In a cohort of patients adhering to a strict maintenance program, a significant proportion of implants exhibiting BOP at 50% or more of scheduled periodontal therapy visits showed deterioration of peri-implant tissues beyond predetermined clinical and radiographic thresholds [10].

Koldsland OC et al., utilised probing depths of ≥4 mm and ≥6 mm to differentiate between varying severities of peri-implantitis [11]. According to Ata-Ali J et al., peri-implant probing is crucial for diagnosing peri-implant disease [12]. Conversely, Misch CE et al., argue that probing around implants may have limited diagnostic value and do not recommend routine probing [13]. However, they suggest using probing as a diagnostic parameter for compromised implants, roughly corresponding to moderate peri-implantitis [14].

While BOP is a well-established indicator of periodontal inflammation in natural teeth, its interpretation around implants remains less clear due to differences in peri-implant tissue anatomy, probing force sensitivity, and variability in clinical protocols. The literature lacks a focused, systematic synthesis evaluating whether BOP alone can serve as a reliable marker for peri-implant mucositis or early peri-implantitis, or if, it should always be interpreted alongside other parameters such as probing depth, bone loss, and suppuration. Moreover, emerging studies suggest that BOP's predictive value for future peri-implantitis is inconsistent, with both false positives and negatives reported.

The present review addresses this gap by critically appraising current evidence, highlighting diagnostic thresholds, and clarifying the influence of patient-related and implant-related factors. Therefore, it can guide clinicians in interpreting BOP findings more accurately, promote standardised protocols, and identify directions for future

longitudinal studies to enhance early diagnosis and prevention strategies for peri-implant diseases.

Data Collection

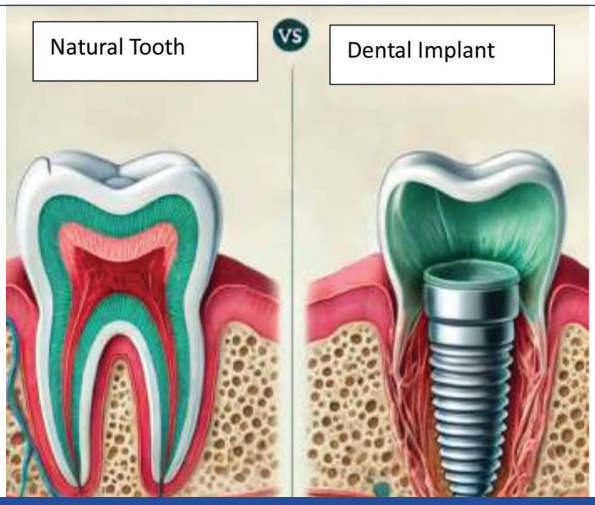
Data collection for present review involved gathering and analysing relevant literature to synthesise insights, identify patterns, and evaluate key findings across studies.

DISCUSSION

Clinical and Histological Differences in Tooth and Implant

There are clinical and histological differences between teeth and implants that influence the response in BOP. These include differences in the orientation of collagen fibers and vascularisation. In natural teeth, collagen fibers run perpendicularly and insert into the cementum, providing structural stability and resistance to probing [15]. In contrast, dental implants have collagen fibers that run parallel to the implant surface, offering reduced resistance to probing pressures and potentially leading to easier bleeding during probing.

Differences in vascularisation also exist due to the presence of the Periodontal Ligament (PDL) in natural teeth, which promotes a dense vascular network supplying nutrients and immune cells to the periodontium. In dental implants, the absence of the PDL results in diminished vascularisation of the peri-implant tissues, which may impair the tissue's capacity to respond to bacterial insults and repair after injury [Table/Fig-1] [16].



[Table/Fig-1]: Represents the difference between natural teeth and peri-implant structures. {Artifical Intelligence (AI) generated}.

