Long-Term Implant Survivability of an Implant Having Direct Contact with Cementum-Like Tissue in a Preexisting Mandibular Intraosseous Lesion with a 16-Year Longitudinal Follow-up

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This case report demonstrates long-term implant stability associated with focal cemento-osseous dysplasia (FCOD). The nature of the cementum-like tissue (CLT) of FCOD around the titanium surface of the implant is described clinically, radiologically, and histologically. A dental implant placed directly into the FCOD lesion was successfully integrated, and the stability was maintained for 15 years. However, at the 16-year follow-up, the implant and a sclerotic mass were removed due to peri-implantitis, and the entire specimen was evaluated with conventional histology and microcomputed tomography. The analysis revealed that the sclerotic mass attached to the implant was CLT that was free of any intervening soft tissue. The implant placed into the FCOD lesion achieved integration similar to that of conventional osseointegration. Rather than bone, the CLT was in direct contact with the titanium surface. The integrity of the union was maintained for up to 16 years.


Osseointegration is defined as the direct contact between bone and implant without the involvement of fibrous tissue.1,2 Generally, the duration of successful integration is directly proportionate to the magnitude of bone-to-implant contact (BIC).1,3 The higher percentage of BIC at the interface should yield longer implant survivability. The optimal bone quality, native or augmented, and enhanced surface texture of implants are critical factors in terms of providing long-term masticatory stability.4–7 However, there is little information about types of tissue other than bone that can potentially integrate with the implant to provide similar long-term support. In an animal study in 1990, Buser and his colleagues reported the formation of periodontal ligament around titanium implants when the implants were placed adjacent to retained root tips.8 Similarly, in 2002, Guarnieri et al reported a continuous layer of cementum and cementocytes on the implant surface but without any blood vessel or collagen fibers, contrary to findings in previously reported experimental animal studies.9 Further studies were indicated to clarify the opposing findings from the animal and human studies.

Cemento-osseous dysplasia (COD) is a fairly common lesion of the jaws originating from the periodontal ligament. Although COD
has no known neoplastic characteristics, the progression of COD occurs through three stages of development: early, intermediate, and late.10 An early or osteolytic lesion may present as well-defined, unicocular, and radiolucent, involving well-vascularized fibrous tissue.10 As the calcification of cementoid deposits increases, COD proceeds to the intermediate or cementoblastic stage, with a mixed radiolucent and radiopaque appearance.11,12 Further calcification leads to an osteosclerotic—or late, inactive—stage where the sclerotic CLT lesion is surrounded by a radiolucent rim.12,13 It is generally an asymptomatic lesion with little expansion or perforation of the cortical bone plate and exists in the upper part of the mandibular canal.10

Although the classification of cemento-osseous lesions of the jaws has long been a matter of debate, in 2005, the World Health Organization referred to this entity as “osseous dysplasia,” with three subsets: periapical osseous dysplasia, focal osseous dysplasia, and florid osseous dysplasia.14 Periapical osseous dysplasia is described as a lesion localized to the anterior mandible with only a few teeth involved, whereas focal osseous dysplasia lesions occur only in a single location of a posterior jaw quadrant.14 Lastly, florid osseous dysplasia has more extensive presentations, occurring bilaterally in the mandible or in all jaw quadrants.11,14 The histopathologic findings of COD are highly similar to that of the fibro-osseous lesion.10 Thus, a definitive diagnosis must be made using histopathology, clinical and radiographic examinations, and a thorough review of medical history.11,15,16

Numerous reports have introduced various adverse effects associated with dental treatment and COD.16–20 It is generally recommended that surgical interventions involving osseous structures such as tooth extraction, periodontal surgery, or surgical implantation be avoided in COD cases.18 Although short-term success has been reported previously, long-term survivability of a dental implant when placed into a COD lesion has not been well documented. This report introduces a case where the implant was placed into a late-stage focal COD (FCOD) in the posterior mandible and was successfully integrated and maintained for 16 years. Subsequent histologic analysis of the block specimen, consisting of the implant with its surrounding tissue, provides further valuable information regarding the nature of the supporting tissue.

