CASE REPORT

Implant-Associated Giant Cell Granuloma: A Case Report of 4.8-Year Follow-up and Literature Review.

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ABSTRACT

Implant-Associated Giant Cell Granuloma: A Case Report of 4.8-Year Follow-up and Literature Review.

Objective. The aim of this case report was to document a case of implant associated central giant cell granuloma (CGCG) and review the literature on implant associated and intrabony lesions. CGCG is most common in females and usually seen in the mandible from anterior to posterior. Based on its clinical, radiological, and histological findings, it can be classified as aggressive and non-aggressive forms. Trauma is considered a major etiological factor for the lesion. Even peripheral giant cell granuloma has been shown as a peri-implant lesion, CGCG has not been reported as an implant-associated pathology. In this case report, we reported that CGCG developed after implant placement in 8 months. 39-year-old female patient with partial edentulism in the posterior mandible presented to our clinic. She had reported that she lost her posterior mandible teeth for more than six years. Initial clinical and radiological examination revealed that she showed localized slight to moderate chronic periodontitis, horizontal ridge deficiency (in the posterior mandible), and cavities. A total of six implants were placed at the same time. At 8-month of the surgery, she showed a radiolucency area #34 area. The lesion was enucleated, and the defect area was filled up with a xenogeneic bone substitute. The healing was uneventful. The histological examination determined the lesion was CGCG. The lesion showed no recurrency for 4.8 years.

KEYWORDS

Central Giant Cell Granuloma, Mandible, Giant Cell Lesions, Dental Implants, Guided Bone Regeneration.

ÖZ

İmplant İlişkili Dev Hücreli Granülom: 4,8 Yıllık Takip Ve Literatür İncelemesine İlişkin Bir Vaka Raporu.

Bu olgu raporunun amacı, implant ilişkili merkezi dev hücre granülom (CGCG) olgusunun belgelenerek implant ilişkili ve intrabony lezyonları ile ilgili literatürü gözden geçirmektir. CGCG en sık kadınlarda görülür ve genellikle önden arkaya doğru daha çok mandibulada görülür. Klinik, radyolojik ve histolojik bulgularına dayanarak agresif ve agresif olmayan formlar olarak sınıflandırılabilir. Travma lezyon için önemli bir etiyolojik faktör olarak kabul edilir. Periferik dev hücreli granülom peri-implant lezyonu olarak gösterilmiştir, ancak CGCG implant ilişkili bir patoloji olarak bildirilmemiştir. Bu olgu sunumunda CGCG'nin implant yerleştirilmesinden sonra 8 ayda sonra geliştiğini bildirdik. Arka mandibula bölgesinde de kısmi dişsiz olan 39 yaşındaki bayan hasta kliniğimize başvurdu. Altı yıldan fazla bir süredir alt arka bölgede kısmi dişsiz olduğunu bildirmiştir. İlk klinik ve radyolojik incelemede lokalize hafif ile orta derecede kronik periodontitis, yatay alveolar kemik eksikliği (arka mandibulada) olduğu gözlemlenmiştir. Aynı zamanda toplam altı implant yerleştirildi. Ameliyatın 8. ayında #34 bölgesine yerleştirilen implant a komşu alanda radyolüsens alanı gözlemlendi. Lezyon enükle edildi ve defekt bölgesi hayvan kaynaklı kemik bio-materyali ile dolduruldu. İyileşme sorunsuz bir şekilde oluştu. Histolojik incelemede lezyonun CGCG olduğu belirlendi. Lezyon 4.8 yıl boyunca takip edildi ve nüks göstermedi.

ANAHTAR KELİMELER

Merkezi Dev Hücreli Granülom, Mandibula, Dev Hücre Lezyonları, Dental İmplantlar, Yönlendirilmiş Kemik Yapımı.

