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Review Article

Failures in Endosseous Dental Implants – A Literature review

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Abstract

Endosseous dental implants have become commonly used in clinical situations. Despite many advances in materials, techniques and implant design, the potential for clinical failure is a significant concern for both dentist and patient. A variety of factors can precipitate failure of an implant, including occlusal overloading, preoperative or post-operative infection and placement of implant in bone with inadequate quality and quantity. This study reviews the literature concerning the success or failure of dental implants and provides the general information to dentist to decide whether to recommend dental implant to patient.

Keywords: Endosseous implants, peri implantitis, osseointegration, periapical radiolucency, implant fracture

1. Introduction

In 1969, Branemark et al published landmark research documenting the successful osseointegration of Endosseous titanium implants. Since then surgical dental implants have had a profound influence on the practice of dentistry. Implants have become the treatment of choice in many, if not most, situations when missing teeth require replacement.

Implant complications can be due to a number of causes including prosthesis instability, implant mobility, occlusal trauma, fractured components, pain, inflammation, infection and neuropathy. Peri-implantitis is considered as one of the important postoperative soft tissue complications associated with implant placement.

An attempt has been made to review various complications related to implant therapy, with a particular emphasis on peri-implant diseases, including its diagnosis and management. The long-term goal of implant therapy is to prevent or to arrest the progression of disease and to achieve a maintainable implant site.[1]

2. Review

Complications following implant placement can be due to several etiology and can be avoided by careful attention to diagnosis, treatment planning, good surgical and prosthodontic training and experience following well-established protocols. However, the authors failed to demonstrate any significant relationship between smoking habits and implant loss.[2]

Etiology of complications

- Diagnosis and treatment planning: Inadequate quality and quantity of bone
- Intra operative: Over heating
- Prosthodontic: Biomechanical leverage, torsion, occlusal overload, poor oral hygiene
- Post-operative: Postoperative swelling, bruising, and discomfort, wound dehiscence, periimplant mucositis, peri implantitis.
- Failure of implant has a multifactorial dimension. Often many factors come together to cause the ultimate failure of the implant.

2.1 Surgical sequelae

Common surgical / postoperative complications, even with straight forward single implant surgery can be minimized with:

- Gentle surgical manipulation of the hard and soft tissues
- Use sterile physiologic saline irrigation during surgery
- Avoidance of over-reflection of flaps
- Preoperative and postoperative analgesics
- Recommendations to use icepacks to reduce swelling
- Intravenous steroids in more major cases

- Pressure applied to the wound postoperatively to control hemostasis and avoid hematoma formation

Complications:

Soft Tissue: Soft-tissue overgrowth, Soft-tissue deficiencies, Persistent inflammation and infection.

Hard tissue: Continuing bone loss (peri-implant diseases)

2.2 Osteotomy procedure

The amount of heat produced in the bone directly related to the amount of bone removed by each drill. ***Eriksson*** [3] reported bone cell death when a temperature of 40^0C was applied for 7 minutes or when a temperature of 47^OC was applied for 1 minute.

Overheating during osteotomy decreased by,

- Abundant external or internal irrigation
- Cooled saline irrigation
- Intermittent pressure on drill
- Pause every 3 to 5 seconds, keep irrigating
- New drill designs, flutes, geometry
- Incremental drill sequence
- Final osteotomy drill should use greater width, greater height and slower speed used

Heat generated during an implant osteotomy [4] related to,

- Pressure and temperature of irrigation
- Amount of bone being prepared
- Drill sharpness and design
- Time of preparation
- Depth of osteotomy
- Pressure of the drill
- Drill speed
- Variation in cortical thickness

2.3 Wound dehiscence

The soft tissue wound may break down in the first following implant installation (e.g. particularly in the severely atrophic mandible). This used to concern clinicians when it led to implant head exposure in submerged implant systems, but because it has now been shown that these implants work in non-submerged protocols it is not thought to be significant *(Figure-1)*. If an implant head becomes exposed in a case that was planned to be submerged, it is important to keep the area clean with antiseptic rinses (such as 0.2 / 0.12% Chlorhexidine).

