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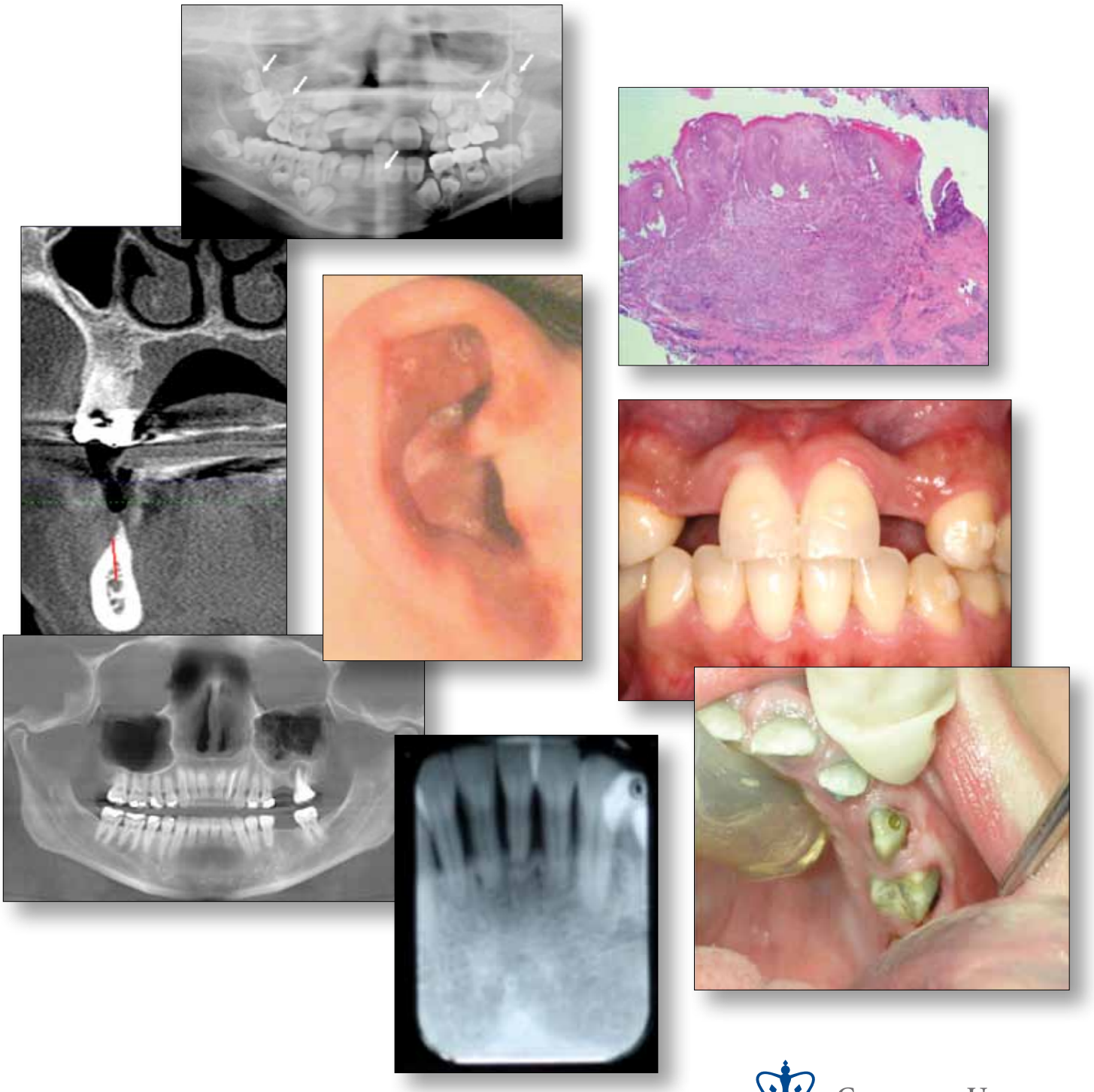
Columbia Dental Review



Columbia University
College of Dental Medicine

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Aim and Scope

The Columbia Dental Review (CDR) is an annual publication of Columbia University College of Dental Medicine (CDM). This journal is intended to be a clinical publication, featuring case presentations supported by substantial reviews of the relevant literature. It is a peer-reviewed journal, edited by the students of the school. The editors are selected on the basis of demonstrated clinical scholarship. Authors are primarily CDM students from pre-doctoral and postdoctoral programs, CDM faculty and residents, and attendings from affiliated hospitals. Peer reviewers are selected primarily from the CDM faculty. Instructions for authors wishing to submit articles for future editions of the CDR can be found on the last page of this journal. Opinions expressed by the authors do not necessarily represent the policies of the Columbia University College of Dental Medicine.

Editors’ Note

Dear Readers,

The Columbia Dental Review was created to give Columbia dental students a voice in current dental research. To this end, our authors, in collaboration with faculty, have researched a wide array of topics covering many different facets of clinical dentistry.

It is with continual research that our profession is able to progress. This publication continues to stay committed to contributing to our field by addressing and highlighting some of the most interesting current topics in clinical dentistry.

On behalf of all the editors and assistant editors, I would like to thank Dr. Letty Moss-Salentijn whose guidance has ensured this publication continues to serve our dental community year after year. I would also like to thank the authors, faculty reviewers, editors, and graphic designer for all their efforts. Without their contributions, this fourteenth volume of the CDR would not have been possible.

Sincerely,

Nicole Madison Lambert ‘10

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Columbia Dental Review 2009-2010

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Dental Defects Connected with Chemoradiotherapy: A Case Report

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Abstract
Developmental orofacial dentoalveolar complications associated with chemoradiotherapy in a 7-year-old child with a history of rhabdomyosarcoma are reported. This report describes, clinically and radiographically, these effects in a child diagnosed at 3 years of age with a lesion primary to the left buccinator. Early evaluation is crucial to determine potential dentoalveolar complications and long-term consequences.

Keywords:
chemotherapy, radiotherapy, dental defects, case

Introduction
Developmental orofacial dentoalveolar complications are associated with both chemotherapy and radiation therapy subsequent to treatment of rhabdomyosarcomas (RMS). RMS are the most common soft-tissue tumors in children. These tumors are derived from skeletal muscle. Cell markers such as desmin, sarcomeric actin, sarcomeric myosin heavy chain and MyoD suggest myogenic cell origin for this tumor.[1] This type of sarcoma accounts for 4-8% of all malignancies in children under 15 years old.[1] The tumor is more common in Caucasians, and most studies show slight gender predominance in male. While RMS may occur in all age groups, it is mostly seen in the first and second decades of life with a peak incidence between ages two and six.[2] The most common sites of this tumor in children are head and neck (35%), genitourinary tract (23%), and extremities (7%).[3] There are three main types of RMS classifed histologically as embryonal, alveolar, and undifferentiated. The alveolar type accounts for 20% and is histologically characterized by clusters of small round cells with hyperchromatic nuclei and eosinophillic cytoplasm that are separated by fibrovascular septae.[2,4-6]

Approximately 65% of children diagnosed with rhabdomyosarcoma survive after receiving multimodality treatment.[7] An important reason for the improved survival rates is well-timed initiation of radiotherapy combined with chemotherapy.[7] Chemoradiotherapy can be used for local control of the primary lesion, to induce regression of tumor size, and to treat tumors not easily accessed for resection in the head and neck area.[2] Chemotherapy consists of combinations of vincristine, actinomycin-D, cylcophosphamide, and doxorubicin. The dose for radiation therapy of rhabdomyosarcoma ranges from approximately 40 to 50 Gy.[7,8]

Dental and orofacial abnormalities are most predominant in children who have received chemoradiotherapy treatment before three years of age, as they do not have a fully developed primary dentition and the permanent dentition is also not yet completely formed. The developing teeth are exposed to radiation during the course of treatment for head and neck sarcomas. More than 85% of survivors of head and neck RMS who receive radiation doses greater than 40 Gy may have significant dental abnormalities.[9] The abnormalities include mandibular or maxillary hypoplasia, increased caries, hypodontia, microdontia, root stunting, and xerostomia.[9] Chemoradiotherapy has a considerable effect on soft and hard tissue growth in the affected regions of the head and face. This leads to facial and dental irregularities that exacerbate during growth. The extensive effect of radiotherapy and chemotherapy on craniofacial skeletal growth must be monitored for all patients undergoing treatment for tumors. Consequential dental and maxillofacial abnormalities can be expected in all cases. Management of the patient calls upon the involvement of different members of a healthcare team including maxillofacial surgeons, dentists, psychologists, dieticians, and speech therapists, along with the patient and the primary caregiver.[10] The following case report describes the orofacial dentoalveolar findings for a pediatric patient diagnosed with rhabdomyosarcoma.

Case Report
A 7-year-old male patient with a history of alveolar RMS, hypothyroidism, and sickle cell anemia presented to the Columbia University Medical Center pediatric dental residency clinic for routine dental care. The child patient had undergone radiation therapy on the left side of the head and neck region at the age of three; the cancer is in remission at the present time. Before he was diagnosed with cancer, the patient developed a proptosis of the left eye and a left submandibular mass. The mass was first noted during a routine visit to the Hematology Clinic of New York-Presbyterian. Computed axial tomography (CT) imaging revealed a 2 x 2.4 x 2.5 cm mass at the angle of the mandible on the left side. Additionally, magnetic resonance imaging revealed an enhancing lesion in the region of the left ethmoid air cells, extending into the left anterior cranial fossa and epidurally along the left frontal lobe. Biopsy confirmed an alveolar rhabdomyosarcoma (chromosome 2:13 translocation). Further evaluation of the lung CT and bone revealed no metastasis.

Following the diagnosis, he received 50.4 Gy radiation to the head and neck region including the ethmoid and sphenoid sinuses and completed the chemotherapy in December 2003. When a CT evaluation in June 2004 revealed a new lytic bony abnormality in the midline of the frontal bone extending to the ethmoid bone, he was re-examined for a cancer relapse. Additional examinations, including a bone scan and chest CT, demonstrated no other evidence of a relapse, and hence a biopsy was deferred. The imaging study in June 2005 reported the lesion as stable and it is presently believed to be caused by an infarct due to the patient's sickle cell disease.

Complications during the patient's cancer treatments included fever, neutropenia, and infections that ranged from G-tube cellulitis to facial cellulitis. He also developed an episode of leg pain that upon extensive evaluation, was attributed to a sickle pain crisis. He previously had several episodes of life-threatening epistaxis, but has not had any occurrences for more than two years. The patient's growth had fallen below average for his age, possibly due to endocrine abnormalities subsequent to cranial irradiation. Currently, his growth is improving since starting supplemental growth hormones and is carefully monitored by an endocrinologist. His prognosis is now excellent; he has been free of the cancer and off of chemoradiotherapy. He continues to be followed with semi-annual scans.

The patient is currently taking folic acid, growth hormone, synthroid, and penicillin. When initially examined at age five, he presented with poor oral hygiene and visually evident rampant dental caries and dental abscesses. Comprehensive dental treatment was accomplished under general anesthesia at the Children's Hospital of New York. No complications followed the procedure, and wounds healed uneventfully. The patient was placed on routine oral hygiene maintenance visits.

The parent and patient failed to follow up regularly and returned to the clinic three years later with a chief complaint of unerupted teeth. There were no signs of facial swelling or lymphadenopathy. Intra-oral examination *(Figure 1A)* revealed normal soft tissue with an absence of any soft tissue pathology or gingival inflammation. Spacing in the dentition was consistent with the child's age, and the occlusal relationship was within normal limits. Although the patient's overall oral hygiene was good with no clinical evidence of caries, his incisors had minor enamel hypocalcification defects. While the remaining dentition was free from any signs of mobility, the central incisors exhibited Grade II mobility. Radiographic evaluation of the patient *(Figure 1B and Figure 2)*, presently eight years old, demonstrated multiple developmental dental defects. These included complete tooth agenesis of the permanent maxillary second premolars and partial odontogenic deficits, such as generalized moderate root stunting, agenesis of permanent first molar roots in both arches, tapering of lower right permanent canine roots, and microdontia of permanent premolars and maxillary second molars. Underdeveloped jaws were also noted radiographically. Future treatment options were discussed and the patient continues to be monitored through his routine dental examinations.

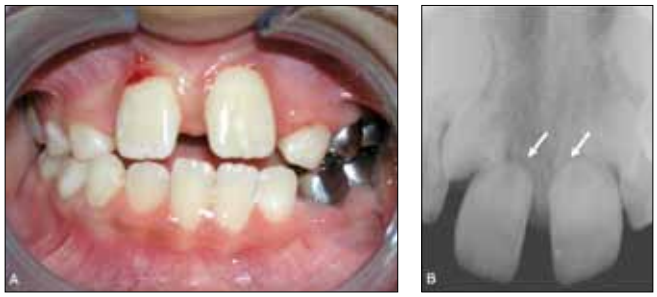

Figure 1
(A) Intraoral photo demonstrating splaying of incisor teeth, and (B) a periapical radiograph revealing root agenesis on incisor teeth resulting in delayed eruption.

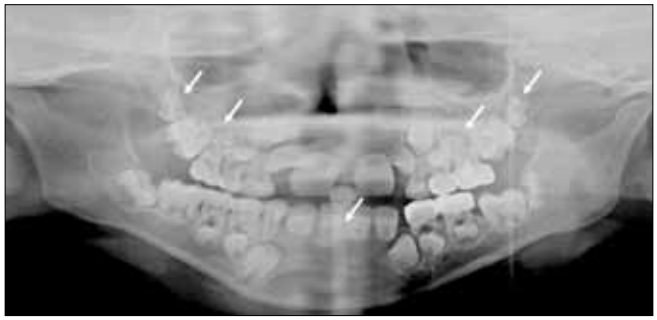

Figure 2
Panoramic radiograph. Note advanced root stunting of incisor teeth, microdontic premolars and second molars, root stunting of the six year molars, and root tapering of lower right permanent canine.

Discussion

RMS are a rapidly growing, aggressive neoplasm in children.[2] Of the three types, the embryonal form is said to have the most favorable prognosis, while the alveolar and undifferentiated forms are unfavorable.[11] The aforementioned patient is unusual because he has an excellent prognosis for the alveolar RS and has been free of the cancer since June 2005. This could be due in part to the early staging of the cancer and the absence of metastases.

The treatment the patient received was based on the tumor stage and clinical presentation (using the pretreatment tumor-node-metastasis (TNM) system).[12] It included both radiation therapy and chemotherapy. Although curative, the chemoradiotherapy for the patient's rhabodomyosarcoma produced long-term side effects. These effects typically include neuroendocrine, dental, thyroid, and cognitive issues. Neuroendocrine dysfunction and clinical hypothyroidism

typically occur during the first 10 years after radiotherapy.[7] This matches the patient's presentation in the case, as he is currently taking growth hormone and synthroid for his delayed growth and hypothyroidism, respectively. Complications of chemotherapy include alopecia, myelosuppression (thrombocytopenia and neutropenia), mucositis, nausea, vomiting, and neurotoxicity. Complications of radiation therapy to the head and neck region include interference with growth of the craniofacial skeletion, limitation of mouth opening, microdontia, hypoplastic enamel, underdeveloped roots, delayed or premature exfoliation of teeth, and dental caries.[2] The rampant dental caries that the patient presented with at age five was due in part to the xerostomia associated with radiation treatment. Radiation greater than 40 Gy that targets more than 50 percent of the salivary gland *(Table 1)* can cause decreased salivary flow, xerostomia, and subsequently increase a patient's risk of dental caries. Radiation around 10 Gy destroys developing roots of the dentition.[9]

Dental defects, secondary to chemoradiotherapy, manifest as certain signs and symptoms. Chemotherapeutic agents can cause microdontia, hypoplastic or hypomineralized enamel, underdeveloped roots, and delayed eruption. Radiation can lead to premature exfoliation of primary teeth and hypoplasia of the maxillary and mandibular jaws, along with root obliteration. The patient's radiographs are precisely characteristic of the aforementioned dental defects. The developing permanent tooth buds were clearly affected by the cancer treatment, leading to hypoplastic enamel, root agenesis, microdontia of the premolars and underdeveloped jaws *(Figure 1B and Figure 2)*. In a study of 17 children with head and neck rhabdomyosarcoma, all 17 had at least one dental abnormality.[7] The abnormalities ranged from microdontia, trismus, mandibular hypoplasia, hypodontia, root stunting, maxillary hypoplasia, xerostomia, and radiation caries.[7] The children with dental abnormalities received radiotherapy anywhere from age 3.4 to 11.5 years with a dose to the orofacial region of approximately 40 to 60 Gy.[7] These findings satisfy both the age bracket and radiation dose range for the patient mentioned above.

