ORIGINAL RESEARCH PAPER
Volume - 11 | Issue - 07 | July - 2022 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CURRENT CONCEPTS AND RECENT ADVANCES IN PERI-IMPLANT DISEASES: - A NARRATIVE REVIEW.

Dentistry

Humaira Siddiqui	Senior Resident, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Laxmikanta Patra	Senior Resident, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Subash Chandra Raj	Associate Professor, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Shaheda Tabassum	PG Student, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Asit Kumar Mishra	PG Student, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Peri Srivani	PG Student, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.
Swoyangprava Sil	PG Student, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007
Abinash Pattanaik	PG Student, Department of Periodontics, SCB Dental College and Hospital, Cuttack, Odisha, India. Pin – 753007.

ABSTRACT

The insertion of the dental implant in order to replace missing teeth in partially and completely edentulous patients is a successful treatment modality for over 50 years now. However, its predictability and applicability for a variety of treatment options is still a clinical dilemma. Peri-implantitis represents a serious condition after implant therapy and affects both hard and soft tissues surrounding an implant. While implant therapy represents a safe treatment option with high long-term success, reported complications are associated with improper treatment planning, surgical and prosthetic replacement, material failure and maintenance. Peri-implant lesions may develop after several years of implant placement therefore regular check-ups and life-long supportive periodontal therapy is a must. The management protocol for peri-implant diseases consists of various conservative and surgical approaches. Peri-implant mucositis and moderate peri-implant lesions can be treated with a conservative approach using non-surgical therapy and local and systemic antibiotics. Resective and regenerative therapy are advocated for more severe lesions to completely eliminate the disease. However, due to the lack of prospective randomized long-term follow-up studies, no ideal implant therapy can be implicated. The aim of this review is to provide an overview of current data and to suggest different treatment modalities for diagnosis, prevention and management of peri-implant diseases.

KEYWORDS

CIST, Peri-implant mucositis (PIM), Peri-implantitis (PI), Peri-implant diseases (PID)

INTRODUCTION

The use of dental implants continues to grow as a popular means of replacing teeth, the insertion of these devices has also heightened the perceptions of dentists and the patient regarding what is necessary to maintain health, comfort, function and esthetics. Microbial overload in the oral environment causes pathological reactions in peri-implant tissues, thereby compromising tissue integrity. Dental implants have been reported to achieve long-term success but, are not immune from complications associated with improper treatment planning, surgical and prosthetic execution, material failure and maintenance. (1)

Natural Tooth Vs Peri-implant Tissue: -

Clinical and histological studies have documented the analogy and disparity between natural dentition and dental implants.(2) The dental implants lack the well-structured anatomy and histology of a natural tooth; with the absence of characteristic structures such as root cementum, periodontal ligament, and bundle bone (alveolar bone proper)(3), and with the parallel orientation of collagen fibres on the implant surface facing the perpendicular insertion of collagen in the cementum of natural teeth. The reduced cellularity and poor vascularity on the implant surfaces make them more prone to initiation and progression of peri-implant disease.(2) The differences between tooth versus Implant support system is given in Table 1.

Table 1. Tooth versus Implant Support Systems

Tooth	Implant
1. Periodontal membrane a. Shock absorber b. Distribution of force around tooth c. Tooth mobility can be related to force d. Mobility dissipates lateral force e. Fremitus related to force f. Radiographic changes related to force (reversible)	1. Direct bone implant a. Higher impact force b. Force primarily to crest c. Implants are always rigid (mobility means failure) d. Lateral force increases strain to bone e. No fremitus f. Radiographic changes at the crest (bone loss; not reversible)
2. Biomechanical design a. Longer force duration (decreases impulse of force) b. Cross-section related to direction and amount of stress c. Elastic modulus is similar to bone d. Diameter related to force magnitude	2. Implant design a. Short force duration (increased force impulse) b. Round cross-section and designed for surgery c. Elastic modulus five to 10 times that of cortical bone d. Diameter related to existing bone
3. Sensory nerve plexus within and around the tooth a. Hyperemia induced by trauma from occlusion causing sensitivity to cold. b.Proprioception (reduced maximum bite force)	3. No sensory nerves a. No precursor sign of slight occlusal trauma b. Decreased occlusal awareness of two to five times (increased maximum bite force functional) c. Functional bite force four times higher
4. Occlusal material: enamel a. Occlusal wear facets, pits & fissures and abfractions.	4. Occlusal material porcelain (metal crown) a. No early signs of force
5. Surrounding bone is cortical a. Resistant to change	5. Trabecular bone surrounds the