Peri-implant Bleeding as a Sign of Inflammation

Peri-implant bleeding is one of the earliest and most noticeable signs of inflammation surrounding dental implants and serves as a key clinical predictor of potential peri-implant diseases such as mucositis or peri-implantitis. This bleeding typically occurs when the peri-implant tissues are gently probed and results from an inflammatory response triggered by bacterial plaque accumulation. Unlike natural teeth, dental implants lack a periodontal ligament (PDL) and rely solely on the peri-implant mucosa for soft-tissue attachment. When this mucosa becomes inflamed, increased blood flow, capillary dilation, and alterations in vascular anatomy occur, rendering the tissues more prone to bleeding [17].

This inflammation-induced bleeding is comparable to that observed in gingivitis around natural teeth, where hyperaemic tissues bleed easily under minimal pressure. Histologically, gingivitis-related bleeding involves superficial inflammation of the gingiva, characterised by infiltration of inflammatory cells such as neutrophils and lymphocytes into the connective tissue without affecting the underlying bone. In contrast, peri-implantitis-associated bleeding involves deeper inflammation extending from the soft tissue to the bone surrounding the implant, resulting in bone resorption and more extensive tissue damage [18].

Early detection of peri-implant bleeding enables timely intervention before more severe signs, such as pocket formation, suppuration, or implant loss, appear. Prompt management of this early inflammation including improved oral hygiene, regular professional cleanings, and, if, necessary, the use of antiseptic or antimicrobial agents-is crucial to preventing progression to peri-implantitis, a condition that can compromise bone integrity and implant stability [19]. Differences between the characteristics of gingivitis and peri-implantitis are summarised in [Table/Fig-2] [20].

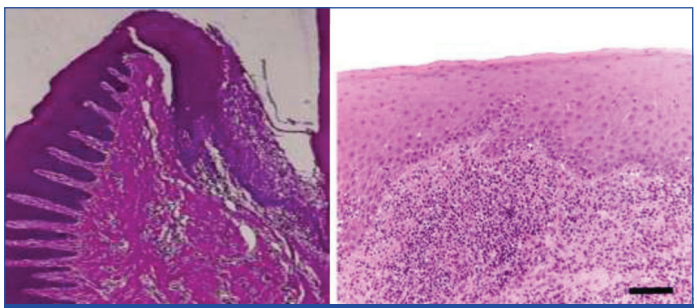
Parameters	Gingivitis	Peri-implantitis
Clinical features	Red, swollen gums Bleeding upon probing or brushing Presence of plaque and calculus No attachment loss Often asymptomatic	Inflammation of the soft-tissues around the implant. Bleeding on probing. Increased pocket depth around the implant. Bone loss around the implant. Possible pain or discomfort.
Histological features	Increased inflammatory cell infiltrate (mainly neutrophils and lymphocytes) Thickened epithelium (hyperplasia) Altered collagen structure Dilated blood vessels	Inflammatory infiltrate (neutrophils, plasma cells). Loss of connective tissue attachment. Resorption of the bone around the implant. Presence of microbial biofilm on the implant surface.
Bacteria associated	*Streptococcus sanguinis* *Porphyromonas gingivalis* *Treponema denticola* *Fusobacterium nucleatum*	*A. actinomycetemcomitans* *P. intermedia* *T. forsythia*

[Table/Fig-2]: Differences between characteristics of gingivitis and peri-implantitis.

The Histology of Peri-implant Bleeding

Histological analysis provides insight into the inflammatory processes occurring within peri-implant tissues and sheds light on the early stages of peri-implant diseases such as mucositis and peri-implantitis. Inflamed peri-implant mucosa exhibits several key features, including increased infiltration of inflammatory cells, predominantly neutrophils, lymphocytes, and plasma cells. These cells infiltrate the connective tissue, particularly around vascular structures, causing dilation and increased permeability of blood vessels. This vascular alteration is characteristic of inflammation and contributes to the hyperaemia and edema observed in affected tissues.

