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Review Article

Peri implantitis- A narrative review

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ABSTRACT

Dental implant therapy has become the prosthetic standard of care in modern comprehensive dental care. With increase in placement of dental implants, increase in prevalence of peri-implant diseases have also been reported. Two entities are described within the concept of peri-implant diseases; peri-implant mucositis and peri-implantitis. Various etiological factors are responsible for the occurrence of peri-implantitis with bacterial biofilm playing the major role. Appropriate diagnosis and timely management of peri-implant mucositis and peri-implantitis is essential to prevent the implant loss. Variety of treatment modalities are available for management of peri-implantitis which can eliminate the disease progression and enable the restoration of optimal implant function. This narrative review provides insight of the prevalence, etiology, pathogenesis, diagnosis and management of peri-implantitis with emphasis on current evidence.

1. Introduction

Dental implant therapy has become the prosthetic standard of care in modern comprehensive dental care. With increase in placement of dental implants, increase in prevalence of peri-implant diseases have also been reported.[1] Two entities are described within the concept of peri-implant diseases; peri-implant mucositis and peri-implantitis. Peri-implant mucositis is the reversible inflammation in the soft tissue surrounding implants. Peri-implantitis is an irreversible inflammatory reaction involving the underlying supporting bone.[2]

The bacteria from dental biofilms are considered as the major etiological factor for peri-implant disease. The associated risk factors for peri-implant diseases are alteration in the balance of host-parasite interaction, cigarette smoking, oral hygiene and history of periodontitis.[3] The other possible factors such as genetic traits and implant surface are under investigation. Porphyromonas gingivalis, Prevotella intermedia and Aggregatibacter actinomycetemcomitans are some of the bacterial species responsible for peri-implant disease. The prosthetic design is also considered as risk factor when the prosthesis obstructs the patient or the dental professional in daily hygiene measures or gaining access to the implant surface.[4]

Peri-implantitis is classified based on severity of the disease as Early, Moderate and Advanced. Different methods have been used to assess peri-implant tissue health and to diagnose these disease entities.[3] The diagnostic methods include evaluation of peri-implant microbiota, peri-implant probing, analyses of peri-implant crevicular fluid or saliva and radiographic evaluation of peri-implant bone loss. The treatment modality includes two types: surgical therapy and non-surgical therapy. Mechanical debridement of the implant surface is the commonest treatment employed for peri-implant diseases.

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This article will provide an overview of prevalence, classification, etiology, clinical features, management and maintenance therapy of peri-implantitis with emphasis on current evidence.

2. Peri-Implantitis

Peri-implantitis is an implant related disease condition which contributes to significant proposition of implant failure. This condition was first described Levignac in 1965.[5] The term peri-implantitis was coined by Mombelli[6] in 1987 who described the disease as equivalent to chronic periodontitis.

According to Frank Schwarz 2017, Peri-implantitis is a pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri- implant mucosa and progressive loss of supporting bone.

3. Prevalence

Prevalence of peri-implantitis has been reported by various authors and they greatly differ based on number of implants & follow up

4. Classification of P eri-Implantitis

Numerous classification systems have been proposed in literature for peri-implantitis. However there is no universally accepted classification so far. The following are the classification systems for peri-implantitis:

1. Froum and Rosen, 2012classified peri-implantitis based on distinct clinical stages as follows,[21]
2. Bogaerde et al 2014 proposed classification of bone defects adjacent to dental implants highlighting the defect anatomy in the progression of the regenerative process[22].
   (a) Closed defects- It is characterized by the maintenance of intact surrounding bone walls
   (b) Open defects- It is the one which lack one or more bone walls.
3. Ata-Ali et al (2015)classified peri-implantitis based on the clinical status.[23]
4. Sarmiento et al (2016)classified peri-implantitis based on etiology.[24]
5. Passi D et al (2016)classified peri-implantitis based on bleeding on probing, probing depth, percentage of bone loss and mobility.[25]
6. According to Rucha Shah et al (2016), Retrograde Peri-implantitis is classified as,[26]
7. According to Sarmast et al 2017, retrograde peri-implantitis is classified as,[27]