2.4 Early implant failure

Most early surgical failures of Osseointegration are due to poor surgical technique or placement of implants into bone of very poor density, or in areas with a lack of bone volume that has only allowed utilization of very short implants (≤ 7 mm). Failure of osseointegration may not be obvious until the surgeon carries out abutment connection surgery or when the prosthodontist tries to load the implant. It is sometimes difficult for the clinician to assess implant stability and occasionally a loose abutment may be misinterpreted as a failed implant. In such cases, the suspect abutment should be tightened and stability reassessed.

Damage to the bone resulting in necrosis, pain and subsequent infection is more likely where hard bone has been prepared with inadequate cooling and/or blunt drills *(Figure- 2)*. This is more likely at deep preparation sites (>13 mm). Such deep preparations are not usually necessary in good-quality bone and should be avoided. The resultant pain can be severe and an infection that is likely to be deep seated can track through the soft tissue. In the anterior region of the mandible the infection may track to the external skin surface, producing a disfiguring sinus tract, fibrosis and scarring *(Figure-3)*.

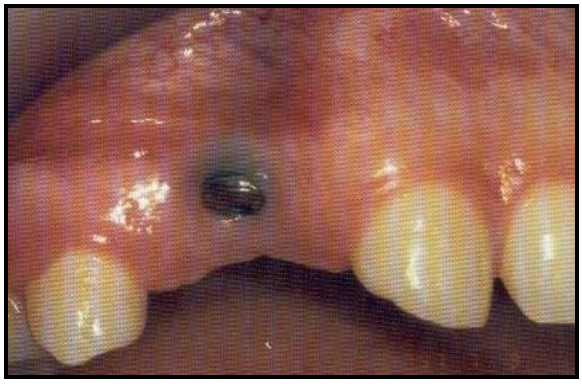

Fig. 1: Spontaneous exposure of previously submerged implant

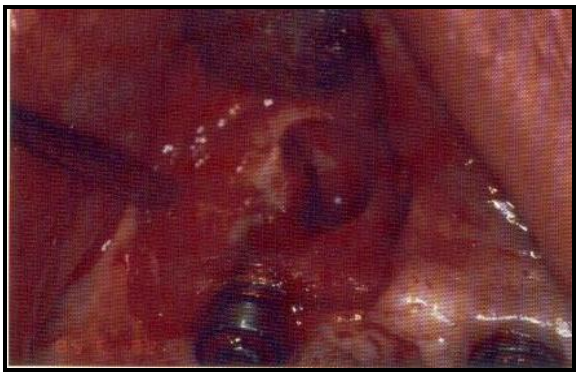

Fig. 2: Surgical exposure of an apical lesion of bone loss, probably caused by inadequate cooling of the end of the drill during preparation of the site

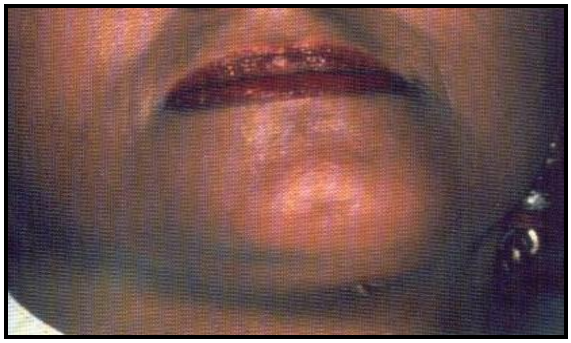

Fig. 3: A sinus tract appearing on the skin at the lower border of the mandible

3. Soft tissue evaluations and problems

3.1 Soft-tissue overgrowth

Soft-tissue proliferation may occur around bridges with poorly designed embrasures and under supporting bars of implant dentures. It may require simple excision if there is adequate attached keratinized tissue apical to it, or an inverse bevel resection to thin out the excess tissue but preserve the keratinized tissue to produce a zone of attached tissue around the abutment *(Figure-4).*