INTRODUCTION

Central giant cell granuloma (CGCG) is considered a fibro-osseous benign lesion of the jaws and accounts for 7% of the osseous benign lesion.[1] Several reports suggest there are three theories for its development 1) reactive origin to a local irritant, 2) development anomaly and 3) neoplastic etiology.[2-4]

CGCG is more commonly found in the mandible and mainly in a young females.[2] It can be seen classified as "aggressive" or "non-aggressive" radiologically, clinically, and histologically (Table 1).[2,4-6]

Table 1

Summarizing clinical and radiological features of aggressive and non-aggressive CGCG.

Differences	Aggressive	Non-Aggressive
Clinically	Symptomatic, Pain	Asymptomatic, No-pain
	Recurrence, rapid growth	Slowly growth, maybe non recurrence
	Usually extra oral swelling	Usually intra oral swelling
Radiologically	Multi or unilocular	Unilocular
	Larger than 2 cm	Smaller than 2 cm
	Perforation expanded cortical bone	Usually intact cortical bone
	Root resorption and displacement	Intact root and no displacement

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CGCG can be seen unilocular or multilocular with wispy-septation, cortical expansion, and perforation in the radiological examination.[5-7] Its treatment is mostly surgical, but recently some medications, including corticoid, interferon, bisphosphonates, or monoclonal antibody have been used for the treatment.[7] The aim this case report is to present a case of CGCG associated with inserted implant in a female and its treatment challenge.

Statement of Clinical Importance

A well-known etiological factor of CGCG is trauma. And it more likely occurs in female mandible. These patients should be closely followed up after implant placement. Surgeons should be aware to properly diagnose and manage them.

CASE REPORT

In 2016, A 39-year-old female presented to our clinic with some dental problems including cavities and missing teeth (Figure 1).

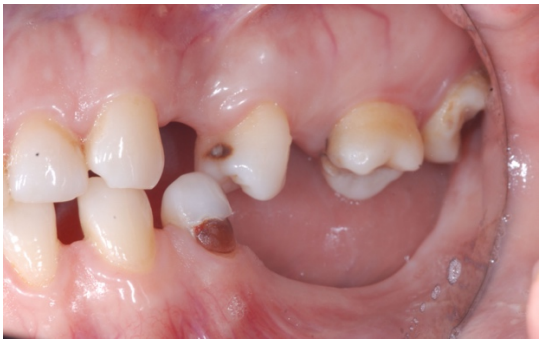

Figure 1.

Initial clinical buccal intraoral view.

She reported no medication in use. She had lost her second premolars and molars in mandible at the left and right side for more than six years. Her CBCT analysis showed she had vertical and horizontal alveolar ridge deficiency in the mandible posterior areas (Figure 2).

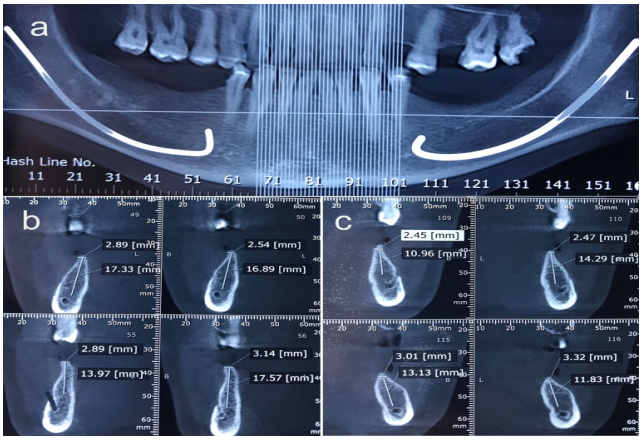

Figure 2.

Panoramic and CBCT analysis. Initial panoramic x-ray (a). CBCT analysis at the right (a) and left (b) side of the mandible showing horizontal alveolar ridge deficiency.