4.1. Classification based on defect morphology

1. **Nishimura et al, 1997** gave another system of classification exists amount of bone loss with shaped of defect associated[28]
   (a) Class 1: Slight horizontal bone loss with minimal peri-implant defects
   (b) Class 2: Moderate horizontal bone loss with isolated vertical defects
   (c) Class 3: Moderate to advanced horizontal bone loss with broad, circular bony defects.
   (d) Class 4: Advanced horizontal bone loss with broad, circumferential vertical defects, as well as loss of the oral and/or vestibular bony wall
2. **Schwarz et al 2019[29] c**lassified peri implant defect depending on the configuration of the bony defect as:
   (a) Class I defect – Intraosseous
   (b) Class II defect – Supra-alveolar in the crestal implant insertion area.
3. **Spiekermann[30] 1**984 characterized peri-implant defect into the type of bone resorption pattern into 5 category.
   (a) Class I – Horizontal,
   (b) Class II – Hey-shaped
   (c) Class III a – Funnel shaped
   (d) Class III b – Gap-like
   (e) Class IV – Horizontal-circular form

4.2. Etiology of peri –implantitis

Though peri-implantitis, like periodontitis is considered a multifactorial disease, two major etiological factors are:

1. Bacterial infection
2. Excessive mechanical stress

4.2.1. Bacterial infection- Role of plaque microbiota

Dental plaque or biofilm describes a structure of highly organized microbial community in a well-defined matrix which is adherent to hard surfaces of the oral cavity. A cause- effect relationship between biofilm formation and peri-implant diseases have been demonstrated in humans.[31]

Peri-implantitis present with the mixed and variable microbial pattern, although predominated by gram-negative bacteria.[27] Microbiota associated with failing dental implants is similar to flora commonly associated with periodontally involved teeth. The microorganisms most commonly related to the failure of an implant are rods and motile forms of gram negative anaerobes and spirochetes. These includes: Prevotella intermedia, Porphyromonas gingivalis(PG), Aggregatibacter actinomycetemcomitans(AA), Bacteroides forsythus, Treponema denticola(TD), Prevotella

Table 1: Prevalence ofperi-implantitis

Author	Follow up	Number of implants	Prevalence (%)
Derks et al (2015)[7]	-	-	42.9%
Aguirre –Zozrano et al (2015)[8]	5.3 years	786	16%
Daubert et al (2015)[9]	10.9 years	225	16%
Konstantinidis et al (2015)[10]	5.5 years	597	6.2%
Derks et al (2016)[11]	9 years	2277	24.9%
Monje et al (2017)[12]	3.9 years	206	7.3%
Schwartz et al (2017)[13]	-	512	7.6%
Tenenbaum et al (2017)[14]	10.8 years	108	12%
Papaspyridakos et al (2018)[15]	5.2 years	457	10%
Katafuchi et al (2018)[16]	10.9 years	168	16.7%
Cosgarea et al (2019)[17]	9 years	-	45%
Francetti et al (2019)[18]	5 years	384	4.6%
French et al (2019)[19]	6.7 years	4591	3.6%
Kerbs et al (2019)[20]	17 to 23 years	274	15%

Table 2: Classification ofperi-implantitis by Froum and Rosen

Early	PD ≥ 4 mm (bleeding and/or suppuration on probing) Bone loss < 25% of the implant length
Moderate	PD ≥ 6 mm (bleeding and/or suppuration on probing) Bone loss 25% to 50% of the implant length
Advanced	PD ≥ 8 mm (bleeding and/or suppuration on probing) Bone loss > 50% of the implant length

PD-Probing Depth

Table 3: Classification ofperi-implantitis by Ata-Ali

Staging	Definition
Stage I	BoP and/or SUP and bone loss ≤ 3 mm beyond biological bone remodeling
Stage II	BoP and/or SUP and bone loss > 3 mm and < 5 mm beyond biological bone remodeling
Stage III	BoP and/or SUP and bone loss ≥5 mm beyond biological bone remodeling
Stage IV	BoP and/or SUP and bone loss ≥50% of the implant length beyond biological bone remodeling

