Simultaneous Removal of Horizontally Impacted Maxillary Canine and Placement of an Immediately Loaded Implant

To the Editor: Epidemiologic data show that, after the third molars, the canines, followed by the premolars, are the most frequently impacted teeth.[1] When impacted teeth are asymptomatic, surgical removal might not be necessary sometimes. Patients, however, seek rehabilitation of the site when the primary canine is lost and the presence of the impacted tooth must be dealt with. Treatment usually requires that either the canine be moved orthodontically to the ridge, when feasible,[2] or the impacted tooth be surgically removed before an implant is placed.[3] Treatment of asymptomatic impacted maxillary canines in adults is inevitable when primary canine becomes lost through extraction or exfoliation or when the impacted tooth becomes symptomatic.[4] The replacement of a single tooth with an implant in the anterior maxilla is a topic of interest for clinicians because of its aesthetic implications.[5] The aim of this article is to present the method of simultaneous removal of impacted maxillary canine and placement of an immediately loaded implant to achieve an aesthetically stable result, with minimal bone resorbtion and shortened treatment period with no incidence of complication.

This article describes a case series of 5 patients where horizontally impacted maxillary canine was surgically removed and immediate implant placement with immediate provisionalization was done. All the patients had undergone thorough clinical and radiologic examination preoperatively. Panoramic radiographs were taken in all the 5 patients and cone beam computed tomography scan was done to further evaluate the position of the impacted canine. After confirmation of the exact position, patient was planned for surgery under local anesthesia. Instead of cutting more amount of bone, the crown was sectioned at cement–enamel junction in all the patients. After the removal of crown portion, the root was removed. After that the osteotomy was prepared and the site for placement of implant was prepared. Touareg-S implants (Adin Dental Implant Systems Ltd., Industrial Zone Alon Tavor, Afula, Israel) of the desired diameters and length were placed. The anchorage and stability of the implant was achieved from the bone in canine pillar region successfully. The residual defect was filled with alloplastic bone graft material (OsteoGen Synthetic Bioactive Resorbable Graft Impladent Ltd.). The closure of the wound was done primarily. Temporization of the implant was immediately done within 48 hours of the procedure in all 5 patients. The crowns were cement-retained acrylic crowns and were kept infraocclusion to prevent excessive forces during the healing of implants.[6]

After 4 months, the patients were clinically evaluated and radiographs were taken to check the radiographic implant osseointegration. Temporary acrylic crowns were removed, impressions were taken, porcelain fused to metal crowns were fabricated and the restorations were successfully delivered to the patients. Implants were evaluated clinically and radiographically at the end of the healing period, at[6] months, and at on an annual recall. The results were that there were no radiolucency noted around the implants, no abnormal reaction at the bone–implant interface, and there was a good consolidation of the graft at the site of removal of the impacted canine.

The removal of impacted canines followed by immediate implant placement and provisionalization minimizes the number of surgical interventions and the waiting time, although increased surgical skill is needed to place the implants. This treatment modality avoids the need for conventional preparation of teeth as part of prosthetic reconstruction or prolonged orthodontic treatment aimed at bringing the impacted canine to the dental arch. Combining the implantation with immediate provisionalization, despite the initial large bone defect caused by the impacted canine extrusion, preserved the alveolar bone and shortened the treatment period.

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Argyrophilic Nucleolar Organizer Regions Count and its Diagnostic Significance in Condylar Hyperplasia

To the Editor: The mandible is a bone having a great risk of developing a deformity resulting in facial asymmetry, being condylar hyperplasia (CH) the most notorious.[1] Recently, investigators have used the argyrophilic nucleolar organizer regions (AgNOR) count to diagnose active CH. Nucleolar organizer regions (NORs) were described in 1930s[2] as nucleolar components containing a set of argyrophilic proteins, which are selectively stained by silver techniques. Argyrophilic nucleolar organizer region dots increase during the proliferative stage of the cells.

Esalami et al[3] compared 13 normal condyles with 9 CH patients using hematoxylin, eosin, and colloidal silver, finding that the amount of AgNOR dots was higher in CH. The statistically significant difference ($P = 0.0001$) was confirmed by Mann-Whitney test. Fariña et al[4] investigated the relationship between SPECT, AgNOR, and histology in 8 CH patients, finding wide variations in condylar histology. An inverse relationship between age and condylar thickness was seen. The relationship between AgNOR dots and age was inverse. This means that the older the patient, the lesser the dots found ($r = -0.65$, $P = 0.08$).

To date, only 2 groups have attempted to establish the diagnostic value of AgNOR in CH. Although Esalami et al[3] concluded that AgNOR count could be utile, Fariña et al[4] found a relative value. Argyrophilic nucleolar organizer region count could play an important role in the diagnosis of CH. To obtain solid conclusions, however, extensive research must be performed using larger samples.

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Parapharyngeal Space Lipoma

To the Editor: Lipomas, in the neck, involving parapharyngeal space are extremely rare. Lipomas arise in the head and neck region, mainly in posterior cervical triangle.[1] In this letter, we aimed to analyze clinic, radiologic, and histopathologic features of parapharyngeal space lipoma (PSL).

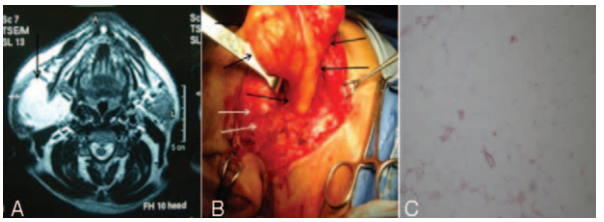

FIGURE 1. A, MRI image of the mass (arrow) is seen (in T1 sequence, lipoma is seen hiperintense). B, The mass which originated parapharyngeal space and, relationship structures are seen (black arrows point out the mass and, white arrows point out parotis tissue). C, Histopathology showing sheets of mature adipocytes, confirming the diagnosis of a lipoma.

A 68-old-male was referred to our clinic with a right parotid area swelling. It had been 9 months since he had recognized the mass. He had not felt any pain or tenderness. On physical examination, a 5×6 cm mass located in the right parotid location was felt, which was soft on palpation. Facial nerve motor function was normal. Other head, neck, and systemic examination findings were normal. On our patient's MRI, on T1 sequences, the mass was seen hiperintense (Fig. 1A). The lesion was excised totally by a transcervical approach under general anesthesia (Fig. 1B). At postoperative 7th months, head and neck examination was normal. Postoperative histopathologic evaluation was reported as lipoma (Fig. 1C).