b. Strong	tooth a. Conducive to change b. Reduced strength

The peri-implant disease can be classified as: -

1. Peri-implant mucositis
2. Peri-implantitis

The term peri-implantitis was introduced in the 1980s (Mombelli A, 1987) to describe inflammatory reactions associated with loss of supporting bone around an implant in function. (1st European workshop on periodontology, Ittingen, Switzerland, 1993).(4-7) Peri-implant mucositis is defined as a reversible inflammatory reaction in the soft tissue surrounding a functional implant.(5,8-10)

Periodontitis versus Peri-implantitis

Periodontitis and peri-implant diseases are similar in etiology, pathogenesis, and risk factors. The dental biofilm plays an important role in the etiology of both of them. The rate and severity of disease progression, and unpredictable response to both surgical and nonsurgical treatment is more for peri-implantitis than periodontitis, due to the reduced vascularity and parallel orientation of collagen fibres.(6,11-14)

CLASSIFICATION OF PERI-IMPLANTITIS

The classification of peri-implant diseases was given by several authors and is mentioned below:-

Speikermann(1995)(15) classified peri-implantitis into four classes depending on the amount of bone loss (Table 2)

Table 2. Speikerman(1995)(15) classified into four types	
Class I	Slight horizontal bone loss with minimal peri-implant defect
Class II	Moderate horizontal bone loss with isolated vertical defect
Class III	Moderate horizontal/vertical bone loss with circular bony defect
Class IV	Advanced bone loss with broad circumferential vertical defect as well as loss of buccal and palatal bony wall.

Schwarz et al (2007)(15) proposed classification of five categories depending on the bone loss around the implant (Table 3.)

Table 3. Schwarz et al (2007)(15) classified peri-implantitis as	
Class A	Buccal or oral dehiscence defect with the position of the implant body within or beyond the envelope
Class B	Buccal or oral dehiscence defect with semicircular bone resorption to the middle of the implant body (position of the implant body within or beyond the envelope.
Class C	Dehiscence defect with circular bone resorption under maintenance of the buccal oral compact layer (position of the implant body within or beyond the envelope).
Class D	Advanced bone loss with broad circumferential vertical defect as well as loss of buccal and palatal bony wall
Class E	Circular bone resorption under maintenance of the buccal and oral compact layer

Froum and Rosen (2012)(16) gave a classification based on the increasing probing depth and percentage of bone loss, which is most widely accepted (Table 4)

Table 4. Froum and Rosen (2012)(16) classification of peri-implantitis as	
Early	PD>4mm (bleeding and/or suppuration on probing) Bone loss< 25% of the implant length
Moderate	PD>6mm (bleeding and/or suppuration on probing) Bone loss 25% to 50%% of the implant length
Advanced	PD>8mm (bleeding and/or suppuration on probing) Bone loss >50% of the implant length

Meffert et al (17) proposed a classification based on the condition of failure on clinical and radiographic status (Table 5)

Table 5. Meffert et al (17) proposed a classification according to the condition of failure on a clinical and radiographic basis.	
Ailing implant	Radiographic bone loss without inflammatory signs or mobility
Failing implant	Progressive bone loss, signs of inflammation and mobility. The peri-implant disease is usually in a reversible state.
Failed implant	Progressive bone loss with clinical mobility and that is not functioning.