BoP- Bleeding on Probing; SUP- Suppuration

Table 4: Classification ofperi-implantitis based on etiology

Origin	Example
Peri-implantitis induced by pathogenic bacteria/biofilm	Plaque, calculus, biofilm, previous host susceptibility to periodontitis (previous/active)
Peri-implantitis induced by exogenous irritants	Residual cement, smoking, impacted food debris
Peri-implantitis induced by iatrogenic factors	Buccal implant placement, inadequate inter-implant distance, overheating during surgical placement, poorly fitting restorations
Peri-implantitis induced by extrinsic pathology	Proximal periapical pathology, proximal carcinoma, latent endodontic lesion post extraction
Peri-implantitis induced by absence of keratinized tissue (AKT)	Absence of attached gingival, lack of keratinized tissue with or without muscle attachment

nigrescens, Peptostreptococcus micros and Fusobacterium nucleatum.[32,33] Some studies have reported that the microbiota of peri-implant cases is different from those periodontal disease. Those studies have detected high numbers of peptostreptococcus and staphylococci species.

The bacterial species of peri-implantitis cases vary between partially and fully edentulous subjects.

Samples from partial edentulous patients have greater gram-negative aerobic species when compared to completely edentulous cases.[34] Periodontal pathogens Aggregatibacter actinomycetemcomitans (AA), Porphyromonas gingivalis (PG) and Treponema denticola (TD) where more commonly reported in partially edentulous cases than completely edentulous cases.[35,36]

Longitudinal studies have found an increase in a number of colony forming units of bacteria, slight additional increase in pathogenic species and increase in proportion of motile organisms especially spirochetes as severity of disease progresses.

Table 5: Classification ofperi-implantitis by Passi D

Stage	Bleeding on probing	Probing depth	Bone loss (%) of implant length	Mobility	Proposed treatment and prognosis
Stage 1	-	2-3mm	10-25%	No mobility	No treatment. Oral hygienic instructions; Prognosis is good
Stage 2	+	4-6mm	25-50%	Grade 1	Vertical defect <2-4mm
			Vertical		Guided bone regeneration, osteoplasty
			Horizontal		Horizontal defect< half of implant height Apically positioned flap, Guided bone regeneration, osteoplasty
			Combination		Combination defect Bone augmentation & Guided bone regeneration Prognosis is fair
Stage 3	+	6-8mm	>50%	Grade 2	Vertical defect 2-4mm
			Vertical		Guided bone regeneration Autogenous bone wedge grafting
			Horizontal		Horizontal defect> half of implant height Guided bone regeneration and augmentation
			Combination		Combination defect Implant removal Questionable prognosis
Stage 4	+	>8mm	>50%	Grade 3	Implant removal Poor prognosis

Table 6: Classification of retrogradeperi-implantitis by Rucha Shah

Class I (Mild)	Extends < 25% of the implant length from implant apex.
Class II (Moderate)	Extends 25–50% of the implant length from implant apex.
Class III (Advanced)	>50% of the implant length from implant apex.

Table 7: Classification of retrogradeperi-implantitis by Sarmast

Class 1	Implant placement resulting in devitalization of adjacent previously vital tooth
Class 2	Implant apex infected by persistent periapical lesion on adjacent tooth/implant
Class 3	Implant apex placed/angulated labially or lingually outside envelope of bone
Class 4	Implant apex lesion developed due to residual infection at placement site

4.2.2. Excessive mechanical stress

It has been postulated that mechanical overloading a contributing factor for peri-implant bone loss and late implant failure. Occlusal overload is influenced by prosthetic design. Implants subjected to occlusal overload results in micro motion at the implant abutment interface and compromises the osseointegration during early healing.[34] The location of implant, the dimension of the implant, proportion of the prosthesis are some of the factors leading to occlusal overload. The occlusal overload also depends on the magnitude, duration, transmitted as lateral or axial stress on to the bone. [34]This creates micro fractures of the bone around the implant and initiate peri-implant crestal bone loss which will further propagate in the presence of microbial plaque.[37]

The excess occlusal can be a consequence of placement of implant in poor quality bone, Placement of an implant in poor quality bone, Placement of an implant is an unfavorable position that does not favor ideal load transmission over implant surface, The patient has a pattern of heavy occlusal function associated with para functional habits, Improper prosthetic superstructure that does not fit the implant precisely.[38]

In specific occlusal overload conditions like bruxism, the habit often results in fracture of the suprastructure, but never affects the marginal bone around implants.