Following a periodontal exam, she was diagnosed with generalized chronic slight periodontitis.[8] The patient was informed of her treatment options, including filling all cavities, implant placements with bone grafting. Risks and benefits were carefully reviewed with the patient. After full mouth scaling and root planing, all cavities were restored. Then, the patient was scheduled for the implant placement and the wisdom tooth extraction. The following areas utilized to place implant; in positions #24 and 26 (3.3x10 mm, Straumann® BLT Roxolid®, Basel, Switzerland), #35 (3.3x8 mm, Straumann® SP Roxolid®, Basel, Switzerland), #37, 44 and 46 (3.3x10 mm, Straumann® SP Roxolid®, Basel, Switzerland) (Figure 3).

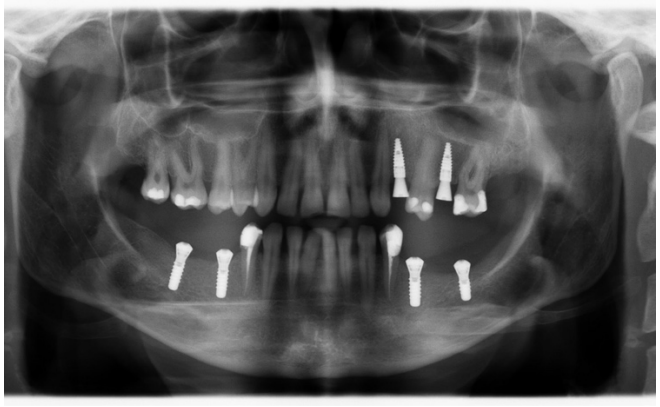

Figure 3.

OPG showing the placed implants after the surgery (day 0).

Exposed implant threads were covered with xenogeneic corticocancellous bone substitute (Apatos, Osteobiol, Tecnos, Italy).[9] The grafted area was covered with resorbable collagen membrane‡. The grafted implants were closed primary, but healing abutments were inserted on the upper implants (Figure 3). The patient was prescribed (started at -1 day) an antibiotic (Augmentin 1 gr: 125 mg of Clavulanic acid + 875 mg of Amoxicillin, twice/day, GlaxosmithKline, Turkey) and asked to use it for a week. Home care instructions were given including mouth rinse with chlorhexidine (0.05%, and twice/day) and clean the healing abutments with a soaked cotton applicator to prevent infection. No clinical complications were observed during the healing time. Post-operative follow-up appointments were scheduled at 1 week, 1 month, and 2 months. After 2 months of the surgery, her implants showed no infection sign and good ISQ values (68-74). Impressions were taken with polyether (Impregum, 3M, USA) by using an open tray technique. Then, implant-supported fixed prostheses (screw-retained) were delivered. Home care instructions were given, including the use of auxiliary hygiene aids for the cleaning of gingival and proximal surfaces. The patient was returned to periodontal maintenance every 3-4 months for continued care and monitoring of her implants and periodontal status. After around 8-month

of loading, the implant placed on #26 area showed some pus formation. Following examination, the crown was removed, and the area was cleaned surgically and left for healing. However, the implant failed in 3 months after the periimplantitis treatment. The implant was removed and left for secondary healing. Then, another implant (3.3x10 mm, Straumann® BL Roxolid®, Basel, Switzerland) was placed after 2-month.

During the regular follow-up (at 8-month of the initial surgery), she reported some discomfort around the implant position of the #35 area. She did not have any pain, had only an intraoral small swelling between implants #35 and 37. Her panoramic x-rays showed a small radiolucency area adjacent to implant #35 (Figure 4).

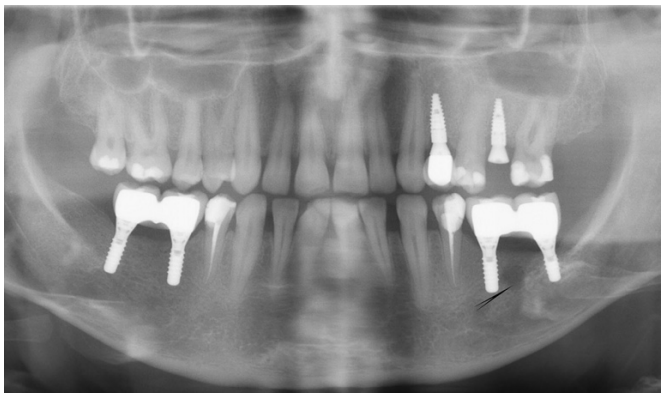

Figure 4.

