

Biologic Complications of Dental Implant: An Update

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Abstract

With the wide use of implants to replace missing teeth, the complications of implant are also being encountered more frequently. Biologic complications are one of the complications in implant. This includes inflammation, recession, dehiscence, periimplantitis and implant failure. The dentist should be aware of these conditions and their etiology and risk factors. Relevant clinical and radiographic findings must be made for the diagnosis of the condition. The treatments should be addressed promptly. The patient must be reinforced on proper implant hygiene maintenance for the success of the implant.

Keywords: complication, implant, peri-implant mucositis, periimplantitis.

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INTRODUCTION

The placement of implants to replace single or multiple missing teeth is one of the most reliable treatment options.¹ With the increase in the number of the implant patients, it is likely that a greater number of complications will be reported. The complications seen with implant placement are surgical complications, technical complications, biologic complications and esthetic and phonetic complications.²

A biologic failure can be defined as the inadequacy of the host to establish or to maintain osseointegration. The inability to establish osseointegration can be regarded as an early failure, whereas the inability to maintain the achieved osseointegration, under functional conditions, may be considered a late loss.³

A biologic complication suggests an increased risk for failure. It may be of temporary significance or may be treatable. It is important to distinguish between failed implants, failing implants, and biologic. Clinically, implant mobility is a failed implant as it shows lack of osseointegration. An implant that shows progressive bone loss but is clinically stable is a "failing" implant.³

Types of biologic complications:

Biologic complications around implant can be categorized as:²

- 1) Inflammation and Proliferation
- 2) Dehiscence and Recession:
- 3) Periimplantitis
- 4) Implant failure

1. Inflammation and Proliferation

Inflammatory sequela is an inevitable response to implant surgery. After implant surgery, the patient should be closely monitored for swelling, fever, bleeding, redness, exudate, necrotic tissue and fistulae. Bleeding, infections, swelling and adverse tissue changes occurs in about 50% of patients, but only less than 1% are severe in nature. These are more common in the anterior segments.⁴ Biologic complications include decubitus ulcers of the mucoperiosteum covering a healing implant, peri-implant mucositis⁵, hyperplastic mucositis⁶ and fistulae.³

The American Academy of Periodontology has defined peri-implant mucositis as a disease that includes

inflammation of the soft tissues surrounding a dental implant, without additional bone loss after the initial bone remodeling that may occur during healing following the surgical placement of the implant.⁷ The estimated prevalence of peri-implant mucositis is 30.7% at the implant level and 63.4% at the subject level.⁸ The probability of increased peri-implant mucositis increases by an odds ratio of 3.8 in case of smokers.⁹

Clinically, peri-implant mucositis sites showed substantially increased BOP (67%).¹⁰ Additional signs may include erythema, swelling, and suppuration. Healthy peri-implant mucosa turns into peri-implant mucositis with the accumulation of bacterial biofilms around the dental implants. Further progression leads to periimplantitis. Diagnosis of peri-implant mucositis requires presence of bleeding and/or suppuration on gentle probing with or without increased probing depth compared to previous examinations and absence of bone loss beyond crestal bone level changes resulting from initial bone remodelling.¹¹

To remove inflammation and proliferation, it is essential to identify and remove or control the etiological factor. Loose abutment connection, retained cement should be identified and treated properly. For the treatment of periimplant mucositis, nonsurgical therapy including supragingival and subgingival debridement with or without adjuncts should be incorporated. It has been observed that chlorhexidine, the administration of azithromycin through the systemic route, and glycine powder air polishing are not effective in the treatment of periimplant mucositis over the long term. The use of toothpaste with 0.3% triclosan was found to be the only effective treatment.¹²

2. Dehiscence and Recession:

On average, the buccal plate is less than a millimeter in thickness.¹³ After extraction, there is further inevitable loss of the alveolar bone. The buccal ridge decreases by ± 2 mm in height.¹⁴ In cases of immediate implant placement with preserved buccal lamella, a recession of the marginal mucosa of an average 0.5 mm has been reported.¹⁵ This should be anticipated by the clinician so that complications can be prevented or managed. The role of immediate implant placement and implant loading protocols show the highest risk of complication¹³ whereas others show little or no statistical difference between immediate and early placement protocols in the aesthetic zone.¹⁶

The position of the gingiva is determined by the position of the underlying bone and the patient's soft tissue biotype. A thin tissue biotype may favor apical displacement of the soft tissue margin.¹⁷ When an implant is affected by peri-implantitis, there is peri-implant bone loss which may eventually result in soft tissue resorption. A vicious cycle may occur as the implant-abutment interface harbors plaque resulting in inflammation, bone loss and gingival recession.¹⁸

In terms of gingival thickness, an adequate width of keratinized gingiva is essential for optimal gingival health.¹⁹ Bone loss and recession give darker appearance to the gingival tissues in the presence of the underlying metal abutment. If the recession progresses, unsightly metal exposure above the gingival margin may occur.¹⁸ The midfacial gingival recession is associated with a buccal shoulder position of the implant (odds ratio, 17.2).²⁰ Mesial and distal papilla recession is associated with ridge recontouring and with bone loss around a periodontally compromised tooth.²⁰

Clinical and radiographic assessment is necessary in cases of wound dehiscence. During the submerged period in the case of a 2-stage procedure, signs such as swelling, fistulae, or persisting pain require immediate attention. It is important to distinguish if the involvement is in the soft tissue compartment or if the hard tissues have also been involved. Soft tissue dehiscence may occur due to complications resulting from residual suture material, poorly seated cover screws, premature wearing of the denture or inadequate relief of the denture on protruding implants.³

Improper implant positioning is another factor for gingival recession. Implants placed too far buccally have shown to cause recession three times as high.¹⁵ Thus, presurgical detailed planning should be emphasized. Soft tissue and bone augmentation should be preplanned and modifications of prostheses should be considered.

