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PERIIMPLANTITIS AND IT'S MANAGEMENT A REVIEW

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ABSTRACT

Implants are considered to be one of the successful treatment options available in the field of dentistry for the replacement of the missing teeth. The prosthetically driven implants improves the clinical outcome in terms of both function and esthetics. Even though implants are considered to be the game changer in the field of dentistry, complications and failures related to implants has become a major concern. Proper diagnosis and treatment planning is mandatory in order to prevent the complications of implant. This review article will pivot mainly on the etiopathogenesis of periimplantitis, risk factors associated, diagnosis, management with different treatment protocols available in the literature.

1. INTRODUCTION

The successful use of Osseo integrated dental implants has dramatically changed dentistry and significantly improved dentists' ability to provide tooth replacement options for patients. Peri-implant tissues are those that occur around Osseo integrated dental implants. They are divided into soft and hard tissue compartments. The soft tissue compartment is denoted "peri-implant mucosa" and it is formed during the wound healing process that follows implant/abutment placement. The hard tissue compartment forms a contact relationship to the implant surface to secure implant stability. The destruction of peri-implant tissues can jeopardize the implant success and survival.[1] Despite implants long-term predictability and success, implant-related complications and failures can happen. Complications can be surgical, biologic, mechanical, or aesthetic.Biologic complications involve the hard and soft tissues that support the implant. Peri-implant tissue changes can be limited to inflammation of surrounding soft tissues or be more significant, such as progressive loss of supporting bone.[1] Peri- implant disease is a collective term used to describe inflammatory processes in tissues that surround implant(s), that is peri- implant mucositis and peri- implantitis (Albrektsson & Isidor 1994).[1]

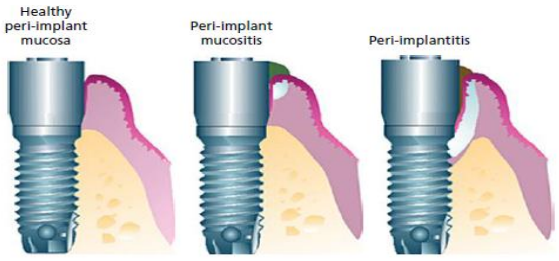

Figure 1: Peri-implant health, Peri-implant mucositis and Peri-implantitis

2. DEFINITION OF PERIIMPLANTITIS

Peri-implant diseases, including peri-implant mucositis and peri-implantitis, were first defined and described at the First European Workshop on Periodontology in Ittingen in 1993.[3]

Peri- implant mucositis is defined as an inflammatory lesion that resides in the mucosa. (Lindhe & Meyle 2008).[1] 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]

3. CLASSIFICATION OF PERIIMPLANTITIS

Froum and Rosen 2012[3]:

Figure 2: Periimplantitis classification

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4. ETIOPATHOGENESIS OF PERIIMPLANTITIS

At the First European Workshop on Periodontology in 1993, two disease patterns associated with oral implants were identified and defined. *Peri-implant mucositis* is a term used to describe reversible inflammatory reactions in the mucosa adjacent to an implant and *peri-implantitis* is defined as an inflammatory process that (i) affects the tissues around an osseointegrated implant in function. Under heavy plaque accumulation and in a time period long enough for the development of infections lesions may progress into the supporting tissues around implants as they do around teeth. It was concluded that the pattern of spread of inflammation was different in periodontal and peri-implant tissues. The lesions in plaque associated periodontitis were limited to the connective tissue, whereas in the peri-implant tissues the lesions involved the alveolar bone as well. In contrast to the periodontal tissues, the peri-implant tissues appeared to be poorly encapsulated to resolve progressive, plaque associated lesions and extend into the marginal bone tissue and may, if they are allowed to progress, lead to the loss of the implant.[4] The contributory role of a lack of keratinized mucosa to the pathogenesis of peri-implantitis has been a topic of dispute. Experimental studies in animals and human longitudinal studies were inconclusive in attributing a significant role to the lack of keratinized mucosa.[4]

5. CLINICAL & RADIOGRAPHIC FEATURES OF IMPLANTITIS

Clinical signs of inflammation including redness, edema, mucosal enlargement, BOP+ with or without suppuration along with increases in PD and radiographic bone loss are commonly used in case definitions for peri-implantitis.[3]