3.2 Soft-tissue deficiencies

The transmucosal part of the implant restoration may emerge through non-keratinized mucosa, particularly in situations where there has been severe loss of bone, e.g. edentulous jaws. Non-keratinized mucosa looks inflamed and more delicate than keratinized tissues and may lack attachment to the underlying bone. This can give rise to soreness and compromised plaque control, particularly in implant denture cases. Persistent soreness and inflammation can be overcome by grafting keratinized mucosa to the site in a procedure that is the same as free gingival grafting using donor tissue from the palate. In other situations, it may give rise to compromised esthetics.

3.3 Persistent inflammation

Persistent inflammation or discomfort may arise due to poor implant positioning. It may require recontouring of the soft tissues to allow patient cleaning, and this may reveal the less than satisfactory esthetics produced by poor planning and execution of treatment *(Figure-5).* In other more severe cases, the only remedy may be to remove the implants or bury them permanently beneath the mucosa *(Figure-6).*

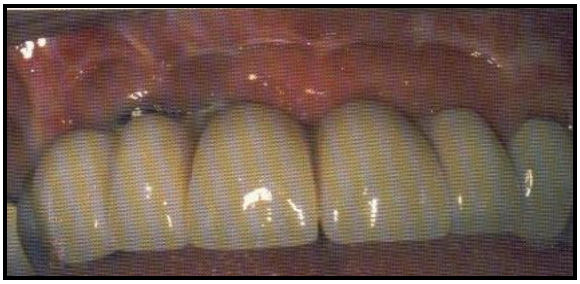

Fig. 4: Soft tissue overgrowth exposing the titanium abutments

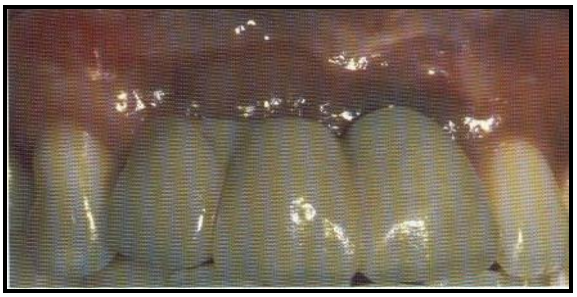

Fig. 5: Persistent inflammation of soft tissues around dental implants

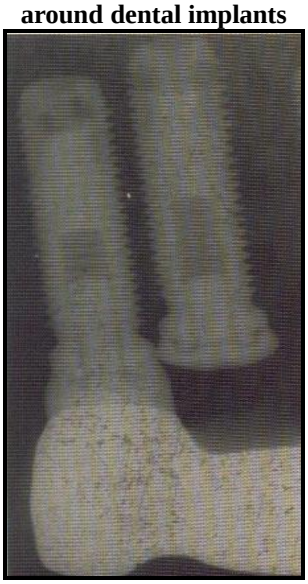

Fig. 6: A radiograph of the bridge shows that there is buried (unused/unconnected) implant beneath the right central incisor unit

4. Peri-implant diseases

A peri-implant disease is a descriptive term used to describe a non-specific inflammatory reaction in the host tissues. "Peri-implantitis" should be distinguished from "peri-implant mucositis" in that the former is defined as, "an inflammatory reaction with loss of supporting bone in the tissues surrounding a functioning implant" *(1st European Workshop on Periodontology)* [1] while the latter involves a reversible inflammation localized to the soft tissues only. Peri-implantitis may display some or all of the following symptoms; bleeding on probing, increased probing pocket depth, mobility, suppuration and pain.

4.1 Classification

Spiekermann (1995) proposed a classification of peri-implantitis depending on the morphology of the peri-implant bone loss **(Table-1)**.