3. Periimplantitis and bone loss

Peri-implantitis is a plaque-associated pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone.²¹

Diagnosis of peri-implantitis requires:

- Presence of bleeding and/or suppuration on gentle probing.
- Increased probing depth compared to previous examinations.
- Presence of bone loss beyond crestal bone level changes resulting from initial bone remodeling.

In the absence of previous examination data diagnosis of peri-implantitis can be based on the combination of:

- Presence of bleeding and/or suppuration on gentle probing
- Probing depths of ≥ 6 mm.
- Bone levels ≥ 3 mm apical of the most coronal portion of the intraosseous part of the implant.

The weighted mean prevalence of peri-implant mucositis and peri-implantitis was found to 43% and 22% in Europe and South and North America, respectively.²² According to

Atieh et al.⁸, the estimated prevalence of peri-implantitis was 9.6% at the implant level and 18.8% at the subject level.

The risk factors for peri implantitis are related to the patient, the prosthesis, the clinician, the implant design and the implant site.²³ Patient related factors are disease control, plaque control, maintenance, smoking and diabetes. The prosthesis related factors are type of restoration, emergence profile and occlusal contacts. The clinician related factor is implant positioning. The implant has to be placed three dimensionally, prosthetically driven and guided. The implant site related factors are tissue phenotype, width of keratinized mucosa and infection. The implant design related factors are collar of the implant, implant surface and the type of implant.²³

Besides these, other factors that need further investigation are occlusal overload²⁴, genetic factors²⁵, rheumatoid arthritis with concomitant connective tissue disease²⁶, increased time of loading²⁷, and alcohol consumption.²⁸

As in periodontitis, the biofilm triggers an inflammatory response in the periimplant mucosa. When cases of peri-implantitis were compared to cases of periodontitis, it was observed that the tissue destruction is more severe in association with peri-implantitis.^{29,30} Unlike natural teeth, dental implants do not have cementum, Sharpey's fibers or periodontal ligament. There is direct contact between bone and implant surface. Thus, infection can progress without impediments from soft to hard tissue. When peri-implant mucositis progresses, the inflammatory process reaches the crest of alveolar bone and osteoclastic bone resorption begins.

It has also been suggested corrosion/tribocorrosion, the presence of titanium particles, and biological complications have an association.³¹ This corrosion/tribocorrosion may cause immune modulation directly or may cause microbiome perturbation indirectly leading to peri-implant inflammation and implant failure.³¹ Thus, further investigation is required to clear the role of ion/particle release in the pathogenesis of peri-implant diseases.

The treatment of periimplantitis includes local debridement, implant surface decontamination, anti-infective therapy, surgical technique and explantation.³² The proper guideline for the treatment of periimplantitis has been given by the CIST "cumulative interceptive supportive

therapy" protocol.³³ This includes a combination of different options such as mechanical cleaning, polishing, oral hygienic instructions, local anti-infective therapy (e.g. CHX), systemic antiinfective therapy, Resective or regenerative surgery based on the pocket depth, plaque, bleeding on probing and radiographic features.

4. Implant loss or failure

Implant failure is defined as the total failure of the implant to fulfill its purpose (functional, esthetic or phonetic) because of mechanical or biological reasons.³⁴ Implant failures can be diagnosed by clinical signs of early infection, pain or sensitivity and clinical discernible mobility. Occasionally, the patient presents with implant mobility in absence of distinct radiographic bone changes.³⁵ In all cases, mobility is the cardinal sign of implant failure.³⁶ Different types of mobility seen are rotation mobility, lateral or horizontal mobility and axial or vertical mobility.⁸ A dull sound on percussion suggest soft tissue encapsulation and a clear crystallization sound suggests successful osseointegration.³⁶

Oral implant failure can be classified as biological, mechanical, iatrogenic and inadequate patient adaptation.³⁶ Biological failures may be early or primary (before loading) and late and secondary (after loading). Early failure is due to failure to establish osseointegration and late failure is due to maintain the achieved osseointegration. Mechanical failures may be due to fracture of implants, connecting screws, bridge frameworks, coating etc. Iatrogenic failures may be due to nerve damages, wrong alignment of implants, etc. Inadequate patient adaptation may be due to phonetical, esthetical, psychological problems, etc. The failed implant should be explanted with meticulous removal of granulation tissue and the use of wider implants with improved surfaces should be used.³⁷ It should be kept in mind that the survival rates are lower than the rates reported for first time single implant placement.³⁷

CONCLUSION

Biologic complications in implants demand rapid and utmost care. Removal of the etiology is important to achieve success. Proper treatment planning should be done along with the anticipation of the possible outcomes to avoid complications. The clinician must be acquainted with the treatment outcomes if such complications are encountered.

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