Epidemiology of peri-implant diseases

The exact prevalence of the peri-implant disease is difficult as there is a lack of specific clinical and radiographic definitions. However, it is important that this disease is prevalent and longer the implant, a greater number of implants affected. (18) *Peri-implant mucositis:* 80% of the subjects with dental implants suffer from mucositis however only 50% of the implants are affected.(12) *Peri-implantitis*: 28-56% of the subjects suffer from peri-implantitis and only 12-43% of the implants are affected. (11) The prevalence of peri-implantitis is increasing as a function of time.(7,8) Koldsland et al. concluded that peri-implant inflammation was a frequent finding with and without peri-implant bone loss on applying different diagnostic thresholds for evaluating its prevalence.(19)

Peri-implant mucosa from health to disease

The aetiology of peri-implantitis is multi-factorial and micro-organisms play an important role in its development.(20) Biofilm plays a significant role in the initiation and progression of peri-implant diseases and is necessary for the development of infection around implants.(21) Peri-implant mucositis is similar to gingivitis in presentation and is reversible.(9) In animal studies experimental peri-implant mucositis disease remission may take longer than three weeks, unlike gingivitis which subsides within 21 days. Similarly, in Human studies, large inflammatory lesions are seen in peri-implant mucositis.(9,22,23) Peri-implantitis, similar to periodontitis, occurs primarily as a result of microbial overload and subsequent host immune response. Many studies have found that the bacterial species associated with periodontitis and peri-implantitis are similar, mainly gram-negative anaerobes including Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythia, Peptostreptococcus micros, Campylobacter rectus and Fusobacterium species. Staphylococcus aureus may also be an important pathogen in the initiation of peri-implantitis.(2,10,24) Connective tissue adjacent to the pocket epithelium is infiltrated by inflammatory cells, B-lymphocytes and plasma cells being the most dominating cell types. Similar markers are upregulated between peri-implantitis and periodontitis, including proinflammatory cytokines such as interleukin-1 (IL-1), IL-6, IL-8, IL-12, and tumour necrosis factor-alpha (TNF-α).(2,10,25) Periodontitis is a self-limiting disease however peri-implantitis has a continuous progression of the disease which leads to additional bone loss. The primary objective for treating peri-implantitis is to eliminate biofilm from the implant surface. (2,10,26,27)

Risk Factors: -

Many risk factors are responsible for the establishment and progression of peri-implantitis.

1). Previous Periodontal Disease

Many studies have indicated that peri-implantitis was a more frequent finding in patients with a history of periodontitis.(28,29) Karoussis et al concluded in a study that when comparing the failure, success, and complication rates of patients who lost their teeth due to periodontitis or other reasons the group with a history of chronic periodontitis had a significantly higher incidence of peri-implantitis (28.6%) than the group with no history of periodontitis (5.8%). (30)

2). Poor Oral Hygiene maintenance.

An implant prosthesis can cause difficulty in proper plaque control. May be due to prosthesis design being clinically evaluated with probing and adequate home-care procedures.(10,31) Reinforcement of oral hygiene habits and regular follow up will help diagnose early disease and is treatment.(10)

3). Residual cement

Incomplete removal of dental cement left in the subgingival space around implants may cause inflammation and disease. (32,33)

4). Smoking:

Many cohort studies and cross-sectional studies frequently have linked smoking to higher implant failures (10,34,35) Klokkevold et al. (34) reported that 78% of the implants in smokers had peri-implantitis, compared to non-smokers was 64%.

Smoking causes: -

- Impaired wound healing.
- Reduced collagen production.
- Impaired fibroblast function.
- Reduced peripheral circulation.

- Compromised function of neutrophils and macrophages. (2,10,35,36)

5). Genetics:

It was observed that polymorphisms of interleukin 1β and its natural specific receptor antagonist IL-1RN significantly influence the incidence of peri-implant bone loss. (10,37)

6). Systemic Disease: -

- Diabetes mellitus: High blood glucose levels can impact tissue repair and host defense mechanisms, as diabetic control affects neutrophil function. (9,38)
- Alcoholism: Peri-implant bone loss significantly related to alcohol consumption daily more than 10g.(10,39)
- Rheumatoid Arthritis (10)

7). Parafunctional Habits like bruxism etc. (40,41)

8). Lack of Keratinized gingiva-

Soft tissue defects or poor-quality soft tissue at the implant site.(41) Majority of human clinical trials indicated that if a wide band of keratinized mucosa is present then there is reduced plaque accumulation, tissue inflammation and probing depths as patients had less discomfort when brushing. (42)