OPG showing (**with an arrow**) a radiolucency area in a close distance the implant #35 at 8 months follow-up.

The patient was evaluated by her physician to exclude her possible malignant metastases in the jaw.10 Screw retained prosthesis was removed. Then, an excisional biopsy was taken following an envelope flap reflection (Figure 5A). The removed lesion was around 0.4 cm and showed some hard tissue (Figure 5B).

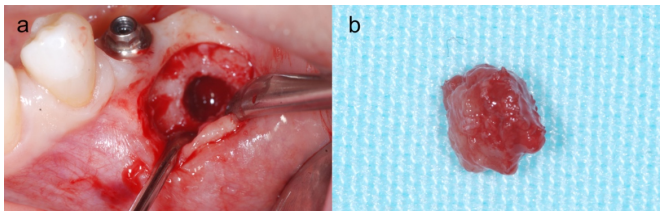

Figure 5.

Surgical approach of the lesion. An envelope full thickness flap reflection in the defect area (a) and the excised lesion (b).

The lesion was immersed into the 10% formaldehyde solution for a pathological examination. The defect area was repaired with xenogeneic corticocancellous bone substitute (Evolution, Tecnos, Italy). The healing was uneventful, and no post-op complication was seen. Histopathological examination revealed the lesion showed a reactive new bone formation, revascularization, multinucleated giant cells (Figure 6A-C).

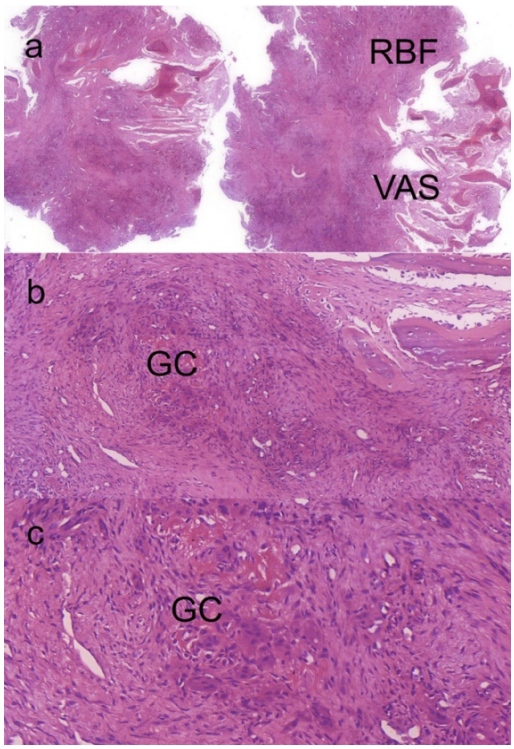

Figure 6.

Histopathology of CGCG showing clusters of multinucleated giant cells (H&E staining). Hypervascularization (VAS) and a reactive new bone formation (RBF) (x4) (a). Hypercellularity of the connective tissue, hyperinflammatory response and several multinucleated giant cells spread across the section and (x20) (b). Multinucleated giant cell embedded in spindle mononuclear cell (x40) (c).

It was diagnosed as a central giant cell granuloma. Intraoral examination demonstrated no peri-implant inflammation, bleeding, or other detectable signs of peri-implant disease at 4.8-year of lesion removal. Soft and hard tissue around implants showed very stably (Figure 7A). Radiologically, it was not observed no crestal bone loss or recurrency of the lesion (Figure 7B).