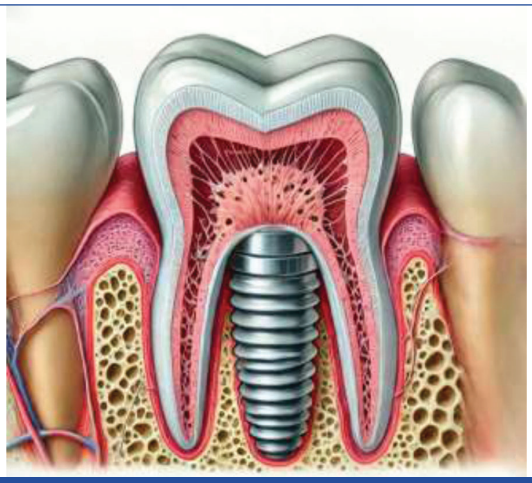
The epithelial and connective tissue barriers differ around implants compared to natural teeth. The junctional epithelium around implants is weaker and longer, forming a less protective seal. This structural difference increases the susceptibility of the peri-implant region to bacterial infiltration. Additionally, the absence of a PDL weakens the connective tissue barrier, reducing vascularity and resistance to mechanical stress, thereby increasing the likelihood of peri-implant inflammation [Table/Fig-3] [21].



[Table/Fig-3]: Histological pictures of gingivitis and peri-implantiti [21].

Because of these differences, the epithelial barrier around implants may exhibit signs of breakdown or ulceration, allowing greater bacterial invasion and perpetuating the inflammatory response.

Additionally, the connective tissue may show collagen fiber degradation, indicating a compromise in the tissue's structural integrity and further exacerbating inflammation. In peri-implantitis, this inflammatory reaction penetrates deeper into the tissues, damaging the bone, stimulating osteoclastic activity, promoting bone resorption, and leading to the formation of granulation tissue within the peri-implant pocket. The peri-implant epithelium may elongate and migrate apically, reflecting the body's attempt to re-establish a barrier against bacterial infection. Bacterial colonies can also be observed in the peri-implant sulcus, indicating ongoing bacterial challenges and biofilm development [22-25]. These histological alterations reflect the tissue's response to bacterial invasion and inflammation, which, if, left untreated, can result in progressive tissue deterioration and implant failure [Table/Fig-4] [26].



[Table/Fig-4]: Diagrammatic illustration of epithelial and connective tissue attachment around dental implant. (AI Generated).

Peri-Implant Bleeding and its Association with Crevicular Fluid, Peri-implant Crevicular Fluid (PICF)

The relationship between peri-implant bleeding and crevicular fluid, particularly PICF, is significant. PICF is an inflammatory exudate that forms in the peri-implant sulcus or pocket, analogous to gingival crevicular fluid in natural teeth. PICF contains various biomarkers, including enzymes, cytokines, and prostaglandins, which reflect the inflammatory state of the peri-implant tissues [27].

Research indicates that elevated levels of biomarkers in PICF-including IL-1β, TNF-α and Matrix Metalloproteins (MMPs)—are associated with peri-implant bleeding and inflammation. These biomarkers play a role in the host immune response and tissue degradation, contributing to the progression of peri-implant diseases. The presence of blood during probing reflects an inflammatory response that promotes PICF accumulation, which can be used to gauge the severity of inflammation. Increased PICF levels are often correlated with higher BOP scores, demonstrating the close relationship between peri-implant bleeding and crevicular fluid production [28].

Bacteria Associated with Peri-implant Beeding

Peri-implant bleeding is largely caused by a complex biofilm of pathogenic microorganisms, which contributes to inflammation and the progression of diseases such as peri-implant mucositis and peri-implantitis. The prevalence of *A. actinomycetemcomitans, P. intermedia*, and *T. forsythia* in peri-implantitis biofilms is generally higher than in periodontitis samples or healthy implants. The interaction of these bacteria induces dysbiotic changes in the peri-implant microbiome, promoting a persistent inflammatory state that clinically manifests as peri-implant bleeding [29].

