Is Off-Axis Tilted Implant a Better Option Than Maxillary Sinus Lift Procedure in Posterior Edentulous Maxilla?

To the Editor: Edentulous posterior maxilla has several anatomic and physiologic limitations such as low density of alveolar bone, increased pneumatization of maxillary sinuses, and faster negative remodeling in absence of continuous pressure.[1] Owing to mechanical and anatomic difficulties, implant treatment in the atrophic maxilla represents a challenge.[2] The techniques of maxillary sinus floor elevation pose a series of inconveniences, such as the need for multiple surgical interventions, the use of bone graft and the long duration during which patients remain without rehabilitation during the graft consolidation and healing interval.[3] The use of tilted implants is being advocated by an increasing number of clinicians.[4–7] They concluded that tilted implants to avoid the maxillary sinus were a successful alternative procedure to more resource-demanding techniques such as bone grafting.

The aim of this study was to compare the sinus lift approach to the tilted implants approach with immediate loading for restoring the posterior maxilla. The comparison was done in terms of shorter surgical procedure, minimal invasive technique, desired primary stability at the time of implant placement, shortening loading protocols such as immediate loading of implants and to investigate whether there is a difference in success rates, survival rates, and peri-implant parameters, including marginal bone level changes between the 2 groups. The sample of the study was taken from the patients operated at Datarkar Institute of Maxillofacial Surgery Nagpur, India from the year May 2009 to May 2013. The records of 100 implants (Adin Dental Implants, Alon Tavor, Afula, Israel) placed in posterior maxillary region were included in this study. These were divided into 2 groups. Group 1 (n = 50) off-axis tilted implants were placed anteriorly or posteriorly to maxillary sinus wall. All implants in this group were immediately loaded by screwed provisional, and the correction of implant angulations was done by transmucosal abutments (TMA) of 17° or 30° (Adin Dental Implants, Alon Tavor). Group 2 (n = 50) direct maxillary sinus lift procedure with simultaneous implant placement (Fig. 1A-B).

The patients were monitored on periodic basis clinically and radio graphically at 3 months, 6 months, and 1 year follow-up after the placement of the prosthesis and once every year for 3 years. During the follow-up period, out of 50 off-axis tilted implants, one implant was lost within10 months of loading, giving a cumulative survival rate of 98.5%. The survival rate of prostheses was 100%. Sinusitis occurred in 2 implant sites (4.1%). The marginal bone resorption was on average (standard deviation) 0.57 mm (0.50 mm), 0.29 mm (0.32 mm), and 0.19 mm (0.11 mm) at 1st, 2nd, and 3rd year, respectively, for the tilted implants.

Out of 50 implants in maxillary sinus augmentation, 3 implants were lost at the end of 6 months from loading, giving cumulative success rate of 92.3%. The survival rate of prosthesis in all the implants was 100%. Sinusitis occurred in 4 implant sites (8%). The marginal bone loss was on average (standard deviation) 0.43 mm (0.44 mm), 0.23 mm (0.28 mm), and 0.12 mm (0.10 mm) at 1st, 2nd, and 3rd year, respectively, for the implants placed in augmented area. After 3 years, the implant cumulative success rate was 98.5% (survival: rate 100%) for the tilted implants and 92.3% (survival rate: 96.5%) for the implants in sinus augmentation procedure, and the prosthesis survival rate was 100%.

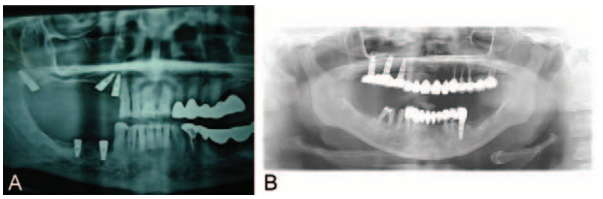

FIGURE 1. (A) Immediate postoperative panoramic X-ray. (B) Panoramic X-ray showing loading of implants after 6 months.

Results of this comparative study indicate that the use of immediately loaded tilted implants for the restoration of the edentulous posterior maxilla is an effective and safe alternative to maxillary sinus floor augmentation procedures. In this study, we recommend the use of tilted implants in the posterior maxillary region with the possible benefits of significant reduction in duration of the surgery and of the total duration of treatment. We also observe that there is less postoperative morbidity, decreased treatment costs. It is possible to successfully load dental implants in the posterior maxilla early or immediately after their placement in off-axis position. A high degree of primary implant stability (high value of insertion torque) and implant surface characteristics play an important role. Also placement of tilted implants in the available bone is easier for the implantologists than additional grafting procedures.

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Diagnosis and Management of Ossified Chronic Subdural Hematoma

To the Editor: A 54-year-old woman presented to our hospital with complaints of a paroxysmal headache during the last 2 years without neurological deficit. She had had meningitis when she was 5-years old. She had suffered from high fever and unconsciousness for about 1 month but recuperated without obvious sequelae. The initial brain computed tomography (CT) showed a plate-like mass with high density over the left hemisphere, with its central part of the lesion being low density (Fig. 1A). Encephalatrophy with mild subdural hygroma at the right side was also noticed. Cranial magnetic resonance imaging (MRI) of the brain demonstrated that the lesion was hypo- to isointense in T1-weighted imaging, hypointense in T2-weighted imaging and not enhanced. (Fig. 1B-D). To address the compression effect of the lesion, left frontotemporal craniotomy was performed. On opening the dura mater, a flat bony mass covering the convexity was discovered, which was removed en bloc in strict accordance with an in situ resection method (Fig. 1E-F). The crust-like specimen was then sent to have microscopic examination, which showed ossified bony structure with hematopoietic tissue (Fig. 1G-H). Postoperatively, the patient was well initially and postoperative CT revealed a complete removal of the subdural mass without any significant abnormalities (Fig. 1I). She was neurologically intact when discharged and in a follow-up 2 months after the dismissal.