ADDITIONAL FACTORS:

1.Biomechanical factors:

Experimental and clinical evidence supports the concept that excessive biomechanical forces may lead to high stress or microfractures in the coronal bone-to-implant contact and thus lead to loss of osseointegration around the neck of the implant.43 Bone remodels in response to the strain.44 Excessive stress can cause microfracture within the bone and eventual bone loss.(43) Although overload is clinically difficult to define and measure, the role of overloading is likely to increase in four clinical situations. (2,41)

1. Implant placed in poor quality bone.
2. Implant's position or the total amount of implants placed not favoring ideal load transmission over the implant surface.
3. Patients having pattern of heavy occlusal function associated with parafunction.
4. Prosthetic superstructure does not fit the implants precisely. (40,41)

Other etiologic factors such as traumatic surgical techniques, inadequate amount of host bone resulting in an exposed implant surface at the time of placement, and a compromised host response can act as co-factors in the development of peri-implant disease. (2,6,10)

2.Technical implant failures:

In clinical reality, fatigue of implant materials, weakness in prosthetic design and dimensions, and other factors may result in implant fractures.(45) Balshi listed three categories of causes that may explain implant fractures: design and material, the non-passive fit of the prosthetic framework, and physiologic or biomechanical overload.(46) The occurrence of implant fractures can be kept to a minimum through the use of quality-controlled implant designs and the consideration of physical principles and biomechanical characteristics of various materials and prosthesis design. Patients with bruxism seem to be at higher risk for such events and therefore need to be screened and informed accordingly. (46,47)

3.Abutment loosening and fracture:

Screw loosening has been reported to occur quite frequently in screw-retained fixed partial dentures. Screw retained single crowns may also be prone to technical complications.(48,49) Abutment screw fractures are less frequent but remain clinical hazards that are not always easily resolved.(50) If the fractured abutment is buried within the internal threads of the implant, it may be impossible to retrieve the screw. Such an implant may then be left buried beneath the mucosa.(51) In some instances, screw retrieval sets can be used successfully to salvage implants with deeply broken fragments.(51) Newer abutment designs and higher torque levels used to insert abutment screws has helped reduce the rate of screw loosening.(52)

4.Esthetic complications:

Implant placement in the esthetic zone requires precise three-dimensional tissue reconstruction and ideal implant placement to develop a natural emergence profile of the implant crown. Sometimes if the amount of available bone does not allow for ideal implant placement and the implant is positioned apical, buccal, or interproximal, an unesthetic prosthetic profile develops the same is true if a bone reconstruction procedure shows a compromised result and the implant placed in an inappropriate position If crown form, dimension, shape, and gingival harmony around the implants are not ideal, the patients are not satisfied with the esthetics. If patients are fussy then implants need to be removed, the case reevaluated and retreated. (52)

DIAGNOSIS OF PERI-IMPLANTITIS

Peri-implant diagnostic procedures can serve several functions (6)

1. Screening for peri-implant disease
2. Differential diagnosis of peri-implantitis and peri-implant mucositis
3. Treatment planning
4. Evaluation of therapy and monitoring.

Peri-implantitis lesions are often asymptomatic and detected at routine recall appointments.(6)

PRIMARY DIAGNOSTIC AIDES

In peri-implantitis, a bony defect develops around the implant extending mostly around the full circumference of the implant.(11,12,15,54,55)

1.Probing, Bleeding, Suppuration:

Peri implant probing depth (PPD) around normal implants is also influenced by the duration of time that the implant has been in function and there is an increase in PPD up to 0.22 mm with each year in function.(53) In peri-implantitis, a bony defect develops around the implant extending mostly around the full circumference of the implant.(11,12,15,55) Bleeding on Probing (BOP) is a mark of active periodontal disease.(56,57) however it cannot be considered as a reliable marker for detection of peri-implantitis(58), as peri-implant tissues have a weak desmosomal attachment and a slight mucogingival shift after implant placement.(59) Suppuration is a sign of inflammatory process in the tissues and hence is a highly specific clinical parameter for a progressive form of peri-implantitis.(57) For peri-implant mucositis it has a low predictability rate in comparison to peri-implantitis, therefore it is a clinical endpoint to disclose progressive peri-implant bone loss.(57,59)