Table-1: Classification of peri-implantitis [5]

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 defects
Class IV	Advanced bone loss with broad, circumferential vertical defects, as well as loss of oral and/or vestibular bony wall

Successful and failing implants showed marked differences in the composition of the associated microflora. Successful implants were

sparsely colonized by gram-positive cocci, whereas failing implants yielded large amounts of gram-negative anaerobic bacteria. Longitudinal studies demonstrated that the normal peri-implant micro flora was established shortly after implant placement. Data from several studies suggested that periodontal pathogens may be transmitted from residual teeth to implants.[6] [7]

4.2 Influence of oral hygiene on long-term success

More bone resorption was noted around fixtures in edentulous patients with poor oral hygiene than in subjects with good hygiene *(Lindquist et al. 1988)*.

Clinical features & frequency

The following signs and symptoms [8] are typical for peri-implantitis lesions:

- There is radiological evidence for vertical destruction of the crestal bone. The defect usually assumes the shape of a saucer around the implant *(Figure-7)*, while the bottom part of the implant retains perfect osseointegration. In some instances wedge-shaped defects develop along the implant. Whether specific patterns of marginal bone loss indicate a specific underlying cause is not known. Bone destruction may proceed without any notable signs of implant mobility until osseointegration is completely lost. Implant mobility [9], or continuous peri-implant radiolucency, indicates the final stage of peri-implant disease, characterized by total loss of the direct bone-to-implant interface. *(Figure-8)*
- Vertical bone destruction is associated with the formation of a peri-implant pocket.
- There is bleeding after gentle probing with a blunt instrument and there may be suppuration from the pocket.
- Tissues may or may not be swollen. Hyperplasia is frequently seen if implant supported overdenture are located in an area with non-keratinized mucosa.
- Pain is not a typical feature of peri-implantitis.

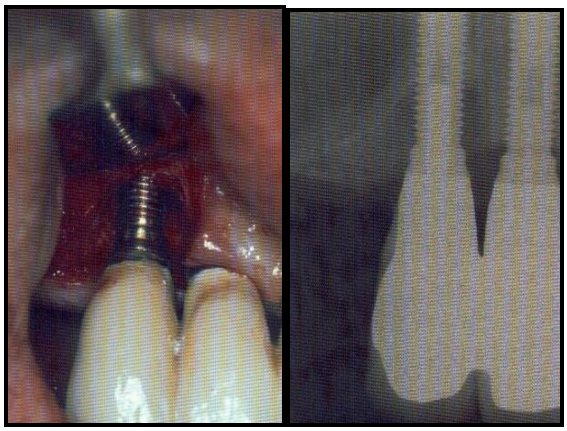

Fig. 7: Peri-implantitis with typical circumferential bone defect (saucer formation)

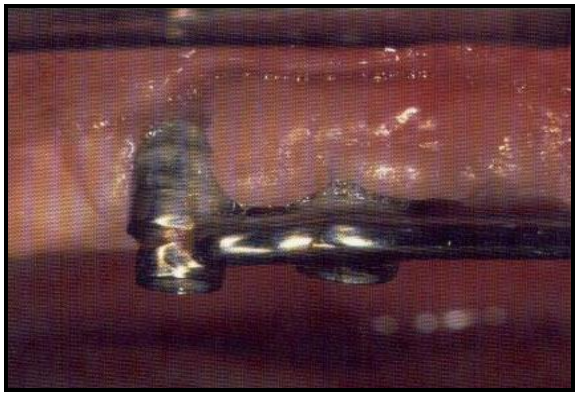

Fig. 8: Loss of osseointegration of distal implant due to high loading

4.3 Preventing peri implantitis

- Massive bacterial colonization of implant surfaces should be avoided to prevent inflammation and infection of the peri-implant tissues and to minimize the risk for implant failure.
- Removal of bacterial deposits is a crucial step in the therapy of peri-implant infections.
- Inducing a change in the local ecology around the implants to impede massive multiplication of potential pathogens assumes high priority to prevent disease recurrence.