Various studies dealing with chronic subdural hematoma (CSDH) showed that the incidence of chronic calcified subdural hematoma ranged from 0.3% to 2.7%, but ossified ones are extremely rare, which are merely reported occasionally worldwide.[1–6] Moreover, although head injury, surgeries, and alcoholism were the cause of CSDH in the vast majority of cases,[7] it was rarely seen as a consequence of intracranial infections. A mild traumatic event is believed to be the cause of CSDH in 60% to 80% of reported cases.[8,9] Traumatic subdural effusion is prone to develop into CSDH,[10] which suggests the role of subdural effusion in the formation of the latter. The same rationale may apply to postmeningitic subdural effusion. Subdural effusions are a well-recognized complication of bacterial meningitis, particularly in infants and toddlers.[11] It is possible, therefore, that in the present case, bilateral subdural effusion formed in the meningitic

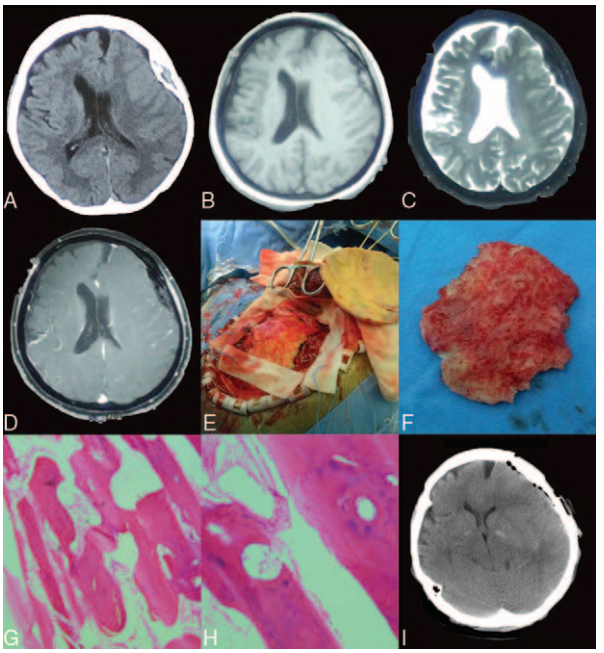

FIGURE 1. (A) The initial CT of the patient. (B) The initial T1-weighted imaging of the patient. (C) The initial T2-weighted imaging of the patient.(D) The initial contrast-enhanced T1-weighted imaging of the patient. (E) The mass revealed on opening the dura. (F) Photograph of the whole structure removed at operation. (G) Histopathology of the subdural specimen, showing a viable, mature, boney tissue, where an outer cortex and an inner trabeculated portion with hematopoietic tissues could be found (H & E, 100). (H) Another histopathological result of the same specimen (H & E, 200). (I) The postoperative CT scan revealed no obvious abnormality. CT, computed tomography; MRI, magnetic resonance imaging.

course and triggered the inflammatory reaction as described above, leading to the onset and propagation of subdural hemorrhage. Moreover, although the pathogenesis of ossification in CSDH is not yet fully understood, as calcified subdural effusion following bacterial meningitis has been reported previously,[5] we proposed that an additional third ossified stage occurred in addition to the CSDH stage in the left side of our present case, but the detailed mechanism of ossification is still in need of investigation.

In terms of management, preoperative diagnosis was often made unequivocally with CT imaging, where a subdural mass resembling a flat bone could be identified and a decompressive removal was routinely carried out with excellent outcome.

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Clinicopathologic, Cone-Beam Computed Tomographic, and Surgical Findings in a Unique Maxillary Hybrid Odontogenic Tumor

To the Editor: Odontogenic hybrid tumors are rare lesions that should be considered in a differential diagnosis of several entities occurring in the jaws.[1,2] To date, only 3 patients of calcifying cystic odontogenic tumors (CCOT) showing areas of an ameloblastic fibro-odontoma (AFO) were published in international literature[2–4]; however, none of these patients were identified in the posterior maxilla area. Thus, this study aimed to discuss the clinicopathologic, tomographic, and surgical aspects of a hybrid lesion (CCOT/AFO) in the posterior maxilla.

A 15-year-old patient was referred for orthodontic treatment. Initial examination showed absence of facial or intraoral swelling; a panoramic radiography showed a maxillary mixed radiolucent/radiopaque lesion in close proximity to the left second molar. A cone-beam computed tomographic (CBCT) examination was performed to evaluate the relationship between the lesion and the unerupted molar, because the orthodontist requested the maintenance of this tooth. A 2 cm well-defined hyperdense mass associated with a malformed and unerupted third molar was observed, which was surrounded by a thin hypodense region. Cone-beam computed tomographic analysis (Fig. 1) provided a clear definition between the presumptive dental follicle of the second molar and the lesion associated with the third molar. After an excisional biopsy, the surgical specimen was sent for histopathological analysis. Microscopically (Fig. 1), a cystic wall partially lined by a stratified oral squamous epithelium with clusters of ghost cells was observed. Island and cords of odontogenic epithelium, dentinoid material, sheet of enamel matrix, mineralized component, cellularized mesenchyme, and tooth-like structures were observed. Thus, the final diagnosis was a hybrid odontogenic neoplasm (CCOT/AFO).