- Tissues may or may not be swollen. Hyperplasia is frequently seen if implants are located in an area with nonkeratinized mucosa or if the suprastructure is an overdenture.[5]
- Pain is not a typical feature of peri-implantitis.[5]
- Vertical bone destruction is associated with the formation of a peri-implant pocket.[5]
- There is bleeding after gentle probing with a blunt instrument and there may be suppuration from the pocket.[5]
- There is radiological evidence for vertical destruction of the crestal bone. The defect usually assumes the shape of a saucer around the implant while the bottom part of the implant retains perfect osseointegration. In some instances, wedge-shaped defects develop along the implant. A continuous periimplant radiolucency indicates implant failure.[5]

6. RISK FACTORS FOR PERIIMPLANTITIS

Risk factors for peri- implantitis can be categorized according to the patient, clinician/treatment procedures, and implant characteristics.[2]

History of periodontitis:

There is evidence that patients with a high susceptibility to periodontitis exhibit a higher risk for periimplantitis.[1]There is strong evidence from longitudinal and cross-sectional studies that a history of periodontitis constitutes a risk factor/indicator for peri-implantitis.[2]

Smoking:

Smoking has been strongly associated with chronic periodontitis, attachment loss as well as tooth loss.[3] In line with the association between history of periodontitis and periimplantitis, it is logical to assume that known modifying factors for periodontitis, such as smoking and diabetes, may also be valid for peri- implantitis.[2]

Diabetes:

Diabetes mellitus comprises a group of metabolic diseases where type 1 describes an autoimmune destruction of insulin producing β-cells and type 2 is characterized by insulin resistance. The global prevalence of diabetes in the adult population is estimated at around 8%,and the disorder has been identified as a risk factor for periodontitis. Available evidence is inconclusive as to whether diabetes is a risk factor/indicator for peri-implantitis.[2]

Poor plaque control/lack of regular maintenance therapy:

As demonstrated in classical studies on periodontal diseases, lack of regular maintenance therapy is associated with tooth mortality and clinical attachment loss at teeth. These findings have highlighted the importance of self-performed and professionally-administered infection control measures in the prevention of periodontal diseases. Other factors related to oral hygiene measures at implants may also be considered. Recently, Souza et al. reported that brushing at implant sites with keratinized mucosa (KM) <2 mm was associated with considerably more discomfort when compared to brushing at sites with KM ≥2 mm.[3] There is evidence that poor plaque control and lack of regular maintenance therapy constitute risk factors/ indicators for peri-implantitis.[2]

Design of suprastructure:

Treatment procedures and the design of prosthesis may also be regarded as a potential risk for peri- implantitis. The neglect of access for self- performed and professional infection control procedures when designing the

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supraconstruction to be connected to implants entails an increased risk for peri- implant disease. It is imperative that the implant supported prosthesis allows appropriate access for infection control.[2]

Implant surface characteristics:

Risk factors for peri- implant disease also relate to specific features of the implants, such as design and surface characteristics. While clinical evidence in this field is weak, data from preclinical studies indicate that implant surface characteristics influence progression of peri- implantitis.It is concluded that rough surface of the implant may influence the onset of peri- implantitis.[2]

Iatrogenic factors:

The Consenus report of the 7th European Workshop on Periodontology recognized that the onset and progression of periimplantitis may be influenced by iatrogenic factors such as "inadequate restoration-abutment seating, overcontouring of restorations or implant-malpositioning".[1] It appears reasonable that the implant position and design of the suprastructure should facilitate access for self-performed oral hygiene and professionally administered plaque removal.[2]

7. DIAGNOSIS OF PERIIMPLANTITIS

It is evident that peri-implant disease should be recognized earlier, to allow intervention before a substantial portion of the supporting bone is lost.

The diagnosis of peri-implantitis requires:[2,5]

1. Evidence of visual inflammatory changes in the periimplant soft tissues combined with bleeding on probing and/or suppuration;

2. Increasing probing pocket depths as compared to measurements obtained at placement of the supra-structure; and

3. Progressive bone loss in relation to the radiographic bone level assessment at 1 year following the delivery of the implant-supported prosthetics reconstruction; and

4. In the absence of initial radiographs and probing depths, radiographic evidence of bone level ≥3 mm and/or probing depths ≥6 mm in conjunction with profuse bleeding represents peri-implantitis.