5. Clinical Features

Peri-implantitis is the inflammation of soft tissue around the implant with loss of supporting bone. It corresponds to periodontitis around natural teeth. Early signs of peri-implantitis include increase in GCF production and bleeding on probing.[39] Two important features that differentiates peri-implantitis from peri-implant mucositis are the presence of peri-implant pocket and attachment loss. Additional clinical features are redness of the tissue, swelling of the tissue, suppuration and mucosal enlargement.[40]

Other important features late stage peri-implantitis are implant mobility and pain on function. The classical feature of peri-implantitis is bone loss. Although large variation exist regarding the amount of bone required to define a case. 8^{th} European workshop of periodontology in 2012 reported

that > 2mm bone loss from the expected marginal level is considered as case definition.[41]

5.1. Guidelines for diagnosing peri-implantitis

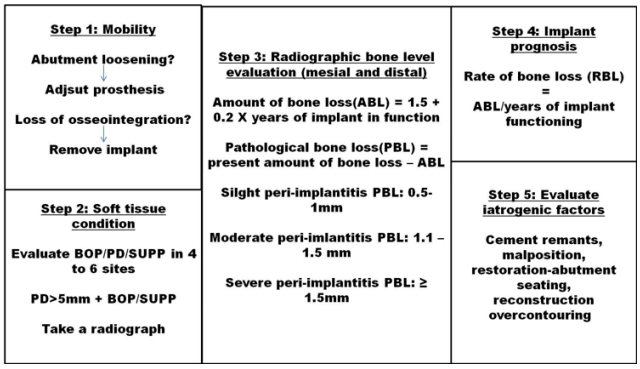

Fig. 1: Guidelines for diagnosing peri-implantitis[42]
BOP-Bleeding on Probing; PD- Probing Depth; SUPP-Suppuration; ABL- Amount of bone loss;
PBL- Pathological bone loss; RBL- Rate of bone loss

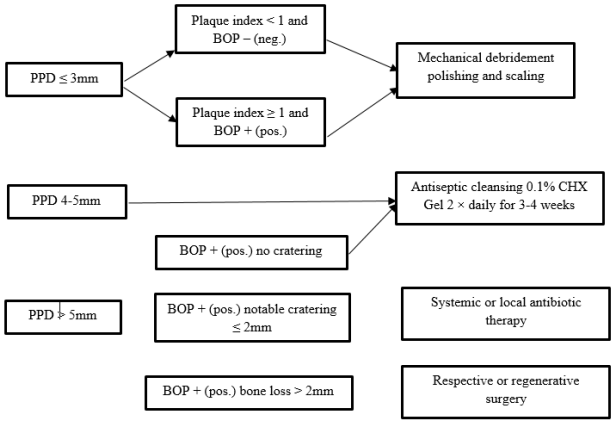

Fig. 2: CIST Protocol

PPD- Probing Pocket Depth; BOP- Bleeding on Probing; CHX-Chlorhexidine; pos- Positive; neg- Negative PPD- Probing Pocket Depth; BOP- Bleeding on Probing; CHX- Chlorhexidine; pos-Positive; neg- Negative
Lang et al (2004) $ modified the CIST protocol and termed it as AKUT concept which is as follows:

6. Management

Based upon the clinical and radiographic diagnosis, certain protocols for preventive and interceptive therapeutic measures have been proposed for implants diagnosed with peri implantitis. Lang et al (2004) reported that the system of supportive therapy is cumulative and consists of few steps. The major clinical parameters used are Presence of biofilm, Presence or absence of bleeding on probing (BOP), Presence or absence of suppuration, increased peri-implant Probing depth and evidence and extent of radiographic alveolar bone loss.

Oral implants without plaque and calculus with healthy peri-implant tissue there may be absence of BOP, suppuration, Probing depth should not exceed greater than 3mm to be considered as clinically stable and these sites should not be exposed to therapeutic measures. The indications of appropriate treatment for the management of peri-implantitis lead to the development of treatment protocols.

CIST (Cumulative Interceptive Supportive Therapy) Protocol by Mombelli and Lang(1998)[43]

7. Maintenance Program

Maintenance therapy after implant placement is of utmost important for effective functioning of implants without any disease. It is classified into two categories.