Table-2: Incidence and Failure Rates in Previous Studies

Authors	Number of years	Incidence/ Failures	Cause
Smedberg et al 1993	2yr follow up	6 %	Peri-implantitis
Weber et al 2006	2yr follow up	11%	Peri-implantitis
Richter et al 1998	5yr follow up	4-15%	Peri-implantitis
Fransson 2005	5-20yrs follow up	27.8%	Peri-implantitis
Ferreira 2006	4yr follow up	8.9%	Peri-implantitis
Maximo et al 2009	3-5 yrs	12.4%	Peri-implantitis
Koldsland 2010	Mean follow up 8.4 yrs	47.1%	Peri-implantitis
Sahm et al 2011	5 yrs	1.26%	Peri-implantitis

It has been speculated that implants with titanium plasma-sprayed surfaces are more susceptible to implant failure due to peri-implantitis than implants with machined surfaces. A study indicated that implants with rough surfaces had a tendency to fail more often than implants due to peri-implantitis with machined surfaces.[10] However,

other studies failed to demonstrate any significant differences among the different implants, indicating that all implants were susceptible to peri-implantitis.[11][12]

More recently, a study using ligature-induced peri-implantitis model in animals reported that after removal of ligatures the progression of bone loss was greater at SLA than at polished sites and thus it was concluded that the progression of peri-implantitis, if left untreated, is more pronounced at implants with a moderately rough surface than at implants with polished surface.[13] Furthermore, in a prospective, multicenter study conducted over a period of 8 years, out of 2359 implants, only 5 implants had to be surgically removed due to a recurrent peri-implant infection.[14]

Table-3: Main diagnostic differences between peri-implant mucositis and peri-implantitis Adapted from *Chen & Derby (2003)* [15]

Clinical parameters	Peri-implant mucositis	Peri-implantitis
Increased probing depth	+/-	+
BOP	+	+
Suppuration	+/-	+
Mobility	-	+/-
Radiographic bone loss	-	+

4.4 Retrograde Peri-implantitis

The term retrograde peri-implantitis has just recently been introduced through several case reports. Synonyms are “apical periimplantitis” or “implant periapical lesion (IPL)”. It is defined as a clinically symptomatic periapical lesion (diagnosed as radiolucency) that develops shortly after implant insertion while the coronal portion of the implant achieves a normal bone to implant interface.

4.5 Incidence

Information on incidence of retrograde peri-implantitis is scarce. A study observed a higher incidence of IPL for implants with rough surfaces when compared to machined implants.[16]

4.6 Screw and abutment connections

Repeated chewing cycles may produce screw loosening, either at the bridge screw level or the abutment level. *(Figure 9 & 10*) Most single tooth restorations have cemented crowns with no direct access to the screw tightening. Therefore, should this be required, removal of the crown is necessary. Abutment loosening and development of a gap between abutment and implant will manifest as a loose prosthesis *(Figure-11).*

Screw loosening is often an important sign of overload due to:

- Poorly fitting prosthesis/non-passive fit [17]
- Poor design, e.g. overextension of a cantilever
- Poor implant/crown ratio [18]
- Inadequate attention to occlusal contacts
- Too few implants/teeth to establish an adequate occlusal table
- Para functional activity [19]

These factors require identification, correction and proper management to avoid this complication. Failure to deal with these problems, particularly in patients who exhibit Para functional activities, may predispose to screw fracture, which manifests as a loose prosthesis and may be detected radiographically.

5. Implant fractures

Fortunately, fracture of an implant is rare. It is more likely to occur with:

- Narrow diameter implants, particularly where the wall thickness is thin
- Excessive load [20]
- Marginal bone loss that has progressed to the level of an inherent weakness of the implant, often the level where wall thickness is thin at the apical level of the abutment screw.

Implant fracture is rarely retrievable, and requires both burying the fractured component beneath the mucosa or its removal *(Figure-12).*The latter can be difficult and traumatic, usually requiring surgical trephining that may leave a considerable defect in the jaw bone.