Diagnostic parameters:

Peri-implant radiography: The preservation of marginal bone height is considered crucial for implant maintenance and is often used as a primary success criterion for implant systems. Vertical bone loss of less than 0.2 mm annually following the implant's first year of service has been proposed as one of the major criteria for success. In the absence of clinical signs of infection, it is recommended to take radiographs 1 year after implant installation and not more than every other year thereafter. Additional radiographs should only be taken to determine the extent of marginal bone loss if clinical parameters (increased probing depth) indicate signs of peri-implant infection.[5]

Peri-implant probing

In addition to the evaluation of bone levels on radiographs, peri-implant probing has been suggested as a useful diagnostic procedure. Probing the peri-implant sulcus with a blunt, straight periodontal probe allows the assessment of the following parameters: peri-implant probing depth; distance between the soft tissue margin and a reference point on the implant (measurement of soft tissue hyperplasia or recession); bleeding after probing; and exudation and suppuration from the peri-implant space. Standardized probes such as the Audio Probe, the TPS Probe or the HAWE Click Probe may be recommended.[5]

Mobility

Implant mobility is an indication for lack of osseointegration.[6] Assessment of implant biomechanics can be done clinically with the help of non-invasive devices like Perio test and Ostell.

PERIOTEST:It is a non-invasive electronic device that provides an objective measurement of the reaction of the Periodontium to a defined impact load applied to the crown of the tooth. It assesses the bone implant contact and provide values which are mainly dependent on the dampening characteristics of the Periodontium. The optimum bone implant contact rigidity should be in the range of -8 to + 50. [6]

Reading	Interpretation
−8 to 0	Good osseointegration, implant can be loaded
+1 to +9	Clinical examination is required, in most cases loading is not possible
+10 to +50	Osseointegration is not sufficient, implant cannot be loaded

Figure 3: Interpretation of Periotest values

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8. RESONANCE FREQUENCY ANALYSIS

It is a non-invasive test in which a small transducer is attached to the implant or abutment. It imposes a series of frequencies to the implant and assess the changes in the resonance frequency. The changes in the resonance frequency indicates the quality and quantity of bone-implant interface. The frequency values are transformed into arbitrary values and it should exceed 56 which indicates the level of bone support and thereby it determines the level of osseointegration.

9. TREATMENT MODALITIES OF PERIIMPLANTIITS

NON- SURGICAL THERAPY:

Mechanical debridement:

Mechanical debridement alone involves the supra and subgingival debridement of the implant surface, the implant neck and the abutment. The main objective is to remove peri-implant biofilm and calculus without altering the implant surface, with the goal of re-establishing a healthy peri-implant mucosa. Different debridement systems have been evaluated, normally in combination with polishing the implant surface and/or the prosthetic components using a rubber cup and a polishing paste or using an abrasive sodium-carbonate air–powder system. Such debridement systems include curettes and ultrasonic devices with polyether-ether ketone-coated tips.[7]

Curettes:

Curettes of different materials have been produced for use specifically to debride implant surfaces. Steel curettes have an external hardness higher than titanium and not indicated for cleaning titanium implants. Titanium-coated curettes have a similar hardness to the titanium surface and thus do not scratch its surface. Carbon-fiber curettes are softer than the implant surface and therefore remove bacterial deposits without damaging the surface, although they break easily. Teflon curettes have similar properties to carbon fiber curettes and they have been proposed for use in combination with air-abrasive systems. Plastic curettes (polytetrafluoroethylene-e) are the most fragile of all curette types and have limited debriding capacity.[7]

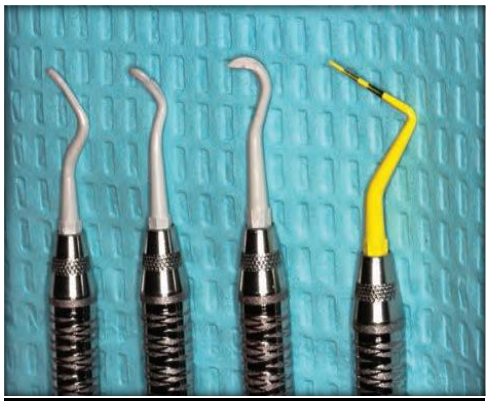

Figure 4: Plastic curettes and plastic Periodontal probe

Ultrasonic devices.