Mucosal Redness (MR): Plaque accumulation corresponds to mucosal redness in natural teeth (59) and implants. (23) In a study it was found that MR was an accurate diagnostic tool to monitor PIM and PI, and was associated with bleeding on probing (BOP).(60)

2. Radiographs: -

Radiographs are taken to detect bone loss. Clinical presentation of inflammation is similar for PIM and PI however progressive loss of bone support distinguishes the former from the latter in the radiograph. Different patterns of bone loss occur around implants and therefore many authors have formulated the implant success index (20) based on the percentage of bone loss concerning the length of the implant. Also, the sixth, seventh and eighth EWP(11,12,14) considered the change in crestal bone levels at baseline as data for diagnosis of peri-implantitis.(61) During the initial days of function, there is 2mm crestal bone loss around implant(61) however more than 2mm is suggestive of pathology.(62) The diagnostic parameters for monitoring peri-implant conditions were assessed by various authors.(63-65) Peri-apical radiographs should be taken perpendicular to the implant body to clearly demarcate the threads of the implant.(6,10) Paralleling and bitewing (Saucer shaped or rounded beaker appearance in radiograph often extending the implant's full circumference) can be taken.(2,66)

CBCT may be considered depending on the location of progressive attachment loss.(66)

3.Mobility:-

Mobility indicates a very advanced case of bone loss or loose crown or broken component.(8,10)

SECONDARY DIAGNOSTIC AIDE

Bacterial culturing, inflammatory markers, genetic diagnostics may be useful in the diagnosis of peri-implant diseases.(2,10)

No single diagnostic tool can be used for the diagnosis of peri-implantitis. (10,67,68)

- Combination of probing data over time.
- Inflammatory status of the mucosa.
- Bleeding on light probing,
- Radiographic changes in bone levels over time, and possibly bacterial.
- PICF (peri-implant crevicular fluid) sample can be used to come to a diagnosis. (3,38)

RETROGRADE PERI-IMPLANTITIS

McAllister et al. (69) in 1992 reported an entity different from peri-implantitis as retrograde peri-implantitis (RPI). It was described as a

clinically symptomatic periapical lesion that develops clinically within the first few months after implant insertion while the coronal portion of the implant sustains normal bone to implant interface. (70)
Exact etiology is unclear, may be attributed to:

- Implant surface contamination
- Residual bacteria in the implant site
- Presence of adjacent endodontic lesions
- Residual root particles or foreign bodies. (69)

As micro-organisms are responsible for this disease, management should be aimed at reducing the microbial load, eliminating inflammation and decontaminating the implant surface in order to preserve supporting bone and do periodontal regeneration if possible. (71)

MANAGEMENT OF PERI-IMPLANTITIS

Surgical and non-surgical techniques have been developed. Treatment will be different for peri-implant mucositis (PIM) and peri-implantitis (PI).(72)For peri-implantitis, both surgical and non-surgical approaches can be used (figure 1).

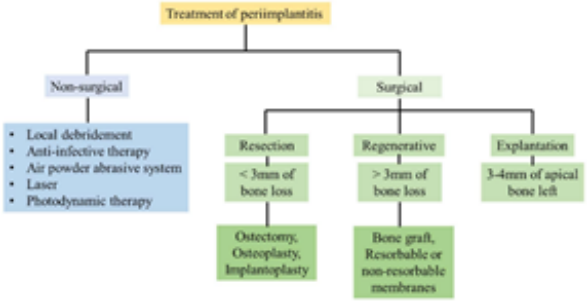

Figure 1. Flow chart determining the treatment peri-implantitis

NON-SURGICAL APPROACH TO PERI-IMPLANTITIS

1.Local debridement: -

Implants should be cleaned by instruments softer than titanium such as polishing with a rubber cup and paste, floss, interdental brushes or using plastic scaling instruments. These do not roughen the implant surface unlike metal and ultrasonic scalers.(67,71,73,74) Eger et al,(75) in their study, examined and proved that ultrasonic scaling of titanium implants with metal tips releases titanium particles in a surface type-dependent manner and aggravate peri-implantitis.
Treatment of peri-implant mucositis (42): -
•Bacterial plaque and calculus should be removed
•Chemical plaque control is achieved with 0.12% chlorhexidine applied topically, every 8-12 hr for 15 days.
•Give oral hygiene instructions.
•Prosthetic design should also be checked and modified to correct design defects and correct biomechanical stress
•Periodic check-ups must be scheduled, reducing the interval between maintenance visits.