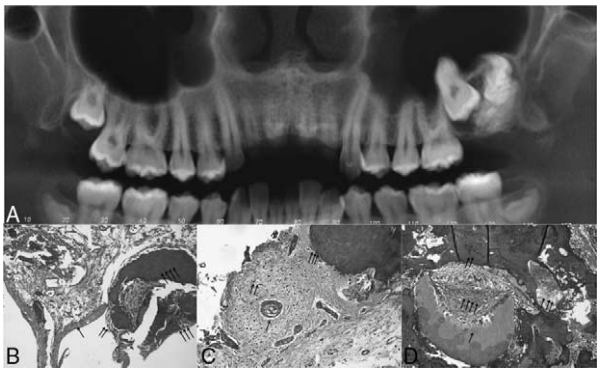

FIGURE 1. A, CBCT image showing a well-defined hyperdense lesion compatible with hard odontogenic tissue and associated with a malformed maxillary third molar. B, CCOT findings (HE stain, ×100): Single arrow describes a cystic wall partially lined by a stratified oral squamous epithelium showing columnar and cubic cells; Double arrow describes cells exhibiting fusiform nucleus and scarce cytoplasm; Triple arrow describes clusters of ghost epithelial cells; Quadruple arrow describes the presence of a dysplastic dentin. C, AFO findings (HE stain, ×200): Single arrow describes follicles of odontogenic epithelium showing ameloblastic arrangement; Double arrow describes a proliferative ectomesenchyme exhibiting fusiform cells with large and ovoid nuclei; Triple arrow describes the presence of a large amount of dysplastic dentin. D, AFO findings (HE stain, ×200): Single arrow describes the deposition of a mineralized dentinoid material; Double arrow describes cementum-like tissue; Triple arrow describes cavities containing enamel matrix; Quadruple arrow describes an ectomesenquimal tissue displayed adjacent to the mineralized material. AFO, ameloblastic fibro-odontoma; CBCT, cone-beam computed tomographic; CCOT, calcifying cystic odontogenic tumors.

The current case was carefully studied for 12 months and a normal bone formation was observed on a follow-up radiograph.

The CCOT can present histologic features associated with other tumors. Among these tumors, odontoma is the most common but may also be associated to the ameloblastoma, adenomatoid odontogenic tumor, ameloblastic fibroma, and rarely, AFO.[2] Currently, we described the first posterior maxillary hybrid odontogenic tumor with features of a CCOT and an AFO. Although extremely rare, it is not unexpected that a CCOT gives rise to an AFO.[5,6]

To date, 3 case reports of mandibular hybrid odontogenic neoplasm with exclusive features of a CCOT and an AFO were described in international literature.[3,4,7] Phillips et al[2] reported this tumor with areas of an adenomatoid odontogenic tumor in the posterior mandible of a 7-year-old boy. Kolomvos et al[1] described a patient of a hybrid odontogenic tumor as a large lesion in a boy of 11 years showing inclusion of tooth 47 whose diagnosis was a CCOT associated with the ameloblastoma, AFO, adenomatoid odontogenic tumor, and fibromixoma odontogenic. This report reinforces the multipotentiality of the CCOT to yield hybrid tumors.

The therapeutic approach for hybrid odontogenic lesions is based on experience of sporadic cases published in literature. In this context, a conservative approach was adopted for the current case. Due to the request of the orthodontist, and analyzing images from CBCT, the planning of the case included the removal of the lesion along with the third molar, and maintenance of the second molar. According to Pontes et al,[8] it is believed that in cases where the teeth impacted by the lesion do not interfere with the surgical removal of the tumor, there is no justification to remove them. In addition, there is still the possibility that the spontaneous eruption of such nonerupted teeth will occur.[9] It, however, is essential to have long-term follow-up in cases where it is opted to have maintenance of the teeth near lesional tissue because of the risk of recurrence, even if it is a rare event. Furst et al[10] reported a patient of an AFO, in a boy of 9 years of age who relapsed after 2 years of surgical treatment probably because of the maintenance of the tooth germ

that was initially associated with the lesion and was maintained after excision of the lesion.

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A Rare Presentation of Epidermoid Cyst

To the Editor: Epidermoid cysts can be present anywhere in the body lined by squamous epithelium, and they occur in approximately 7% of the head and neck region.[1]

A 30-year-old male patient applied to our outpatient clinic and complained of a slow-growing mass placed in the right preauricular region for 10 years. On examination, a 2×2 cm cystic and nontendered mass was revealed.

Complete blood count, comprehensive metabolic and serologic panel, chest radiography, and electrocardiography showed no abnormality. The patient underwent surgery to totally excise this mass. Macroscopically, the mass had a thin-walled capsule surrounding, with a cheesy white material inside (Fig. 1A–B). Histopathologic evaluation showed that the lesion was an epidermoid cyst (because histopathologic material do not have skin appendages)