Neoplasms arising in the parapharyngeal space are rare, accounting for less than 1% of the tumors of the head end neck. Most of them are benign.[2] And they include salivary gland tumors forming the majority, followed by tumors of neurogenic origin.[3] Lipomas, in the neck, involving parapharyngeal space are extremely rare.[4] With this manuscript, we have aimed that the lipomas should be kept in mind in differential diagnosis of the parapharyngeal masses.

Magnetic resonance imaging (MRI) is a very useful method for definite differential diagnosis and operative plan in preoperative period.

The symptoms of the PSL depend on mass effect (change from asymptomatic (especially initially) to respiratory distress).

Treatment of the PSL is totally surgical excision to prevent recurrence. Of course, surgical option to a parapharyngeal mass varies, according to the location, dimension, and malignancy potential of the tumor. The transcervical approach may be used for tumors up to 8 cm.[3] We chose transcervical approach in our patient because dimension of the mass was less than 8 cm. The transcervical approach is a precious approach because it is providing perfect exposure of the neurovascular structures.

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Modification of the Vestibular Mucoperiosteal Flap Technique for Closure of Oroantral Fistula

To the Editor: There are many surgical techniques for closure oroantral fistula, such as the use of biodegradable polyurethane foam,[1] Bichat buccal fat pad,[2] septal cartilage graft,[3] and palatal rotation flaps.[4] However, the use of a mucoperiosteal flap remains the procedure of choice of the closure of an oroantral fistula.[1] This procedure offers greater patient comfort and better results in comparison to other techniques.[5] On the other hand, a flawed execution of the method may result in the recurrence of the fistula. The aim of this article is to describe a modification to the marginal flap technique for fistula closure that promotes more efficient healing and minimizes recurrence.

Before the surgical closure of an oroantral fistula, decontamination of the maxillary sinus must be performed through daily irrigation with 0.9% saline solution 3 days before surgery, together with the use of 500 mg of amoxicillin administered orally every 8 hours (Fig. 1A). The use of antibiotic therapy should be continued for 12 days following the surgery.[6]

The surgical procedure might be performed under local anesthesia with a vasoconstrictor (2% lidocaine with epinephrine [1:100,000]). Before the mucoperiosteal flap an incision around the fistula should be made, detaching it and suturing the edges with inverted knots using 4-0 mononylon thread. The knots of this suture remain buried in the interior of the oroantral fistula, preventing exposure of the knots. Following, a trapezoidal mucoperiosteal incision should be made on the alveolar ridge in the vestibular region near the fistula. Then, scarification of approximately 5 mm of the alveolar face of the flap is then performed, following by the complete displacement of the flap (Fig. 1B). Several periosteal incisions must be made in the flap to allow it to cover the fistula without tension, and allow that the edge of the flap be inserted under palatine mucosa. Next to the fistula, the palatine mucosa must be detached. Then, the alveolar portion of the flap is inserted below the previously detached palatine mucosa. The surgeon must make sure that the all scarified area of the flap should be positioned under the detached palatine mucosa. Isolated U-shaped sutures with 4-0 mononylon thread should be placed to stabilize the flap in position (Fig. 1C). The stitches should be removed 10 days after surgery (Fig. 1D). The placement of the scarified tissue under the palatine flap leads to a more effective primary wound closure reinforces the stabilization of the flap and diminishes the possibility of its displacement and recurrence of the oroantral fistula.

Following surgery, the patient must be instructed not to perform any actions that would tend to displace the repositioned flap, such as sneezing with one's mouth closed or blowing one's nose. This care leads to a better result and prevents the displacement of the flap and recurrence of the fistula.

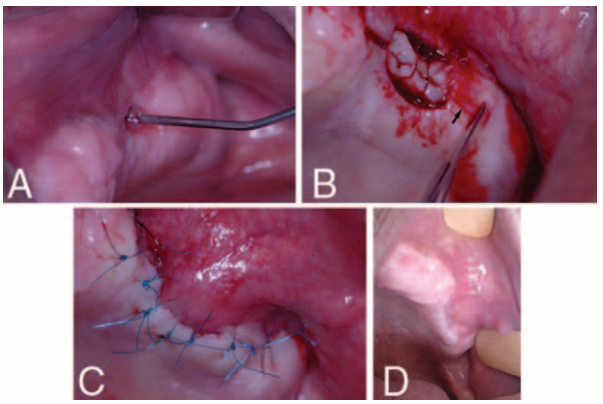

FIGURE 1. (A) Preoperative irrigation with 0.9% saline solution. (B) Filled fistula, trapezoidal incision, and scarification of 5 mm of flap (arrow). (C) Insertion of scarified portion of flap under palatine mucosa and U-shaped suture with mononylon thread. (D) Healing of flap 10 days following surgery.

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Root Particle of Maxillary Premolar in Facial Cutaneous Fistula as the Rare Complication of Exodontia

To the Editor: Extraction is unquestionably the most common oral surgical procedure and it is supposed to be the simplest one, when compared with other major oral surgeries. It, however, is not free from the complications. Root fracture, root or tooth displacement may develop unless proper assessment of the patient has been

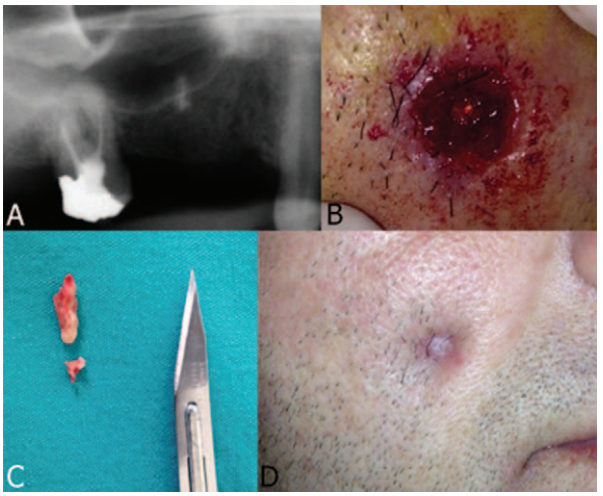

FIGURE 1. A, The panoramic radiography revealed the fractured endodontically treated root superposed on the floor of the maxillary sinus. B, After a while, tooth particles were seen within the granulomatous tissue, and they were removed. C, There were 2 fragments with sharp edges, and a particle of guttha-percha was seen in one of it. D, The patient was followed up postoperatively and showed significant improvement after 3 weeks.

done.[1,2] If the fractured root tip or the whole tooth is retained for a long time in its displaced space, infection and eventually abscess and a cutaneous fistula may develop.[3,4] Here, we report a case of an cutaneous fistula which contains root particles of upper first premolar that was previously attempted to extract, and accidentally displaced into the deep buccal tissues. As we know, there is no such case in the literature.