2.Anti-infective therapy: -

a) Local or systemic antibiotic therapy.(72,73)
b) Chemical decontamination using citric acid or EDTA or hydrogen peroxide, tetracycline, minocycline, saline and saline-soaked cotton pellet, or 35% phosphoric acid gel, in combination with mechanical debridement for eliminating hard and soft deposits.(18,71)
c) In localized peri-implant problems local drug delivery devices work effectively like minocycline spheres and chlorhexidine chips.(73)

3.Air powder abrasive system:-

Air powder abrasive system (AP) features the use of an abrasive powder, generally, sodium bicarbonate, and sodium hydrocarbonate, or amino acid glycine, propelled by a stream of compressed air to remove biofilm or extrinsic stains from teeth.(18) AP system causes detoxification of implant surface and can improve clinical outcomes, however causes subcutaneous emphysema around teeth and implants, hence it has to be used cautiously and cannot be recommended routinely. (18)

4.Laser decontamination:-

Laser decontamination is based on its thermal effect, where proteins are denatured and cause cellular necrosis to achieve complete surface decontamination.(71) Diode, CO2, and erbium-doped yttrium, aluminium, and garnet (Er:YAG) lasers are suitable for implant irradiation because of their hemostatic properties, selective elimination of calculus and bactericidal effects. The degree of energy absorption by titanium is low, and there is no significant temperature increase of the implant body.(76) Another literature review found that laser combined with chlorhexidine or saline solution achieved a greater percentage of re-osseointegration.(71,77)

5.Photodynamic therapy

Photodynamic therapy is based on the application of photosensitive dyes activated by a light with a specific wavelength to kill bacteria. Three basic elements included are visible light, nontoxic photosensitizer, and oxygen. Oxygen is transformed into ions and radicals which are highly reactive kill micro-organisms has a better bactericidal effect than laser alone.(18) The disadvantage of PDT is that the dye cannot differentiate between bacteria and host cells and hence adjacent tissue is also affected. Advantages are no adverse effects or resistance development to target bacteria as seen in systemic antibiotics. (18,78)

Factors that influence the PDT effectiveness are (18)

- Light absorption by the bacteria
- Wavelength of the laser
- Time of laser exposure
- Area to be stained
- Organic matrix of the biofilm.

SURGICAL APPROACH

In cases of advanced bone loss, in spite of initial treatment, a surgical approach is required to eliminate the infection and restore lost bone.(72,79)

The surgical approach can be resective or regenerative depending on the type of bone defect and morphology.(72)

I) RESECTION TECHNIQUES:

Used in moderate (< 3mm) horizontal suprabony defects or vestibular dehiscences in the non-aesthetic zone. The procedure includes ostectomy or osteoplasty after raising an apically positioned flap and implantoplasty.(72,79) The main aim is to achieve good soft tissue morphology and reduced pocket depth for optimum oral and implant health.(72)

The resection technique involves (72)

1. Removal of supragingival plaque.
2. Surgical access.
3. Removal of granulation tissue.
4. Detoxification of implant surface.
5. Correction of the bony architecture.
6. Modification of implant surface roughness.
7. Implementation of plaque control.