A recent systematic review and meta-analysis identified the main bacterial species consistently associated with peri-implantitis, including *Staphylococcus epidermidis*, *Fusobacterium nucleatum*, *T. denticola*, *T. forsythia*, *P. intermedia*, and *P. gingivalis*, while *Aggregatibacter actinomycetemcomitans*, *Staphylococcus aureus* and *Campylobacter rectus* were not consistently implicated [30].

Role of Bleeding in Assessing Healing around Dental Implants

The presence or absence of bleeding during routine clinical tests is an important indicator of peri-implant tissue health. The absence of bleeding typically reflects stable, healthy peri-implant tissues and suggests successful implant integration and healing. Conversely, bleeding may indicate chronic inflammation, infection, or mechanical complications such as implant overloading, especially when accompanied by clinical signs like increased probing depth or suppuration.

Monitoring bleeding throughout the healing process-from surgery to follow-up visits-provides valuable insight into tissue repair and implant longevity. A decrease in bleeding over time generally indicates proper healing. Persistent bleeding, however, warrants further investigation and potential intervention to prevent progression to more severe peri-implant diseases [31].

Limitations of Bleeding Assessment in Peri-implant Diseases

While BOP is a widely used clinical technique for monitoring peri-implant tissue health, it has several limitations in detecting and managing peri-implant disorders. One of the most significant drawbacks is that BOP is a non specific marker of inflammation [32]. Although bleeding often indicates inflammation, it does not provide specific information regarding the severity or extent of the condition.

The BOP can be influenced by various factors unrelated to peri-implant disease, including probing force, patient anxiety, and recent oral hygiene practices, potentially leading to false-positive results. Furthermore, bleeding may occur due to mechanical stress during probing rather than underlying inflammation, which can result in misinterpretation. Conversely, the absence of bleeding does not necessarily indicate healthy peri-implant tissues; peri-implantitis can occasionally be present without bleeding, particularly in chronic inflammation when tissues become fibrotic and less prone to bleed, resulting in false-negative outcomes.

Additionally, BOP does not differentiate between different forms of peri-implant diseases, such as peri-implant mucositis and peri-implantitis, nor does it identify the underlying cause, whether microbiological or biomechanical. Due to its lack of specificity, BOP should not be used in isolation to diagnose peri-implant disorders but rather as part of a comprehensive assessment that includes radiographic evaluation, probing depth measurement, and examination of clinical signs such as suppuration and tissue recession [33].

Recent Advances

A cross-sectional study conducted in 2024 concluded that peri-implant sites exhibit a higher prevalence of BOP compared to periodontal sites in patients receiving supportive periodontal care. Clinically, this suggests that practitioners providing supportive care to patients with dental implants should anticipate a higher prevalence of BOP around implants compared to natural teeth [34].

Regarding biofilm management around dental implants, recent research highlights that nanotechnology offers innovative solutions for drug delivery and biofilm disruption, providing new opportunities for the development of antimicrobial surfaces and healthcare devices [35].

Patient-related factors also influence BOP. Recent studies revealed that the likelihood of a peri-implant site bleeding upon probing is associated with Probing Depth (PD), implant position, and the patient's gender [36.37].

CONCLUSION(S)

In conclusion, BOP around dental implants is an important clinical indicator of peri-implant tissue health. It serves as an early marker of inflammation, aiding in the diagnosis of conditions such as peri-implant mucositis and peri-implantitis. BOP reflects underlying histological changes, including increased inflammatory cell infiltration and tissue degradation. Although its prognostic value may vary due to anatomical differences between peri-implant and periodontal tissues, BOP assessment remains essential for early intervention. Early detection and management of BOP can halt the progression of peri-implant diseases and contribute to the long-term success of dental implants.