It must be noted that the patient presents with sickle cell anemia in his medical history. Sickle cell disease is a blood disorder characterized by morphologically changed erythrocytes and a defective form of hemoglobin due to an amino acid substitution mutation. Systemic manifestations in patients with sickle cell anemia include dentoalveolar

Table 1

Dental and Oral Complications Secondary to Chemoradiotherapy*

Complication	Signs/Symptoms	Treatment
Abnormal Dental Development *Chemotherapy:* Vincristine, actinomycin D, cyclophosphamide, 6-mercaptopurine (6-MP), procarbazine, nitrogen mustard (HN2) *Radiation:* Generally 10 Gy can obliterate developing roots	Microdontia Hypoplastic or hypomineralized enamel Underdeveloped roots Delayed eruption Premature exfoliation of primary teeth Hypoplasia of jaws	Dental examination every 6 months with attention to early caries, periodontal disease, and gingivitis, and baseline panoramic and bitewing radiographs (age 5-6 years) Careful evaluation before tooth extraction, endodontics and orthodontics, topical fluoride, antibiotics as needed for patients at risk for infection
Xerostomia, Stomatitis *Radiation:* >40 Gy and >50% of gland irradiated	Decreased salivary flow Xerostomia Altered taste perception Caries Candida	Dental examination, salivary flow studies, attention to early caries, periodontal disease Encourage meticulous oral hygiene, saliva substitution, prophylactic topical fluorides, dietary counseling regarding avoidance of fermentable carbohydrates, nystatin for oral candidiasis, pilocarpine

**Adapted from Schwartz et al. 9*

complications, impaired growth, and delayed skeletal maturation. The dentoalveolar sequelae include enamel hypoplasia, delayed tooth eruption, and impaired dentin mineralization.[13] These traits resemble the dental problems seen in the patient. Due to the broad overlap of dental manifestations between sickle cell disease and chemoradiotherapy, it is important to note that chemoradiotherapy may not be the sole cause of the patient's dental complications.

According to general guidelines on dental management of pediatric patients receiving chemotherapy and/or radiation,[14] early and definitive dental intervention can minimize the risks for oral complications. Education about oral hygiene and optimal care can prepare the parents to deal with the acute and long-term effects of therapy to the orofacial region. After cancer therapy is completed, periodic evaluations of the patient are recommended at least every six months.[14] The patient in this case report failed to return for routine follow-ups until unerupted teeth were noticed. Potential dental treatment plans for a child who has received chemoradiotherapy must include a thorough assessment and a discussion with caregivers about the potential dental disturbances caused by the cancer therapy.

In order to curtail dental defects it is ideal to reduce the radiation to healthy oral tissues. This can be accomplished through the use of lead-lined stents, prostheses and shields.[14] Although high-intensity courses of treatment for head and neck sarcomas are often necessary, studies suggest it is possible to decrease radiotherapy for certain patients without compromising survival, in hopes of decreasing long-term side effects.[15]

Conclusion

Chemoradiotherapy can lead to various abnormalities in both the primary and permanent dentition. Head and neck rhabdomyosarcoma treatment often directly involves high doses of radiation to the orofacial region. Consequently, one should expect dental and maxillofacial abnormalities in long-term cancer survivors of childhood malignancies. Early evaluation is necessary to determine the potential dental abnormalities and long-term consequences for children receiving chemoradiotherapy. This case demonstrates several signs indicative of developmental orofacial dentoalveolar complications associated with chemoradiotherapy as reviewed in the literature. It is anticipated that continued routine dental examinations, management by a multidisciplinary medical team, and semi-annual scans will help ensure the patient remains free of cancer.

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Misdiagnosis of Gingival Squamous Cell Carcinoma Presenting as a Periodontal Lesion of the Anterior Palate

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Abstract
Due to its close proximity to periodontium and teeth, gingival squamous cell carcinoma can easily resemble a benign lesion which can lead to a misdiagnosis. This is a case report of an 87-year-old female with no history of alcohol or tobacco use in which proper diagnosis of gingival squamous cell carcinoma took nearly 6 months despite repeated visits to her general dentist.

Introduction
Oral Cancer accounts for less than 3% of all cancers in the United States, though it is the eighth most common cancer in males and the fifteenth most common in females[1]. Approximately 94% of all oral malignancies are squamous cell carcinoma (SCC). While the exact etiology of oral SCC remains unknown, some of the most common risk factors include the use of tobacco, consumption of alcohol, the chewing of areca (betel) quid, syphilis (tertiary stage), *candida albicans* infection, oncogenic viruses, and immunosuppression[1,2].

Oral SCC may have a varied clinical appearance. However, studies have shown erythroplakia to be the earliest manifestation of oral SCC in the United States and Europe, especially in smokers and consumers of alcohol[3]. It may appear as an exophytic mass with a granular, papillary, or verrucous surface. It may also appear ulcerated[4].

The stage of diagnosis is the most important predictor of long-term prognosis[5]. For this reason, delayed detection of oral cancer results in a low five year survival rate when compared to other types of cancers[6]. The most common sites for oral SCC include the lateral tongue, floor of the mouth, and soft palate. Other less frequent sites include gingiva, buccal mucosa, labial mucosa, and hard palate[1]. When affecting the gingiva, the mandibular molar region is generally the most common site, with most cases occurring in edentulous areas[7].

Gingival SCC is one of the most devastating malignancies due to its common invasion into underlying bone[8]. Since the lesion is usually very close to teeth and periodontium, most patients with gingival SCC visit a general dentist first. Thus, dentists play a crucial role in early detection and management of gingival SCC[9].

The following is a case of an 87-year-old female who presented with a gingival lesion which was incorrectly attributed to her periodontal condition rather than gingival SCC.

Case Report
An 87-year-old female presented to a general dentist on 9/9/2007 requesting to be seen for comprehensive care. She reported the loss of a restoration on tooth #8 as well as gingival soreness in the region of #8. The patient's past medical history included hypertension, arthritis, and Paget's disease. Medications included verapamil, atorvastatin calcium, meclizine, and acetaminophen. Due to atypical clinical presentation and a lack of risk factors for oral SCC, the patient's lesion was attributed to local etiology, and she was diagnosed with generalized moderate chronic periodontitis. She was treatment planned for four quadrants of scaling and root planing along with restorative treatment.

Between the dates of 10/2/07 and 11/1/07 the patient was seen three times by her general dentist for scaling and root planing of the upper right quadrant because there was no improvement in the gingival lesion around tooth #8.

On 2/15/08 the patient presented to Columbia University College of Dental Medicine with the chief complaint of swollen and sore palatal tissue in the region of teeth #8 and #9 *(Figures 1, 2)*.

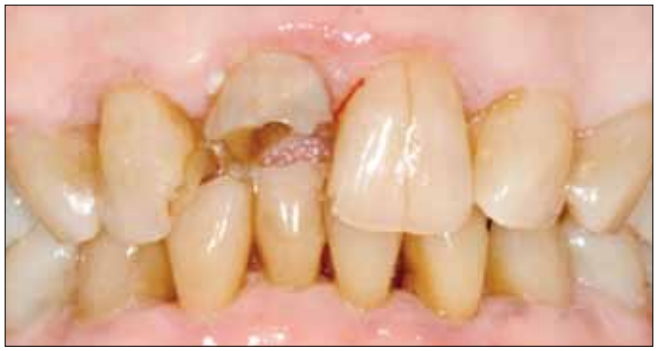

Figure 1
Fractured tooth #8 on clinical exam when patient presented on 2/15/2008 to the periodontics department at Columbia University College of Dental Medicine.

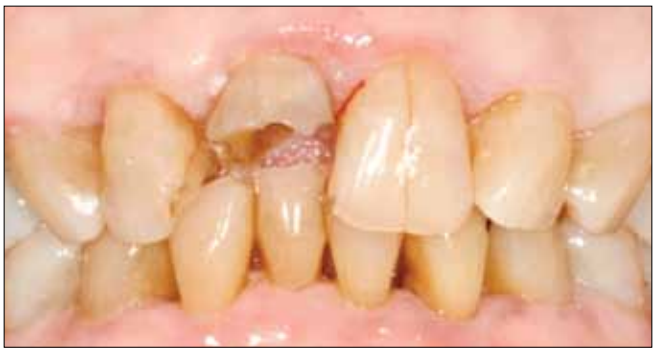

Figure 2
Palatal gingival lesion between teeth #8 and #9.

The patient reported an increase in pain around the gingival region of tooth #8 for the past several months. The patient took antibiotics prescribed by her general dentist in 1/08 and reported that the antibiotics did not help and her pain persisted. A periapical radiograph of the region was taken and no periapical radiolucency was noted *(Figure 3)*.

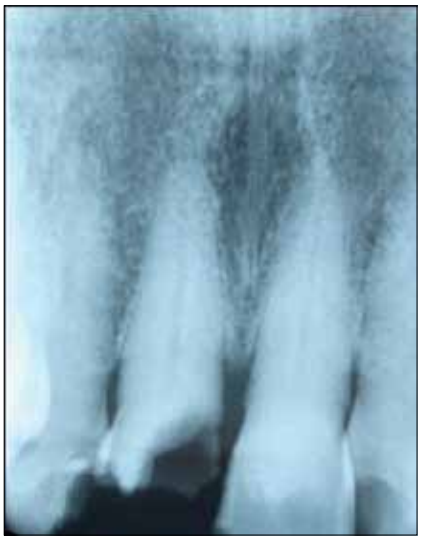

Figure 3
Radiograph showing no periapical radiolucency or endodontic lesion around tooth #8 or #9

The patient was referred to the post doctoral periodontics clinic for further evaluation. Intraoral examination revealed a granulomatous and erythematous lesion extending from the gingival margin to approximately 1 cm onto the hard palate from the distal of tooth #8 to the distal of #9. Palatal probing depths were 5 mm on both tooth #8 and #9, and the marginal gingiva was erythematous and edematous with bleeding on probing present. A moderate amount of plaque and calculus was present. The patient was diagnosed with chronic pyogenic granuloma. Scaling and root planing of teeth #7-#10 was performed with a gingival flap, which allowed a biopsy of the lesion to be taken in order to confirm the diagnosis *(Figures 4, 5)*.

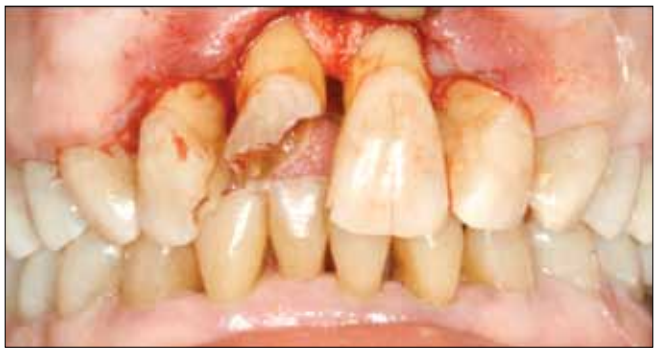

Figure 4
Gingival flap, buccal view.

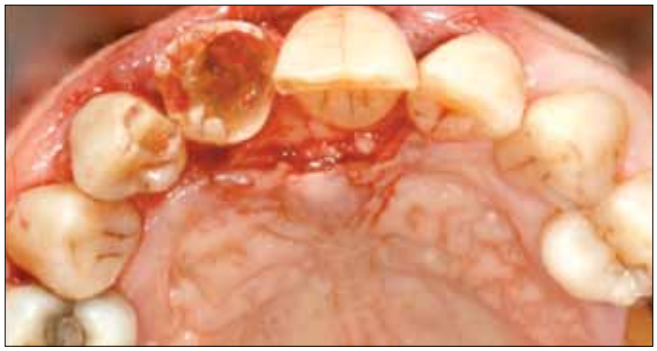

Figure 5
Gingival flap, palatal view.

Submarginal and sulcular incisions were carried out on the palatal region of teeth #7-#10, which allowed the removal of the abnormal granulomatous tissue for biopsy. Similar incisions and flap design were created on the buccal side of teeth #7-#10, followed by thorough debridement of the area. Sutures were placed and healing occurred by secondary intention.

Histologic examination revealed curved pieces of soft tissue covered by atypical and hyperplastic stratified squamous epithelium with overlying parakeratotic material. Epithelium was dyskeratotic and demonstrated bulging of rete pegs *(Figure 6)*.

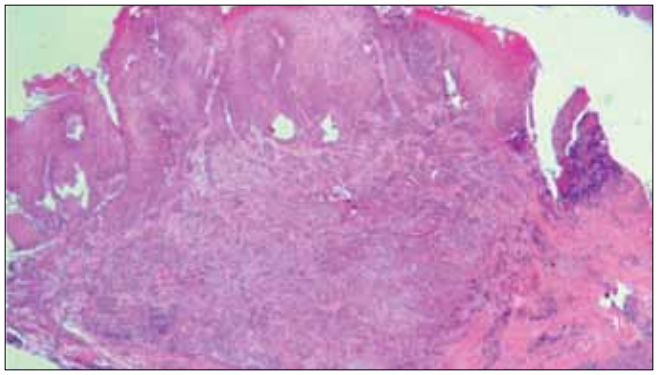

Figure 6
Photomicrograph (H&E, 40x) revealed hyperplastic and hyperkeratotic epithelium with bulging rete pegs. Strands of invasive squamous cells were also present.

Islands and strands of invasive squamous cells exhibiting pleomorphic cellular features were identified. These invasive islands deeply infiltrated the underlying fibrous connective tissue *(Figures 7, 8)*. Also noted were fungal hyphae and spores, consistent with candida albicans.

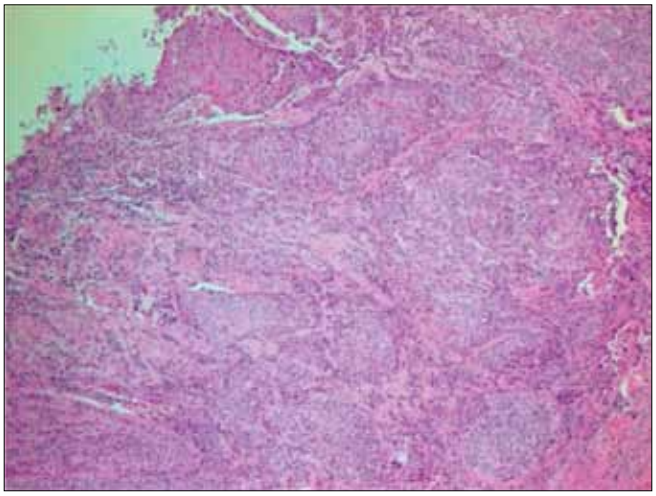

Figure 7
Photomicrograph (H&E, 100x) showed invasive islands deeply infiltrating the underlying fibrous connective tissue.

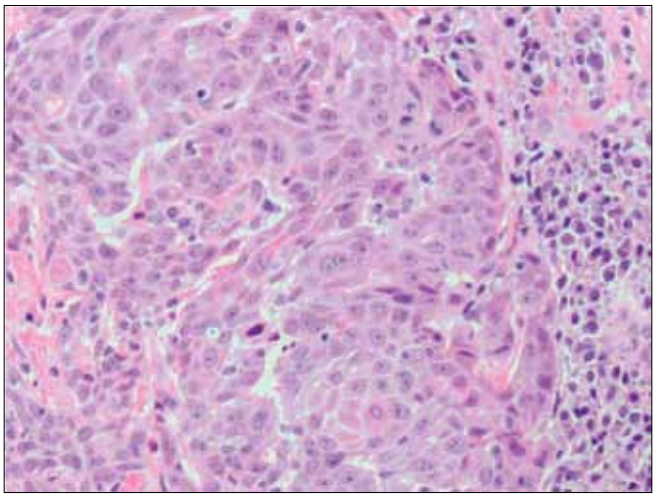

Figure 8
Photomicrograph (H&E, 200x) with squamous cells exhibiting pleomorphic cellular features including enlarged and hyperchromatic nuclei with prominent nucleoli, increased mitotic activity, and chronic inflammatory cell infiltrate.

