Cystic epithelial lining seen around a dental implant: A case report of a rare finding

Yazad Gandhi^{a,*}, Neel Bhatavadekar^b

^a Consultant Maxillofacial Surgeon, Saifee Hospital, Mumbai, India

^b Clarus Dental Specialities, Pune. Adjunct Faculty: University of North Carolina at Chapel Hill, USA

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ABSTRACT

Dental implants have been successfully used for over 30 years, with a low complication rate. Peri-implant pathologies have recently seen an increase in incidence.

This case report elucidates a rare case implant failure that occurred shortly after prosthetic loading, where the clinical signs mimicked peri-implant disease and attending bone loss, but the final histological report confirmed presence of a cystic lining around the implant. The manuscript also emphasizes the possible causes and management of such complications.

To the best of our knowledge, this represents one of the first publication of an evident peri-implant cystic lining in a case exhibiting no radiographic evidence of the same.

1. Introduction

The concept of osseointegration, introduced by Brånemark, has revolutionized the clinical practice of dentistry. There are numerous documented studies that prove their success and long term predictability, thereby increasing the use of dental implants as a preferred alternative to traditional prostheses to rehabilitate partial and complete edentulous cases. Dental implants have successfully fulfilled the objectives of restoration of form, function and esthetics.

Reported success rates of dental implants are high. Nevertheless, failures that mandate immediate implant removal can and do occur.

Implant success is known to be influenced by the diameter of fixtures, the type of bone and the length of fixtures. Diameter of implants and the bone type are important especially in the posterior region of the mouth where they encounter functional loads. Length of the fixture is important when we deal with primary stability and also when we look at excessively long fixtures being placed in dense mandibular bone.

Peri-apical lesions and cystic osseous cavitation associated with implants have been reported. Literature also has mention of a case of Squamous Odontogenic Tumor arising adjacent to a dental implant.¹

Biological and biomechanical, factors have a cumulative impact on the final amount of bone loss seen and success of implants.² It is important for clinicians to understand all of these factors in addition to their relative contributions and interactions.

Long term success depends on a biological balance between

inflammatory and regenerative processes, ultimately with the latter as a dominant component.³

It is considered normal to see a settling of crestal tissues as some may term it after exposure of dental implants in the oral environment but clinicians need to understand the subtle difference between this non-pathologic cause of reduction in bone height and that due to inflammatory causes.

This case presents a noteworthy cause of multiple implant failures which would easily be confused with the common causes of peri-implant crestal bone loss had it not been for the histopathological examination showcasing a cystic lining around the implant.

2. Case report

A healthy, non-smoker, male patient 45 years consulted this facility for replacement of a missing molar. Upon clinical and radiographic examination a vertical fracture was found in tooth # 36 (mandibular left first molar) with a history of endodontic treatment.

The treatment plan formulated was to extract the roots, graft the socket and intervene at second stage to place the implant (Fig. 1).

The root remnants were extracted under local anesthesia and the socket thoroughly debrided. Autogenous bone harvested from the mandibular retromolar region was used to graft the socket and Collaplug (Collagen Fleece, ACE Biologics) was placed to seal the socket (Fig. 2).

* Corresponding author.

E-mail address: info@completedentalcare.co.in (Y. Gandhi).

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Fig. 1. Baseline presentation.

Fig. 3. First implant placed.

Fig. 2. Post-extraction before placement of first implant.

Fig. 4. First implant at second stage surgery.

After 3 months, the re-entry was made and a tapered internal Laser-lok implant (5.8 × 9mm), (Biohorizons, Birmingham, AL) was placed as a submerged unit (Fig. 3). After 3 months, the implant was uncovered (Fig. 4), ISQ values of 68 were measured and a hybrid cement-screw retained porcelain fused to metal crown (cemented on the model with resin cement which was buffed and polished then inserted as a screw retained unit) was inserted and torqued to 35Ncm. A week prior to his routine 4 monthly follow-up, the patient reported with discomfort on mastication. Upon clinical examination the screw retained unit exhibited second degree mobility bucco-lingually with a peri-implant probing depth of 6–7 mm (Fig. 5).

No other area of the oral cavity was found affected neither by gingivitis nor periodontitis suggesting satisfactory oral hygiene.

The screw-cement crown-abutment complex was disconnected from the implant and a reverse torque of 15 Ncm was applied to retrieve the implant. The osseous defect was debrided thoroughly and a mixture of

autograft and allograft (Lifenet Inc, USA) was placed and covered with a resorbable collagen membrane (Biogide, Geistlich Pharma) (Fig. 6).

In the interim period tooth #35 (mandibular left second premolar) showed a flare up with tenderness to percussion and frequent bouts of moderate to severe pain. An old silver amalgam restoration was present in the tooth. An endodontic opinion was sought and treatment initiated by the endodontist as it was not amenable to any conservative restorative procedure.

4 months later, a tapered internal laser-lok implant 5.8 diameter and 10.5 mm in length (Biohorizons, Birmingham, AL,) was placed submerged and left for 3 months (Fig. 7).

Thereafter the implant was uncovered, ISQ levels of 77 measured and the implant was loaded with the same type of prosthesis (cement-screw retained) as the first time.

After 6 weeks of receiving the prosthesis, the patient reported back with 2° mobility in the implant crown. Radiographs revealed unzipping of bone around the implant (Fig. 8)

After clinical examination, the screw retained unit was reverse torqued at 20 Ncm and removed and a gingival former placed. Upon

Fig. 5. First failing implant.

Fig. 7. Implant placement (second attempt).

Fig. 6. Socket graft after first failure.

Fig. 8. Second implant failure.

application of reverse torque of 20Ncm the patient experienced slight discomfort which may possibly be due to micromotion of the implant in the bony envelope. A cone beam scan was obtained which showed peri-implant loss of bone down to the junction of middle and apical 3rd (Fig. 9).