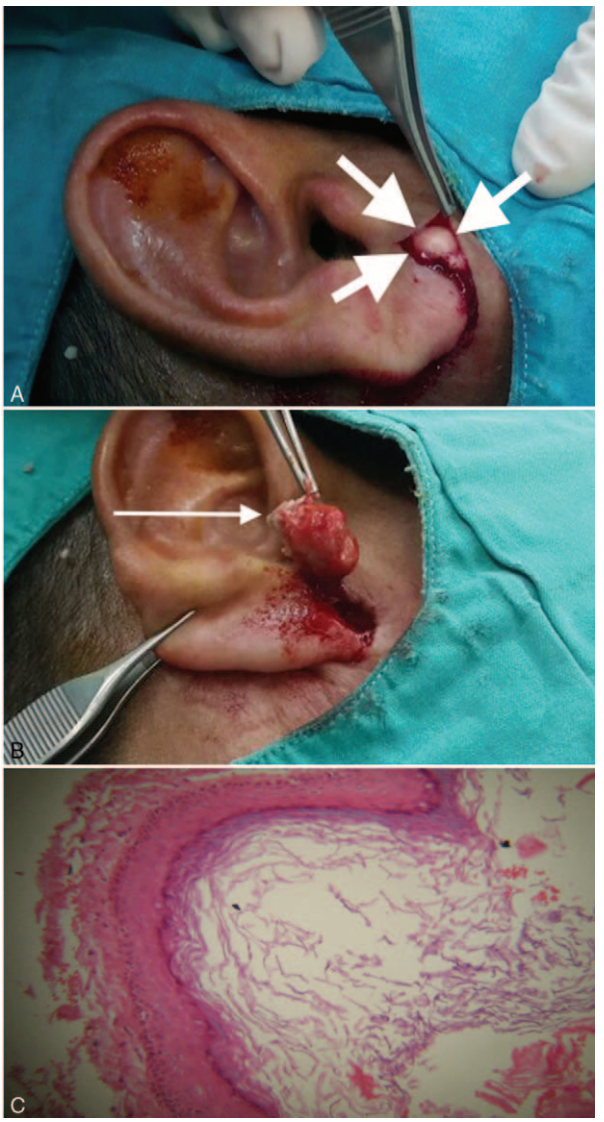

FIGURE 1. A, Intraoperative view of the preauricular mass (white arrows). B, The mass with a cheesy white material inside (white arrow). C, Histopathologic picture shows a cystic cavity lined by keratinised stratified squamous epithelium, with lumen containing abundant keratin content.

(Fig. 1C). If the dermis of cyst contains sebaceous glands, eccrine glands, hair follicles, and mature chondroid elements, the histologic diagnosis is "dermoid cyst."[2] Our patient's pathologic specimen did not have skin appendages such as sebaceous glands, eccrine glands, hair follicles, and mature chondroid elements, so the histopathologic diagnosis was reported as an epidermoid cyst.

A cystic mass in the preauricular region is mostly a Warthin tumor, lymphoepithelial cyst, epidermal keratinous cyst, cystic lymphangioma, lipoma, teratoma, and abscess. The differential diagnosis of these preauricular masses should be confirmed with histopathological examination. With this paper, we have aimed that the epidermoid cysts should be added to this list.

Total surgical excision, for the treatment of epidermoid cysts, reduces the risk of recurrence.

Radiographic studies may lead to suspicion of the anomaly but are not diagnostic. The diagnosis of dermoid cyst is confirmed histopathologically. Complete surgical excision is enough for the treatment of choice.[3] There is rare malignant transformation in the epidermoid cyst.[4]

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Mandibular Osteomyelitis Caused by Periodontal Treatment in HIV Patient

To the Editor: Oral conditions related with HIV infection may identify a person who is unknowingly infected with HIV, and could be used in staging, classifying, and predicting the progression of the acquired immunodeficiency syndrome (AIDS).[1–3] Osteomyelitis of the jaw has been documented as one of the possible presenting manifestations of HIV infection. We report a case of osteomyelitis and its management in an HIV patient, caused by a periodontal treatment. A 40-year-old man was referred to the Oral and Maxillofacial Surgery Department (OMFS) of "São Lucas" hospital in Porto Alegre, Brazil for an evaluation of a gradually evolving bone exposition of the anterior mandible. The patient was diagnosed with HIV infection 4 years earlier, and refused to undergo highly active antiretroviral therapy (HAART). The patient began self-treatment with lemon juice, acupuncture, and homeopathic treatment. After 3 years he was admitted to "São Lucas" hospital referring persistent cough, high fever, and weight loss. After clinical and laboratorial evaluations the patient was diagnosed with tuberculous pericarditis. The patient was treated clinically, and started HAART, after his health condition improved he was referred to the Oral and Maxillofacial Surgery Department for an evaluation. The patient presented a significant bone exposure in the vestibular area of the anterior inferior incisors, which started 1 week after a routine periodontal treatment, 2 months before the admission (Fig. 1). The exposure was initially small and had gradually increased in size with inferior incisor mobility. The computed tomography (CT) scan revealed an ill-defined, radiolucent, and osteolytic lesion in the anterior alveolar area measuring 5×3 cm involving the mandibular anterior teeth. The diagnosis was osteomyelitis, confirmed by incisional biopsy and culture. The culture was positive for *Staphylococcus aureus*. After clinical improvement, the patient underwent general anesthesia for mandibular marginal resection. An intrasulcular incision was performed in the mandibular arch and after mucoperiosteal elevation, osteotomies were performed between the first premolar and canine bilateraly, all necrotic bones were removed until the bleeding bone was evident. In a 1 year follow-up there were no signs of recurrence. It is well known that HIV infection can predispose patients to oral infections although there are few reports regarding osteomyelitis on HIV patients.[1,3] Dental procedures such as dental extractions could facilitate bacterial contamination and bone infection.[4] Dinkar and Prabhudessai reported a case of tuberculous osteomyelitis in which a carious deciduous molar was the portal of entry for the bacilli.[4] The authors pointed the extraction socket as being the portal of entry for *S. aureus* infection.[1] In the current case it seemed to be related to a conventional periodontal treatment, even though the patient had

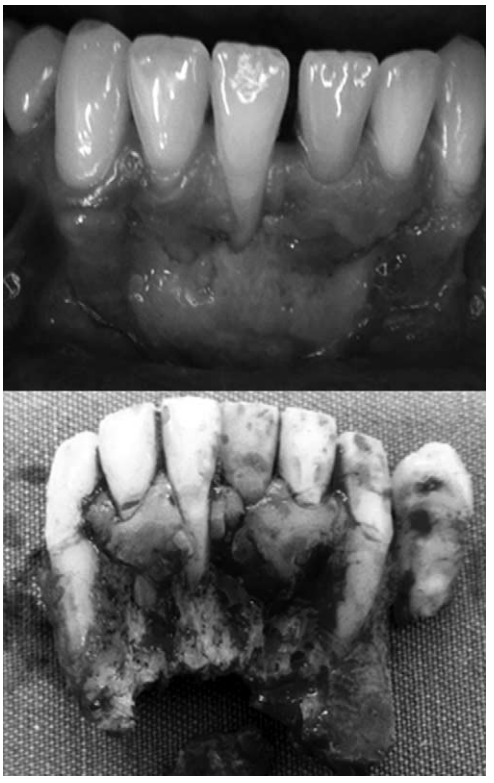

FIGURE 1. Bone exposure in the vestibular area of the anterior inferior incisors and resected specimen.