A diagnosis of moderately differentiated squamous cell carcinoma of the anterior palatal gingiva was made. The patient was referred to an otolaryngologist for resection of the anterior maxilla from teeth #6-#11. Resection took place on 3/28/08, at which time Iodoform packing and a skin graft from the patient's thigh was placed over the resected area *(Figure 9)*. A surgical obturator was delivered, and three ligature wires were placed to retain the obturator.

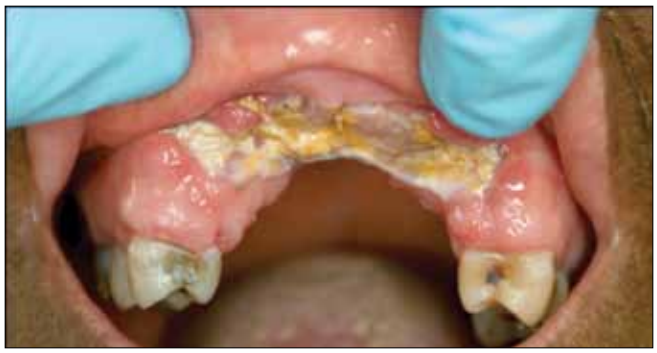

Figure 9
Skin graft placed after resection of anterior maxilla.

On 4/4/08 the ligature wires were sectioned and the surgical obturator was removed. The interim prosthesis was delivered *(Figure 10)*.

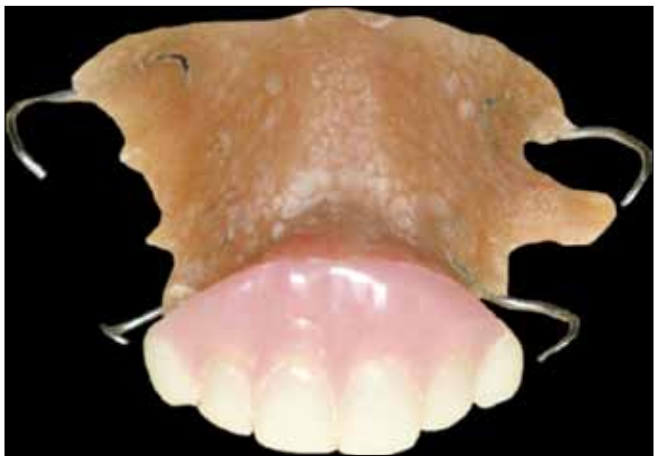

Figure 10
Interim prosthesis.

Discussion

Over the past 40 years despite advances made in diagnosis, the overall five year survival rate for oral SCC has remained relatively constant at around 50%[5]. Several reasons may account for this: 1) high risk patients do not seek medical attention, 2) oral cancer examinations are not frequently performed, and 3) existing lesions are often overlooked by the general dentist. Delays from the onset of signs/symptoms to clinical diagnosis are also common[10].

Gingival carcinomas in particular are usually painless and are most frequently found in the keratinized mucosa of the posterior mandible. If the tumor presents on the maxillary ridge it can extend onto the hard palate. Tumors in dentate areas are easily mistaken for periodontal disease or pyogenic granuloma. Gingival carcinomas have a tendency to destroy underlying bone, thus causing tooth mobility. Of all intraoral carcinomas, gingival SCC is least associated with tobacco smokers and has a higher frequency in females[1].

As is the case for many patients with oral SCC, a surgical resection was necessary to remove the tumor. This may be very devastating both from a psychological and physical standpoint. The rationale for creating a surgical obturator has three purposes. The first purpose is to maintain function. The obturator acts as a matrix for the surgical dressing and allows the patient to swallow and speak normally. The second purpose is to maintain hygiene. The obturator separates the surgical site from the oral cavity. Finally, the obturator helps the patient maintain their self image so they can continue to function socially[11].

Conclusion

The gingival SCC in our patient presented as generalized moderate chronic periodontitis. After a flap was created for debridement, the lesion was biopsied due to abnormal appearance of granulomatous tissue. A biopsy of this lesion could have easily been overlooked because clinically the lesion could have been attributed to local etiology or chronic periodontitis. Also, common etiology, risk factors, and typical location for oral SCC were not present in our patient. Overall, this case report is a reminder of the importance of oral cancer screening and appropriate referral if the diagnosis is questionable.

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Maxillary Sinusitis Due to Apical Rarefying Osteitis

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Abstract
Odontogenic infections are common occurrences, but the incidence of sinusitis seen with these infections accounts for approximately 10%-12% of cases of maxillary sinusitis.[1,2] If a periapical infection of a maxillary tooth violates the Schneiderian membrane, infection will likely spread into the sinus, leading to sinusitis. A thirty-one year old woman in good general health presented for a Cone Beam Computed Tomography (CBCT) scan to evaluate potential dental etiology of her left-sided Bell's palsy. The CBCT scan revealed the presence of an apical radiolucency associated with an endodontically treated tooth #15. The left maxillary sinus was filled with a significant amount of inflammatory tissue in a bubble-like pattern. However, no perforation of the cortical floor of the sinus was noted between the apical inflammatory lesion and the maxillary sinus. Therefore, although causality could not be established between the two lesions, clinical and radiographic information indicated a relationship. Management of this condition required concomitant therapy of the odontogenic infection and sinusitis.

Introduction
Periapical inflammatory lesions come about as the result of chronic infection or trauma to the pulpal tissues and the resultant necrosis of the dental pulp. Toxins produced by pulpal necrosis can then cause chronic or acute apical inflammatory lesions, such as a periapical granuloma, radicular cyst, or apical abscess.[3] Diagnosis of periapical granuloma, also known as chronic apical periodontitis, can be made when there is granulation tissue at the apex of a nonvital tooth. The lesion may be either chronic or subacutely inflamed. The lesion first develops as an acute apical periodontitis, where neutrophils release prostaglandins, which activate osteoclasts that resorb the surrounding bone. As the body continues to wall off the infection, chronic inflammatory cells begin to dominate the host response. The lymphocytes release mediators stimulating osteoclasts and fibroblasts, which histologically will appear as inflamed granulation tissue surrounded by a fibrous connective tissue wall. As a result of these actions, chronic lesions are often asymptomatic. Bone resorption can be detected radiographically as a periapical radiolucency, which can be discovered on routine radiographic examination. The affected tooth will generally reveal a loss of apical lamina dura.[4] The lesion can be ill-defined, showing a gradual transition from the surrounding normal trabecular bone into the abnormal bone pattern of the lesion. Alternately, it may have a well-defined periphery with a corticated border, attributed to the stimulation of osteoblastic activity in the surrounding bone.[5] Due to the variation in size, the radiographic appearance is not sufficient to confirm a diagnosis of a periapical granuloma, since periapical granuloma can transform into a cyst or an abscess (and vice versa) without a radiographic change.[4]

The Schneiderian membrane is the thin epithelial lining of the maxillary sinus. In the rare event that the Schneiderian membrane is perforated by a dental pathosis that has crossed the cortical boundary of the sinus, a maxillary sinusitis can manifest.[1] Odontogenic sinusitis accounts for only about one tenth of all cases of maxillary sinusitis.[1,2] In addition to periapical infection, sinusitis related to odontogenic causes also occur when the Schneiderian membrane is violated by other pathologic lesions of the jaws and teeth, maxillary (dental) trauma, or by iatrogenic causes such as complications of implant placement or maxillofacial surgery.[1] The healthy maxillary sinus contains a normal bacterial flora that may include a combination of aerobic and anaerobic bacteria. Due to local or systemic factors, a bilateral maxillary sinus infection may develop, leading to the thickening of the sinus membrane and improper drainage caused by the blockage of the ostium.[4,6] In the event of focal areas of inflammation within a single sinus, a unilateral sinusitis may occur, which can be attributed to an odontogenic source.[4] Maxillary sinusitis of odontogenic origin is usually chronic.[6]

Computed tomography (CT) is currently the modality of choice for evaluating the presence and extent of disease as well as any anatomic predisposing factors in patients with symptoms of chronic maxillary sinusitis.[7] CBCT is a recent technology that was first developed for angiography in 1982 and later applied to maxillofacial imaging. CBCT uses a divergent or "cone-shaped" source of ionizing radiation and a two-dimensional area detector fixed on a rotating gantry to acquire multiple sequential projection images in one complete revolution around the area of interest.[5] Although soft tissues cannot be differentiated, hyperplastic tissue in the sinus can usually be easily visualized on CBCT scans.

Case Report
A 31-year-old female presented to a private periodontist's office with a complaint of left-sided facial numbness and a feeling of "fullness" in the left side of her face. The symptoms were of recent onset. Previous medical history was otherwise unremarkable. Clinical examination revealed a marked "droop" to the left side of her face. She was unable to smile on the left side. A tentative diagnosis of facial nerve paralysis was made after eliciting the Bells's sign. A CBCT scan was prescribed to evaluate potential dental or otologi-

cal etiology of her left Bell's palsy and to seek a causative factor for the feeling of "fullness". Previous dental history was significant for endodontic treatment of an upper left second molar and multiple restorations. The CBCT scan was sent to Columbia University College of Dental Medicine for consultation and radiology report.

The CBCT scan viewed in panoramic reconstruction, as well as multiplanar reconstructions, revealed the presence of an apical radiolucency associated with endodontically treated tooth #15 *(Figure 1)*. It was approximately 1 cm in its greatest dimension and was surrounded by a corticated border. The lesion appeared to have caused remodeling of the floor of the sinus superiorly in the region *(Figure 2)*. The maxillary sinus was filled with a significant amount of inflammatory tissue in a bubble-like pattern *(Figure 3)*. Non-contributory findings included several restored teeth. The

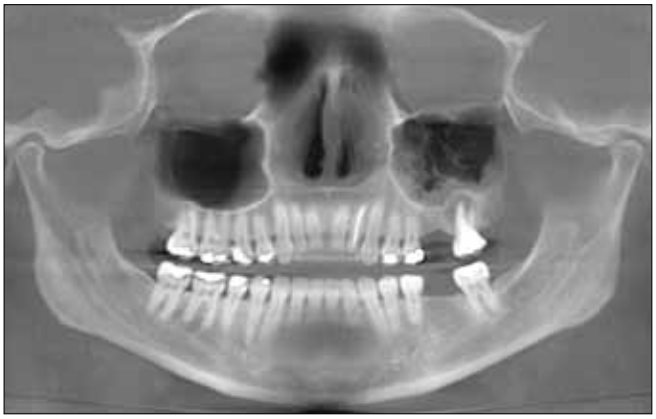

Figure 1
CBCT Panoramic reconstruction. Apical inflammatory lesion associated with tooth # 15 and maxillary sinusitis can be seen on the patient's left side.

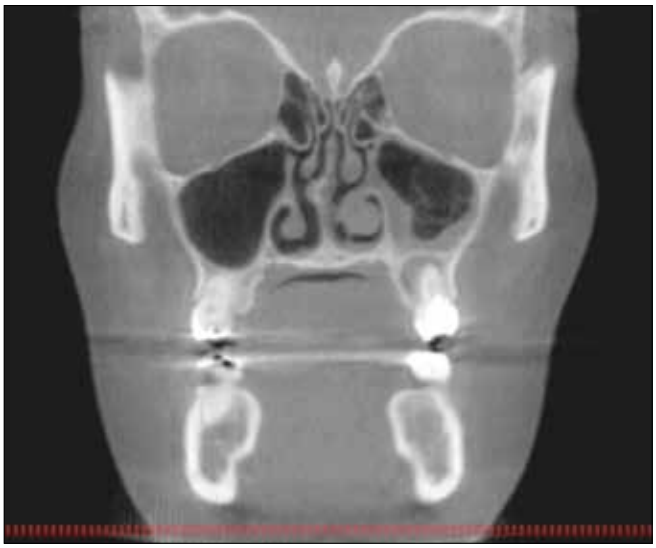

Figure 2
CBCT Coronal reconstruction. Remodeling of the floor of the left maxillary sinus is noted.

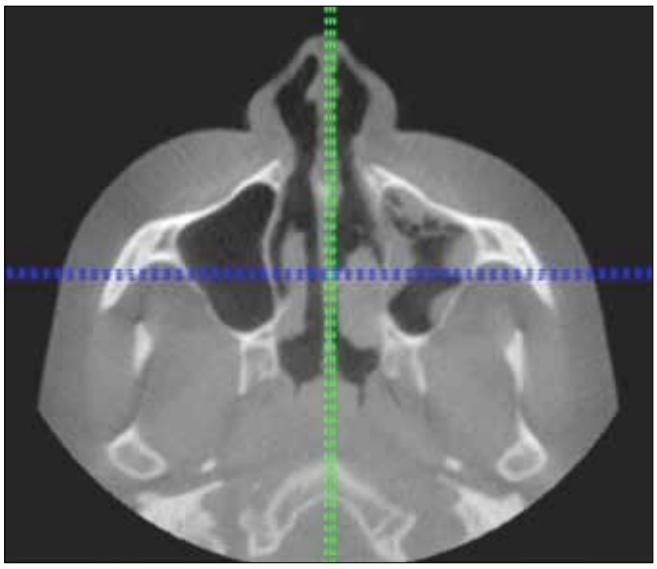

Figure 3
CBCT Axial reconstruction demonstrating hyperplastic soft tissue in the left maxillary sinus.

contralateral maxillary sinus, ethmoidal air cells, and the sphenoid sinus were normally aerated. No communication was noted between the apical inflammatory lesion and the maxillary sinus. The diagnoses of apical rarefying osteitis and acute sinusitis were made based on the clinical and radiographic information.

The patient was placed on antibiotics for the sinusitis and the offending tooth #15 was extracted uneventfully. The patient was referred to a neurologist, who was able to confirm the diagnosis of unilateral Bell's Palsy. The patient was kept on follow-up.

Discussion

Odontogenic infections are common in occurrence, but the incidence of sinusitis seen with these infections is extremely low.[1,2] Most infections will spread along the path of least resistance, which is generally through the thinner bone of the lateral wall of the maxillary alveolus and present as facial or intraoral swellings and abscesses.[1] The thick cortical bone of the floor of the maxillary sinus usually serves as an effective barrier, preventing the direct penetration of odontogenic infections into the maxillary sinus. As in this case, when odontogenic infections spread into the sinus, the second molar is often involved due to its root having the closest distance to the sinus floor (mean distance of 1.97mm). Also, it is common for maxillary posterior teeth to be associated with sinusitis, because as the maxillary sinus expands during development the maxillary teeth roots may protrude into the forming sinus cavity, resulting in the root apices being surrounded by sinus mucoperiosteum (Schneidarian membrane).[2] Root canal therapy of a maxillary tooth is also a potential cause of maxillary sinusitis,

due to instrumentation that may introduce bacteria close to the sinus cavity, or extrusion of material used in root canal therapy into the sinus.[1,6] Whether the sinusitis is due to the spread of odontogenic infection or caused iatrogenically via root canal therapy, a direct communication exists with the sinus. This case is unique in that, although a mucositis is apparent in the left maxillary sinus, a perforation of the sinus membrane or extrusion of material from the previous endodontic treatment of the tooth is not apparent.

Radiology is an important tool in establishing the diagnosis. The advent of CBCT greatly facilitates access to the internal morphology of soft tissue and skeletal structure, and causes no magnification errors because of geometric distortions.[8] Although a panoramic radiographic view is helpful for evaluation of the maxillary teeth to the sinus, CBCT is better suited to visualize bone and soft tissue outlines with multiplanar reconstructions. In this study the initial purpose of ordering the CBCT was to evaluate a potential dental or otological etiology of the patient's left-sided Bell's Palsy. Since the significant findings of the CBCT examination were confined to the maxilla (the alveolus and the sinus), it can be inferred that the apical rarefying osteitis was incidental to the Bell's Palsy and no direct connection between the two can be made. Therefore, while the CBCT findings did not confirm the diagnosis of Bell's Palsy, the three-dimensional images gave the radiologist the ability to view communications between the maxillary sinus and the maxillary teeth, as well as mucosal changes of the sinus.[7,9] In cases of apical rarefying osteitis, the radiographic term used to describe periapical inflammatory lesions, a "halo shadow" may be noted within the maxillary sinus. This "halo shadow" is the result of an inflammatory periosteal reaction, which results in a thin layer of new bone produced by the inflamed periosteum within the maxillary antrum.[5] In the presented case a "halo shadow" is present, but an oro-antral communication is not noted.