Though the implant was not very stable in the bone and could have been retrieved by reverse torque method it was deemed prudent to use a trephine to remove the implant along with peri-implant granulation tissue and some surrounding bone for histopathological examination. The tissue was stored in 10% formaldehyde prior to histopathological examination. The area was inspected for any residual granulation tissue following which a bone scraper (Safe Scraper, Metahosp, Italy) was used to collect autogenous bone shavings which was mixed with xenograft (Bioss, Geistlich Pharma) and covered with a resorbable collagen membrane (Biogide, Geistlich Pharma). Primary closure was achieved using 5,0 Prolene (Ethicon, Johnson & Johnson). Histopathologic examination revealed a cystic lumen lined by non-keratinized stratified squamous epithelium covering chronically inflamed connective tissue (Fig. 10).

The patient was free of any risk factor that could result in an implant failure and the screw-retained unit was thoroughly checked both times for occlusal contacts (light marks in heavy bite and no marks in light

Fig. 9. CBCT of second failing implant.

Fig. 10. H & E analysis of biopsy.

bite). There was adequate keratinized soft tissue both buccally and lingually as well.

The patient was recalled for follow-up as per routine protocol.

A cone beam CT scan obtained after 4 months revealed satisfactory bone fill (Fig. 11).

3. Discussion

Despite high success rates reported with endosseous titanium implants, failures do occur.

There are a variety of biologic causes for peri-implant lesions mentioned in literature, (a) heat induced osseous necrosis (b) contamination (c) residual root remnants (d) residual osseous pathology (e) foreign bodies and (f) contaminated graft particles to mention a few.

Peri-implant tissue inflammation may be limited to the soft tissue or progress to involve bone. This recognition of this subtle difference goes a long way in successful mitigation of the inflammatory condition.

Evidence in literature suggests that microbiology at sites with peri-implant disease is different to that of healthy implant and tooth sites, in the same individual as well as between different individuals.⁴

It is postulated, that microbial flora around dental implants are similar to flora that is native to the oral cavity, and that teeth in the mouth can act as reservoirs for seeding of microbial flora into peri-

implant tissues.^{5,6,7}

Crestal bone loss around osseointegrated implants can be attributed to a several reasons either acting in combination or as lone standing causes. Currently, in spite of improved techniques and science, implant failure, even with its decreasing incident rates, is still considered by many clinicians a major risk in implant treatment.⁸

The plethora of host factors that may cause peri-implant bone loss may be influenced by the host's genetic profile, medical history, periodontal status, oral hygiene, and smoking.^{9,10,11}

It is well documented that diabetics are at higher risk for periodontitis and more prone to peri-implant infection.¹²

Remodelling of crestal bone to allow establishment of a “biologic width” in peri-implant mucosal tissues is considered an important factor in early crestal bone remodelling in endosseous dental implants and should not be confused with pathologic bone loss.¹³

Advances in scientific research have led to certain surface modifications to reduce crestal bone die-back and unzipping of bone after exposure to the oral environment.

Incorporation of etched surfaces, grooves HA coated surfaces and Laser-lok surfaces¹⁴ are a few among the many surface modifications available.

A case of periapical implant infection has been reported in literature by Wang et al.¹⁵

There have been reports in literature of cystic lesions developing around endosseous dental implants but they were cases where the lesions did not mimic peri-implantitis and were osteolytic lesions with frank radiolucency either after implants were in function.

In one such case the implant was placed in close proximity to the radiolucency because the region apical to the implant was taken as the anterior border of the sinus when actually it was a pathological osteolytic lesion.¹⁶

It would be difficult to speculate regarding the cause of the cyst but it is worthy noting that this occurred around an integrated endosseous titanium implant and subsequently lead to bone loss and failure.

Plausible causes would include, titanium allergy, residual granulation tissue after tooth extraction.

Cell rests of Malassez remain in the form of a meshwork of cells around the root of a tooth under normal conditions. These cells may persist even after tooth extraction and have been shown to exist in alveolar bone many years after extraction.¹⁷

Inflammatory mediators around a radicular apex may stimulate the rests of Malassez and the epithelium forms the resultant cystic lining.¹⁸

An alternative explanation for the presence of epithelial cells that may proliferate into a cystic lesion around a titanium dental implant would be surgical implantation in the osteotomy prior to or with insertion of the implant.

Frisch et al 2001 suggested that displaced cells lose their capacity to multiply once separated from connective tissue and undergo apoptosis.¹⁹

Another possible source of epithelial cells around a titanium implant may be from a residual sinus lining around the pre-existing tooth, but in our case there was no such infective lesion noted.²⁰

Kochaji 2017 proposed a new entity for an inflammatory odontogenic cyst on an osseointegrated implant where he reported a periapical radiolucent lesion on radiographic examination.²¹

To the best of our knowledge we report this as the only documented case till date where a cystic lesion with histopathologic characteristics mimicking that of a dental cyst was detected without the evidence of periapical radiolucency on a 2 dimensional radiograph.

4. Conclusions

This case clinically and radiographically mimicked the condition of peri-implantitis related bone loss but histopathology examination reports a cystic lumen lined by non-keratinized stratified squamous epithelium covering chronically inflamed connective tissue. This was not

Fig. 11. The final bone fill at 4 months.

visible clearly even on 3 dimensional radiographic scans and had it not been for the histopathologic examination the cause for this repetitive failure would have remained in doubt.

This case report brings to light the importance of histopathologic examination of peri-implant tissue in cases of implant failure and also the multitude of reasons speculated to have caused proliferation of the cystic lining which need further investigation.

Declaration of competing interest

The authors whose names are listed below certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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