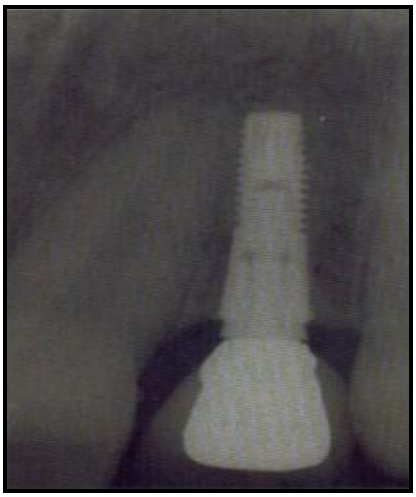

Fig. 9: A radiograph and clinical photograph showing failure to seat the cemented crown fully

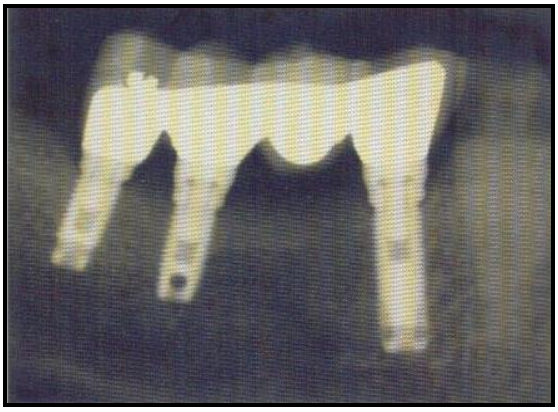

Fig. 10: Radiograph of a Posterior Mandibular Bridge. The Loose Bridge Screw in the Distal Abutment Is Visible

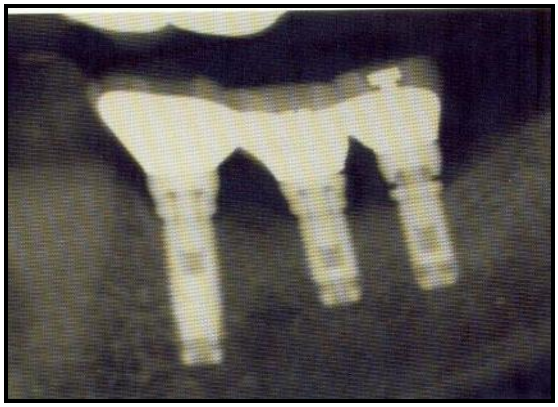

Fig. 11: A radiograph of a posterior mandible bridge showing failure to seat the distal abutment

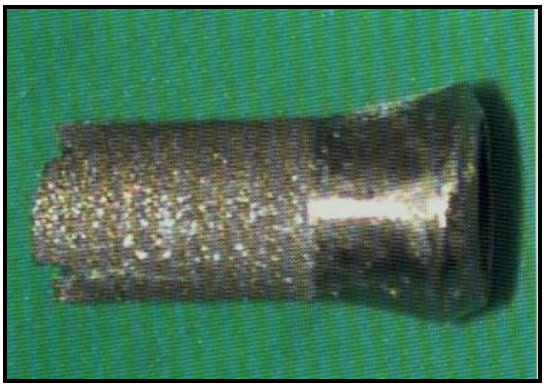

Fig. 12: A fractured implant

6. Summary and Conclusion

Since the introduction of the concept of osseointegration, the success of dental implants has increased dramatically because of better understanding of bone response and the improvement in bone loading concept. Failures can be identified during recall visit and an early intervention is always possible if regular check-up are undertaken. One needs to identify the cause not just to treat the present condition but also as a learning experience for future treatments. Proper data collection, patient feedback, and accurate diagnostic tool will help point out the reason for failure.

As well said, it is not how much success we obtain, but how best we tackle complex situations and failures, that determine the skill of a clinician. No, doubt, failures are stepping stones to success but not until their etiologies are established and their occurrence is prevented.

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