In this case, while the radiographic findings do not reveal oro-antral communication, the maxillary sinusitis is most likely due to the odontogenic inflammatory lesion. Management of this condition requires concomitant management of the dental origin and the associated sinusitis to ensure complete resolution of the infection.[2] In order to eliminate the source of the infection, extraction or root canal therapy of the infected tooth is recommended.[10] However, if root canal therapy is unsuccessful, it is advisable that the tooth be extracted. For this patient, this was the option selected, since tooth #15 had previously been endodontically treated. It is recommended that antibiotic therapy effective against oral flora and sinus pathogens be taken for 21 to 28 days.2 The oral flora implicated in maxillary sinusitis of odontogenic origin is similar to that of usual oral and jaw infections of odontogenic origin, which is typically a combination of aerobic and anaerobic bacteria including streptococci, *Bacteroides, Veillonella, Corynebacterium, Fusobacterium, Peptostreptococcus,* and *Eikenella* species.[1] A chronic sinusitis has a greater percentage of anaerobic bacteria, mainly because the obstructed ostium and resultant inflammation of the sinus produce changes in the Schneiderian membrane and reduce the oxygen tension within the sinus. The antibiotic of choice is still amoxicillin, but with increased resistance due to ß-lactamase-producing bacteria alternative antibiotic therapy is now used (eg. amoxicillin-clavulanic acid, cephalexin, cefoxitin, ceftriaxone, azithromycin, clindamycin).[1] Along with antibiotic therapy, the use of systemic and local intranasal decongestants also aids in reducing mucus production, altering the environment of the sinus cavity, and improving ciliary function. Saline nasal sprays also aid to mechanically loosen bacteria and alleviate side effects of nasal mucosal dryness.[1,2]

Conclusion

Concomitant apical rarefying osteitis and unilateral maxillary sinusitis are uncommon events. Without direct evidence of perforation of the cortical boundary of the sinus and communication between the sinus and the apical inflammatory lesion, it is difficult to assign causality. Nonetheless, the proximity of the two lesions in the presented case, along with the absence of detectable lesions in the other paranasal sinuses in this patient do suggest a relationship. CBCT imaging provides three-dimensional viewing of the affected regions, along with accurate measurements and correct anatomic relationships between adjacent structures.

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Idiopathic External Root Resorption: A Case Study

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Abstract
External root resorption is an uncommon occurrence in dentistry and there are very few cases and sparse literature in the area of generalized idiopathic resorption. It is the purpose of this article to highlight a clinical case presented to the post-doctoral Periodontics clinic at Columbia University, College of Dental Medicine. Through this, the etiology, characteristics and possible treatment will be highlighted to describe the process of idiopathic external root resorption.

Introduction
External root resorption has been described as early as 1930.[1] Since that time, the etiology has been determined to be primarily due to traumatic injury. This includes injury to the root surface caused by trauma, orthodontic treatment, periapical inflammation, and neoplastic disease of the jaw.[1] However, there has been little documentation and literature associated with idiopathic external root resorption. There are even fewer reports of *generalized* external root resorption in which resorption affects the entire dentition. In general, idiopathic root resorption can be described as resorption in the absence of any of the traditional etiologies described above. There may be numerous nontraditional reasons for root resorption, including a genetic predisposition and perhaps maybe undetected minor trauma. However, these nontraditional etiologies have not been conclusively studied or documented.[2] Thus, it is clear that external root resorption without any external inducing factors remains unclear to this date.[3]

Although the causes of idiopathic root resorption are not clear, the pathophysiology has been extensively studied and described. External root resorption, whether from trauma or idiopathic reasons, is due to an inflammatory response. The inflammatory response includes the presence of cytokines, proteinases, collagenases and multi-nucleated osteoclasts that resorb the cementum and dentin of the root, causing the root to be blunted and lose its natural anatomy.[4] Additionally, the hard tissues are usually protected by layers of osteoblasts, cementoblasts and the periodontal ligament. The loss of the periodontal ligament, from sources such as inflammation, causes the exposed cementum to become chemotatic to clastic cells.[4,5,6]

In general, external root resorption can be subdivided into three types. This includes *surface resorption, replacement resorption associated with ankylosis and inflammatory resorption*.[2] The first, *surface resorption,* is when a denuded root surface has osteoclast-activating factors that attract osteoclasts and cementoclasts, causing resorption of the external surface of the root. However, besides resorption at the sites, there is a cyclic balance of resorption and deposition from nearby cementoblasts that will try to repair the damage.[2,4] External root resorption occurs as a result of an imbalance, in which resorption activity exceeds that of deposition.

The second, *replacement resorption with ankylosis,* occurs when surface resorption stops and bone cells invade the site and establish themselves in the area and thus form bone on the external surface of the root. This prevents the normal reparative cells of the periodontal ligament from depositing at the site of resorption and causes the fusion of the tooth to the bone (anklyosis).[2] Because of this ankylosis, the bony area of the tooth as well as the surrounding alveolar process are subject to bodily turnover processes, which continually lay new bone around the root surface.

The last process, *inflammatory resorption,* can further be divided into two types; *peripheral inflammatory root resorption* (PIRR) and *external inflammatory root resorption* (EIRR). Peripheral inflammatory root resorption is due to destruction by cementoblasts through cementoclast-activating factors derived from the periphery of the root. External inflammatory root resorption is caused by a necrotic pulp that stimulates the external clastic cells. In both PIRR and EIRR, the osteoclasts act as specialized macrophages to remove the infected calcified tissue from the body.[6] Thus, it is clear from the above descriptions that external root resorption is a stalwart reaction to the root surface of the dentition and that it rarely occurs in the absence of a specific etiologic factor.

Case Presentation
A 45 year old female presented to Columbia post-doctoral Periodontic clinic with a referral from St. Luke's Hospital. The patient's chief complaint was that "her teeth were loose." Her dental history included 13 different restorations and a history of loose teeth. She denied having any previous history of orthodontic treatment or any factors that are normally associated with external root resorption. The patient reported scaling done bi-annually and had acceptable hygiene (brushes twice a day, but does not floss regularly). The past medical history included hypercholesterolemia and asthma as a child, but the patient denied any significant systemic health issues. She is currently medicated with Tricor, Calcium, and Vitamin D supplements. She has no known drug allergies and does not smoke or consume alcohol.

Upon examination, both extraoral and intraoral examinations showed all hard and soft tissues to be within normal

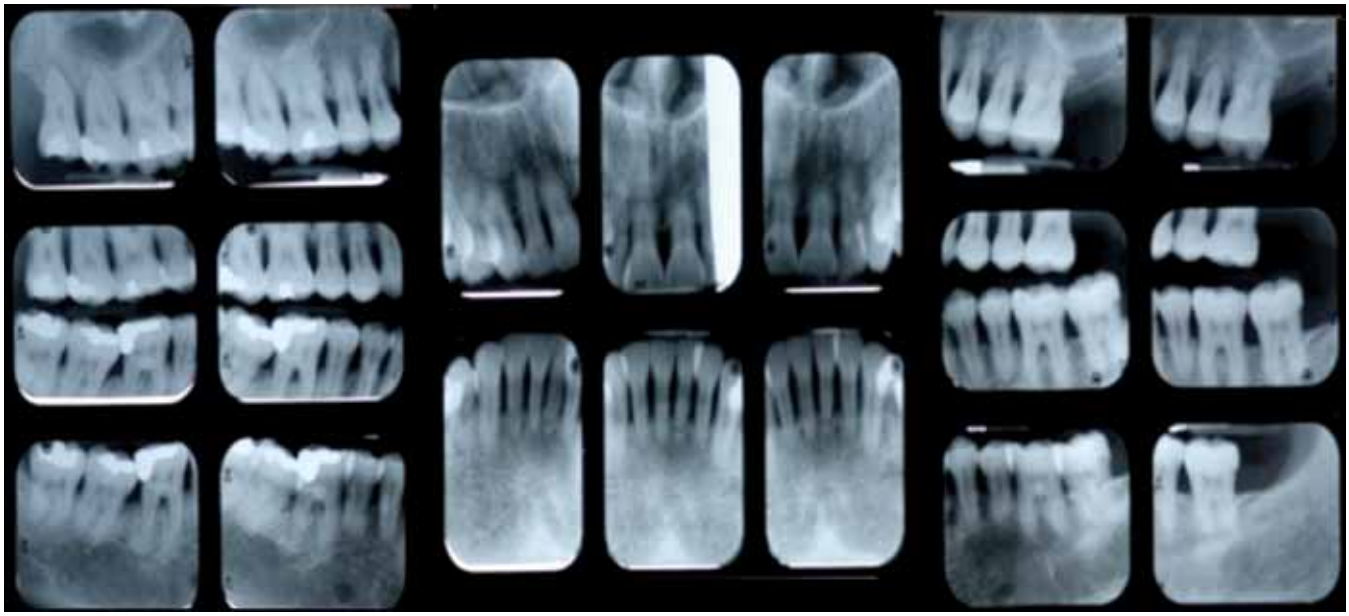

Figure 1-1
A full-mouth-series of the patient showing generalized bone loss, with furcation involvements on most molars. Additionally, the root apices are blunted and have lost their original anatomy and form.

limits. Her oral hygiene was evaluated and diagnosed as poor because of generalized moderate plaque buildup. The amount of plaque had led to the development of generalized, pink-red gingival that was not swollen. Periodontic examination revealed 20 percent of sites with pocket depths of 5mm or greater in the posterior regions, furcation involvements on roughly 60 percent of molars, bleeding-on-probing at approximately 50 percent of all sites and mobility of a majority of her teeth.

A full-mouth-series of radiographs was taken and shown in Figure 1-1. It is evident from the full-mouth series that every tooth has undergone extensive loss of root structure.

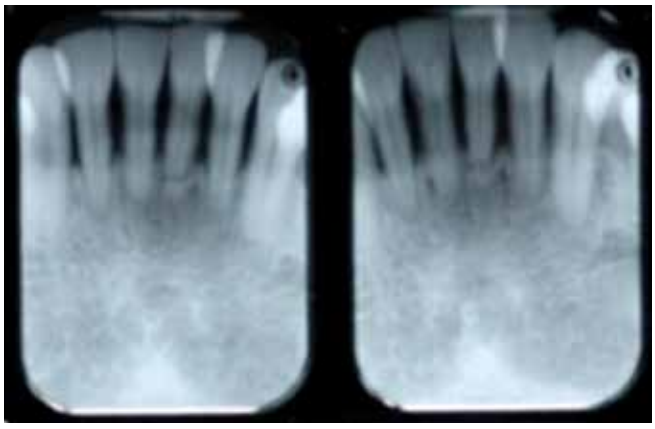

Figure 1-2
Periapical radiograph of the anterior mandibular segment of patient presenting with idiopathic external root resorption. It is observed that the patient has decreased amounts of bone with blunting of the apices of the teeth as indicated.

Specifically, it can clearly be shown that generalized blunting of roots, a decrease in root length and a loss of anatomy. These issues are generalized, having effects on all teeth in the dentition. To illustrate this, Figure 1-2 shows these features in the anterior mandibular region and Figure 1-3 shows this in the left posterior region.

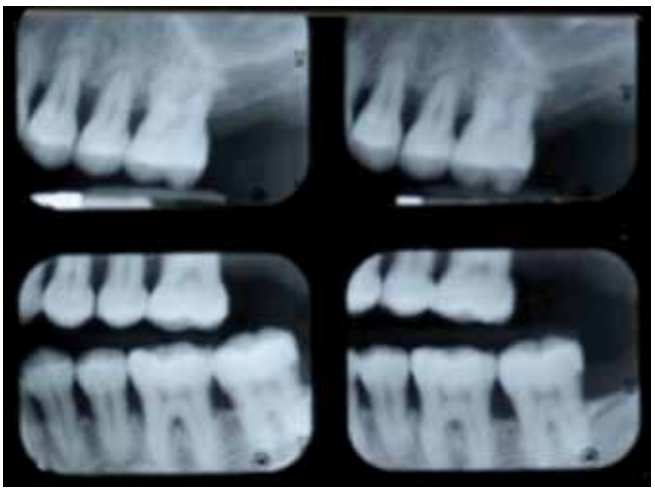

Figure 1-3
Periapical radiographs and bitewings of the posterior left segment of patient presenting with idiopathic external root resorption. It is observed that the patient has decreased amounts of bone with blunting of root tips, loss of root length and loss of anatomy.

Based on the clinical and radiographic evaluations, the patient's prognosis was determined that she would eventually lose all her teeth and become edentulous. Currently, there was not any specific treatment modality to stop her con-

dition but rather, aggressive periodontal scaling and root planning was planned to possibly help arrest the inflammatory response that was causing the root resorption.

Discussion

Generalized idiopathic root resorption is a rare and uncommon occurrence in dentistry. Despite the fact that most practitioners will never observe this phenomenon first hand, it is still important to document these cases and note any possible treatments. One of the earliest cases of idiopathic external root resorption was described in 1930, where a 36-year-old female had generalized progressive cervical root resorption. Strangely, it was then believed that this patient's root resorption was caused by a "functional hepatic disturbance."[7] The treatment to halt the root resorption was dietary modification until the liver returned to normal. Although the determined etiology in 1930 was unsound, the notion of a link between systemic illnesses and root resorption is accurate.[1,4,8,9] To clarify, previous literature has shown that systemic illnesses associated with external idiopathic root resorption include hypophosphatasia, hyperparathyroidism, renal disease, hepatic disease, bone dysplasia, Papillon Lefevre syndrome, endocrine disorders and so forth.[1,4,8,9] However, the patient who presented to the Columbia dental clinic did not have any systemic features that have been documented with external root resorption. Additionally, the patient did not have any of the more common etiological factors that are associated with external root resorption such as excessive pressure, orthodontic treatment, occlusal trauma, impacted teeth, periradicular infection or even tooth bleaching.

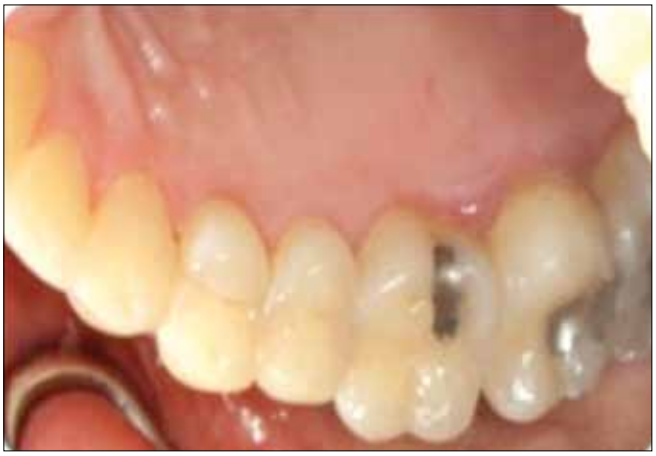

Figure 2-1
A photograph of the patient's upper right posterior dentition, which did not show any signs of adverse loading or observable etiological factors.

Thus, according to her medical and dental history, there are no known systemic or common etiologic factors associated with this patient's condition. What is present, however, does not seem to be associative with her disease. Primarily, the patient's poor periodontic health and generalized plaque build-up could play a role in the external root resorption. However, similar levels of plaque in other patients do not cause such generalized destruction. Moreover, this patient's dentition did not show any signs of adverse occlusal loading or wear, as illustrated in *Figure 2-1*. One notion that has been discussed in Saravia's study in 1989, is that there could be a genetic predisposition towards external bone resorption.[9] It is conclusive that the cause of her root resorption cannot be isolated to identifiable causes. This is also in accordance to Kerr and his conclusion that despite lab examinations and histologic studies, there has been little evidence of a direct causative pathology.1

Despite the fact that the source of the patient's root resorption is unresolved, the condition of idiopathic root resorption can be characterized and therefore aid in the identification and diagnosis in other patients. From previous literature, idiopathic root resorption can be characterized by several factors. According to Kerr, predisposing factors include female gender, being in the age range of 30-40, and perhaps high levels (high spectrum of normal) of alkaline phosphatase.[1] However, other researchers have described cases ranging in age from 14-39 and external root resorption dominance in males by a ratio of 11:1.[4] Clearly, larger studies must be conducted to resolve this discrepancy. Furthermore, there may be a genetic relationship. Multiple studies have shown a "tentative genetic association" whether by an autosomal dominant inheritance pattern or recessive pattern.[4,9] But these studies were small and not deemed of statistical significance. Nevertheless, these may aid in diagnosing a patient by asking them if "such an event has occurred in their families." Clinically, these patients usually present with normal-appearing dentition and periodontia, tooth mobility, and a lack of periodontal inflammation.[4] Furthermore, Cholia and his peers have stated that idiopathic root resorption is usually associated with the premolar and molar areas. Yet this case is an exception and shows root resorption to be affecting her entire dentition. Radiographically, there may be a loss of anatomy of the root structure with blunting of the apices with absence of periapical radiolucenies. Histologically, clastic cells are present in abundance. These characteristics may aid practitioners in recognizing idiopathic external root resorption and provide their patients with some treatment options.

