FAILURE IN OSSEOINTEGRATION – A REVIEW

Suman Yadav*1, Mayank Agrawal2, Anubhav Sharma3, Tarique Anwar4, Mallika Sethi5 and Himanshu Thukral6

1MDS, Oral and Maxillofacial Surgeon, Azamgarh, UP.
2MDS, Orthodontics and Dentofacial Orthopedics, Private Practitioner and Consultant, Hathras, U.P.
3Senior Lecturer, Department of Public Health Dentistry, I.T.S Dental College, Muradnagar, Ghaziabad, U.P.
4MDS3 Year, Oral and Maxillofacial Surgery, Shree Bankey Bihari Dental College, Ghaziabad.
5Associate Professor, Department of Periodontics and Oral Implantology, I.T.S Dental College, Muradnagar, Ghaziabad, U.P.
6Oral and Maxillofacial Surgeon, CEO Sarita Dental Care, Delhi.

ABSTRACT

Oral implantology (implant dentistry) is the science and discipline concerned with the diagnosis, design, insertion, restoration, and/or management of alloplastic or autogenous oral structure to restore the loss of contour, comfort, function, esthetic, speech, and/or health of the partially or completely edentulous patient. Osseointegration, a term coined by Branemark and co-workers in early 1960s, represents a direct connection between bone and implant without interposed soft tissue layers. The aim of the present review is to discuss various factors responsible for loss of oral implants. The factors contributing to failure of osseointegration have been identified as medical status of the patient, smoking, bone quality, bone grafting, irradiation, bacterial contamination, lack of preoperative antibiotics, degree of surgical trauma, and operator experience. Furthermore, it appears that implant surface properties, roughness and premature loading influence the failure pattern.

INTRODUCTION

Dental implants have been a successful treatment alternative for restoring missing teeth. The concept of failure beyond the loss of integration has included esthetic, functional and
phonetic reasons. With high patient expectations successful implant integration does not necessarily result in a satisfied patient. A better understanding of the factors associated with implant failures will facilitate clinical decision making and may enhance implant success.[1]

Implant failure is routinely categorized as a surgical complication and it is frequently argued that implant failure before functional loading has minimal impact on prosthodontic outcomes because the loss of one or more implants can be compensated for either by the placement of or by modification of the treatment plan and/or the prosthesis design.[1]

In studies with follow up of more than 5 years, delayed implant loss (loss of osseointegration) becomes nearly as frequent as early implant loss. Implant failure either late or early, directly impacts the prosthodontic phase of treatment by altering the type of prosthesis planned or by necessitating modification or replacement of an existing prosthesis.[2]

Early implant failures are the result of events that may jeopardize or prevent osseointegration from occurring and include among others:
- Improper preparation of the recipient site which results in undue hard tissue damage such as necrosis of the bone.
- Bacterial contamination and extensive inflammation of the wound that may delay healing of the soft and hard tissues.
- Improper mechanical stability of the implant following its insertion.
- Premature loading of the implant.

Late failures occur in situations during which osseointegration of a previously stable and property functioning implant is lost. It was suggested (Proceedings of the 3rd European Workshop on Periodontology; Flemig & Renvert, 1999) that such late failures are often the result of excessive load and/or infection.[2]

The success of dental implants is highly dependent on integration between the implant and intraoral hard/soft tissue. Initial breakdown of the implant-tissue interface generally begins at the crestal region in successfully osseointegrated endosteal implants, regardless of surgical approaches (submerged or non-submerged).[3] Early crestal bone loss is often observed after the first year of function, followed by minimal bone loss (0.2mm) annually thereafter. Six plausible etiologic factors are hypothesized, including surgical trauma, micro gap, biologic width, implant crest module, occlusal overload, infection and peri-implantitis.[4]
Crestal bone loss to or beyond the first thread of titanium screw implants, characterized by “saucerization”, is often observed radiographically around certain implant types. Studies have shown that submerged titanium implants had 0.9mm to 1.6mm marginal bone loss from the first thread by the end of first year in function, while only 0.05mm to 0.13 mm bone loss occurred after the first year.\cite{5}

1. **SURGICAL TRAUMA**

Surgical Trauma has been regarded as one of the most commonly suspected etiologies proposed for early implant failure. Implants which fail due to surgical trauma are often surrounded by fibrous connective tissues or have an apical extension of the junctional epithelium. Heat generated at the time of drilling, elevation of the periosteal flap, and excessive pressure at the crestal region during implant placement may contribute to implant bone loss during the healing period.\cite{6}

2. **MICRO-GAP**

In implant dentistry, there are two basic approaches to place endosseous implants, including submerged (two stage) and non submerged (one stage) implants. In most two stage implant systems, after the abutment is connected a micro gap exists between the implant and abutment at or below the alveolar crest. In non submerged implant designs, the implant itself extends above the alveolar crest level therefore; such a micro gap does not exist at the level of the bone.\cite{7}