A 30-year-old male patient came to our clinic complaining about the pain, swelling, and cutaneous fistula on his right cheek. Tracing back the history, the patient had his upper right first premolar extracted approximately 8 months ago in a different hospital. He mentioned that the extraction was somewhat difficult for the dentist at that time, and after surgery he felt mild discomfort on the right side of his face. He said he had felt swelling and pain in the same region including under the right eye. The condition was resembling the canine space abscess. These schemes of symptoms repeated over and over again and medications provided only temporary relief. After that, white-colored pus was discharged from the swelling approximately 3 months before visiting our clinic. He told that the fistula has never healed since that time.

Upon examining his oral condition, a mild swelling with tenderness on middle part of his right cheek and a cutaneous fistula with retracted skin were noted. On intraoral examination, healed upper first premolar area was evident and there was no sign of inflammation. The panoramic radiography showed the fractured root (Fig. 1A). Surgical intervention did not result in manifesting the particle. This finding suggested that the fractured root was actually in the soft tissues rather than in the alveolar bone or maxillary sinus floor. Afistulectomy and removal of the root particle were planned. Ring block anesthesia was performed, and after circumferential excision of the fistula margins, attempts were made to remove the dental particles through cutaneous fistula. In our first attempt, we managed to retrieve 2 fragments close to the external orifice of the fistula (Fig. 1B-C). Rest of the granulation tissues were removed and the fistula tract was irrigated. Then it was sutured in layers. Postoperative course was uneventful (Fig. 1D).

When it comes to the issue of displacement complications during extraction, the literature often presents the cases of third molar. There have been many published case reports of displaced teeth, but these circumstances are still seen as rare complications.[5] Despite the fact that we have little information about how the first extraction attempts were made, some clues let us make some predictions. The tooth whose root was fractured and displaced was the endodontically treated upper first premolar. Maxillary first premolar teeth generally have very thin 2 roots and are subject to fracture easily and, moreover, being endodontically treated makes it more likely to happen.[1,2] These may be the reasons why the tooth was fractured, but it seems fractured root tip was further displaced into the deep buccal tissues. Excessive force and incorrect use of the elevator may lead to this type of complication. If the fragment is not visible and the dentist has lack of experience, we recommend that the practitioner should halt the procedure and refer the patient to an oral and maxillofacial surgeon. As we noted before, when we surgically seek the fragment in the alveolar bone, we could not find it. At this situation, we preferred to seek the particle in the cutaneous fistula but we recommend computerized tomography (CT) scan or at least occlusal radiography to identify the exact position. To the best of our knowledge, this is the first case report of a retrieval of the tooth fragment from the cutaneous fistula on the cheek.

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Pediatric Nevus Sebaceous of the Scalp: A Reconstructive Challenge

To the Editor: Nevus sebaceous of Jadassohn (NSJ) is a hamartoma with epidermal, follicular, sebaceous, apocrine elements that occurs most frequently on the scalp. It is frequently associated with multisystem disorders and carries a small risk of development of malignancy. The treatment options are complete excision, photodynamic therapy, carbon dioxide laser resurfacing, and dermabrasion. None of these treatment modalities ensures complete removal of the lesion and therefore, a risk of recurrence or malignant transformation always exists. Reconstruction is mostly accomplished using local rotational flaps that pose an intriguing challenge to the surgeon in case of larger defects in pediatric patients. A clinically and histopathologically diagnosed patient of NSJ in a 2-year-old patient was referred to our center for further management by a dermatologist. The lesion was round, approximately 10 cm in diameter, located on left parieto-occipital region (Fig. 1A). Surgical excision and reconstruction using

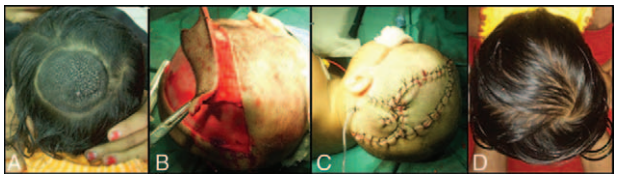

FIGURE 1. A, A large NSJ in the left parieto-occipital region. B, Insufficient STF pedicled V-Y advancement flap. 1C, Complete closure using STF pedicled V-Y advancement and OA pedicled flaps. D, 1-year postoperative result. NSJ, nevus sebaceous of Jadassohn; STF, superficial temporal fascia; OA, occipital artery.

superficial temporal fascia (STF) pedicled V-Y advancement flap was planned after tracing the vessel using hand held doppler (Fig. 1B). The procedure, however, could not be accomplished because of inadequacy of the flap and intraoperatively it was decided to use an occipital artery (OA) pedicled flap in addition. The postablative defect was finally closed without tension using both the flaps (Fig. 1C). The postoperative period was uneventful and the result was cosmetically acceptable with optimal hair growth (Fig. 1D). Both STF pedicled V-Y advancement and OA pedicled flaps are known to produce aesthetic results.[1,2] The defects of scalp should be ideally replaced with hair-bearing scalp not just for aesthetic appeal but also to reconstruct the defect with a tissue of the same histology. The choice of the flap depends on the site of defect. Unilateral rotation scalp flaps based on temporal vessels is an excellent choice.[3] In patients of larger defect this, however, may be inadequate. Use of additional OA pedicle flap in conjunction proves to be a viable alternative option for reconstruction of larger defects. To the best of the knowledge of the authors, this is the largest defect ever reconstructed in a pediatric patient using STF pedicled V-Y advancement and OA pedicled flaps in the existing literature.

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Bilateral Antrochoanal Polyp

To the Editor: The antrochoanal polyps (ACP) are benign neoplasms of the nasal cavity, which originate from maxillary sinus mucosa, prolapse into the nasal cavity by growing through the natural or accessory ostium extending to the choana, nasopharynx, and sometimes to the oropharynx. It was first described by Gustav Killian in 1906.[1] ACPs are generally unilateral. Bilateral antrochoanal polyps are extremely rare with few reports in the literature. In this article, we presented a patient with bilateral ACP.