an excellent oral hygiene and good periodontal health; however, osteomyelitis can occur spontaneously.[1] Although the most likely portal of entry of the offensive microorganisms may have been through the periodontal tissues a hematogenous spread of the microbes may be contemplated in an immunossuppressed state.[1] Although the dental routine treatment may have caused the initial contamination of healthy periodontal tissues, oral health must be encouraged, and routine dental treatment must be established for the HIV patients.[1] In the case presented, the patient omitted the information about being HIV positive for the periodontist, and the treatment was performed with a low CD4 cell count that may have contributed to bone dissemination of the infection. Antibiotic prophylaxis could be considered in the dental treatment if the CD4 cell count is below 200/mm^3 for invasive procedures such as extractions and periodontal treatment, although invasive procedures may not be indicated in the presence of severe immunosuppression.[2]

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Mandibular Reconstruction for a Neglected Langerhans Cell Histiocytosis by Using Free Osteocutaneous Fibula Flap

To the Editor: Langerhans cell histiocytosis (LCH) is a rare proliferative disorder of the Langerhans cell and formerly known as histiocytosis X. Histiocytosis X was first described by Lichtenstein in 1953.[1] Langerhans cell histiocytosis is divided in to 3 clinical entities based on the extent of tissues involved and the severity of the presentation: eosinophilic granuloma of bone; Hand–Schüller–Christian disease, a multifocal variant of eosinophilic granuloma characterized by the classic triad of bony lesions, exophthalmos, and diabetes insipidus; and Letterer–Siwe disease, an acute progressive multisystem disorder that generally affects infants.[2] The estimated incidence is 4 to 5 cases/million in children and 1 to 2 cases/million in adult population.[3,4] The exact etiology of LCH is unknown. Genetic, immaturity of the immune system, neoplasm, or some kind of infectious disease are the theories behind the disease. Abnormal immune response to viral infections especially to HHV-6 is a new theory for pathogenesis of LCH.[5] Skeletal lesions are commonly located in the pelvis, ribs, skull, long bones, vertebrae, and facial bones. Langerhans cell histiocytosis affects both mandible and maxilla but mandible is affected twice as often as the maxilla. The incidence in the jaws is 7.9% and the body and angle are the most affected sites.[1,6] Oral manifestations of LCH are periodontal destruction with gross gingival recession and alveolar bone loss, typically involving a small group of teeth and delayed dental healing after the extraction of teeth. These manifestations may be easily mistaken with common dental disorders such as periodontitis or periodontal dental progress.[7,8]

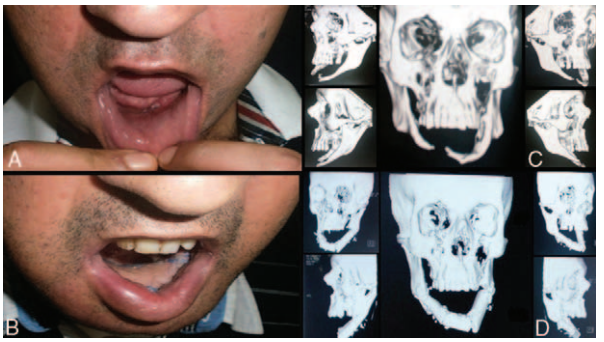

FIGURE 1. A, Preoperative intraoral view. B, Postoperative intraoral view. C, Preoperative three-dimensional CT images. D, Postoperative three-dimensional CT images. CT, computed tomography.

A 36-year-old man diagnosed with LCH was referred to our clinic with complaints of severe mandibular pain, feeding difficulty, and pathologic mandibular fracture (Fig. 1A). Computed tomography (CT) scans showed alveolar bone destruction between 2 angular areas and symphyseal fracture (Fig. 1C). The tumor was excised while the patient was under general anesthesia. An osteocutaneous free fibula flap was harvested and adapted to mandibular defect. Patency of the anastomosis was evaluated with flat panel angiographic CT on postoperative follow-up (Supplementary video). There were no problems on postoperative follow-up (Fig. 1B-D).

Langerhans cell histiocytosis may involve maxillofacial bones and lesions can be diagnosed in early stages and treated with minor surgical interventions. Delayed lesions can be presented with extensive bone lesions. Pathologic fractures and severe pain may occur in mandibula. Osseous or osteocutaneous free fibula flap is the main option for both functional and aesthetic reconstruction of mandibular defect.

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Near-Total Pediatric Parotidectomy for Refractory Chronic Sialadenitis

To the Editor: Traumatic injuries of parotid gland and stensen duct are rare. The treatment depends on the extent of injury and the time elapsed. In case of children, the diagnosis is even more difficult and can easily be overlooked. If it is left undiagnosed or neglected for a long period, it may lead to chronic sialadenitis (CS).[1] Chronic sialadenitis is initially managed conservatively, and if required complex surgical procedures in the form of superficial and near-total parotidectomy are carried out ultimately to cure the condition. Total pediatric parotidectomy is technique sensitive and has never been reported in the existing literature for the management of refractory cases of CS secondary to parenchyma/ductal injury. A 5-year-old male, clinically and radiologically diagnosed case of CS secondary to traumatic injury to stensen's duct with a chief complaint of foul discharge from the mouth, was referred to our department for further evaluation and management. The history revealed a barbed wire injury on the left side of the face 2 years back followed by recurrent pus discharge intraorally. Magnetic resonance imaging (MRI) revealed CS and strictures along the course of the duct (Fig. 1A). Multiple attempts were made to conservatively manage the condition along with dilatation of the stensen's duct. On the basis of long-standing presentation of refractory CS, superficial parotidectomy (SP) along with ductal ligation was carried out under general anesthesia (Fig. 1B). Postoperatively, antibiotics and analgesics were administered. The histopathological examination of the excised superficial lobe confirmed the clinical and radiological diagnosis of CS. Transient facial palsy subsided within 4 weeks, but paresthesia over the pinna persisted. After a month, suppurative discharge started from the suture line. Antibiotics were readministered, but there was no improvement in the condition. Magnetic resonance imaging was repeated after 6 months to evaluate the status of the deep lobe, which revealed active secretion and no change in size, fibrosis, or shrinkage. Deep lobe parotidectomy was then carried out as a last resort (Fig. 1C).