Treatments described in the past literature include modification of any existing adverse occlusal loading, endodontic treatment of the affected teeth and possible inhibition of the clastic cells responsible for the resorption process. To begin, the most non-invasive solution is to remove any adverse loading or trauma inducing factors. However, most idiopathic root resorptions are rarely so simple and it is often that the reasons for this resorption are complex and undisclosed.

A more invasive procedure has been described in past literature where root-canal therapy could halt external root resorption.[5] This was also shown to halt root resorption due to trauma or even from avulsed teeth.[5] Endodontic literature has shown that the high pH of the calcium hydroxide used in endodontic treatment, can permeate through the dentinal tubules to the root surface and can change the root surface environment to prevent inflammation.[5] It is thought that calcium hydroxide is beneficial for root resorption because of its high calcium ion concentration which promotes healing, calcification and remineralization.[5,10] Additionally, the alkaline pH of calcium hydroxide stimulates matrix formation by formative cells, as well as neutralizes the acidic products of the resorptive cells.[5,10] Previous studies have shown that root canal therapy has halted the external root resorption process and patients present as post-operatively asymptomatic. However, in this case, the practicality of performing root canals on all her teeth does not seem like a viable option.

Another modality proposed for external root resorption is inhibition of the clastic cells in the resorption process. Clastic cells, such as osteoclasts and fibroclasts can be inhibited via calcitonin. This is similar to the action of calcitonin products, such as Cibacalcin, which reduce bone turnover in the body in conditions such as osteoporosis.[4] Another option would be stimulation of osteoprotegerin (OPG) production which is known to inhibit osteoclast activity. Such treatment options have yet to be researched extensively and there are no studies to confirm that they would work effectively on a patient.

Conclusively, with all these proposed treatment modalities, there has yet to be an established treatment option. Perhaps with more cases of generalized idiopathic root resorption, more research on appropriate treatment can be further studied. As a result of the current level of research, the prognosis for the patient was described as poor as it was expected that she would lose her remaining teeth. Her idiopathic root resorption presents as an interesting case to review the pathophysiology, possible etiologies, predisposing factors and possible treatment.

Conclusion

To conclude, there is much that is unknown about generalized idiopathic external root resorption. Past studies and literature have provided very little information about this rare phenomenon. The presentation of this 40-year old female to PG periodontics at Columbia University provided a valuable insight into this occurrence and re-established the stance that further study is required in order to understand and properly treat this seemingly untreatable condition.

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Ramsay Hunt Syndrome presenting as TMD: A Case Report

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Abstract
Ramsay Hunt syndrome (RHS) results from the reactivation of the varicella zoster virus in the pre-auricular region that is associated with facial paralysis. Additional symptoms may include tinnitus, hearing loss, nausea, vomiting, vertigo, synkinesis, and nystagmus. Temporomandibular Joint Disorder (TMD) is a term that covers a range of clinical problems that involves the masticatory muscles, the temporomandibular joint (TMJ), and the surrounding anatomy. This article presents a case report that describes a patient with RHS who presented with pre-auricular pain and was diagnosed with TMD.

Introduction
James Ramsay Hunt, a professor at Columbia University, first described Ramsay Hunt syndrome (RHS) as varicella zoster virus (VZV) oticus in conjunction with peripheral facial nerve paralysis.[1] Closely associated with Ramsay Hunt syndrome are symptoms of tinnitus, hearing loss, nausea, vomiting, vertigo, synkinesis, and nystagmus.[2] These symptoms typically present unilaterally.[3] RHS is second to Bell's palsy as the most common cause of atraumatic peripheral facial nerve paralysis.[4] Primary VZV infection can lead to dormant cranial nerve infection. Ramsey Hunt syndrome results from a reactivation of the VZV in the geniculate ganglion;[5] therefore, a positive history of VZV infection or chicken pox is essential for diagnosis. Reactivation of VZV may lead to deep facial pain that radiates to the ear, followed by the appearance of a vesicular rash on the geniculate region of the ear.[6]

Standard treatment of RHS includes administration of Acyclovir (250 mg three times daily IV or 800 mg five times daily PO) and Prednisone (1mg/kg/day PO for 5 days followed by a 10 day tapering).[7] Early diagnosis and treatment are the most important predictors for successful recovery. Recent studies have shown that compete recovery from RHS symptoms occurred 75% of the time when patients with RHS were treated within 3 days of reactivation compared to 30% complete recovery when patients received treatment 7 days post reactivation.[7] Moreover, 50% of patients who did not receive treatment in the first 3 days progressed to complete loss of facial nerve response.[7]

TMD is a major form of non-odontogenic orofacial pain.[8] There are a number of different types of TMD, all of which involve the masticatory muscles, the TMJ, and/or the surrounding structures. The most prevalent symptom of TMD is pain. The pain is usually limited to the muscles of mastication and the pre-auricular area and has been described as jaw pain, earache, headache, and facial pain. The pain may be exacerbated by chewing and other jaw movements. Many patients with TMD have clicking or popping sounds when they open and close their jaw.[9] Up to 75% of the world's population has at least one sign of joint dysfunction and up to one third have at least one symptom.[10,11] The most common form of TMD is articular disc displacement, which occurs when the disc is abnormally positioned in relation to the condylar head.[9] Anterior displacement is the most common position.[12] This type of TMD is not usually associated with pain or limited jaw movement and therefore does not typically require treatment.[13]

According to the literature there have been no cited cases of RHS manifesting with TMJ pain.[14] We report a patient in whom pain due to RHS in conjunction with signs generally associated with TMD, led to a delay in correct diagnosis.

Case Report
A 43-year-old woman presented to the Columbia University Center for Oral, Facial, and Head Pain, with a 4-day history of gradually increasing right-sided TMJ pain, which was most severe in the pre-auricular region. She had no fever, vomiting or neurologic symptoms. Chewing and pressure resulted in exacerbation of pain. Two days prior to presentation she had been diagnosed with TMD and was prescribed Ibuprofen (600mg TID) and Valium (5mgs) for pain management. Her past medical history was significant for childhood varicella infection.

On examination, the patient reported pain in the right pre-auricular area upon active mouth opening and left laterotrusive movement. Active mouth opening was restricted to 43mm with slight deviation to the right side. Maximum opening could be stretched to 46mm and was associated with an increase in pain. Palpation of the right TMJ capsule was also associated with an increase in pain. The pain was described as burning, stinging, and stabbing. There was an audible reciprocal clicking of the right TMJ that was eliminated by 1mm of opening. Additionally, there was pain on palpation of the right masseter and temporalis muscles, as well as pain to light touch in the right pre-auricular area. Cranial nerves II-XII were otherwise intact; pupils were equal

and reactive to light, and strength and sensation were normal. Erythema and slight rash were present over the concha and antihelix of the right ear. (Figure 1) The diagnosis of herpes zoster was made and the patient was prescribed oral acyclovir. Further diagnoses of TMJ disc displacement and capsulitis, as well as myofacial pain was made, and treatment was deferred until after the herpes zoster infection could be managed.

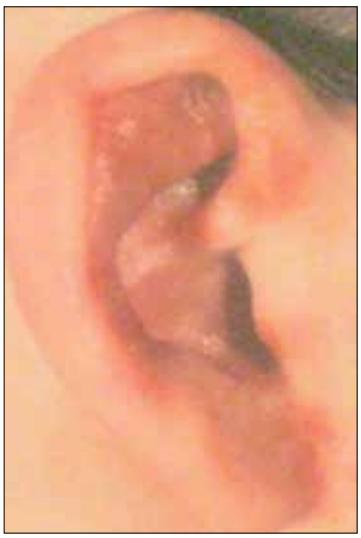

Figure 1
Erythema and slight rash were present over the concha and antihelix of the right ear

Four days later, the patient returned to the clinic with worsening symptoms. She had not taken the prescribed acyclovir and reported increased pain in the right pre-auricular region, inability to close her right eye, vertigo, and tinnitus. There were vesicles present over the concha and anti-helix of the right ear and there was right-sided facial weakness.

The diagnosis of Ramsay Hunt syndrome was confirmed and the patient was admitted to the hospital for intravenous acyclovir and steroids. MRI with contrast revealed mild linear enhancement of the right internal auditory canal consistent with enhancement along the 7th and 8th cranial nerves and throughout the remainder of the 7th cranial nerve.

At 1-month follow-up, the patient's symptoms, including the vesicles, facial weakness, TMJ pain, and myofacial pain, had resolved. All that remained of her initial complaint was the right-sided TMJ clicking, which did not require treatment.

Discussion and Conclusion

Ramsay Hunt syndrome is diagnosed clinically as peripheral facial nerve palsy in association with zoster otitis.[3] This diagnosis is based on patient history of previous VZV infection and neurological examination.[3] It has long been held that this syndrome is due to reactivation of the VZV in the geniculate ganglion resulting in various neuropathic symptoms from the nerves leaving this ganglion.[5] Various studies have reported RHS in association with concurrent VZV infections in CN VIII, IX, X, XI, XII and upper cervical nerves that stem from widespread contamination of VZV via nerve anastomoses[2] or connecting blood vessels.[15]

The difficulty with diagnosing RHS is usually due to its similarity to Bell's palsy, especially when the vesicular rash is absent. This form is known as RHS zoster sine hepete and in recent studies has been shown to account for up to 19% of Bell's Palsy diagnoses.[16] Fortunately, there are few negative consequences of misdiagnosing RHS for Bell's palsy as studies show that both are treated effectively with prednisone and acyclovir.

However, missing a diagnosis of RHS due to TMD, which is a rare occurrence, can have negative consequences. As was stated earlier, early detection and treatment is paramount in decreasing the probability of permanent neuropathies associated with RHS. RHS and various forms of TMD may include symptoms of pain in and around the ear. Although this case manifested the hallmark signs of RHS, it is unique in that the patient presented with TMJ pain as well. It is likely that the patient's TMJ disc displacement existed prior to her RHS presentation and accordingly it continued after her symptoms had resolved. It is possible that pain from the localized inflammation associated with RHS was worsened by jaw movement, leading to muscle guarding and subsequently more pain. The pain was located in the masticatory muscles and around the TMJ, both of which are common features of TMD. These symptoms may mask the diagnosis of RHS; however, TMD is not associated with allodynia, rash, vesicles, or facial nerve weakness. These clinical signs and symptoms are not usually associated with TMD and should prompt the clinician to consider alternate sources for a patient's pain complaint. While conservative management, including NSAIDs, is often the first step in managing TMD symptoms, in a case such as this, an incomplete diagnosis could delay proper treatment and thereby negatively affect the patient's prognosis for full recovery. After the correct diagnosis of RHS was determined and treated, all symptoms, including the myofacial pain and TMD associated pain had resolved. The clinician treating patients with TMD must be aware of conditions that should be included in the differential diagnosis, especially when there are signs or symptoms that are not explained by problems involving the temporomandibular joint or surrounding musculature. These characteristics must be fully examined and analyzed to obtain a proper diagnosis and have the appropriate treatment applied.

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VACTERL Association: A Dental Case Study

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Abstract
VACTERL association is an association of congenital anomalies, which occur together with enough frequency that their simultaneous presentation cannot be attributed to random chance. This case describes the dental findings of a 3-year-old male with VACTERL association that presents with bilirubin-discolored teeth, gingival overgrowth, and abundant calculus. In this report we consider the unique dental challenges that face patients with this association, as well as various methods in approaching their oral health care.

Introduction
'Associations' are used to describe the existence of a specific set of malformations that tend to occur together more frequently than can be attributed to chance. One such 'association' is VATER association, in which patients present with a non-random group of congenital anomalies that include: defects of the vertebrae (v), anal atresias (a), tracheoesophageal fistulas and atresias of the esophagus (te), and renal and radial limb abnormalities (r). Recently, it has been suggested that VATER association be expanded to include congenital heart lesions (c) and limb defects (l), and can thus be referred to as VACTERL association.[1] Diagnosis requires the presence of at least three of the previously listed elements.[2] Cleft lip and palate also present more often than would be expected in patients with VACTERL association.[3] The degree of involvement of any one element of VACTERL association is case-dependent, and consequently, each patient is truly distinct.[2,4] The underlying causes of this association remain to be elucidated.[1]

Early documentation of this group of associated birth defects appeared more than 30 years ago,[5-9] and the incidence of each associated component has not been well quantified in contemporary literature. The vast spectrum of anomalies that exist in VACTERL association make it very difficult to create a precise definition for the association and thus to develop studies with the appropriate patient populations.[10] In addition, there are many VACTERL-like cases that present features of the association, which may have actually resulted from other syndromes or single gene disorders such as Feingold, Charge, Townes-Brocks, Pallister-Hall and 22q11 deletion syndromes, as well as Fanconi anemia.[11] These factors make it very difficult to obtain accurate information and statistics for this association.[10]

VACTERL association affects about 1 in 5,000 live births and has been hypothesized to originate from the midline developmental field due to errors in blastogenesis.[1,12] The etiology remains unclear but is believed to be multifactorial. Certain chromosomal defects and deletions have been found in patients with VACTERL association but, to date, no single chromosomal abnormality has been implicated. Additionally, it has been proposed that exposure to certain environmental factors during pregnancy, such as sex hormones, can also influence this association.[3]

Patients can often be identified as having either the 'cranial' or 'caudal' phenotype of the association. The cranial phenotype often presents with esophageal atresia, defects of the preaxial limbs, and malformations of the thoracic vertebrae. In contrast, the caudal phenotype frequently includes defects of the lower vertebrae, renal malformations, anal atresias, and possible genetic anomalies.[2]

Children born with this association often require urgent surgical intervention immediately after birth because of the foregut and hindgut anomalies.[12] Approximately 70 percent of patients with VACTERL association are affected by esophageal atresia with tracheo-esophageal fistula (EA/TEF).[13] Several complications are possible after correction of EA/TEF, such as respiratory problems – respiratory arrest, apnea, bradycardia, and aspiration – leading to numerous bouts of pneumonia.[10] Up to 75 percent of patients with VACTERL association have been reported to have congenital heart disease. The most common heart defects seen with the association are ventricular septal defects (VSD), atrial septal defects, and Tetralogy of Fallot (TOF). Less common defects are truncus arteriosus and transposition of the great arteries. Patients may have a murmur at birth, however, absence of a murmur does not rule out congenital heart disease.[13] If a patient is suspected of having the association, a consultation with a pediatric cardiologist is recommended to determine whether antibiotic prophylaxis according to the American Heart Association guidelines is required.[13] Patients with VACTERL association do not typically present with learning disabilities or growth abnormalities, nor do they show dysmorphic facial features.[11] The following case presents interesting dental findings in a pediatric patient with VACTERL association.

Case Report
A 3-year-old male patient with a medical history significant for VACTERL association, Tetralogy of Fallot (TOF), gastroesophageal reflux, and asthma presented to the pediatric dental residency clinic at Columbia University Medical Cen-

ter for comprehensive dental care. The patient's past surgical history included a liver transplant and corrective TOF repair in 2006. The patient had a tracheotomy and gastrostomy tube. He was taking cyclosporine and antibiotic prophylaxis, which is recommended prior to all dental procedures that involve perforation of oral mucosa, manipulation of gingiva, or treatment of apical regions of the teeth.

Oral examination *(Figure 1A-C)* revealed a normal complement of primary teeth with evidence of generalized tooth discoloration, early childhood dental caries, generalized calculus accumulation, and gingival overgrowth. Extra-oral examination exhibited findings that were within normal limits. The patient was unable to have any dental treatment in an ambulatory setting due to his acute stress reaction and medical condition. Consequently, dental treatments were to be performed in the operating room under general anesthesia to eliminate infection and prevent unnecessary pain or prolonged suffering.