Case Report

A healthy, 39-year-old man presented to a clinic in June 2001 for the removal of residual roots and surgical placement of dental implants. The patient was a smoker, occasionally consuming alcohol, and had no systemic disease that interfered with implant therapy. Radiographically, sufficient volume of bone existed above the mandibular canal for placement of an implant 10 mm or more in length (Fig 1a), but the width of the alveolar ridge was deficient (Figs 1b and 1c). In addition, a circular sclerotic mass 1.0 cm in diameter was observed in the edentulous ridge of the right posterior mandible (Fig 1a). The superior border of the sclerotic lesion was located 2 mm below the ridge crest, and the inferior border was 2 mm above the mandibular canal. It was a solitary, unilateral lesion with a well-defined periphery of approximately 2 cm. The lesion presented with a globular, cementum-like radiopacity and was also surrounded by a radiolucent rim (Fig 1a). There was no visible confirmation of any cortical expansion or perforation. The mass was highly radiopaque due to its advanced mineralization. The patient did not report any prior symptoms associated with the lesion. All of the above findings were indicative of the lesion being FCOD despite the lack of any histologic verification.

Under local anesthesia and following the specific surgical guidelines provided by the manufacturer, an external hex implant (3i T3, Zimmer Biomet) with a 4.0-mm diameter and a 10-mm length was placed at the mandibular right first-molar site. A buccal dehiscence defect and the exposed threads of the implant were grafted with Bio-Oss (Geistlich) without any membrane, and the overlying flap was closed with a 4-0 nylon suture. A panoramic radiograph showed that the implant was placed in the center of the FCOD (Fig 1d). Postoperatively, three tablets of 500 mg cefadroxil per day was prescribed for 10 days. Over-the-counter analgesics and a twice-daily chlorhexidine rinse were also recommended. A provisional complete denture was delivered 1 week after surgery, and the
The patient was closely monitored for 6 months, during which time no adverse events were noted. After 6 months postsurgery, proper osseointegration was verified, and the final mandibular fixed prosthesis was delivered. Subsequently, the patient was placed on a 6-month recall program, although the interval was not always consistent due to the patient’s personal circumstances.

In conjunction with visual inspection, a series of panoramic radiographs were taken at 3, 6, 10, 14, 15, and 16 years after the surgery (Figs 2a to 2d). Due to the development of peri-implantitis, cone beam computed tomography (CBCT) scans were also obtained at the 16-year follow-up visit to monitor the status (Fig 2e). However, further progression of peri-implantitis was evident on the CBCTs, and the concomitant clinical symptoms of gingival edema, gingival bleeding, pus discharge, and masticatory discomfort led to the decision for the removal of implants at the mandibular right second-premolar and first-molar sites. Under local anesthesia, a full-thickness flap was reflected for access, and the implants were gently removed with forceps. On the
removed first-molar implant, a mass of surrounding tissue was still tightly adhered to the surface (Fig 3a). The remaining defect was thoroughly debrided, and guided bone regeneration was utilized with Osteon 3 collagen (Genoss) and a collagen membrane (Genoss). The overlying flap was sutured without tension.

A radiograph was taken of the entire specimen (Fig 3b). Then, the block was fixated in neutral buffered formalin solution (Sigma-Aldrich) and was sent for microcomputed tomography (micro-CT; SkyScan 1173, Bruker) and histopathologic examination. The specimen underwent a dehydration process in ethanol solution, and the undecalciﬁed specimen was embedded in plastic resin (Technovit 7200, Kulzer) for micro-CT analysis. The scanner used (Skyscan 1173) had a tube voltage of 130 kV and a resolution of 14.91 μm (intensity 60 μA) (Fig 3c). The block of a polymerized specimen was cut in the direction of the implant’s long axis using a diamond cutter (EXAKT 300, EXAKT). Using a grinding system (EXAKT 400 CS, EXAKT), the thickness of the specimen was adjusted to about 50 μm for histopathologic examination (Fig 4). The osseointegration was evaluated in terms of BIC based on two-dimensional histologic images. One of the undecalciﬁed sections was stained with h&e (Figs 4a to 4c), and the other was stained with Goldner trichrome to highlight the distinct mineralized cemental tissue (Figs 4d to 4h). Histologic analysis was performed using a light microscope (BX51, Olympus).

**Results**

Prior to the 15-year follow-up appointment, there were no obvious clinical or radiologic changes in the FCOD lesion. When the patient presented to the clinic for the 15-year follow-up, the panoramic radiograph showed an early sign of bone loss around the second-premolar implant, but the first-molar implant seemed to be stable without any visible changes (Fig 2c). Conversely, the CBCT showed a distinct lesion contour around the first-molar implant that differed from the panoramic radiograph. The FCOD lesion was surrounded by a radiolucent rim that was separated from the adjacent bone. The patient returned 1 year later due to clinical symptoms, including gingival
edema, gingival bleeding, pus discharge, and masticatory discomfort. In the panoramic radiograph and CBCT scan, the radiolucent rim that used to enclose the FCOD lesion was further widened and the sclerotic mass had begun to resorb (Figs 2d and 2e).