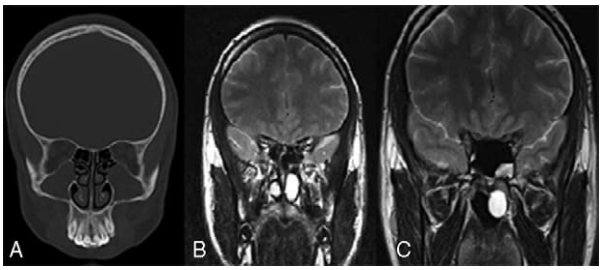

FIGURE 1. A, Coronal CT showed loss of aeration at both maxillary sinuses. B, T1-weighted MRI showed elongation of polyps to choana. C, T1-weighted MRI showed hyperintense appearance of the polypoid mass protruding though the oropharynx. CT, computed tomography; MRI, magnetic resonance imaging.

A 20-year-old boy referred to our clinic with an increasing headache, which started with a feeling of pressure in the face 6 months ago. In last 2 months, persistent rhinorrhea and decreased sense of smell were begun. On oropharyngeal examination, a cystic polypoid mass, protruding to the oropharynx was observed. Fiberoptic endoscopic examination of the nasal cavity revealed a polypoid mass at the right nasal cavity, protruding from maxillary ostium and extended to the choana. On the left nasal cavity, the polypoid mass originating from maxillary ostium was extended to oropharynx. A computerized tomography (CT) was performed, which revealed aeration loss in both maxillary sinuses and polypoid masses protruding toward the nasopharynx at both nasal cavities. (Fig. 1) The polyps were removed by functional sinus surgery. Histopathologic examination revealed benign polyps.

Antrochoanal polyps originate most commonly from the posterior wall of the maxillary sinus.[2] Although the pathogenesis of the lesion is not clear yet, they are believed to occur by the expansion of cysts into the maxillary sinus mucosa.[3] They have a higher prevalence in children and are more common in men than women.

Owing to its benign character, ACP do not damage the surrounding soft or bony tissues. Because of obstruction of nasal cavity, it may cause smelling and breathing disorders. Patients may also complain with rhinorrhea, epistaxis, snoring, foreign body sensation, halitosis, and headache.[1]

The diagnosis of the disease depends on endoscopic examination of nasal cavities, CT findings, and histopathologic results. The differential diagnosis of ACPs should include juvenile angiofibroma, nasal glioma, meningoencephalocele, inverted papilloma, mucocele, mucus retention cyst, Tornwalt's cyst, grossly enlarged adenoids, lymphoma, and nasopharyngeal malignancies.[4]

The treatment of the disease is surgery. Conservative approaches, such as polypectomy, are recommended in patients under 8 years old. Although polypectomies are preferred to prevent the development of teeth and paranasal sinuses, recurrence is often observed after polypectomy. Functional endoscopic sinus surgery (FESS), the most common surgical treatment method, is the gold standard method.[1]

Although ACPs are common in otolaryngology practice, bilateral ACPs are extremely rare. In a patient with ACP, it must be kept in mind that, there may be another polyp at the opposite side.

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Vallecular Cyst Causing Dysphagia and Stridor

To the Editor: A 42-year-old man presented with progressive dysphagia and stridor for 3 days. The patient had no medical history. On physical examination, the patient was irritable and had stridor. Oxygen saturation and vital signs were normal. All clinical findings were normal on the nasal and oropharyngeal examination. The flexible laryngoscopic examination revealed a mobile supraglottic mass that caused subtotal obstruction of the laryngeal inlet. The mass was approximately 3 cm in diameter, of a yellowish color with prominent overlying vasculature. Because of the mass, the epiglottis and vocal folds could not be visualized (Fig. 1A). Magnetic resonance image demonstrated a cystic lesion approximately 35×30 mm in size, causing the subtotal narrowing on the airway, hypointensity in T1A-weighted images, and hyperintensity in T2A images, located on the vallecula without invasion of surrounding tissue (Fig. 1B).

Having been clinically diagnosed with a vallecular cyst following transoral fiber-optic intubation, the patient then underwent direct laryngoscopy and cyst excision. Endoscopic laryngoscopy techniques are used firstly for upper airway lesions. A laryngoscope was used to expose the cyst, which was then totally resected with microforceps. The surgery was uncomplicated and the patient's symptoms were alleviated postoperatively. A histopathologic examination of the removed mass confirmed the diagnosis of a vallecular cyst. Follow-up at 3 years showed no recurrence of the cyst.

Vallecular cysts are usually asymptomatic lesions in adults. Asymptomatic cysts can become infected and cause acute symptoms.[1] There are reports in the literature of infected vallecular cysts causing dysphagia, stridor, acute airway obstruction, globus, and obstructive sleep apnea syndrome in adults.[1,2]

Endoscopic laryngoscopy techniques are used firstly for upper airway lesions. Diagnosis of laryngeal cysts can be confirmed by computed tomography (CT) or magnetic resonance (MR) imaging studies that may reveal the exact extension of the lesion.[3]

Treatment of a vallecular cyst is surgical excision via direct laryngoscopy. Surgical intervention via external approach carries more significant morbidity. Aspiration of the cysts is not recommended because of high recurrence rate.[3] Patients with huge vallecular cysts, present an extreme difficulty in securing the airway for anesthesiologists. Blind intubation attempts might rupture the cyst. The use of flexible fiber-optic bronchoscope might increase the success of airway management.[4]

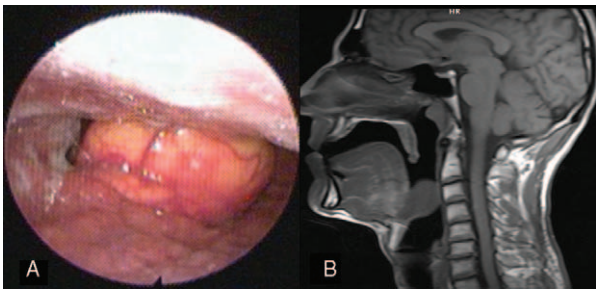

FIGURE 1. A, Flexible laryngoscopic view of huge vallecular cyst. B, Sagittal T1 magnetic resonance image showing a vallecular cystic mass subtotal narrowing the airway.

We report a vallecular cyst causing stridor and dysphagia. An endoscopic and radiologic (CT/MRI) examination should be performed for patients who present with persistent stridor and dysphagia.

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Intradiploic Epidermoid Cyst in the Skull

To the Editor: The incidence of cranial epidermoid cysts is <1% among all cranial tumors. Most of them are located in the intracranial space, for example, the cerebellopontine angle or the suprasellar space. Intradiploic epidermoid cysts are rare and it was first described by Müller in 1838.[1,2] From a review of related literature, such cysts can be located in any part of the skull, and a male predominance has been noted. In this article, the authors present the case of an intradiploic epidermoid cyst and review the pathogenesis of intradiploic epidermoid cyst.