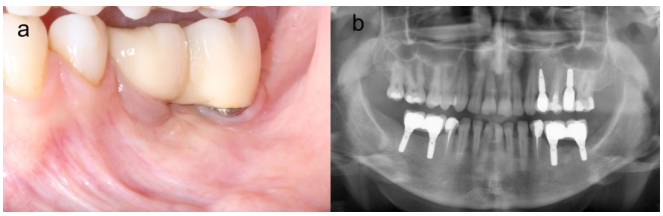

Figure 7.

Clinical (a) and OPG (b) view of the lesion showing no-recurrency at 4.8 years follow-up (b).

The overall clinical outcome was evaluated by Visual Analogue Scale (VAS) by questioning whether the patient satisfies peri-implant soft tissues, implant function, implant crown appearance, and post-surgical periods. The VAS scores were measured to the nearest mm by a ruler. Each question was scored on a 100 mm ruler (0: extreme dissatisfaction; 100: extreme satisfaction). She was very satisfied by the treatment aesthetically and functionally after the 5.3-year of initial implant placement.

DISCUSSION

CGCG is first defined by Jaffe in 1953.[11] It is considered as a non-neoplastic lesion, resulting in osteolysis, and seen mostly mandible in females.[6,12] Its etiological factor remains unknown. Jaffe suggested it was associated with trauma, whilst others have suggested it is instead associated with the inflammatory response[13] or pregnancy.[14] The non-aggressive form usually doesn't show the cortical bone perforation and its recurrence rate is low. The aggressive form of CGCG is characterized by a high recurrence rate, shows rapid growth, cortical bone perforation, pain, root resorption, and massive anatomical destruction.[3,14,15]

Since CGCG shows the low incidence and non-specific radiological features, it can be easily misdiagnosed clinically. The patient's age, sex, histopathology, and response to treatment should be considered for the clinical diagnosis of CGCG. The CGCG differential diagnosis can be made with a tumor such as a bone giant cell tumor (GCT). The pathological character of CGCG consists of multinucleated giant cells clustered around hemorrhagic foci, yet the multinucleated giant cells of GCT seem to be densely packed. The multinucleated giant cells of GCT are larger contain more nuclei than that of CGCG.[12,16] New bone formation and collagen deposition are seen in CGCG but not in GCT and the age at onset of CGCG has earlier than that of GCT.[12] CGCG and GTC have mostly seen the jaws and the long bone, respectively.[15] Importantly, in contrast to GCT, CGCG metastasis has not been reported.[12]

The primary site in this report was the mandible in a female following an implant insertion, which has not been reported. The patient did not show any clinical symptoms, including pain or facial asymmetry. However, it was seen a cortical bone perforation once a full-thickness flap was reflected. According to the literature, cortical bone perforation might indicate the lesion recurrency whereby it is required a radical excision in addition to curettage due to 13% to 22% the recurrence of GCCG in a short time point after the surgery.[17] In this case, surgical curettage, not resection of a part of the mandible, has been performed in the involved area. This successful management of the lesion could be due to catching it at an early stage. Filling up the lesion with xenogeneic bone substitute could help the surgeons to be able to track the lesion recurrency since xenogeneic bone substitute shows radiopacity and can't resorb for a long time.[18] In our present case, a conservative treatment over a resective treatment was preferred. The result of treatment was successful for a long time.

Conclusion

This report indicated implant-supported fixed restoration (especially in the mandible) in women needs to be followed up closely. Catching CGCG at an early stage could result in a more conservative surgical approach and reduced patient morbidity.

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Conflict Of Interest

This paper was not supported by any grant. Dr. Eskan is employed adjunct faculty at the NSU. Dr. Eskan reports no conflicts of interest related to this study.

Authors' Contributions

MAE and YEB have made substantial contributions to complete all surgical parts and collecting all parameters from the subjects. MAE has been involved in analyzing, interpreting, and supervising the study.

Patient Privacy

A written patient consent statement was received.

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