Implantoplasty: -

Indicated to completely flatten/smoothen the exposed implant threads using diamond stones with adequate coolant to grind the plasma spray coatings or implant surface with final polishing achieved with rubber disks.(80,81) It is mainly done to reduce the surface roughness on the implant surface which causes more plaque accumulation.(82) Implantoplasty can be combined with regenerative surgery and sub-epithelial connective tissue graft (SCTG). Schwarz et al., did a study where he did a 6-month follow up for 10 cases treated with implantoplasty, surface decontamination with saline-soaked cotton pellets and xenograft with a collagen membrane, and SCTG and showed a significant reduction in PD, CAL, and soft tissue recession.(83) The drawback of this technique is that it causes gingival recession and exposure of abutment and implant surface which leads to unesthetic appearance and food impaction. However, pockets are eliminated and desirable reattachment is gained. Therefore, implantoplasty cannot be used as monotherapy in many clinical situations.(18)

II) REGENERATIVE SURGERY

Regenerative therapy requires decontamination of implant surface followed by use of bone graft (autogenous bone, demineralized freeze-dried allogenic bone, bovine inorganic bone and hydroxyapatite), in combination with resorbable or non-resorbable membranes, for guided bone regeneration (GBR). This has been used successfully over the years for the treatment of peri-implantitis.(72,79) Collagen membranes are placed to stabilize the clot and prevent the migration of epithelium and connective tissue into the defect during surgery.(84) Most common post-operative surgical complication after membrane placement is the exposure of the same leading to infection.(74,84) This procedure is done when decisions for prosthetic preservation or

esthetics are to be considered.(72,79) Heitz-Mayfield et al. (85) in their study concluded that open flap debridement is done under systemic antibiotic therapy resulted in a reduction in peri-implantitis in 90% of cases for the short term (one year), however bleeding on probing persisted in nearly 50% of these cases. Schwarz et al. (86) concluded in their study that guided bone regeneration yielded greater improvements in clinical parameters. Schwarz et al.(87) in another study found termination of peri-implant bone loss and reduced bleeding on probing from 80% to 34%. Froum et al.(20) also showed that surgical regeneration was effective in arresting peri-implantitis and reduced bleeding on probing. Peri-implantitis lesions are not always favourable for regeneration like thin facial and lingual walls presenting complete loss of surrounding bone leaving regeneration with questionable prognosis.(88) Aljateeli et al. (89) proposed a decision tree (figure 2) based on the defect morphology and suggested that an apically positioned flap with implantoplasty is the treatment of choice in one wall defect. The decision tree for etiology and management of peri-implantitis.

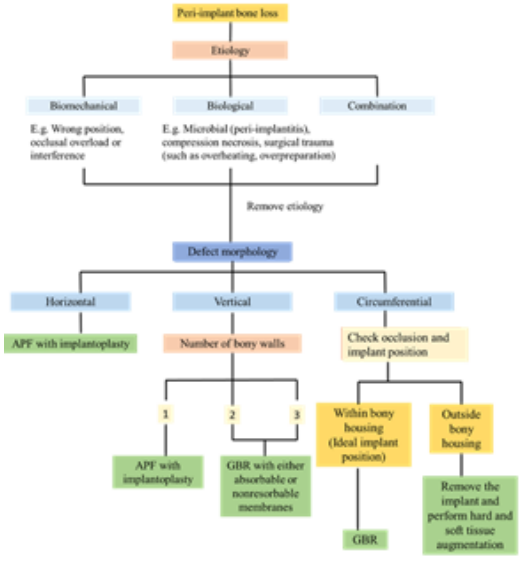

Figure 2. Decision tree on etiology and management of peri-implantitis

Another summary was given by Aljohani M et al (2020) and Mishler O P (2014) for the management of peri-implant diseases (figure 3).(90,91)

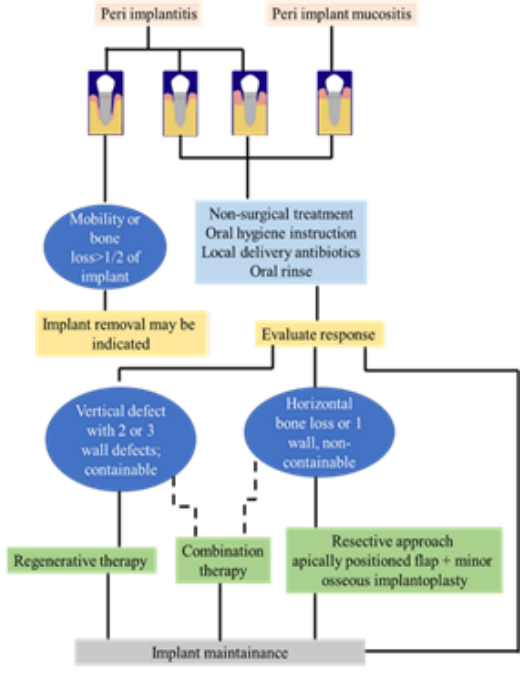

Figure 3. A summary of the management for peri-implant diseases.