CLINICAL REPORT

A 20-year-old man, without a history of major trauma, presented with sudden onset of headache involving nausea and vomiting, 5 days after a traffic accident. He had initial loss of consciousness, but regained consciousness soon after the accident. On admission, the skull x-ray showed a round-shaped with sclerotic margined lesion over the temporoparietal area (Fig. 1A). Further examination using computed tomography and magnetic resonance imaging (MRI) of the brain revealed a 25×36 mm round mass, extra-axial bone in location, at left frontotemporoparietal area with focal brain compression (Fig. 1B-E). The patient underwent left temporoparietal craniotomy with tumor excision (Fig. 1F). The pathology revealed an epidermoid cyst. The postoperative course was uneventful and the 1-year follow-up image showed no evidence of recurrence.

DISCUSSION

The epidermoid cysts are slow-growing in nature, which causes the gradual progression of erosion and expansion of the inner and outer tables of the skull with new bone formation around the margin.[3] This characteristic makes the diagnostic features of radiolucent lesion with sclerotic margin in x-ray findings. The differential diagnosis includes dermoid cysts, eosinophilic granuloma, fibrous dysplasia, osteomyelitis, and metastatic lesions. Dermoid cysts cannot be differentiated from epidermoid cysts by using plain film alone; however, the dermoid cysts occur frequently in the periorbital area. Epidermoid cysts appear usually with a definite and sclerotic border, whereas osteomyelitis and fibrosis dysplasia may present with some degree of sclerosis. This is in contrast with lesions of eosinophilic granuloma and metastatic lesions, which are not usually rimmed by sclerosis. Magnetic resonance imaging is the diagnostic tool of choice, which demonstrates low signal intensity on T1-weighted MRI (Fig. 1C), and high-intensity on T2-weighted MRI (Fig. 1D).[3] The best way to differentiate epidermoids from others is by using diffusion-weighted imaging, which shows an intense signal (Fig. 1E).

The pathogenesis of intradiploic epidermoid cysts has been postulated as congenital and acquired mechanisms. Congenital acquisition may develop from the ectopia or entrapment of ectodermal tissue during the third to fifth week of embryogenesis.[1] The acquired type develops following head trauma with epidermal tissue being implanted into the calvarial bone marrow.[2,4] Our patient denied any prior trauma to that area, and the tumor was located within the temporoparietal suture, which is suggestive of a congenital origin.

Intradiploic epidermoid cyst is a slow-growing lesion and the symptoms signs are related to the tumor size. For small lesions, patients were often asymptomatic and diagnosed incidentally.[2,3] As the tumor growth progresses, patients may have symptoms signs related to mass effect such as focal neurologic deficit, increase intracranial pressure, malignant transformation, and meningitis. Inflammatory reaction of the adjacent meninges is elicited in case wherein the tumor ruptured.[4] With regards to cosmetic concerns, neurologic deficits and malignant transformation related to the cyst progression, early surgical excision is the treatment of choice and a subsequent cranioplasty is recommended, particularly for the very large intradiploic epidermoid cysts associated with significant bony defects. Total removal of these cysts is associated with a very good long-term prognosis. The recurrence rate is around 8.3% to 25.0%, if the cyst wall cannot be completely removed.[1]

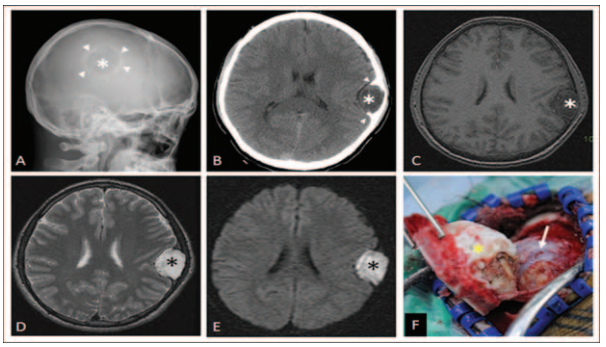

FIGURE 1. A, Skull lateral view showed osteolytic lesion with sclerotic margin (arrow head) over left temporoparietal area. B, Brain computed tomography without contrast demonstrated an intradiploic mass with peripheral hyperostosis. Magnetic resonance imaging images revealed hypointense on T1-weighted image C, hyperintense on T2-weighted image D, and hyperintense on diffusion-weighted image E. F, Intraoperative picture showed the removal of the tumor (*) with adjacent skull bone and the compressed underlying dura (arrow).

CONCLUSIONS

Intradiploic epidermoid cysts are benign skull lesions, which can grow to reach an enormous size causing the neurologic deficits and may also undergo malignant transformation.[4] For the majority of cases, a skull x-ray is a good screening test, which will generally show an osteolytic lesion with sclerotic margin. Computed tomography and MRI of brain are helpful in diagnosis and evaluation for surgical resectability of tumors. The total removal of these cysts is associated with a very good long-term prognosis.

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Fish Bone Induced Sialolith in Warthon Duct

To the Editor: Submandibular sialadenitis associated with stone of Wharton duct is quite common but the incidence of foreign body associated sialolithiasis is scarce, very few patients have been reported in the literature.[1] We present a case of a 45-year-old man who had sialadenitis originated from fish bone induced sialolith.

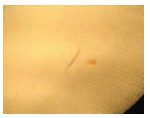

FIGURE 1. Excised Warthon duct stone and fishbone that serve as a scaffold for sialolith.

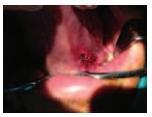

FIGURE 2. After marsupialization, intraoperative view of the placed tupe into the Wharton duct.

A 45-year-old man suffering from a recurrent sore swelling on the left submandibular area, the diameter of which was increasing with chewing for 6 months. There was no history of a fishbone prick during the regular habit of eating fish as he could remember. On the physical examination swollen left submandibular gland was noted. During the intraoral examination, stone was palpated in the left Wharton duct. Purulent drainage was observed from the opening of the duct when compressing. Ductal dilatation in the left submandibular gland was revealed on the ultrasound examination. Therefore, the diagnosis was made as a left submandibular sialolithiasis. The patient was operated under general anesthesia after acute sialadenitis was recovered with the medical treatment. Circa 2 cm long incision was performed to the mucosa of floor of the mouth covering the left Wharton duct and after the incision of the duct a stone with dimensions of 5×2 mm was removed. After the crushing of the stone fish bone nidus was seen together with the sialolith (Fig. 1). Marsupialization of warthon duct lumen was done and stent was placed into the channel (Fig. 2). All symptoms relieved after the oral intervention. Stent was removed at 7th day postoperatively. Postoperative histopathological examination was reported as cartilage-like organic material (Figs. 3 and 4). Postoperative follow-up was normal at 6 months.