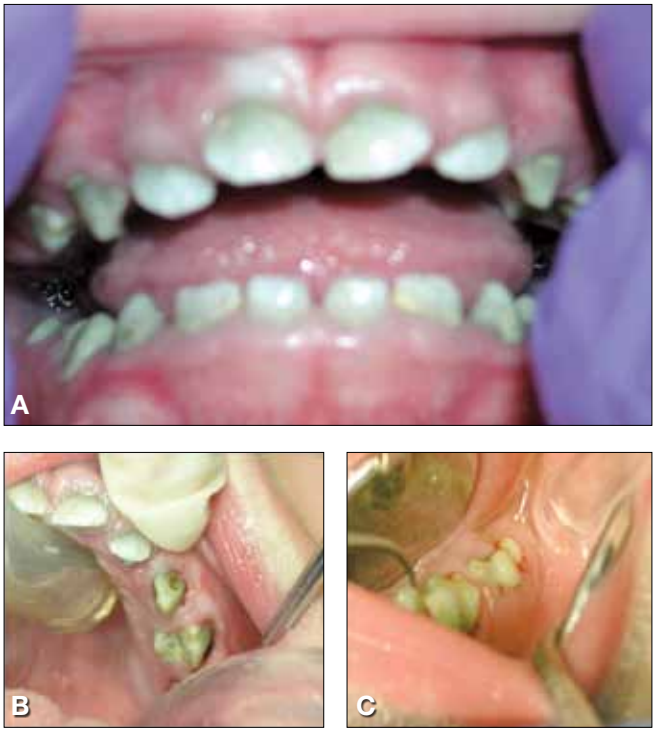

Figure 1
(A) Frontal view reveals green intrinsic staining of all primary teeth caused by hyperbilirubinemia during dentin development. Gingival soft tissue reveals generalized inflammation in response to calculus build-up. (B) Upper left quadrant of a patient with VACTERL association revealing calculus on the occlusal surfaces of molar teeth. Gingivitis in response to calculus accumulation and generalized staining of the teeth and can also be seen. (C) Gingival overgrowth caused as a negative side effect of cyclosporine use along with superimposed gingival inflammation is visible here.

Following medical clearance, the patient received comprehensive dental care under general anesthesia (Children's Hospital, New York). The treatment included: extra-oral and intra-oral examinations, dental radiographs, dental prophylaxis, scaling, multiple dental restorations, and gingivectomies at four sites. Radiographs revealed and confirmed normal developing teeth. No post-treatment complications were noted. After the procedures were performed in the operating room, the patient presented for follow-up at the pediatric dental residency clinic. Recent extra- and intra-oral examinations revealed intact dental restorations and uneventful healing of wound sites.

Discussion

When treating patients with VACTERL association, it is essential to identify associated defects and treat them accordingly. This patient had congenital cardiac anomalies and tracheo-esophageal fistula, and can thus be identified as having the cranial phenotype of this association. As a result of being born with a TEF, he also presented with a gastrostomy tube. It has been postulated that when a gastrostomy tube is placed in a patient with EA or TEF, the pressure of the lower esophageal sphincter can be compromised, leading to gastroesophageal reflux (GERD).[14] The patient's medical history was significant for GERD, which increased his risk for dental caries. GERD may cause enamel erosion and is associated with higher S. mutans counts, of 106 CFU's/ml or above, causing patients to have notably higher dmft scores than patients without reflux.[15]

Green intrinsic staining of all of the primary teeth was noted in the patient's intra-oral examination. Green pigmentation likely resulted from hyperbilirubinemia which occurred in conjunction with the patient's hepatic problems and liver transplant. Bilirubin, one of the breakdown products that results from degradation of red blood cells, causes jaundice at high blood concentrations.[16] The bilirubin is permanently trapped in dentin during the tooth maturation/mineralization process, producing green stained bands that appear on the teeth.[17]

Furthermore, due to liver transplantation, this patient must take cyclosporine, an immunosuppressant used to prevent organ rejection. Cyclosporine use can cause gingival overgrowth and is associated with many oral lesions, such as hairy leukoplakia, and increased occurrence of viral and fungal infections. Of all oral problems, gingival overgrowth is the most common.[18] This patient's gingival overgrowth was exacerbated by increased plaque and calculus accumulation.

Treatment options for both hyperbilirubinemia and gingival overgrowth do exist. To conceal the green staining associated with hyperbilirubinemia and improve esthetics, resin crowns and resin veneers often serve as the best options. Whitening does not decrease the green pigmentation because it is confined to the dentin.[17] Gingival overgrowth can also be reduced by improved oral hygiene, gingivectomies, and the use of antimicrobial rinses.[18]

Patients with VACTERL association should be seen at frequent intervals for scaling and prophylaxis in order to maintain oral health. Parent-assisted toothbrushing and flossing should be emphasized to decrease plaque accumulation. Since fungal infections are common in those taking cyclosporine, topical application of antimicrobial rinses may help to prevent these infections. With regular and frequent oral examinations, oral health can be better maintained and problems can be addressed promptly.

Conclusion

Due to the wide range of manifestations of VACTERL association, the exact incidence within the population is still unknown.[1] Except in cases with severe defects, patients with VACTERL association can lead normal productive lives.[11] Hence, the dentist must be aware of the oral manifestations of a patient's underlying medical condition and the necessary modifications in treatment of such patients with extensive medical histories, such as VACTERL association.

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Chin Block Bone Graft in Patient Congenitally Missing Maxillary Lateral Incisors Prior to Implant Placement: A Case Report

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Abstract
Insufficient alveolar bone can hinder successful implant placement, compromising implant positioning and stability. Various regenerative procedures are available to repair hard tissue defects. Ridge augmentation techniques include guided bone regeneration, and ridge expansion. The types of bone grafts include synthetic bone substitutes, xenografts, allografts, and autogenous bone grafts. Because of their biocomptability and osteogenic, osteoconductive, and osteoinductive potential, autogenous bone grafts are the gold standard of bone graft materials. In this case report, an autogenous chin block graft was used to resolve maxillary horizontal ridge deficiency at sites of congenitally missing #7 and #10 prior to implant placement. A two-stage approach was employed with implant placement six months after ridge augmentation surgery. On average 6 mm of alveolar bone width was gained. Implants were placed in an ideal position and were stable after placement.

Introduction
Safety and aesthetics of implant placement may be compromised if sufficient bone height, width, or density is not available. Trauma, tooth loss, or infection can contribute to insufficient bone volume, preventing the successful placement of implants. According to clinical evidence, a minimum of 5 to 6 mm alveolar width is required for placement of implants.[1] In particular the anterior maxilla displays less dense bone and smaller volume of bone than the mandible, often necessitating a bone graft prior to implant placement.[2,3] These bone augmentation procedures may include socket preservation, horizontal or vertical ridge augmentation, and sinus augmentation.

The type of bone augmentation procedure and material used often depend on the type of alveolar ridge deformity. Seibert has classified the types of alveolar ridge defects into 3 categories: a Class I defect is described as a loss of bucco-lingual width, Class II describes a loss of apico-coronal ridge height, and Class III describes a loss of both apico-coronal height and bucco-lingual width.[4]

There have been various procedures described to increase vertical and horizontal dimensions of alveolar bone as well as density. Ridge augmentation techniques include particulate grafting, membrane use, block grafting, and distraction osteogenesis, either alone or in combination.[2,5] The technique chosen often depends on the extent of the defect and the specific procedures to be performed.[5] Bone grafts can be categorized as autograft, allograft, xenograft, and alloplast. Synthetic graft materials are defined as alloplasts. An allograft is a graft from a non-identical member of the same species, often cadaveric bone, while a xenograft is a graft from a different species, often bovine. Autogenous bone, on the other hand, is from the same individual and requires bone to be harvested at the time of surgery from a second surgical site. The surgery can be somewhat invasive and as a result, some patients prefer the use of allografts or xenografts as an alternative.

Many clinicians view an autogenous bone graft as the gold standard since it is osteogenic (has the ability to form bone), osteoconductive (has the ability to serve as a scaffold for bone regeneration) and most importantly, osteoinductive (is capable of inducing bone formation).[6] Autogenous bone has been harvested from a wide range of sites including the anterior and posterior crests of the ileum, calvarium, tibia, fibula, scapula, ribs, maxillary tuberosity, mandibular retromolar area, ramus, and mandibular symphysis. While intraoral sites have the advantage of being less invasive, extraoral sites, such as iliac crest, cranium or tibia, are necessary if bone defects exceed 2cm.[5] If bone defects are less than 2cm, intraoral sites such as mandibular symphysis and ramus are preferred. One clinican even reported using maxillary tuberosity for bone grafts explaining that in situations of tuberosity overgrowth, it served as a large volume of bone easily harvested with few complications.[7] In addition to the ease of intraoral harvest, grafts derived from intramembranous bone (such as part of the ramus and the mandibular symphysis) have less resorption than endrochondral bone.[3,5] Since healing of bone grafts is dependent on angiogenesis and revascularization, corticocancellous blocks are preferred to cortical blocks since revascularization occurs faster in the former.[5] The most common intraoral sites described are the mandibular symphysis and ramus from which corticocancellous bone is harvested.[1]

Most successful ridge augmentation methods include the placement of a membrane on top of a graft, preferably an autogenous bone graft, to help guide bone growth. This technique is called guided bone regeneration and is being used successfully by many clinicians to achieve favorable ridge augmentation results.[1]

Chin Block Bone Graft in Patient Congenitally Missing Maxillary Lateral Incisors Prior to Implant Placement: A Case Report

Ridge augmentation and implant placement techniques can occur via a one-stage simultaneous approach or a two-stage approach. During the simultaneous approach the implant is placed in the same visit as the bone graft, while in the two stage approach, the bone graft is allowed to heal and the implant is placed six months later. Both treatment options are being used and while time and money may be saved with single stage therapy, the two stage technique has reported better positioning, stability, and integration of the implant.[8]

Case Report

A 29-year-old male was referred to Columbia College of Dental Medicine for an implant consultation *(Figure 1)*. His medical history was noncontributory. The patient presented with congenitally missing maxillary lateral incisors #7 and #10 and was interested in having implants placed. He had completed Invisalign treatment leaving sufficient mesio-distal space for implants and wore a flipper.

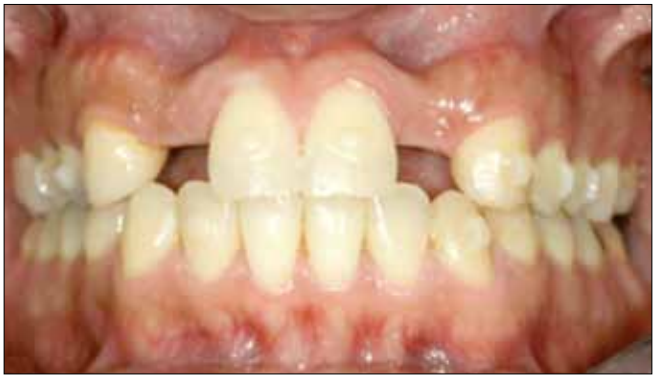

Figure 1
Initial record. Arrows show large labial ridge defects at site of congenitally missing lateral incisors (#7 and #10).

The patient was referred for cone beam computed tomography scan (CBCT) constructed using Xoran and Vision software. After analysis of the CBCT scan the patient was diagnosed with bilateral horizontal ridge defects at sites #7 and #10 *(Figure 2).* The patient had sufficient vertical height for placement of implants. The defects were diagnosed as Class I *(according to the Seibert classification).*[4]

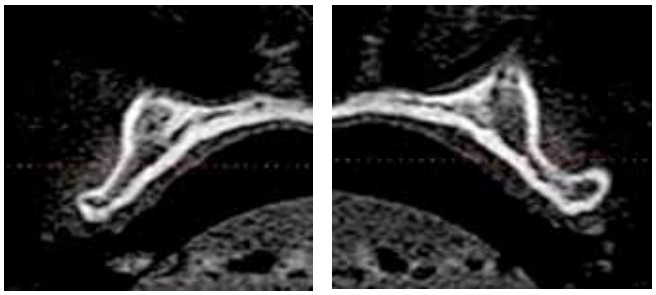

Figure 2
Initial CBCT pre-op prior to grafting of #7 (left scan) and #10 (right scan). Arrows show large labial defect. Width of defect at site #7 measures 2.3 mm and at site #10 measures 2 mm.

The patient was then treatment planned for a two stage approach to lateral ridge augmentation with autogenous bone grafts from the mandibular symphysis. Full thickness flaps were reflected from below teeth #22 to #27 and two 10 x 7 mm corticocancellous bone blocks were harvested 5mm apical to mandibular roots *(Figure 3)*. Harvest sites were filled in with Bio-Oss.

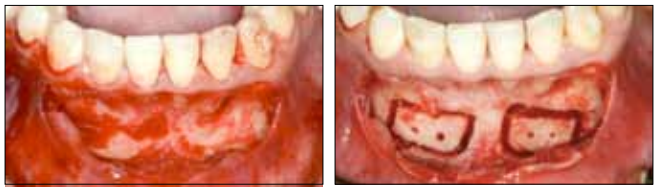

Figure 3
Left image shows donor site after full thickness flap. Right image shows donor site with two 10 x7 mm blocks designed.

Maxillary recipient sites were prepared for placement of grafts with a full thickness flap from teeth #5 to #12. The recipient sites were decorticated and bone blocks were shaped and fixed with 1.5 x 7mm fixation screws *(Figure 4).* Autogenous particulate bone and Bio-oss® were placed around both blocks to fill any voids. An absorbable extracellular collagen matrix membrane, Dynamatrix®, was placed over both block grafts and the flaps were replaced.

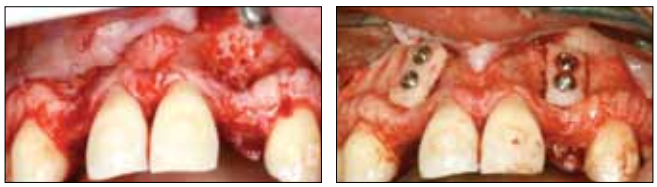

Figure 4
Left image shows decortications of recipient site. Right image shows placement of chin block graft in sites # 7 and #10 with fixation screws

The patient was followed up at regular visits and grafts appeared stable both clinically and radiographically. *(Figure 5)*

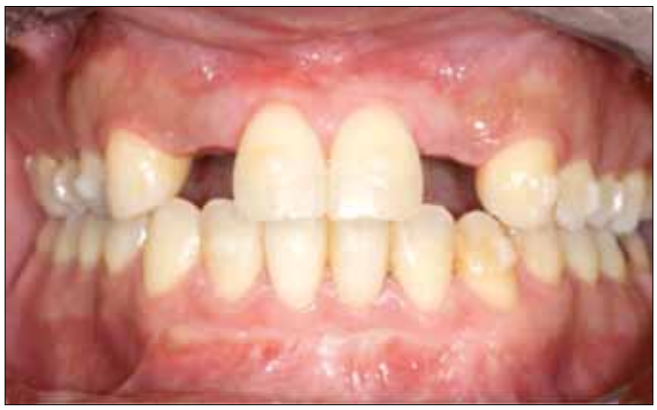

Figure 5
Two months post-op. Labial ridge defects have been greatly reduced. On average 4-5mm bone has been gained.

Six months later, prior to implant placement, a new CBCT was taken to evaluate the graft sites. Sufficient lateral augmentation was achieved *(Figures 6 and 7)* and two Straumann® implants were placed in the site for #7 and #10.

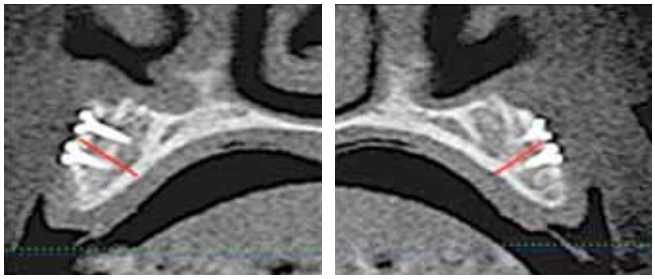

Figure 6
CBCT at 6 months post-op prior to implant placement of site #7 (left scan) and #10 (right scan). Width at site #7 measures 3 mm and at site #10 measures 2 mm.