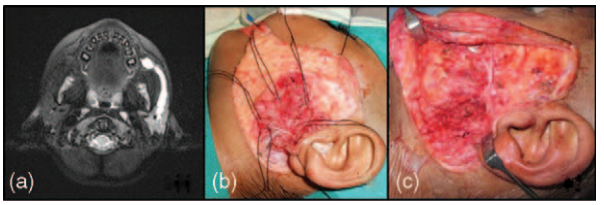

FIGURE 1. A, T2-weighted fat sat axial image shows a dilated with partially beaded appearance of the left-sided stensen's duct in its entire length. B, SP along with ductal ligation. C, Deep lobe parotidectomy.

The histopathological examination of the excised deep lobe confirmed the earlier diagnosis. Postoperatively, transient paralysis of the marginal mandibularis and lower buccal branch of the facial nerve was observed and is still persisting. However, the improvement is marked in the related motor function of the affected nerve. The patient is completely asymptomatic and on regular recall visits.

In the genesis of CS, the primary pathogenic event is believed to be a decrease in the secretion rate with ductal stasis and ascending infection secondary to a stone, stricture, mucous plug, injury to the duct or papilla, or ductal compression by a tumor. In long-standing cases of CS, there are repeated episodes of suppuration, which can lead to dilatation of ducts, acinar atrophy, periductal inflammation, and progressive replacement of the secreting glandular element by the inflammatory infiltrate.[2] In the present case, etiology was barbed wire injury and because of longer duration of symptoms, the more extensive and severe was the involvement of the gland. The general principle of management of CS is to start with conservative treatment, but cases with recurrent episodes must be treated surgically. After the failed conservative treatment, ligation of the parotid duct along with SP was carried out to limit the ascending infection and fibrose the remaining deep lobe. It is not usually necessary to remove the deep lobe, which accounts for only one-fifth of total parotid mass, as this undergoes spontaneous atrophy following SP and duct ligation.[3] Parotidectomy remains the mainstay treatment for both pediatric and adult benign/malignant lesions of the parotid gland. But the extent of removal of parotid parenchyma is justified depending upon the involvement of the 2 lobes. Although pediatric parotidectomy rarely reported, it has a limited negative impact on patients' quality of life. Till date the indications were neoplasms, branchial arch anomaly, vascular lesions and CS of unknown origin.[4] In the present case, refractory CS secondary to stensen's duct injury in a 5-year-old boy was cured with near-total parotidectomy as a last resort similar to what is advocated for adults. To the best of author's knowledge, this is the first case in literature where such a radical approach was used in a pediatric case to completely cure CS.

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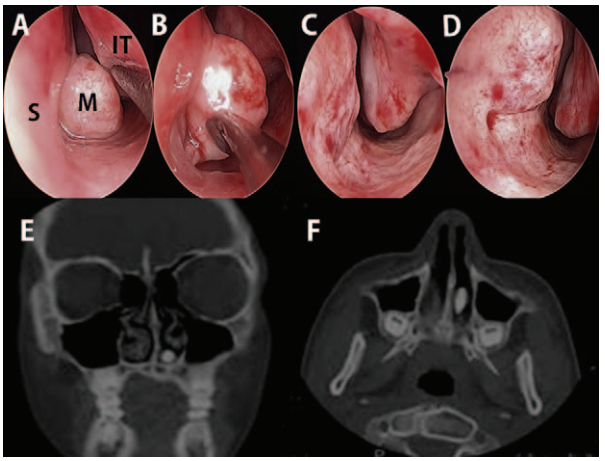

FIGURE 1. A and B, After the submucosal inferior turbinectomy and outfracture surgery to secure the space, nasal endoscopy revealed a nontender, hard, smooth, and movable mass located in the nasal floor between the nasal septum and the midportion of inferior turbinate (S, nasal septum; M, mass; IT, inferior turbinate). C and D, After the complete removal of intranasal tooth, we found that the tooth was fully erupting from the nasal septum and fully covered with the nasal mucosa but, not attached to the septum or the inferior turbinate. E and F, Paranasal sinus CT revealed normal bone continuity of facial skeleton with a radiopaque tooth-like structure on the nasal floor of left side located approximately halfway between the anterior and posterior portion of the naris. CT, Computed tomography.