Sialolithiasis is the most common cause of obstructive sialadenitis and it constitutes %50 of the major salivary gland diseases.[2,3] Submandibular gland is the most affected gland due to its anatomic features and secretion characteristics.[4,5] Stones are located frequently in the distal third of the Wharton duct. Sialoliths consist of many different organic (glycoproteins, mucopolysaccharides, cellular debris) and inorganic substances (calcium carbonates, calcium phosphates).[4,6] The etiology of sialolithiasis is almost always endogenous.[3] But, few cases have been reported about sialolithiasis arising from foreign body, especially fish bone.[2,6,7] Reported foreign bodies in Warthon duct are quite varied such as paper pin, tooth brush bristle, bird feathers, splinters of wood, hairs, piece of metal, sliver of fingernail, and fish bone.[1–3,5] As in our case these foreign bodies may lead to stone formation. There are 2 hypotheses about foreign bodies associated with sialolith development. One of them and the most accepted one is that, foreign bodies entering the duct via traumatic way and cause stone formation with recurrent sialadenitis or serve as a nidus for the deposition of saliva salt. The other is the retrograde entrance of foreign body. But, this way is extremely rare due to Warthon ducts course and physiological features of submandibular gland.[3,8] Presented case had a history of eating fish regularly. But, there is no history of a fish bone prick he could remember. As the treatment of recurrent sialadenitis, intraoral sialolithotomy and marsupialization is the most preferred intervention modality for sialoliths when it is located in the distal third of the gland and is easily palpated.[1,4] If dense fibrosis occured due to recurrent infections, transcervical approach should be applied. The prognosis is usually well and there is generally no recurrence of sialoliths in intraoral sialolithotomy approach.[4]

As a result, foreign bodies may cause sialolith formation rarely and intraoral interventions to sialolith can provide better results in appropriate cases.

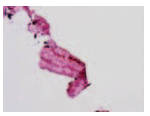

FIGURE 3. Cartilage-like organic material (hematoxylin-eosin × 40).

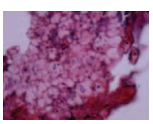

FIGURE 4. Cartilage-like organic material (hematoxylin-eosin × 400).

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Choroid Plexus Carcinoma in the External Ventricle of an Adult

To the Editor: Choroid plexus tumors (CPTs) are uncommon neoplasms, which are derived from choroid plexus epithelium.[1] The CPTs account for 0.4% to 0.6% of central nervous system tumors; however, choroid plexus carcinomas (CPCs) constitute

20% to 30% of CPTs.[2] It is noticeable that CPC occurs predominantly in pediatric population and is located in the ventricle.[3,4] Here, we present a case of CPC of the external ventricle of a 59-year-old man.

A 59-year-old man, who had a history of alalia for nearly a month, was admitted to our hospital. General physical examination was unremarkable. Neurologic examination was entirely within normal limits. Magnetic resonance imaging (MRI) showed a mass with a cyst in the temporoparietal lobe. The mass was isosignal intensity on T1- and T2-weighted images showed a wide, diffusely hypersignaling intense area (Fig. 1A-B), and the gadolinium injection induced homogeneous enhancement of the cyst wall of the tumor (Fig. 1C). A left temporoparietal craniotomy was carried out and gross total removal of the tumor was performed (Fig. 2A-B). Intraoperatively, following a left temporoparietal cortical incision, the tumor was exposed. It was reddish-tan in color, moderately vascularized and adhesive to surrounding brain. Histologic examination revealed focal papillary structure, obvious pleomorphism and invasion of the tumor cells into the surrounding brain (Fig. 3A). Immunohistochemical examination demonstrated that tumor cells were reactive for S-100 protein, cytokeratine, epithelial membrane antigen (EMA), epithelial membrane antigen (EGFR), and vimentin (Fig. 3B-F), but not reactive for glial fibrillary acidic protein (GFAP) (Fig. 3H). Ki-67 proliferation index was 60% (Fig. 3G). The tumor was diagnosed as a CPC. The patient received chemotherapy and radiotherapy in the postoperative course. Six months after the operation, the tumor, however, recurred on follow-up MRI (Fig. 1D-E). The patient underwent the second surgery, and there were no intraoperative complications. Until the manuscript submitted, this patient is still alive.

Within the family of CPTs, they are typically classified as choroid plexus papilloma (CPP) and CPC, respectively.[3] Choroid plexus carcinomas (CPCs) are the low degree of pathologic differentiation type of the CPTs, and be a highly aggressive malignant tumor.[4] In the literature, nearly all the cases of CPC occur predominantly in the ventricle.[1] Few studies, however, have reported that the CPC is located in the external ventricle of an adult.

The differential diagnosis of CPC should include the following: CPP, astrocytoma, and metastatic carcinoma.[5] Neuroradiologic features are nonspecific in CPC.[5] When the tumor invades the parenchyma or presents with metastatic features it may suggest the diagnosis of CPC. But some CPPs also have the feature of cerebral

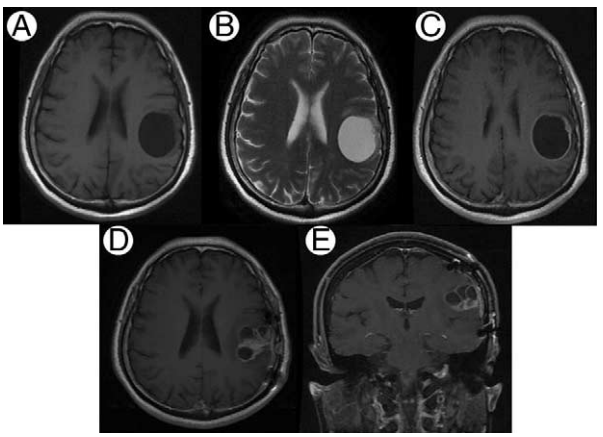

FIGURE 1. The MRI of the preoperative and recurrent tumor. A, T1-weighted axial MR image shows a cyst in the temporoparietal lobe. B, T2-weighted axial MR image shows a cyst in the temporoparietal lobe. C, After Gadolinium administration, T1-weighted axial MR image shows enhancement of the cyst wall. D-E, 6 months later, Gadolinium-enhanced axial and coronal T1-weighted MR image shows the recurrence of the tumor. MR, magnetic resonance.