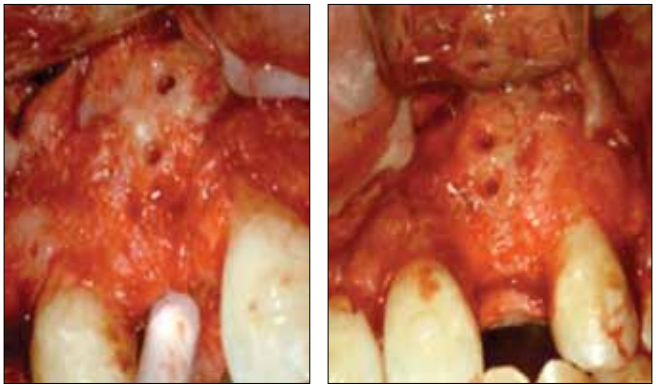

Figure 7
Bone graft 6 months post-op prior to implant placement

Three millimeter implants were placed at both sites and measured 10 mm in length at site #7 and 12 mm at site #10 in accordance with local anatomy *(Figure 8).*

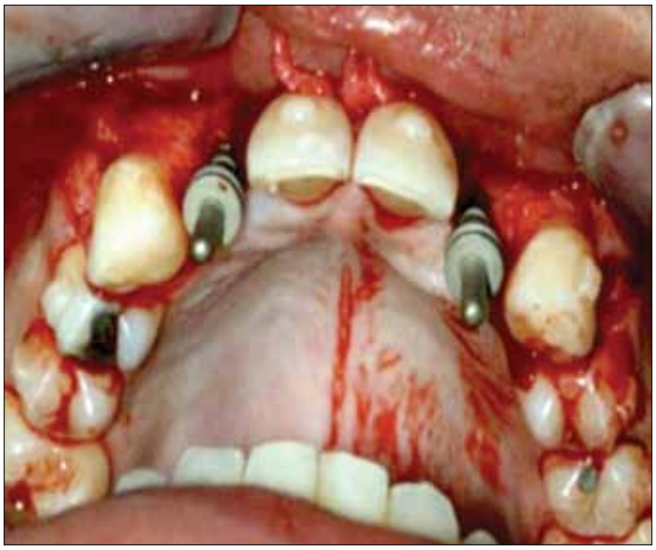

Figure 8
Placement of implants in grafted sites #7 and #10.

At site #7, bone width increased from 2.3 mm to 9.3 mm and at site #10, bone width increased from 2 mm to 7.5 mm. Therefore, 7 mm of bone was gained at site #7 and 5.5 mm at site #10. The flap was replaced and the patient was told to return for loading of implants after osseointegration had been completed. On average, 6mm of horizontal bone was gained after bone graft placement and subsequent bone resorption.

Discussion

Practitioners use personal preference and anatomic considerations when choosing between mandibular symphysis grafts and ramus grafts, although there are advantages and disadvantages associated with each. Misch found that the ramus was a more advantageous graft site than the chin because of less donor site deformation and less postoperative complaints of sensory disturbance.[9] On the other hand, harvesting ramus blocks risk inferior alveolar nerve paresthesia or anesthesia, injury to the long buccal nerve, significant postoperative discomfort, bleeding and swelling among other complications.[7] Chin graft technique offers ease of access, good bone quality, low morbidity of donor site, and minimal graft resorption;[1] it is especially useful if larger grafts are needed.[9] Risks associated with chin graft harvest include lower incisor tooth numbness, temporary or permanent mental nerve injury, incisor injury, lingual cortex fracture and perforation into the lingual soft tissue, and uncomfortable scarring in the lower vestibule.[6] Patients reported some degree of sensory disturbances after chin and ramus grafts, 16% and 8.3% respectively.[7] Tolstunov claims that many of the risks associated with chin and ramus graft techniques are not present with the maxillary tuberosity technique and if sufficient bone is present, it should be a more widely used harvest site due to its lower risk of complications.[7] In this case, mandibular symphysis grafts were chosen due to easy access and low morbidity of the graft sites.

There has been a great deal of debate regarding the degree of graft resorption after placement. Clinicians agree that all types of grafts display some degree of resorption over time, especially if bone is not loaded with implants after approximately four to six months.[7] Resorption rates of 0-25% have been reported at the time of implant placement and up to 60-70% at the time of abutment connection.[5] Some clinicians have reported less resorption with the use of a membrane and employ membranes to maximize regenerative potential.[5] In this case a combination approach was used to maximize regenerative potential. A membrane and particulate bone were used in combination with the block graft for an improved outcome.

Success rates of implants in grafted sites differ greatly among sources. Initial success rates began at 50% but have increased dramatically over the years with many clini-

cians reporting survival rates between 81.2% and 100%. 1,6 Some have reported higher success rates with delayed implant placement after bone graft and also delayed implant loading. Factors such as smoking and uncontrolled diabetes can also greatly diminish success rates.[7] Therefore, patient selection is very important in implant success.

Coordinating treatment with patients' expectations is just as important as patient selection when treatment planning a patient for a bone augmentation procedure. Although autogenous bone is the gold standard, it is not the standard of care because a patient's medical conditions or refusal to undergo a more invasive surgery may limit treatment options. In our case, the patient was very healthy and had no preconceived notions regarding graft materials. The patient was informed that considering his good health and large horizontal defect, an autogenous block bone graft would be the best. In situations of such a large defect, a particulate graft would not have sufficient underlying bone volume to fill in the entire defect and would probably require a second graft to fill in any remaining concavity. An autogenous block bone graft can cause some morbidity at the host site, but since our patient was healthy and had a large volume of bone at the donor site, we chose to use autogenous bone. In combination with some particulate bone and a membrane, the block graft provided a rather predictable and stable outcome.

Conclusion

In the absence of sufficient bone, it may not be possible to achieve successful implant placement. In these situations, a bone graft may be necessary to achieve favorable placement and stability of implants. This case report has shown autogenous block bone grafts can be harvested from the mandibular symphysis and can be successfully grafted onto the maxillary buccal alveolar ridge at sites #7 and #10. The block grafts showed substantial improvement in the horizontal width of bone allowing for ideal positioning of dental implants. This case has shown that ridge augmentation can be successfully accomplished with subsequent implant stability while using a relatively atraumatic intraoral surgical site and low morbidity to the donor site.

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Use of Block Allografts in Pre-Implant Alveolar Ridge Augmentation: Three Case Reports

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Abstract
As dental implants become the increasingly desired alternative for replacement of missing teeth, the need to maximize the success of bone graft methods has increased in kind. The allogenic block graft is a relatively new option for bone grafting that eliminates the autograft's need for a second surgical site but provides the same benefits of physical bulk, stability, and structure while being in virtually unlimited supply. It also aims to add vertical height as well as horizontal width. This case report series details the use of allografts on various alveolar ridge locations. In all instances of allograft placement discussed here, the end result was an augmentation of bone comparable to that typically achieved with the use of other techniques and materials. However, two of the allografts discussed in this report presented the complication of graft exposure, which is not usually experienced with autografts. Various ways of mitigating this risk have been proposed, one of which yielded successful results in the last case described below, indicating that further investigation into the optimal methods for using cadaveric block grafts is worthwhile.

Introduction
Many of the patients who desire dental implants lack the necessary alveolar bone width and/or height to retain an implant securely over the long-term and ensure osseointegration. Several sources of bone graft material are available to address the problem, however all have some drawbacks. Autografts—harvested from intraoral sites such as the mandibular symphysis or ramus, and extraoral sites such as clavicle, ribs, or iliac crest—have to date been considered the gold standard, because they are readily adopted by the recipient site[1] and provide osteoconductive, osteoinductive, and osteogenic properties that encourage bone growth.[2,3] However, for the patient, the prospect of exposing two surgical sites, with the accompanying discomfort, cost, time, and increased risk of infection, paresthesia, or fracture is not always an attractive option.[1] Common alternative methods for bone augmentation include but are not limited to guided bone regeneration, short and narrow implants to avoid ridge augmentation, sinus lift for posterior maxilla, and ridge split for horizontal deficiency. However, these alternatives also bring potential complications that might not make them suitable for all patients.

Use of the cadaver block graft is a relatively new method of bone grafting that provides one additional option to patients and clinicians. These allographic blocks are corticocancellous or cancellous segments of bone that have been dehydrated via various methods, sterilized, and treated to remove antigenic potential and can be fixated to deficient alveolar ridges with fixation screws to increase bone thickness.

The cases that follow discuss the advantages and disadvantages found when using allograft blocks to augment the mandible or maxilla prior to implant placement.

Case Report 1
A 26 year old non-smoking female presented to the Columbia University Post-Graduate Periodontics Clinic for ridge augmentation of the maxillary anterior region in anticipation of implant placement after extraction of tooth #9. This tooth was severely compromised with an anterior fistula and sensitivity to percussion. Her medical history was non-contributory to her condition. After a complete diagnostic workup, including photographs, radiographs, and a CT scan, it was decided to use a block allograft to increase the thickness of the ridge. Upon extraction of #9, socket preservation was performed in order to increase osseous tissue and support a more esthetic outcome. The socket was filled with particulate allograft material (Puros) and covered with a resorbable collagen membrane (Biomend Extend). A connective tissue pedicle was harvested from the palate and placed over the socket *(Figure 1)*.

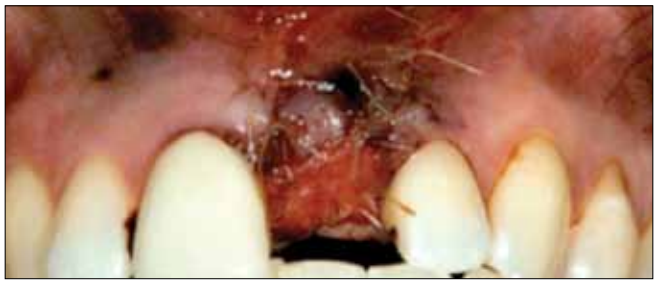


Figure 1
Case 1: The socket was filled with Puross and covered with a layer of Biomend Extend membrane

Figure 2
Case 1: CT pedicle necrosis

One week post operation, there was evidence of connective tissue pedicle necrosis *(Figure 2)* and loss of some particulate allograft in the socket. Both a horizontal and vertical defect was present (residual Class III Seibert defect), significantly compromising any future implant restoration esthetics. Thus, it was decided to augment the ridge with a block allograft.

Six weeks before the placement of the block, a frenectomy was performed to decrease the flap tension anticipated after placement of the bone graft. Sulcular and mid-crestal incisions were made over the edentulous ridge from # 6-12. A full thickness flap was raised and the defective area was visualized and measured to be approximately 11 mm *(Figure 3).*

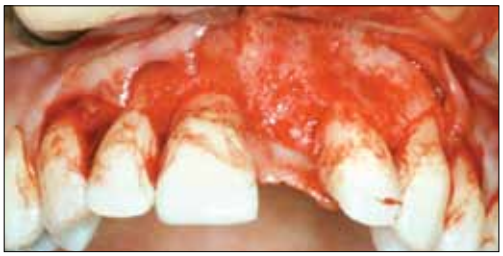


Figure 3 *(left)*
Case 1: Incision and full thickness flap

Figure 4 *(right)*
Case 1: block allograft shaped to fit the recipient site

The block was shaped to the size of the defected area and to fit passively over the ridge *(Figure 4)*. The buccal bone was decorticated to encourage vascularization and bone-block integration. Bone screws of 10.5 mm and 7.5 mm were inserted into the crestal and buccal aspects respectively. Particulate allograft (Allo-Oss) was placed to fill the voids around the block *(Figure 5)* and a resorbable collagen membrane (Ossix Plus) was placed over the block. Periosteal releasing incisions were made and the flap was coronally positioned. The connective tissue graft (CTG) was secured under the flap and the tissue was sutured to obtain primary closure. The CTG covered the 2 mm dehiscence between the buccal and palatal flaps *(Figure 6)*.

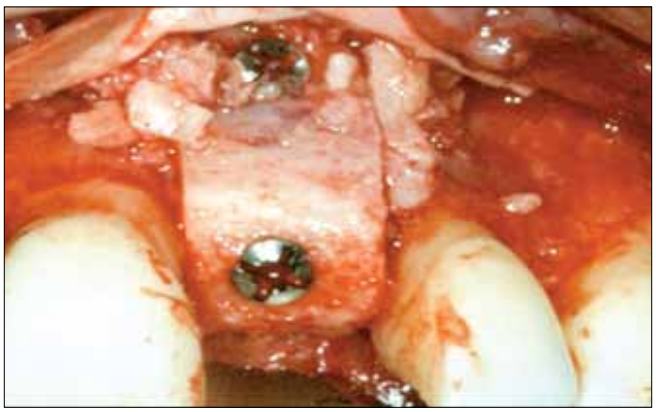


Figure 5
Case 1: block graft inserted

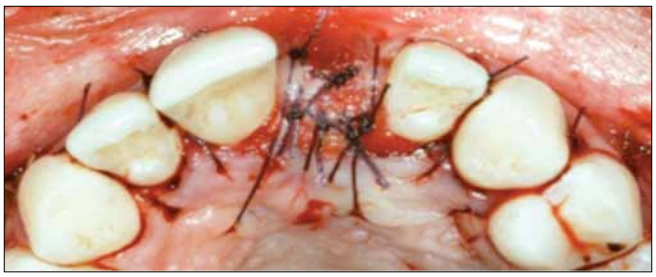


Figure 6
Case 1: Primary closure of hard and soft tissue grafts

The patient was given an interim prosthesis and care was taken to relieve any pressure the prosthesis had on the soft tissue, which could potentiate resorption. Two weeks after the surgery, the block graft and screw became exposed coronally, but the patient was not in discomfort. After another four weeks, the block was completely exposed *(Figure 7)*, thus it was decided to attempt to cover the exposed area with a connective tissue pedicle graft rotated from the left palate.

Figure 7
Case 1: Six weeks post-op, block exposed.

This attempt was unsuccessful and led to complete necrosis of pedicle flap. At this point it was decided to leave the block exposed and re-evaluate from week to week, reducing exposed bone at each visit until healthy vascularized bone was reached. Soft tissue closure was finally achieved after six months *(Figure 8)*. The final CT scan showed that bone thickness in the area had increased to 6.5 mm after integration of the block graft *(Figure 9)*. The implant was the placed successfully *(Figures 10, 11)*.

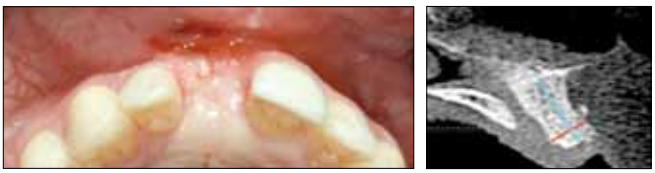
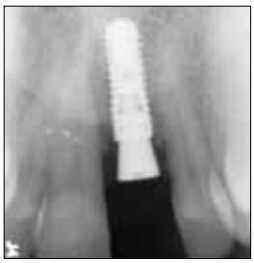
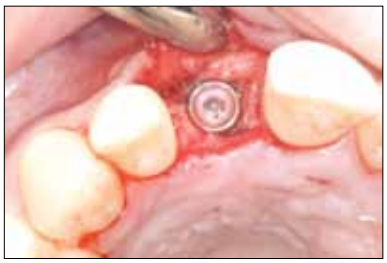


Figure 8 *(left)*
Case 1: Soft tissue closure achieved.

Figure 9 *(right)*
Case 1: Final CT scan shows adequate horizontal and vertical bone (6.5x14.5mm)

***Figures 10** (left) **and 11** (right)*
Case 1: Successful implant placement at 6 months post-graft

Case Report 2

A 56 year old female presented to the Columbia University Post-Graduate Periodontics Clinic for an implant consultation as she was edentulous in multiple areas, including the region of #28-30 *(Figure 12)*. The patient had no significant medical history and was a non-smoker. The diagnostic work-up which included a CT scan indicated that there was insufficient bone width in the lower right mandible to accommodate implants and a bone graft would be necessary.

Prior to placing the bone graft, a connective tissue allograft would also be needed to better accommodate the bone block material, deepen the vestibule, and ensure keratinized gingiva for primary closure. The patient was given multiple options including the use of an autograft harvested from her mandible or the allogenic block graft and she elected to proceed with the allograft block.