Fully Erupted Intranasal Tooth Mimicking Benign Tumor

To the Editor: Ectopic eruption of a tooth into the nasal cavity (intranasal tooth) is a rare clinical entity.[1] Although an intranasal tooth is not difficult to diagnose and mostly diagnosed during routine clinical or radiologic examinations, it may be easily missed because of the lack of symptoms and the variable clinical presentations.[2] The intranasal tooth may often be partially or completely covered by nasal mucosa, facilitating ongoing mineralization, and acting as an underlying source of infection, which can result in accompanying debris, rhinolithiasis, granulation tissue, and purulent materials around the intranasal tooth.[3]

PATIENT PRESENTATION

A 12-year-old boy was referred to the otolaryngology clinic with left-sided nasal obstruction and mucoid discharge because of an intranasal benign mass during the last 2 months. He had no history of nasal trauma or sinonasal disease. Nasal endoscopy revealed a $1.8 \times 1.5 \times 1.4$ cm-sized, nontender, hard, smooth, movable mass located in the nasal floor between the nasal septum and the midportion of inferior turbinate (Fig. 1A-B). There was hardly any change in the size of the mass on nasal decongestion. Paranasal sinus (PNS) computed tomography (CT) revealed normal bone continuity of facial skeleton with a radiopaque tooth-like structure on the nasal floor of left side located approximately halfway between the anterior and posterior portion of the naris (Fig. 1E-F). This tooth-like structure was covered with soft tissue density granulation tissue. We achieved to remove the nasal mass via transnasal endoscopic surgery. Because of the mass size, we performed the submucosal inferior turbinectomy and outfracture surgery to secure the space. The operative findings suggested that the tooth was fully erupting from the nasal septum and fully covered with the nasal mucosa but, not attached to the septum or the inferior turbinate (Fig. 1C-D). The patient did well postoperatively and healed uneventfully.

DISCUSSION

Although the etiology of intranasal teeth is controversial, it has been attributed to obstruction at the time of tooth eruption secondary to crowded dentition, persistent deciduous teeth, or exceptionally dense bone. Other proposed pathogenetic factors include: a genetic predisposition; developmental disturbances, such as a cleft palate; rhinogenic, or odontogenic infection; and displacement as a result of trauma or cysts.[4]

This report raises 2 interesting points in diagnosing and treating the completely erupted intranasal tooth. First, the diagnosis of an intranasal tooth can be easily made on the basis of clinical and radiographic findings. Clinically, intranasal teeth are seen most frequently on the floor of the nasal cavity, and are often an ivory white mass without any covering of nasal mucosa, or a protruding reddish tumor-like lesion surrounded by granulation and necrotic debris.[2,5] In the current patient, because the mass, however, was fully erupted and covered with nasal mucosa with no attachment to the nasal septum or the lateral wall of the nose, we clinically believed it to be intranasal benign tumor rather than intranasal tooth. So, benign tumors, such as osteoma, odontoma, and enchondroma, and malignant tumors, such as chondrosarcoma and osteosarcoma, were included for the differential diagnosis rather than radiopaque foreign body, anterior rhinolith, any inflammatory lesions because of syphilis, tuberculosis, or fungal infection with intranasal calcification.[6,7] Computed tomography is the most useful method to evaluate the depth of the eruption site and location of the tooth in accordance with other teeth. The CT findings of tooth-equivalent attenuation and a centrally located cavity (pulp cavity) are highly discriminating features that help to confirm the diagnosis.[8]

Second, the treatment of the intranasal tooth is early extraction, when diagnosed because of the potential morbidity, including external deviation of the nose, nasal septal abscess, and oronasal fistula.[2] Surgical methods depend on the involvement of structures

adjacent to the tooth and potential complications arising from extraction of the tooth. When a nasal tooth is dislocated in the nasal cavity, it is easier to remove it by endoscopy approach, but when the tooth has bony socket in the floor of the nose, it may be extremely difficult to extract.[6] Recently, extraction of the intranasal tooth under endoscopic guidance has the advantages of good illumination, clear visualization, and precise dissection.[2,7] In the current patient, we believed that the mass might be easily removed by endoscopic surgery because the intranasal tooth was fully erupted. We, however, performed the submucosal turbinectomy and outfracture surgery because the mass size, was larger than the nasal patency.

CONCLUSIONS

Although an intranasal tooth, especially fully erupted tooth, is uncommon, it may be confused with other intranasal benign tumors. We should keep in mind this disease entity when encountering patients presenting with nasal obstruction and intranasal benign mass.

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Experimental Research on Intranasally Administered Dexmedetomidine

To the Editor: Dexmedetomidine is a relatively new, highly selective central α2 agonist. It has sedative, pro-anesthetic, pro-analgesic, and anxiolytic effects.[1] Dexmedetomidine has been investigated for this purpose and it was shown to be effective at inducing preoperative sedation and anxiolysis.[2] Intranasal (IN) administration is relatively easy and convenient, it also diminish first liver passage. It has also been demonstrated that IN administration of dexmedetomidine is efficacious and well tolerated in healthy volunteers.[3] The aim of this in vivo study was to evaluate the effects of IN dexmedetomidine on rat nasal mucosa via cytotoxicity compared with saline.

The rats were randomly divided into 5 groups. Group 1 (n = 7) received intraperitoneal (IN) saline, group 2 (n = 7) received intraperitoneal (IP) dexmedetomidine, group 3 (n = 7) received IN saline, group 4 (n = 7) received IN dexmedetomidine, and also group 5 (n = 7) were control group. Dexmedetomidine and saline were administered via IP and IN routes at the doses of 100 μg/kg and 1cc for the study groups, respectively. On the 6th hour of the administration, the rats were sedated with ketamine (80 mg/kg) and xylazine (5 mg/kg) through IP injection. The nasal septal mucosal stripe tissue was removed from the underlying cartilage after sedation. Antigen retrieval was performed for Ki-67 (10 minutes, citrate buffer pH: 6, 700 Watt microwave oven). The sections were removed from the microwave, cooled, and washed in distilled water. They were then stained for Ki-67, using automated method (Ventana, Tuscon, AZ). Ki-67 index was calculated as the number of positive nuclear immune staining in a total of 1000 cells in randomized areas. ANOVA and LSD tests were used for comparison between the groups, and the level of statistical significance was set at $P < 0.05$. All data were analyzed with SPSS 15.0 for Windows.