Ultrasonic devices with polyetheretherketone- coated tips have been used to debride the implant surface. The polyether-ether ketonecoated tip is a modified tip made of a high-tech plastic material and has a stainless-steel core. It debrides the implant surface easily and is comfortable for the patient.[7]

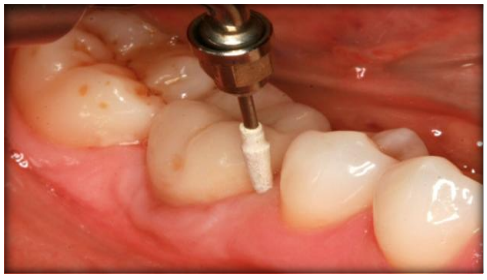

Figure 5: PEEK ultrasonic tip

Air-abrasive systems:

Standard powdered air-abrasive systems are based on the air-spray of sodium bicarbonate. They are used for polishing and for removing tooth stains, but cannot be used for implant instrumentation because they may damage hard and soft tissue as a result of their high abrasiveness. Recently, a powered air abrasive system, based on a low-abrasive amino-

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acid glycine powder, has been demonstrated as an effective method of biofilm removal from the root surface, without damaging hard and soft tissues and it has been recommended for debriding implant surfaces. It uses a specially designed nozzle, consisting of a thin, flexible, plastic tube (length: 1.7 cm; diameter: 0.8 mm at the tip) that is fitted with three orthogonally orientated holes. This specific design is associated with the horizontal exit of the air–powder mixture and reduced pressure, thus preventing the formation of emphysema in the adjacent tissue. The hand-piece (Air-Flows EL-308/A; EMS Electro Medical Systems, Nyon, Sweden) should be guided in a circular motion, from coronal to apical, parallel to the implant surface in a noncontact mode, and the instrumentation time at each aspect (i.e. mesial, distal, vestibular and oral) should be limited to 5 s, as recommended by the manufacturer. It has also been recommended to place the nozzle in the pocket mesially, lingually, distally and buccally, for ca. 15 s in each position. The nozzle should be moved with a circumferential movement around the implant, attempting to cover the entire exposed implant surface.[7]

Vector system:

Another modification to the conventional ultrasonic device is the Vector system in which the horizontal vibration is converted by a resonating ring into a vertical vibration, resulting in a parallel movement of the working tip to the surface.[9]

Lasers:

The use of lasers has been proposed in the treatment of peri-implantitis as a result of their anti-infective, physical and ablation properties. The erbium doped yttrium aluminium garnet laser is the laser that has shown the highest potential for use in the treatment of peri-implantitis as a result of its ability to remove subgingival plaque and calculus efficiently without significantly damaging the implant surface. This laser is used for peri-implantitis treatment with a special hand-piece containing a cone-shaped sapphire tip, which should be used in a parallel and semicircular motion around the circumference of the pocket. The laser should be set with an energy of 100 mJ and a frequency of 10 Hz.[10]

Photodynamic therapy:

It combines the use of a diode laser (with a wavelength of 660 nm and a power density of 100 mW for 10 s in each pocket) with phenothiazine chloride dye (for 3 min), followed by irrigation with 3% hydrogen peroxide.[11]

Antibiotics:

Different protocols using locally or systemically delivered antimicrobials have been evaluated: (i) a single-unit dose of 1 mg of minocycline and 3 mg of poly(glycolide-co-dl-lactide) placed submucosally at each treatment site, at treatment and 30 and 90 days after treatment; (ii) a single dose of 1 mg of minocycline microspheres; (iii) 1 mg of minocycline microspheres at treatment and 180 and 270 days after treatment; (iv) or topical irrigation with a solution containing 8.5% by weight of doxycycline and 37% by weight of poly-DL-lactide dissolved in a biocompatible carrier of N-methyl-2-pyrrolidone. Systemic antibiotics have been also used, but there are no controlled clinical trials evaluating their effect.[12]

10. SURGICAL THERAPY

Moreover, the histopathology of the peri-implant lesions is characterized by the presence of an inflammatory cell infiltrate extending apically in direct contact with the bone crest and leading to loss of osseointegration. As additional objectives of the treatment of periimplant infection, the treatment of peri-implantitis should aim for bone regeneration and the attainment of re-osseointegration. This phenomenon of direct bone-to-implant contact on a previously contaminated implant surface is termed as re-osseointegration.

The rationale of the surgical treatment of peri-implantitis is therefore twofold: to improve implant surface cleanability; and to modify the anatomy of soft and hard peri-implant tissues in order to obtain re-osseointegration.[13]

Decontamination of the implant surface:

When the implant surface is exposed to oral biofilms, it becomes contaminated and, in order to promote healing, decontamination of the surface is mandatory.