REFERENCES

[1] Mühlemann HR, Son S. Gingival sulcus bleeding-a leading symptom in initial gingivitis. Helv Odontol Acta. 1971;15:107-13.
[2] Lenox J, Kopczyk R. A clinical system for scoring a patient's oral hygiene performance. J Am Dent Assoc. 1973;S6:849.
[3] Greenstein G, Caton J, Poison AM. Histologic characteristics associated with bleeding after probing and visual signs of inflammation. J Periodontal. 1981;52:420.
[4] Hassell TM, Germann MA, Saxer UP. Periodontal probing: Investigator discrepancies and correlations between probing force and recorded depth. Helv Odontol Acta. 1973;17(1):38-42.
[5] Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing. An indicator of periodontal stability. Journal of Clinical Periodontology. 1990;17:714-21.
[6] Joss A, Adler R, Lang NP. Bleeding on probing. A parameter for monitoring periodontal conditions in clinical practice. Journal of Clinical Periodontology. 1994;21:402-08.
[7] Berglundh T, Persson L, Klinge B. A systematic review of the incidence of biological and technical complications in implant dentistry reported in prospective longitudinal studies of at least 5 years. J Clin Periodontol. 2002;29(Suppl 3):197-212.
[8] Mombelli A, Müller N, Cionca N. The epidemiology of peri-implantitis. Clin Oral Implants Res. 2012;23(Suppl 6):67-76.
[9] Jepsen S, Berglundh T, Genco R, Aass AM, Demirel K, Derks J, et al. Primary prevention of periimplantitis: Managing peri-implant mucositis. Journal of Clinical Periodontology. 2015;42(Suppl.16):S152-S157.
[10] Luterbacher S, Mayfield L, Brägger U, Lang NP. Diagnostic characteristics of clinical and microbiological tests for monitoring periodontal and peri-implant mucosal tissue conditions during supportive periodontal therapy (SPT). Clinical Oral Implants Research. 2000;11:521-29.
[11] Koldsland OC, Scheie AA, Aass AM. Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. J Periodontol. 2010;81:231-38.
[12] Ata-Ali J, Ata-Ali F, Bagan L. A classification proposal for peri-implant mucositis and peri-implantitis: A critical update. The Open Dentistry Journal. 2015;393-95.
[13] Misch CE, Perel ML, Wang HL, Sammartino G, Galindo-Moreno P, Trisi P, et al. Implant success, survival, and failure: The International Congress of Oral Implantologists (ICOI) Pisa Consensus Conference. Implant Dent. 2008;17:5-15.
[14] Lang NP, Berglundh T; Working Group 4 of Seventh European Workshop on Periodontology. Periimplant diseases: Where are we now?-Consensus of the Seventh European Workshop on Periodontology. J Clin Periodontol. 2011;38(Suppl 11):178-81.
[15] Padial-Molina M, Suarez F, Rios HF, Galindo-Moreno P, Wang HL. Guidelines for the diagnosis and treatment of peri-implant diseases. Int J Periodontics Restorative Dent. 2014;34:e102-11.
[16] Dhir S, Chauhan S, Sinha A, Sharma S. A focus on soft-tissue changes around implant vs natural tooth. Int J Sci Stud. 2021;9:4-10.
[17] Smeets R, Henningsen A, Jung O, Heiland M, Hammächer C, Stein JM. Definition, etiology, prevention and treatment of peri-implantitis-a review. Head Face Med. 2014;10:34.
[18] Schwarz F, Derks J, Monje A, Wang H-L. Peri-implantitis. J Periodontol. 2018;89(Suppl. 1):S267-S90.
[19] Sakka S, Idrees M, Alissa R, Kujan O. Ailing and failing oral implants: Initial therapy and surgical management. J Investig Clin Dent. 2013;4:207-10.
[20] Sangeeta Dhir B, Lanka Mahesh B, Gregori MK, Vandana K. Peri-implant and periodontal tissues: A review of differences and similarities. Compendium of Continuing Education in Dentistry. 2013 Jul/Aug;34(7):e69-e75.