Micro-CT findings of the specimen show that the sclerotic mass attached to the implant is similar to dense bone: the bone mineral density is very high, and the trabecular pattern is not visible. There is no gap in the interface between the FCOD lesion and the titanium surface. A very high percentage of tissue-to-implant contact, similar to BIC, can be seen (Fig 3c).

Histologically, the sclerotic mass attached to the titanium surface can be interpreted as cementum-like tissue (CLT) (Figs 4a to 4c). The outline of the FCOD lesion is spherical in...
shape, and it is surrounded by loose connective tissue. For the Goldner trichrome–stained samples, a dark green color indicates distinct mineralized tissue (Figs 4d to 4h). If there is an immature tissue–like osteoid, it will be stained with red color. There is no blood vessel or cellular fibrous stroma, and no vivid cell element can be observed (Figs 4c, 4f, and 4h), and this shows that the FCOD is sequestered via the limitation of blood supply. The sclerotic mass is histologically identified as the late stage of an FCOD lesion. A direct interface between the implant surface and CLT is formed without any intervening soft tissue and shows a very high percentage of contact, similar to BIC, and the result is very consistent with findings from the micro-CT analysis. At the crestal portion near the implant platform, peri-implantitis–induced bone resorption can be observed. The loose connective tissues around the FCOD lesion seem to be affected by peri-implantitis (Figs 4e and 4g), leading to gradual progression of bone loss.

Discussion

Fibro-osseous lesions are non-neoplastic lesions in the jaw that contain a diverse mineralized substance that may be bony or cementum-like in appearance and can be characterized by varying degrees of bone replacement by other fibrous tissues.15,21,22 The COD is a type of fibro-osseous lesion in the jaw that can be classified into early (stage I, radiolucent), intermediate (stage II, mixed radiolucent-radiopaque), and late (stage III, radiopaque) stages according to radiologic and histopathologic appearances.11,12 The development and maturation of the lesion is self-limiting and FCOD generally does not require a biopsy or any treatment until it shows signs and symptoms of infection.23–25

Adverse events, such as infections, may occur after the exposure of cemental mass, either by the natural resorption of the edentulous ridges or the extraction of teeth or roots in close proximity.18 Waldron et al reported poor socket healing and sequestrum formation after tooth extraction near the COD lesion.18 Osteomyelitis, a more serious complication, has also been reported.16,17 The progressive deposition of CLT seems to increase the chance of secondary infection and osteomyelitis.12,13 Moreover, these side effects may occur because of the direct exposure of the highly hypovascular tissue to the oral cavity.17,24,26 These effects may negatively influence the long-term survivability of dental implants in the FCOD lesions; for that reason, Sukegawa et al suggested removal of such lesions prior to implant placement.24

Placing an implant into COD lesions of an early or intermediate stage may result in failed osseointegration due to limited BIC, and the implant failures reported by Oliveira et al19 and Gerlach et al20 were presumed to be due to the immaturities of COD content. On the contrary, a late-stage COD lesion has been shown to contain a higher percentage of CLT than early- and intermediate-stage lesions,2,13 as the progressive deposition of cementum-like materials is the hallmark of lesion maturation.16 The late-stage lesions continue to enlarge, coalesce, and undergo further substantial radiopacification.16,22 Thus, the greater chance of successful integration of an implant placed in the late-stage lesion can be better explained by the increased CLT-to-implant contact.

Micro-CT analysis is a nondestructive radiographic procedure providing a high-resolution, three-dimensional image that can be used for an accurate determination of hard tissue volume and its content.27,28 It can also quantitatively assess osseointegration. The present specimen shows that the sclerotic mass attached to the titanium surface is very similar to dense bone in that the mineral density seems very high and the trabecular pattern is not visible. There is no gap in the interface between the FCOD lesion and the titanium surface. A very high percentage of tissue-to-implant contact, similar to BIC, is clearly visible. Histologic sections also demonstrate direct contact between the implant and the sclerotic mass, which appears to be CLT in a late-stage FCOD lesion. The interface is without any intervening soft tissue, and there is a very high percentage of CLT-to-implant contact, similar to BIC. The result is very consistent with the findings from the micro-CT analysis.

Cementum is similar to bone in composition, but the morphologic appearance is different. For example, no bone marrow is present within the cementum,29 and cementum is avascular and has many empty
scopic level. 1 The current case con-
direct contact with bone at a micro-
BIC is widely known as the
percentage of the implant surface in
only after sufficient treatment of sur-
In addition, because of the pres-
invasion. tal infections and inflammation.
most of the tissue was filled with highly
calciﬁed CLT. The hypovascularity
and hypercalcification of FCOD may
increase vulnerability to surrounding
insults, such as peri-implantitis.