Intranasal dexmedetomidine usage increased the mucosal proliferation as compared with control and IP saline group numerically; however, this increment was not statistically significant ($P > 0.05$). Average of ki-67 values was 9.57 (SD ± 1.9) in group 1; 15 (SD ± 3.23) in group 2 (Fig. 1); 19.29 (SD ± 4.63) in group 3; 15.86 (SD ± 0.88) in group 4; and 9.86 (SD ± 1.61) in group 5. Intranasal saline usage increased nasal mucosal proliferation statistically, as compared with control group ($P = 0.02$) and IP saline group ($P = 0.02$). Also, IN saline usage did not increased nasal mucosal proliferation statistically, as compared with IN and IP dexmedetomidine usage ($P > 0.05$).

Dexmedetomidine, buserelin, desmopressin, oxytocin, and calcitonin are drugs used in IN way. The low permeability of peptide and marked mucosal peptidase activity require suitable absorption adjuvant which has low local toxicity to increase the systemic update. In general, enzyme inhibitors and permeation enhancers need to be coadministered for successful delivery of these biopharmaceuticals. Classes of enhancers have been associated with adverse effects.[4] Buserelin is GnRH analog and uses intranasally. Oghan et al[5] showed that nasal buserelin usage had no any adverse effects on nasal mucosal tissue in rabbits. α2 Adrenergic receptors are of predominant importance for the mediation of the regulatory effects of catecholamines on several renal functions, including renin release, glomerular filtration, and Na and water excretion.[6] Cussac

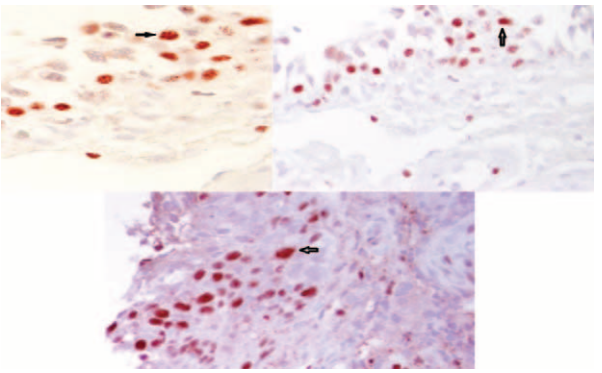

FIGURE 1. In group 2 (IP dex.) and group 3 (IN dex.) Ki-67 proliferation indexes were 15%, and also, it was 25% in group 3 (×400).

et al[7] demonstrated that the α2 adrenergic receptors activates the extracellular signal-regulated kinase pathway and stimulates the proliferation of epithelial cells derived from the proximal renal tubule of rat and pig. The study showed that systemic administration of α2 agonists stimulate the proliferation of epithelial cells derived from the proximal renal tubule and modulate regeneration of tubular cells. Although dexmedetomidine is α2 agonists, in the current study we found similar proliferative effect of dexmedetomidine on nasal mucosal tissue in rats via IN and IP administrations as compared with IN saline usage.

Iirola et al[8] demonstrated that dexmedetomidine is rather rapidly and efficiently absorbed after IN administration. Compared with intravenous administration, IN administration may be a feasible alternative in patients requiring light sedation. Their study also indicates high bioavailability of IN usage of dexmedetomidine.

The strength of our study is the assessment of cytotoxicity of IN dexmedetomidine for the first time. However, usage of one staining method was the limiting point of the study. The results presented are preliminary and the literature concerning this issue is poor. As a result the current study showed that IN administration of dexmedetomidine has not any additional proliferative effect as compared with IN saline. Further studies are needed to support the convenience and safety of this drug.

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Dental Implant Placement in Patients With Osteoporosis

To the Editor: Even though dental implants have a high rate of success on long term, failures are still present. There are some risk factors associated with dental implant failures, such as: smoking, radiotherapy, diabetes, and osteoporosis.[1,2] Especially, osteoporosis has been subjected to controversy about its importance and effects on dental therapy outcomes. Using dental implants for patients with osteoporosis is still debated in dental literature[3] because of quality of local bone, which is a key factor that determines success of dental implant[4] and because of effect of bisphosphonates used in treatment of osteoporosis that could induce osteonecrosis of the jaws.[5]

Osteoporosis is recognized as a common skeletal disorder characterized by reduced bone mass and modification of bone architecture, which leads to increased bone fragility and increased fracture risk.[6] This condition is associated with a decrease in bone quality and quantity and successful dental implant osseointegration depends partially on the recipient site.[7] Furthermore, low bone mineral density and consequently bone loss is significantly associated with periodontitis.[8]

There are relatively few clinical studies that report outcomes of dental implant treatments in patients with osteoporosis and relationship between osteoporosis therapy and outcomes of endosseous implant treatments.

The aim of this study is to assess osseointegration of dental implant in bone with low density. A total of 573 implants were placed for subjects with total or partially maxillary edentulism; 341 implants for subjects (group A) with osteoporosis, and 232 for subjects without osteoporosis (group B). All subjects required bone augmentation techniques. For all subjects, bone density was assessed pre- and postoperatively at 12 months. In both groups, women are prevalent: 92.59% in group A and 83.72% in group B. Main cause of tooth loss is periodontitis (61.11%) in group A, when in group B, there is a balance between periodontitis (41.86%) and caries (44.18%). There is a statistically higher association between osteoporosis and periodontitis. The frequency of complete edentulism was similar 31.48% in group A and 30.23% in group B. The mean number of implants per subject is 6.31 in group A and 5.39 in group B. Failure appeared for 27.78% subjects and 7.33% implants in group A and for 6.98% subjects and 2.59% implants in group B and is related with achieving initial stability. The rate of failure is statistically higher for group A, but there is a significant statistically association between strontium ranelate and low rate of implant failure for subjects with osteoporosis.

According to our findings in this study, it is reasonable to place endosseous implants with bone augmentation in subjects with

osteoporosis, with good success rates even if there are patients with poor bone quality at the time of implants' placement.

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