Implant countersinking below the bone crest was recommended in the Branemark surgical protocol to minimize the risk of implant interface movement during bone remodeling, and to prevent implant exposure during healing. Implant countersinking is also used to accommodate the wider implant platform in Branemark implants or its clones and to enhance emergence profile for implant prostheses at the expenses of the crestal bone. The countersinking performed for the above purposes places the abutment- implant micro gap below the crestal bone.\cite{8}

Even though a micro gap does not exist in non-submerged implants, crestal bone loss during the first year of function in non-submerged implants has been reported, being equivalent or slightly less than submerged implants. However, stable alveolar bone crest levels from 1 year up to 8 years after implant placement were reported in non submerged implants.\cite{9}
3. BIOLOGIC WIDTH (BIOLOGIC SEAL)

In natural teeth, the dentogingival junction consists of 3 components: the gingival sulcus, the epithelial attachment, and the connective tissue attachment. The dimensions of the dentogingival junction were studied in human skulls by Gargiulo et al. and Vacek et al. Gargiulo et al. reported that the average value of sulcus depth was 0.69 mm, and the average values for the epithelial attachment and connective tissue attachment were 0.97 mm and 1.07 mm, respectively. The biologic width included the latter two, the epithelial attachment and connective tissue attachment, which was 2.04 mm.\(^{[10]}\)

Likewise, around dental implants, the epithelial attachment (or zone) and connective tissue attachment (or zone) exist comprising the biologic seal around dental implants that acts as a barrier against bacterial invasion and food debris ingress into the implant-tissue interface. The epithelial attachment in both implant and natural tooth is composed of hemidesmosome and basal lamina, whereas collagen fiber direction in the connective tissue attachment is different, being parallel to implant surfaces and perpendicular to natural teeth. Table describes differences noted between implant and tooth.\(^{[11]}\)

<table>
<thead>
<tr>
<th>Tooth</th>
<th>Implant</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Connection</strong></td>
<td>Cementum, bone and periodontal ligament</td>
</tr>
<tr>
<td><strong>Junctional epithelium</strong></td>
<td>Hemidesmosomes and basal lamina (lamina Lucida, lamina densa zones)</td>
</tr>
<tr>
<td><strong>Connective tissue</strong></td>
<td>Perpendicular fibers</td>
</tr>
<tr>
<td><strong>Vascularity</strong></td>
<td>More</td>
</tr>
<tr>
<td><strong>Probing depth</strong></td>
<td>&lt; 3mm in health</td>
</tr>
<tr>
<td><strong>Bleeding on probing</strong></td>
<td>More reliable</td>
</tr>
</tbody>
</table>
Cochran et al. performed a study on loaded and unloaded non-submerged titanium implants and found that the dimensions of the implant/gingival junction remained constant over time up to 12 months after loading. The dimensions were comparable to the dentogingival tissues as described by Gargiulo et al. After 12 months of loading, the values were 0.16 mm for the sulcus depth, 1.88 mm for the junctional epithelium, and 1.05 mm for the connective tissue attachment. The biologic width reported in the study was 3.08 mm, including the sulcus depth, epithelial attachment, and connective tissue attachment. Later, Hermann et al. histometrically evaluated the dimensional change of the biologic width around non-submerged implants. They observed that each dimension of the sulcus depth, epithelial attachment, and connective tissue attachment changed over time, but within the overall biologic width dimension.¹²

Based upon these findings, it is apparent that early implant bone loss, in part, is from the processes of establishing the biologic width. The amount of bone loss and location of the biologic width may be associated with thickness of soft tissue around implants, location of the junction between rough and polished surfaces in non-submerged implants, and location of the micro gap in submerged implants. However, the reformation of the biologic width may not solely satisfy the cause of early crestal bone loss.¹³

4. CREST MODULE CONSIDERATIONS

The crest module of an implant body is defined as the transosteal region of the implant and serves as the region which receives the crestal stresses to the implant after loading. This region of the implant is often not designed for load bearing instead is usually designed to minimize plaque accumulation and acts a transition zone to the load-bearing structure of the implant body in submerged implants. With regard to the concept of preventing plaque accumulation, two problems may be observed. First, since toothbrush bristles cannot enter a sulcus on a routine basis more than 1 mm, and the tissue height above the implant body in submerged implants is usually 2.5 mm or more, the implant crest module does not provide an environment favorable for hygiene to remove plaque. Second, a smooth crest module may actually contribute to the crestal bone loss.¹⁴

Cortical bone is strongest to compressive loads, 30% weaker to tensile forces and 65% weaker to shear forces compared to compressive forces. Mish and Bidez claimed that a smooth, parallel-sided crest module may result in shear stresses in this region, and that an angled crest module of more than 20 degrees with a surface texture that increases bone
contact might impose a slight beneficial compressive and tensile component to the contiguous bone and decrease the risk of bone loss.\cite{15}

It has been clinically observed that bone is often lost to or below the first thread in some types of submerged implants after loading. Bone grows above the threads during healing as often demonstrated at stage II surgery, but after prosthesis loading, the bone loss down to the first thread is often noted after first year of prosthesis loading. Yet, in many submerged implant systems, the distance between the implant systems, the distance between the implant platform and the first thread varies, ranging from 1 to 3 mm (e.g. 1.2 mm in the Branemark system and 3 mm in many screw–vent implant systems). Therefore, the bone loss is probably not related to a specific anatomic length, but may be in part related to crest module design. Also, it can be hypothesized that the bone loss may slow down at the first thread because the first thread changes the shear force of the crest module to a component of compressive force to which bone is the most resistant.\cite{15}