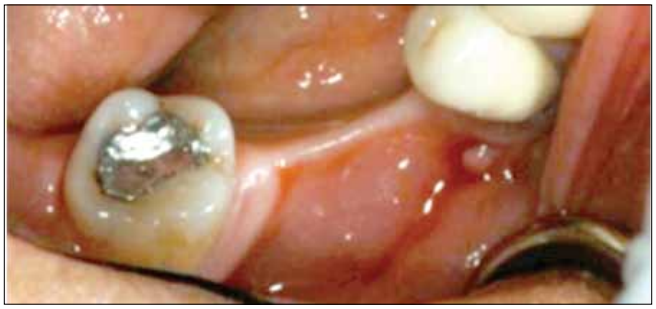


Figure 12
Case 2: Baseline mandibular alveolar ridge, buccal view

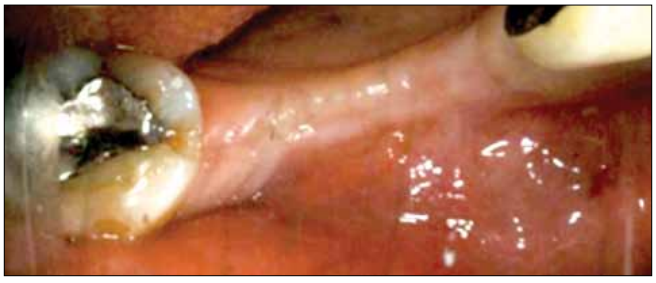


Figure 13
Case 2: Complete healing of alloderm graft in mandible; increased attached gingiva seen compared to baseline

In order to create the needed increase in attached gingiva and vestibular depth it was decided to perform a vestibuloplasty with the use of acellular dermal matrix (alloderm) a few weeks before the block graft. The lower right mandible healed after five weeks without incident *(Figure 13)*.

Three and a half months after the alloderm graft of the lower right mandible was placed, the patient presented for placement of the block graft. A lingual crestal incision was made between teeth #27-31. A sulcular incision was then made with one vertical release and a full thickness flap was raised. After dissection around the mental nerve, the buccal plate was decorticated for better graft integration *(Figure 14)*.

Figure 14
Case 2: Decorticated recipient site

The block was shaped to match the curvature and size of the recipient site and two pilot holes were drilled into the block. Initial stability was not achieved with one of the pilot holes, so a new one was made mesially to secure the block with bi-cortical stabilization. Screws of 10.5mm length were screwed in the pilot holes *(Figure 15)*.

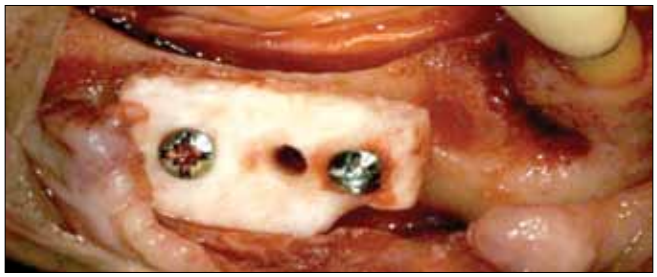


Figure 15
Case 2: Allograft secured by screws at recipient site

Voids were filled with particulate cortico-cancellous allograft (Allo-Oss) and a resorbable collagen membrane (Ossix-Plus) was placed over the block graft. Primary closure was achieved. At the one week follow-up visits, the tissue at the crest of the alveolar ridge was thinning and the patient experienced paresthesia in the tissue anterior to the mental foramen. At two, four, and ten week follow-ups, there was exposure of the block, but the tissue appeared clinically healthy *(Figure 16)*.

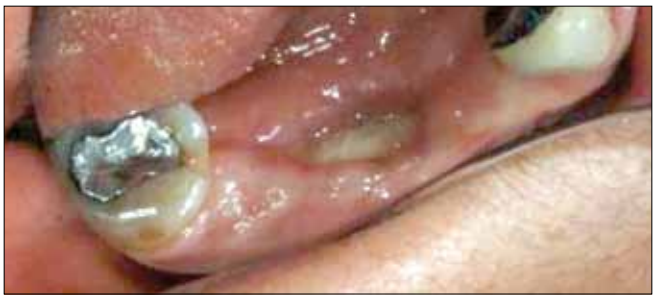


Figure 16
Case 2: Allograft tissue dehiscence at six weeks post-op

Each time the patient presented with exposure, a small amount of the exposed bone was removed until at three months, closure was achieved. In the CT scan done prior to implant placement, the horizontal knife-edged ridge previously present had been augmented considerably and the vertical bone thickness had increased from 8 to 10 mm, enough to house a standard 10 mm implant *(Figure 17)*.

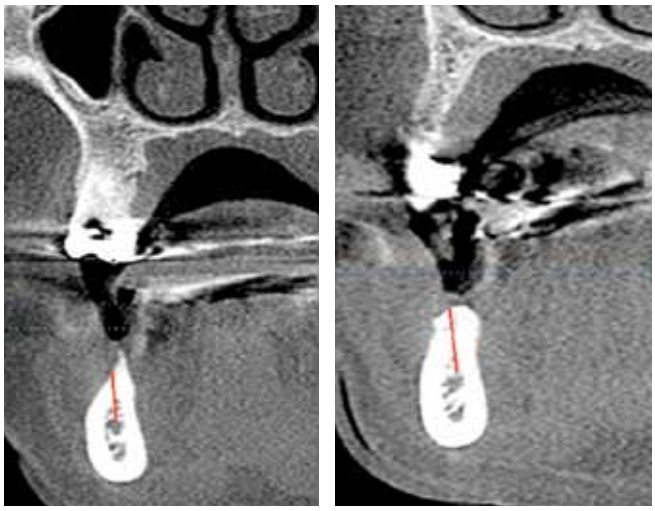


Figure 17
Case 2: Pre- and post-op CT scans showing increased horizontal and vertical bone

Case Report 3

A 40 year old female presented to the Columbia University Post-Graduate Periodontics clinic for replacement of a missing tooth #9 that had been lost several years previously due to trauma (Fig. 18). She was a non-smoker with non-contributory medical history. Examination and a radiographic work-up were done, including a CT scan. The treatment plan for this site was to perform a frenectomy then graft the site with an allogenic block graft covered with autogenous Platelet Rich Plasma (PRP) prior to implant placement. The creation of PRP involves withdrawing and centrifuging the patient's own blood to achieve a high concentration of autogenous platelets and growth factors; this plasma is later placed in the surgical site to enhance healing. The patient agreed to this plan.

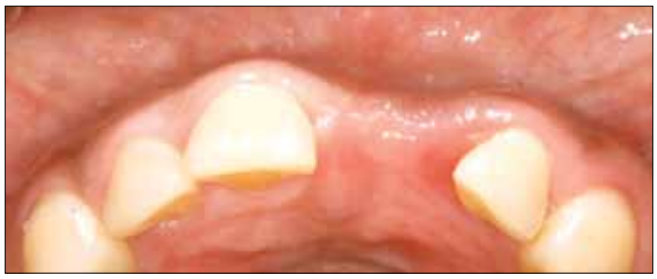


Figure 18
Case 3: Baseline maxillary ridge of #9 area

Two months after the necessary scaling, root planing, and frenectomy, the patient presented for placement of the block bone graft. As PRP was going to be used with the bone graft, 53 mL of the patient's blood was drawn and processed according to protocol. A full thickness flap was elevated from #6-11 *(Figure 19)*. The recipient site buccal plate was decorticated to expose growth factors in blood, as was done in the previous cases. The block graft was shaped to fit the recipient site and also was decorticated.

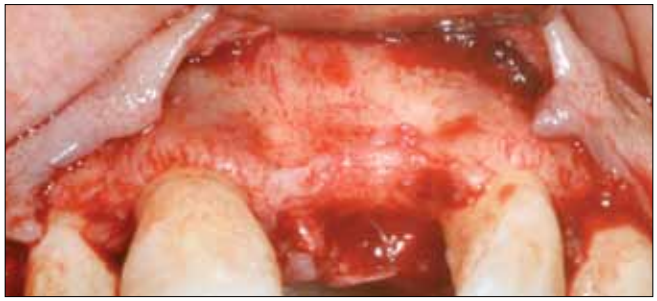


Figure 19
Case 3: Recipient site for block allograft

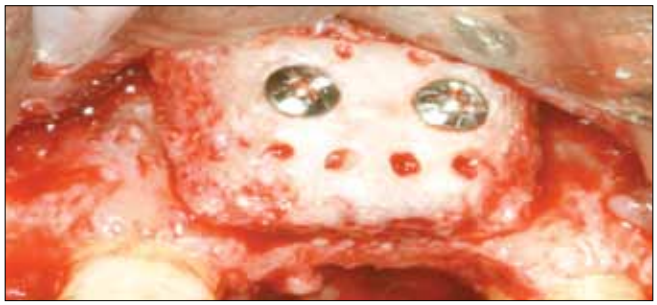


Figure 20
Case 3: Block graft with holes through to native bone

Two guide holes were placed in the block graft and it was then soaked in a PRP and saline solution for approximately 30 minutes. The graft was secured to the recipient site with two screws and several holes were placed through the graft to further increase surface area exposed to the vascular bed *(Figure 20)*. A mixture of freeze dried bone allograft, demineralized freeze-dried bone allograft, and PRP was placed around the block graft to fill the voids and a PRP membrane was placed to cover the entire block graft.

At the one and two month follow-up visits, healing proceeded uneventfully and no graft exposure was noted *(Figure 21)*.

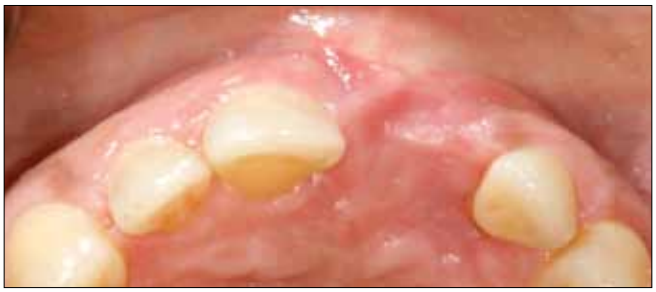


Figure 21
Case 3: Site of block graft two months post-op

Eight months after the placement of the allograft the patient presented for implant placement. The implant pre-surgery CT scan indicated that the alveolar ridge width at the site of the graft had increased by approximately 3.5mm, thus sufficient bone was now present to place an implant *(Figure 22)*. A 4x11.5 mm implant was placed at the site of the bone graft *(Figures 23, 24)*.

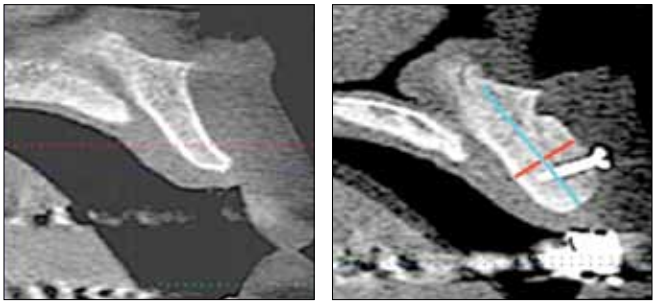
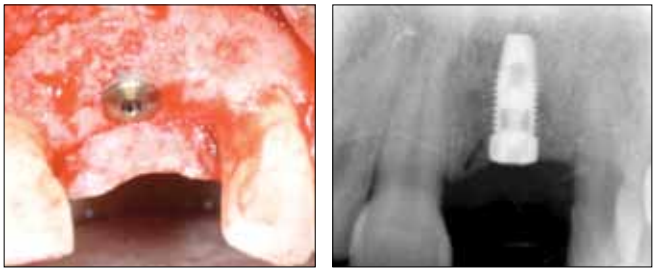


Figure 22
Case 3: Before and after CT scans



Figures 23, 24
Case 3: Implant placement at site of block graft, prior to osseointegration

Discussion

The allografts used as described above showed clear advantages over alternative methods of both soft tissue and bone grafting. In each case, attached gingiva was increased prior to placement of the block allograft. The primary aim of this was to allow for a tension-free closure once the bone graft was in place and to increase keratinized gingiva surrounding the future implant to minimize future recession and maximize esthetic results.

Studies in the literature describe the advantages of using alloderm rather than a free gingival graft for vestibuloplasties, which in addition to those same advantages of bone allografts (no second surgical site, less morbidity) also include the prevention of transferring cells that could transmit viruses.[3] Although the addition of the alloderm graft added three and a half months to the patient's treatment and despite the issue of some expected sloughing of superficial epithelium during healing in the first few weeks due to poor vascularization, this was easily managed.

Similarly hard tissue allografts were successfully used to augment deficient alveolar bone both vertically and horizontally, allowing adequate width to house a dental implant. Previous case studies have shown graft dehiscence to be a particular problem when vertical augmentation is attempted.[4] Unlike cases that use autogenous bone to graft deficient ridges, the patients here did not need to endure the time, risks, costs, and discomforts associated with creating a second surgical site to harvest their own bone.[5]

Once the tissue was healthy and the vestibule deepened enough to proceed with the bone allograft, the allograft block was placed and primary closure was achieved. However, graft sites in cases 1 and 2 above experienced problems with block exposure.

Other clinicians have had success with allograft blocks without experiencing problems with graft exposure.[1,6] There are several possible variables that may contribute to whether or not block allografts become exposed. First, the type of bone in the allograft, either all cancellous or cortico-cancellous may influence exposure. A purely cancellous block graft with a lower density than cortical bone may be more amenable to a rapid vasularization and integration.[1] In addition, cancellous bone gains mechanical strength during the repair process, where as cortical bone is weakened during repair.[2] In similar cases described by Wallace and Lyford et al., several alveolar ridge sites were grafted with cancellous allograft blocks covered with cortical bone particles on multiple patients, and throughout the healing period in all sites, graft exposure was never a problem.[1,2] Nevertheless, cases of cortico-cancellous allografts have also been reported in humans without dehiscence, so most likely the type of bone in the allograft is only one factor potentially contributing to exposure, if it does at all.[5,6,7]

Other factors potentially influencing soft tissue dehiscence are the location of the initial flap incision (crestal vs. vestibular incisions) and the way in which the block graft is processed (freeze-dried, fresh-frozen, solvent-dehydrated, etc.). Processing techniques could affect the graft's mechanical strength and persistence of bone morphogenic proteins.[4,8] The kind of membrane placed on top of the

graft (resorbable vs. non-resorbable, or both, the graft contouring prior to placement, and pressure placed on the tissue by a temporary prosthesis[1,6] may impact soft tissue dehiscence as well.

Although extensive efforts were made to ensure tension free primary closures via soft tissue grafts prior to block graft placement, perhaps an even greater excess of soft tissue is needed to account for the tissue that will possibly experience resorption; proper soft tissue closure techniques are crucial to dehiscence prevention. Case 3 described above also incorporated holes placed entirely through the width of the block graft to encourage vascularization, and this may have contributed to the fact that no exposure was seen in this case. Some cases described in the literature prepared the recipient bed with an indentation that matched the size of the block graft so that it fit snugly in the site and thus increased the surface area of the graft exposed to native bone.[5] This also may have improved vascularization, preventing exposure. However, this was not done in the cases above so as to maximize preservation of native bone; it is uncertain whether sacrificing the already deficient native bone is worth the increased surface area contact. Clearly, use of the block allografts is extremely technique-sensitive.[6]

Lastly, some success has been found with soaking the allograft or the covering membrane in a preparation of the patient's platelet-rich plasma (PRP).[5,7] This was a procedure that was not done in the first two cases described but was incorporated into the last case above as well as several cases in the literature, none of which had problems with block graft exposure. Use of PRP has been somewhat controversial, as there are some studies that indicate that the growth factors presumed to be helpful in osteogenesis are actually very short-lived in PRP and do not last long enough to have a significant impact.[7,9] However, there is also evidence to the contrary, as PRP has been found to enable a more predictable flap adaptation and closure, improve hemostasis, and promote epithelial development and hemostasis when appropriate delivery methods are used to administer PRP in a time-controlled manner.[7,9] Some studies have shown that surgical sites enhanced with PRP heal at a rate two to three times faster than surgical sites without PRP.[10] Given that use of PRP in certain instances may improve outcomes and that the clinical cases that used PRP appeared to have less complications, this is certainly a factor that should be explored in the future via randomized controlled trials of alveolar ridge allografts.

Patient selection is also very important in these cases. Some important selection criteria for patients include: patient compliance/ motivation (since strict adherence to post-op instructions and return for follow-up visits is crucial), negative history of smoking, good oral hygiene, and lack of significant systemic diseases.

Conclusion

Cases presented above and in the literature indicate that there is a demand for reliable alternatives to autografts for augmenting wide spans of alveolar ridge prior to implant placement. Block allografts that can be formed into various shapes show promise in becoming a viable solution to this need. However, to date, there are no known randomized controlled trials on humans that consider variables such as the amount of bone being replaced, use of soft tissue graft prior to block placement, graft bone type, graft preparation method, incision type, suture type, or PRP use to quantify differences in the outcomes seen in autografts vs. allografts. Such studies should be done and would prove beneficial in eliciting a repeatable method for securing good results in allograft use.

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