Mechanical decontamination:

Mechanical decontamination consists of the physical removal of hard- and soft-tissue deposits on the contaminated exposed implant surface. Instruments for mechanical debridement usually include curettes, ultrasonic devices with special tips and air–powder abrasive systems.[13]

Implantoplasty:

A second, and more aggressive, approach has been proposed, consisting of smoothening of the implant surface (and thus removing the rough surface of the implant), resulting in a polished smooth surface more amenable for oral-

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hygiene practices.[13]This procedure, termed 'implantoplasty', is carried out with burs and stones under copious irrigation because there is an important rise in temperature and an extensive local contamination with titanium.

Chemical decontamination:

The rationale for the use of chemical treatments is to disinfect/decontaminate the implant surface by direct application of appropriate substances. Citric acid, hydrogen peroxide, chlorhexidine and/or saline have been utilized.[13]

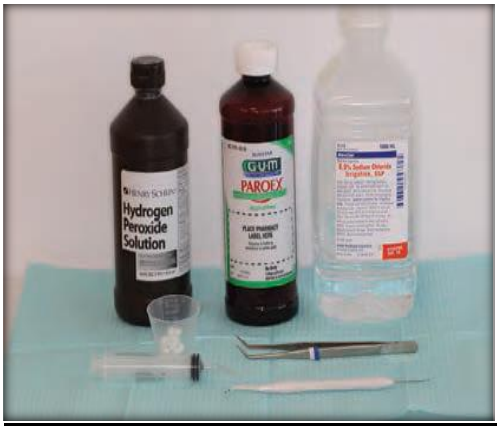

Figure 6: Chemical decontamination

Lasers:

Lasers have also been used to decontaminate the implant surface. Schwarz et al. noticed that erbium lasers yielded significant advantages in terms of bleeding on probing and clinical attachment level; however, no differences were noted when compared with conventional mechanical treatment. No differences between the use of lasers and conventional treatment were also noted with a CO2 laser as adjunct to both resective and reconstructive techniques.[13]

Surgical techniques:

Access flap surgery:

The objective of this flap surgical intervention is to conserve and to maintain all the soft tissues around the affected implant and to focus mainly on the decontamination of the implant surface. Usually, intracrevicular incisions are made around the affected implants and mucoperiostal flaps are raised both buccally and palatally/lingually. Degranulation of the peri-implant inflamed tissues is best accomplished with titanium curettes and implant surface decontamination is performed using one of the methods previously described. Finally, the flaps are repositioned and adequately sutured.

As this technique aims to maintain the position of the soft-tissue margin around the implant neck, this can only be attained when the peri-implant bone loss is shallow.[13,14]

Apically positioned flaps:

This surgical approach has been advocated in order to enhance self-performed oral hygiene and reduce the pockets around the affected implants. Technically, a reverse beveled incision is designed dependent on the probing pocket depth and the width and the thickness of the peri-implant mucosa. Vertical releasing incisions may be needed in order to position the flap apically. Mucoperiostal flaps are raised both buccally and palatally/lingually. The collar of the affected tissues is then removed andthe implant surfaces are thoroughly decontaminated. Often osteoplasty, carefully performed using bone chisels, is needed. Finally, the flaps are sutured in order to leave the previously affected part of the implant exposed to the oral cavity. In order to smoothen the exposed part and to decrease the postsurgical contamination of the implant surface, implantoplasty has been suggested. This technique may be indicated for peri-implantitis with suprabony defects or a one-wall intrabony defect. It is obviously a technique chosen mainly for nonesthetic areas.

Peri-implant regenerative therapy:

Regenerative approaches have two main objectives:

To support the tissue dimensions during the healing process, avoiding recession of the mucosa. To enhance the chance of obtaining re-osseointegration, using reconstructive and regenerative techniques/materials.In this technique, intracrevicular incisions are often performed in order to maintain the total amount of soft tissues. After elevation of buccal and lingual periosteal flaps, degranulation of the defect is performed using titanium instruments. After decontamination of the implant surface, a graft is placed around the implant, filling the intrabony component of the defect. Grafting may be performed with either autologous bone or bone substitutes. The graft may be covered with a

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resorbable or a nonresorbable membrane. Finally, the flaps are coronally positioned and sutured in order to determine healing with either a nonsubmerged or a submerged approach.[13,14]

Peri-implant resective therapy:

The type of osseous defect should be identified before deciding on the treatment modality. Apically-displaced flap techniques and osseous resective therapy are used to correct horizontal bone loss and moderate vertical (< 3 mm) bone defects and to reduce overall pocket depth. Full-thickness or split-thickness flap management is utilized to access the surgical area. To improve the follow-up maintenance of an implant site, surfaces which are smooth and clean coronal to the bone level are preferred. Therefore, implant surfaces with threads, roughened topography, or hydroxyapatite surfaces are modified with high-speed finishing burs and polished

to produce a smooth, continuous titanium surface (implantoplasty). The implantoplasty is performed before any osseous resective therapy is initiated and with profuse irrigation.[13,14]

11. TREATMENT PROTOCOLS IN PERIIMPLANTITIS

Cumulative Interceptive Supportive Therapy (CIST)[6]:

This system is cumulative in nature and includes four steps which should not be used as single procedures, but rather as a sequence of therapeutic procedures with increasing antibacterial potential depending on the severity and extent of the lesion.

The major clinical parameters to be used have been discussed above and include the assessment of the following:

1. presence or absence of dental plaque;
2. presence or absence of bleeding on gentle probing (BOP);
3. presence or absence of suppuration;
4. periimplant probing depth; and
5. evidence of radiographic bone loss.

This protocol is divided into four stages of treatment sequence as shown in the flowchart.

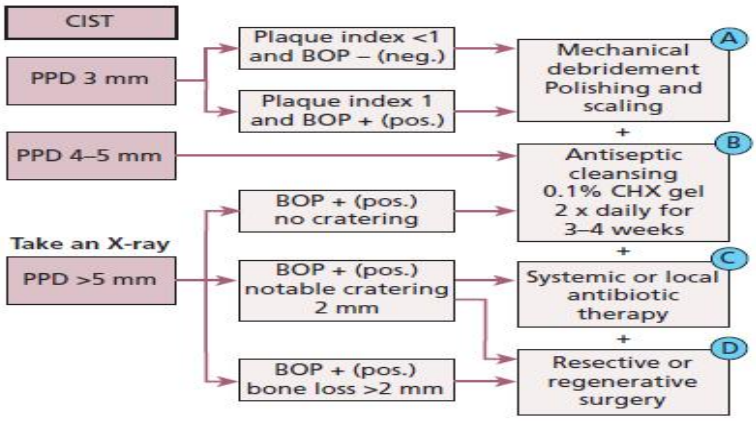

Figure 7: CIST Protocol

In 2004 it was modified and called AKUT concept by Lang et al. The basis of this recall of the implanted patient and repeated assessment of plaque, bleeding, suppuration, pockets, and radiological evidence of bone loss.

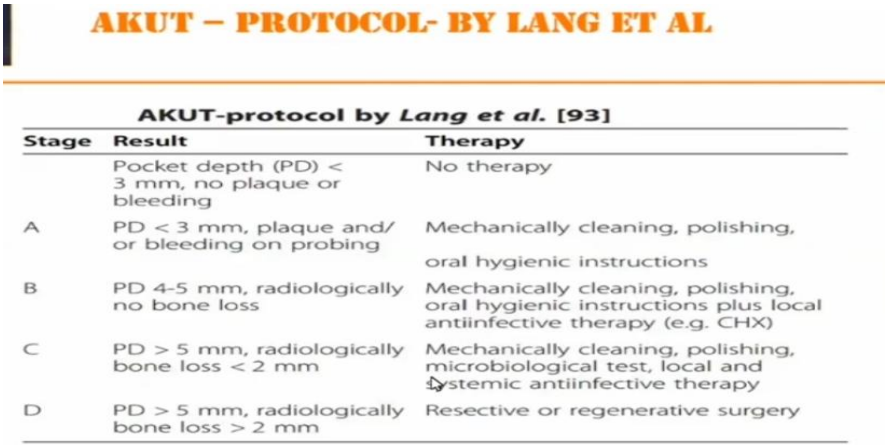

Figure 8: AKUT Protocol

12. CONCLUSION

Although implants offer a highly predictable treatment option for the replacement of missing teeth, there are variety of implant complications such as surgical, biological, mechanical, prosthetic and esthetic. Careful diagnosis and treatment planning is essential in order to prevent the complications in the near future. Thus, successful implant treatment requires a team of clinicians and technicians dedicated to excellence in surgical and prosthetic aspects.

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