Despite the hypovascular na-
ture without any cellular element,
the implant that was placed into a
late-stage FCOD lesion in the pres-
cent case report achieved success-
ful integration with the surrounding
CLT. This phenomenon of direct
contact between an implant’s tita-
nium surface and a tissue type other
than bone has not been well under-
stood. BIC is widely known as the
percentage of the implant surface in
direct contact with bone at a micro-
scopic level. 1 The current case con-
firms that a similar connection can
be achieved with the CLT without
any soft tissue intervention. A high
percentage of CLT-to-implant con-
tact was also evident in both the mi-
cro-CT and undecalcified histologic
sample. Although the implant in this
case successfully integrated with
the surrounding CLT and supported
masticatory function for more than
15 years, upon secondary infec-
tion, the CLT was also vulnerable to
further resorption, similar to the re-
sponse of bone to peri-implantitis.
For these reasons, the authors be-
lieve that implants should be placed
only after sufﬁcient treatment of sur-
rounding endodontic or periodon-
tal infections and inﬂammation.
In addition, because of the pres-
ence of immature tissue, implants
should only be placed into a late-
stage FCOD lesion where the CLT
is highly calcﬁed. A well-controlled
maintenance program should also
be implemented to prevent and
control potentially harmful infective
agents from the surrounding teeth,
implants, and gingiva.

Conclusions
The present case is an uncommon re-
port that demonstrates successful in-
tegration of a dental implant placed
into a late-stage FCOD lesion. The
direct contact of the CLT to the im-
plant was maintained and provided
masticatory function for 15 years
without any complications. When
presented with peri-implantitis, the
tissues behaved similarly to the bone
supporting titanium implants.

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References
1. Albrectsson T, Bränemark PI, Hansson
HA, Lindström J. Osseointegrated ti-
nium implants. Requirements for
ensuring a long-lasting, direct bone-to-
implant anchorage in man. Acta Orthop
2. Albretsson T, Zarb G, Worthington P,
Eriksson AR. The long-term efﬁcacy of
currently used dental implants: A review
and proposed criteria of success. Int J
3. Steigenga J, Al-Shammari K, Misch C,
Nociti FH Jr, Wang HL. Effects of im-
plant thread geometry on percentage of
osseointegration and resistance to
reverse torque in the tibia of rabbits.
4. Buser D, Shenk RK, Steinemann S,
Fiorellini JP, Fox CH, Stich H. Inﬂuence
of surface characteristics on bone in-
tegration of titanium implants. A histo-
morphometric study in miniature pigs.
5. Gottfredsen K, Nimb L, Hjørging-Hansen
E, Jensen JS, Holmén A. Histomorpho-
metric and removal torque analysis for
TiO2-blasted titanium implants. An ex-
perimental study on dogs. Clin Oral
6. Jensen T, Schou S, Gundersen HJ,
Forman JL, Terheyden H, Holmstrup P.
Bone-to-implant contact after maxillary
sinus ﬂoor augmentation with Bio-Oss
and autogenous bone in different ratio
7. BenicGl, Hämmerle CH. Horizontal bone
augmentation by means of guided bone
regeneration. Periodontology 2000
8. Buser D, Warrer K, Karring T. Formation
of a periodontal ligament around tita-
nium implants. J Periodontol 1990;61:
597–601.
9. Gunnari R, Giardino L, Crespi R,
Romagnoli R. Cementum formation
around a titanium implant: A case re-
port. Int J Oral Maxillofac Implants
10. Eversole R, Su L, ElMofy S. Benign ﬁbro-
osseous lesions of the craniofacial com-
plex. A review. Head Neck Pathol 2008;
11. Summerlin DJ, Tomich CE. Focal ce-
mento-osseous dysplasia: A clinicopat-
ologic study of 221 cases. Oral Surg
12. Singer SR, Mupparapu M, Rinaggio J.
Fibrod cemento-osseous dysplasia and
chronic diffuse osteomyelitis: Report of
a simultaneous presentation and review
136:927–931.
13. Kawai T, Hiranuma H, Kishino M, Jikko
A, Sakuda M. Cemento-osseous dyspla-
sia of the jaws in 54 Japanese patients:
A radiographic study. Oral Surg Oral
Med Oral Pathol Oral Radiol Endod
14. Barnes L, Eveson JW, Reichart P,
Sidransky D. Pathology and Genetics of
Head and Neck Tumors. World Health
Organization Classiﬁcation of Tumors.
15. Waldron CA. Fibro-osseous lesions of
51:828–835.