Charalampakis et al. (92) reported that smoking and early disease development were related to higher rates of relapse of peri-implantitis while surgical therapy had lower relapse rates. He concluded that peri-implantitis is not hard to treat but patients have to be kept on follow-up as there are more chances of relapse. There is little evidence from four clinical trials that more complex and expensive therapies were more beneficial than the control therapies which consists of subgingival mechanical debridement. Follow up more than one year suggested recurrence of peri-implantitis in up to 100% of the treated cases for some of the tested intervention, therefore for long term treatment success, the importance of plaque control and oral hygiene cannot be over-emphasized.(93) Lopez et al (94) in their study found that open flap debridement with HYBENX® as a chemical desiccant was effective in the treatment of peri-implantitis. HYBENX® allows selective removal of microbes and tissue debris. Consists of a mixture of acidified phenolics (60% sulfonated phenolics, 28% sulphuric acid, 12% water). Its principal action is Desiccation Shock Debridement Technology (DSD). Further studies are required to understand the role and benefits of HYBENX®

Cumulative Interceptive Supportive Therapy (CIST):

Mombelli and Lang et al. proposed a systematic approach for monitoring tissues around implants in the prevention and treatment of peri-implantitis. This systematic protocol, referred to as Cumulative Interceptive Supportive Therapy (CIST) (figure 4), contains four treatment modalities. Each step of the procedure is used in a sequential manner with increasing antibacterial intervention, combined with surgical resective/regenerative treatment (A+B+C+D).(6)

A. If plaque and/or an increased tendency of the peri-implant tissues to bleed is detected, then the implants are mechanically cleaned using a rubber cup and polishing paste. Oral hygiene practices should be checked, and the proper plaque control technique should be instructed and reinforced.
B. In the presence of pus, or if first signs of peri-implant tissue destruction are detected (pockets in the range of 4 to 5 mm and slight bone loss) regimen A should be combined with the application of a local antiseptic.
C. If the peri-implant sulcus allows more than 5 mm of penetration of a periodontal probe then a radiograph is taken. If there is clear evidence of bone loss, then a microbiological sample is taken. Should there be evidence of an anaerobic flora, the patient is given treatments A and B, and, in addition, is placed on systemic antimicrobial therapy.
D. If the bone destruction has advanced considerably, surgical intervention to correct the tissue morphology or to apply guided bone regeneration techniques may be necessary. Such treatment would, however, only be given in addition to the other measures (A, B and C).

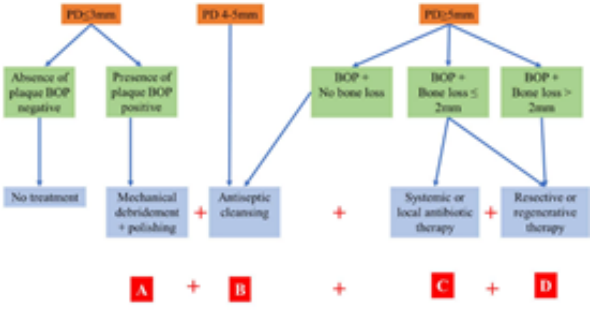

Figure 4. Flow chart determining CIST protocol

III) EXPLANTATION:-

If there is advanced bone loss and mobility then implants cannot be saved, has to be removed. Removal of the implant with trephines causes significant bone removal from buccal and lingual cortices and may damage the adjacent tooth. An alternative approach is to let the bone loss progress and when 2-3mm bone is present around the implant it can be extracted with extraction forceps.(73)

CONCLUSION:-

The clinical diagnosis of peri-implant mucositis and peri-implantitis is becoming more conventional, necessitating effective therapeutic treatments. Comprehensive treatment planning is paramount with regular recall and maintenance necessary to detect and intercept problems early. Instructions in oral hygiene and smoking cessation advice should be mandatory.

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