[21] Kormas I, Pedercini C, Pedercini A, Raptopoulos M, Alassy H, Wolff LF. Peri-implant diseases: Diagnosis, clinical, histological, microbiological characteristics and treatment strategies. A narrative review. Antibiotics (Basel). 2020;9:835.
[22] Lindhe J, Berglundh T, Ericsson I, Liljenberg B, Marinello C. Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog. Clin Oral Implant Res. 1992;3:09-16.
[23] Schou S, Holmstrup P, Stoltze K, Hjørting-Hansen E, Kornman KS. Ligature-induced marginal inflammation around osseointegrated implants and ankylosed teeth. Clinical and radiographic observations in cynomolgus monkeys (Macaca fascicularis) Clin Oral Implant Res. 1993;4:12-22.
[24] Marinello CP, Berglundh T, Ericsson I, Klinge B, Glantz PO, Lindhe J. Resolution of ligature-induced peri-implantitis lesions in the dog. J Clin Periodontol. 1995;22:475-79.
[25] Carcuac O, Abrahamsson I, Albouy JP, Linder E, Larsson L, Berglundh T. Experimental periodontitis and peri-implantitis in dogs. Clin Oral Implant Res. 2013;24:363-71.
[26] Berglundh T, Gislason O, Lekholm U, Sennerby L, Lindhe J. Histopathological observations of human periimplantitis lesions. J Clin Periodontol. 2004;31:341-47.
[27] Javed F, Al-Hezaimi K, Salameh Z, Almas K, Romanos GE. Proinflammatory cytokines in the crevicular fluid of patients with peri-implantitis. Cytokine. 2011;53:08-12.
[28] Akman AC, Askin SB, Guncu GN, Nohutcu RM. Evaluation of gingival crevicular fluid and peri-implant sulcus fluid levels of periostin: A preliminary report. J Periodontol. 2018;89:195-202.
[29] Sahrmann P, Gilli F, Wiedemeier DB, Attin T, Schmidlin PR, Karygianni L. The microbiome of peri-implantitis: A systematic review and meta-analysis. Microorganisms. 2020;8:661.
[30] Carvalho ÉBS, Romandini M, Sadilina S, Sant'Ana ACP, Sanz M. Microbiota associated with peri-implantitis-A systematic review with meta-analyses. Clin Oral Implants Res. 2023;34:1176-87.
[31] Al-Nawas B, Kämmerer PW, Morbach T, Ophoven F, Wagner W. Retrospective clinical evaluation of an internal tube-in-tube dental implant after 4 years, with special emphasis on peri-implant bone resorption. Int J Oral Maxillofac Implants. 2011;26:1309-16.
[32] Rebeiz T, Nasr L, Kassir AR, Menassa G, Chakar C. Assessment of the association between the Implant Disease Risk Assessment (IDRA) tool and peri-implantitis: A retrospective cohort study with up to 8 years of follow-up. International Journal of Oral and Maxillofacial Surgery. 2024;53:845-52.
[33] Linkevicius T, Puisys A, Steigmann M, Vindasiute E, Linkeviciene L. Influence of vertical soft-tissue thickness on crestal bone changes around implants with platform switching: A comparative clinical study. Clin Implant Dent Relat Res. 2015;17:1228-36.
[34] Outatzis A, Nickles K, Petsos H, Eickholz P. Periodontal and peri-implant bleeding on probing in patients undergoing supportive maintenance: A cross-sectional study. Clin Oral Investig. 2024;28:633.
[35] Dash A, Ragavendran C. Innovative approaches to combating dental biofilms: Nanotechnology and its applications. Biocatalysis and Agricultural Biotechnology. 2024;2024:103406.
[36] Farina R, Filippi M, Brazzioli J, Tomasi C, Trombelli L. Bleeding on probing around dental implants: A retrospective study of associated factors. J Clin Periodontol. 2017;44:115-22.
[37] Lucarini G, Zizzi A, Rubini C, Ciolino F, Aspriello SD. VEGF, microvessel density, and CD44 as inflammation markers in peri-implant healthy mucosa, peri-implant mucositis, and peri-implantitis: Impact of age, smoking, PPD, and obesity. Inflammation. 2019;42:682-89.

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