5. **EXCESSIVE LOAD**

Forces applied to the restoration placed on implants are, at least in part, transferred to the bone (Skalak 1985). It is realized that though “excessive load” may be difficult to define and may vary from one subject and site to the next, factors such as occlusal force (trauma from occlusion) in relation to size of implant, surface features of implant and quality of the host bone must obviously be considered. Clinical studies have indicated that peri-implant bone loss may be associated with load. A few experimental studies have indicated that a relationship may exist between load and bone loss. But other investigators have been unable to confirm this association.\cite{16}

6. **INFECTION**

The host response to biofilm formation on the implant includes a series of inflammatory reactions which initially occur in the soft tissue but which may subsequently progress and lead to loss of supporting bone. The tissue destruction in the bone compartment starts in the marginal i.e. neck region, of the implant and crater like bone defects develop and become visible in the radiograph.\cite{17}

Peri-implant mucositis is a term used to describe reversible inflammatory reactions in the mucosa adjacent to an implant. Peri-implantitis is defined as an inflammatory process that (1)
affects the tissues around an osseointegrated implant in function, and (2) results in loss of supporting bone (Albrektsson & Isidor 1994).\[18-20\]

The prevalence of peri-implantitis in man is difficult to estimate but may vary between 2% and 10% of all implants inserted (Esposito et al 1998, Mombelli & Lang 1998). Clinical studies have documented that peri-implantitis may lead to implant failure and loss.\[21\]

7. PERI-IMPLANT MUCOSITIS

The response of the gingival and the peri-implant mucosa to early and more long-standing periods of plaque formation was analyzed both in experiments in animals and in man.

In experiments in the dog, Berglundh et al (1992) and Ericsson et al (1992) compared the reaction of the gingival and the peri-implant mucosa to 3 weeks and 3 months of de novo plaque formation. The mandibular premolars in one side of the mandible were extracted leaving the premolars on the contralateral side as controls. After 3 months of healing, fixtures (Branemark system) were inserted and another 3 months later, abutment connection was performed in a second-stage procedure. The animals were placed in a careful plaque control program to allow for ideal healing of the mucosa at the implants and to prevent gingivitis from occurring in the tooth segments of the dentition. Four months after abutment connection, the dogs were examined clinically and samples from the minute plaques that were present on the marginal portion of the implant and tooth surfaces were harvested. The plaque control program was terminated and the animals given a soft diet, which allowed gross plaque formation. Re-examinations, including clinical assessment and sampling of plaque bacteria from teeth and implants as well as biopsy were performed after 3 weeks and 3 months.\[18\]

During the course of the study, it was observed that similar amounts of plaque formed on the tooth and implant segments of the dog dentition. The composition of the two developing plaques was also similar. It was therefore concluded that early microbial colonization on titanium implants followed the same patterns as that on teeth (Leonhardt et al.1992).\[23\] With increasing duration of plaque buildup (3 months) on the dog model, the lesions in the peri-implant mucosa expanded more and progressed further apically than was the case in the gingiva. The composition of the lesions in the two tissues, the gingiva and the peri-implant mucosa, differed mainly with respect to their content of fibroblasts. Thus, the lesion in the
peri-implant mucosa was found to have a much smaller number of fibroblast than the corresponding compartment in the gingiva.\textsuperscript{[19]}

It may be anticipated that in an inflammatory tissue lesion of long standing, periods of breakdown and periods of repair interchange. The gingival lesion in the dog model retained its size unchanged between 3 weeks and 3 months of plaque exposure. It is suggested therefore, that in the gingival lesion, the amount of tissue destruction that occurred during a breakdown phase was more or less fully compensated by tissue buildup occurring during a subsequent phase of repair. In the lesion within the peri-implant mucosa, the tissue breakdown that occurred during the 3 months of plaque exposure was not fully recovered by reparative events. The small number of fibroblasts present in this particular lesion may simply have been unable to produce enough collagen and matrix during the reparative phase. This reduced buildup resulted in an additional propagation and spread of the inflammatory cell infiltrate in the peri-implant mucosa.

In a study done by Pontoriero et al (1994), in human volunteers, it was observed that plaque formation (amount and composition) and the soft tissue response to plaque i.e. Inflammation, developed in a similar manner in the tooth and implant segments. It was also found that in a given individual, the early soft tissue response to plaque seems to be similar in the mucosa at implants and in the gingiva of the tooth.

Hence in conclusion the Peri-implant mucosa seems less effective than the gingiva in encapsulating plaque – associated lesions.\textsuperscript{[22-23]}

REFERENCE

13. Rothman S. Use of prosthesis generated CT information for diagnosis and treatment planning In.Rothman S. ed Dental applicant of CT Berlin Quintessence, 1998; 177-239.