1. At home implant care
2. Professional hygiene care[44]

8. Summary and Conclusion

Use of dental implants for replacement of missing teeth has become the norm in oral rehabilitation in recent days. Though implants have shown effective and predictable results, increase in use of dental implants has also been associated with occurrence of implant failures of which the most common cause is peri-implantitis. Peri-implantitis is progressive destructive chronic diseases of bacterial or mechanical origin that affects hard and soft tissues surrounding the implant.

The purpose of this paper is to review and report the available evidence for various aspect of peri-implantitis; prevalence, etiology, pathogenesis, management and maintenance.

On analyzing the literature for prevalence of peri-implantitis, the prevalence of peri-implantitis at implant level ranged between 6.5% -47% and prevalence at patient level ranged between 18.8% - 47%.

Looking at etiology, dental plaque and associated micro-organisms has been universally identified as the primary etiologic agents. The second factor identified as etiology is the mechanical factors. Apart from these, a number of risk factors have been associated with peri-implant diseases like local factors (parafunctional habit, local anatomic factors, etc.), systemic factors (age, genetics, medical conditions, etc.), behavioral factors (oral hygiene maintenance, patient compliance).

On analyzing the effects of systemic diseases on periodontitis diabetes was the most commonly investigated

Table 8: AKUT protocol

Stage	Result	Therapy
	PD<3mm, no plaque or BOP	No therapy
A	PD<3 mm, plaque and/or BOP	Mechanical cleaning, polishing
B	PD 4-5 mm, no radiographic bone loss	Mechanical cleaning, polishing, OHI + local antiinfective therapy
C	PD> 5mm, radiographic bone loss <2 mm	Mechanical cleaning, polishing, microbiological test, local and systemic anti‑infective therapy
D	PD > 5 mm, radiographic bone loss >2 mm	Resective or regenerative surgery

PD- Probong Depth; BOP- Bleeding on Probing; OHI- Oral hygiene instructions

Table 9: Aids for implant maintenance

At home implant care	**Professional hygiene care**
Brushing	Scaling and curettage
Soft manual tooth brush	Plastic instruments
Motorized tooth brush	Plastic instruments reinforced with graphites
Sonic tooth brush	Gold-plated curette
End tufted brush	Ultrasonic or sonic scalers covered with plastic sleeve
Interproximal cleaning/circumferential cleaning	**Polishing**
Floss, foam tips, Disposable wooden picks, Interproximal cleaners	Rubber cup with non-abrasive polishing paste E.g.: aluminum oxide, low abrasive dentifrice
Locally applied therapeutics	**Locally applied therapeutics**
Chlorhexidine digluconate	Arestin, Periochip, Atridox
Water irrigation	**Subgingival irrigation**
Hydro floss	Antiseptic agents such as peroxide, chlorhexidine using a plastic irrigation tip.

risk factor. Patients with uncontrolled diabetes were found to be at a higher risk peri-implantitis when compared to controlled diabetes and healthy sites.

On analyzing the evidence for microbiologic profile of peri-implantitis, conflicting results have been reported, but the following micro-organisms were more prevalent Porphyromonas gingivalis (PG), Prevotella intermedia, Treponema denticola (TD), Tannerella forsythia, staphylococcus species, Epstein-Barr virus and human cytomegalo virus 2.

On analyzing the diagnostic evidence of peri-implantitis numerous studies have reported the effects of peri-implantitis on inflammatory cytokines. According to evidence IL1β and TNFα were the most studied pro-inflammatory cytokines. The levels of theses cytokines were found to be significantly higher in peri-implantitis sites when compared to healthy sites. Other cytokines like IL-7, IL-6 and IL-10 have also been investigated but strong association have not been reported.

Looking into treatment of peri-implantitis both non-surgical and surgical interventions have been analyzed. Non-surgical therapy included alteration of implant surface, soft tissue debridement and adjunctive use of anti-microbial, lasers and host modulatory agents. Non-surgical therapy is effective only in eliminating the local etiologic factors and might not the effective in osseous defects. Surgical interventions include reflection of flap, debridement and use of various bone grafts material and membranes. Surgical approach with placement of bone grafts in combination with GTR membranes have been proven to be most effective treatment option with long term predictable result.

With increase in awareness and demand for replacement of missing teeth with dental implants, peri-implant diseases are also increased. Therefore, understanding the etiology, pathogenesis and strategies for treatment for the same should be given equal importance.

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The authors declare no potential conflicts of interest concerning the authorship and publication of this article.

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