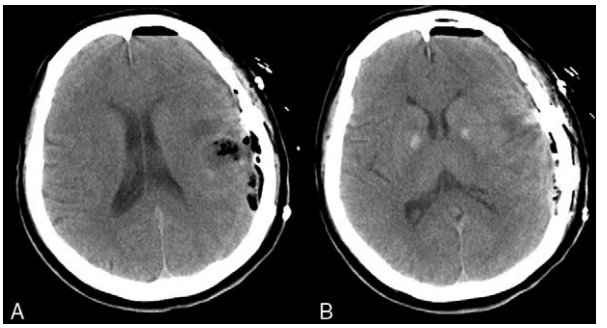

FIGURE 2. A-B, The postoperative CT scan of the patient. CT, computed tomography.

edema and invasion, whereas some carcinomas do not.[5] According to Purva Gopal et al, CPC is distinguished histologically from CPP based on increased nuclear to cytoplasmic ratios, increased mitotic figures, nuclear pleomorphism, the presence of necrosis, and distortion or blurring of the papillary structure of CPP by sheets of cells in the malignant counterpart.[1] A few intracranial metastatic carcinomas were misdiagnosed as primary CPC. Therefore, the importance of differentiating primary CPC from metastatic tumors is emphasized.[6] In this case, the CPC should be differentiated from astrocytoma, because of the similar neuroradiologic features. The astrocytoma is believed to arise from neurepithelium and the GFAP is stained positive.[3] In this case, the GFAP stained, however, is negative of the tumor.

The MRI characteristics of CPC are nonspecific, but is valuable in the diagnosis.[5] It is reported that CPCs are frequently much less homogenous because of cyst formation, hemorrhage, and with invasion of adjacent brain parenchyma.[7]

Immunohistochemical study is useful in the diagnosis of CPC. It was reported that positive immunoreactions ranges in CPP or CPC are 83% to 100% for cytokeratin, 40 % to 94% for S-100 protein, and 16% to 88% for vimentin.[8] Glial fibrillary acidic protein (GFAP) is often negative.[5,9] Positivity for S-100 and transthyretin, however, is typically less than that seen in CPP.[5]

Because of the local recurrence and metastasis, the prognosis of CPC are extremely poor.[1] In the management of CPC, gross total removal is the surgical goal, and in some cases of recurrence, additional surgical resections should be performed.[1,5] The early use of radiotherapy may extend the survival. Unfortunately, radiation is generally avoided in the pediatric population because of severe long-term sequelae.[1,5] Adjuvant chemotherapy after resection

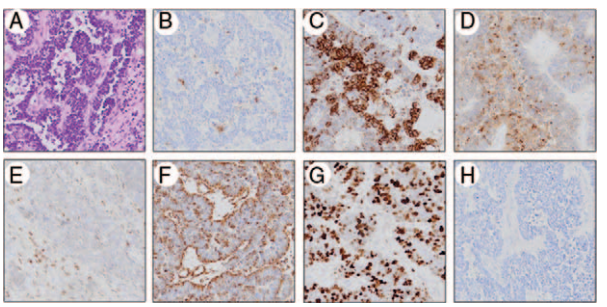

FIGURE 3. Photomicrographs of choroid plexus carcinoma. A, The H&E stain shows poorly structured papillary pattern and high cellularity, nuclear atypia. B-G, Separately, immunostaining with S-100, cytokine, EMA, EGFR, Vimentin, and Ki-67. Positive staining in tumor cells. H, Immunostaining with GFAP. Negative staining in tumor cells. All the images with an original magnification of × 100 are shown. EGFR, epithelial growth factor receptor; EMA, epithelial membrane antigen; GFAP, glial fibrillary acidic protein; H&E, hematoxylin and eosin.

remains controversial.[1,10] Some studies demonstrated that chemotherapy contributes to long-term survival, whereas others have not shown any benefit.[1] Owing to their rarity, there is currently no established protocol for the treatment of CPC.[5] We need more cases and multiinstitution experiences to establish the standard therapeutic measures.

In conclusion, CPCs are extremely rare in adults, and rarely occurred in the external ventricle. Gross total resection is needed to reduce the recurrence and prolong the survival time. The current accepted treatment for CPC is gross total resection with adjuvant therapy.

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An Unusual Deadly Craniofacial Trauma Case Due To Hot Liquid Plastic Infusion

To the Editor: Although various unusual foreign bodies have been reported, penetrating injury of the craniofacial structures caused by hot liquid plastic infusion has not been reported in literature.[1–5] We report how unusual craniofacial trauma case and the affected site and extension of the trauma could be so different from the expected damage depending on the type of injury.

A 45-year-old male presented with blunt head trauma and penetrating plastic material injury in the upper half of his face due to an industrial accident. Physical examination showed unstable vital signs and evidence of central neurologic injury. During the facial examination, penetrating injury that was caused by plastic material was observed. The injury included the left maxillary area, nasal field, and bilateral ocular structures. Computerized tomography (CT) scans demonstrated skull base, anterior wall of the left maxilla, nasal and ethmoid bone fractures, and subdural hematoma were observed, but extension of the foreign material could not be evaluated exactly (Fig. 1).

The patient was taken to surgery and underwent the transcranial hematoma drainage. During the hematoma drainage, extensive cortical brain contusion was observed. We participated in the operation as a plastic surgery team to extract the foreign material and reconstruct the facial structures. With dissection among the bony and soft tissues, we tried to mobilize the foreign material, but could not extract the material from the nasal and maxillary area.

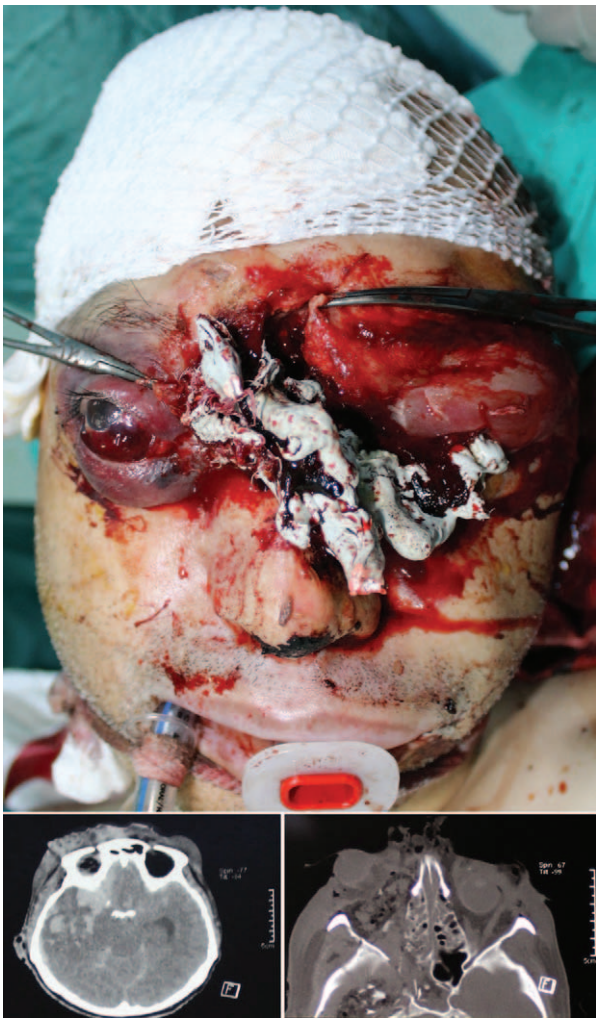

FIGURE 1. The first appearance of the patient when brought to the emergency service by ambulance. He presented with blunt head trauma and penetrating plastic material injury in the upper half of his face due to the industrial accident (above). Computerized tomography scans of the patient (below).

This case showed us that high-temperature liquid plastic spread to the retrobulbar area and solidified in this field. High-temperature property of the liquid plastic damaged the globe and extraocular tissues, and penetrated the brain tissue through optic canal and orbital fissures. And it also caused thermal damage in brain tissue. The anchor effect of the solid plastic also prevents the extraction of the solid plastic from the orbital area. After invasion among the facial fractures, solidifying of the liquid plastic caused the anchor effect. This anchor effect prevented the extraction of the foreign material.

In conclusion, penetrating injury with high-speed hot liquid plastic material can cause more extensive damage that we expect. Routine radiologic examination can be insufficient in this type of injuries. Fractures of the craniofacial structures can be detected easily with fine-cut CT scan, but the injury may extend beyond the detected fracture site. We could identify the extent of the trauma during the operation. In the present case, this atypical trauma resulted in the death of the patient. Total extraction of the solid plastic material was impossible in this patient, and this can be explained in the anchor effect of the plastic material.

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Surgical Treatment and Immediate Reconstruction of Keratocystic Odontogenic Tumor

To the Editor: The keratocystic odontogenic tumor (KCOT) is a benign but aggressive lesion with high recurrent rate. The diagnosis of KCOT depends on histopathologic findings. Histologically, KCOTs have parakeratinized stratified cell layers, thickened squamous epithelium, and formations of islands of odontogenic epithelium.[1] The appropriate treatment protocol remains controversial and is still a challenge for the oral and maxillofacial surgeons.[2] Various surgical options, such as decompression, marsupialization have been described.[3,4] Treatment should be based on many clinical and radiographic parameters, such as extent aggressiveness, size of the lesion, and age and performance status of the patient.

A 22-year old boy was referred to the Department of Maxillofacial Surgery of University Hospital "Città della Salute e della Scienza", Turin, Italy because of a 2-month history of intense mandibular pain. Oral examination revealed no sign of swelling. The patient's chief complaint was intensive pain located in the right side of the mandible. The lining mucosa was intact without sign of infection. Radiographic assessment revealed a loculated, large, radiolucent area extending from the body to the right mandibular ramus, up to the sigmoid notch, involving the unerupted wisdom tooth. A computed tomography (CT) scan was obtained and revealed a multiloculated fluid lesion with sclerotic margins (Fig. 1). An incisional biopsy was performed in the reported lesion under local anesthesia, and the specimen revealed a diagnosis of KCOT with parakeratinized stratified cell layers and thickened squamous epithelium. A surgical procedure was then planned: under general anesthesia an extraoral approach was performed to expose the right mandibular body and ramus. The inferior alveolar nerve was identified, gently dissected, and thus preserved (Fig. 1). Incision was taken till the periosteum at the inferior border of the mandible. The dissection was continued subperiosteally to expose the planned resection portion. A marginal ostectomy was then performed because of the excised lesion and to remove the wisdom tooth. The residual cavity was drilled and a local antibiotic irrigation was carried out.

After the resection, the reconstruction began with harvesting of a 4×2 cm corticocancellous bone graft from the anterior iliac crest. The iliac crest graft was simultaneously performed by a separate team. The harvested graft was then fixed by means of a titanium plate 1 mm with screws and a titanium locking plate 2.4 mm with screws was added for load bearing (Synthes, Oberdorf, Switzerland) (Fig. 1). The skin was then closed in layers. A submandibular suction drain was placed. No intraoperative complications were noted.

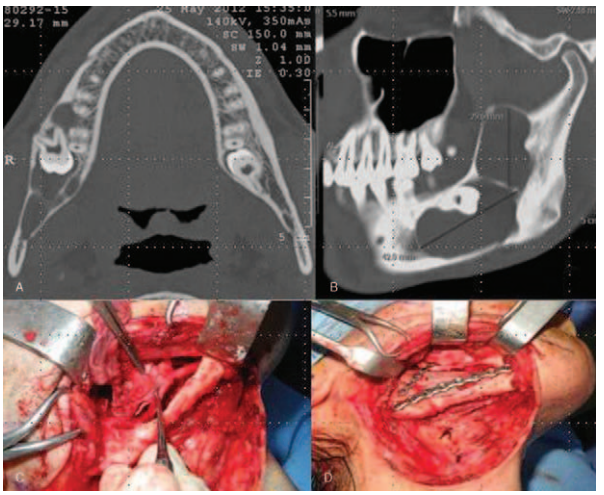

FIGURE 1. Computed tomography scan shows an extensive multiunilocular and hypodense image, axial (A) and sagittal (B) views. The lesion involves the body and the ramus of the right mandible with the presence of the unerupted wisdom tooth. Intraoperative view: the inferior alveolar nerve was identified, dissected, and preserved (C), the gap was reconstructed by iliac crest bone graft, fixed with titanium plates and screws (D).

The whole surgical specimen was sent for final histologic examination, which confirmed KCOT. The postoperative course was uneventful.

In this case, a cervicotomic access was performed not to achieve a better approach to the surgical field and identify the inferior alveolar nerve, reducing the healing time of bone graft. According to Pogrel et al,[5] we immediately performed the bone graft, with the aim of reducing the fracture risk and the risk of infection typical of an intraoral exposure.

At 2-years follow-up, the patient showed no signs or evidence of recurrence and there was no radiographic evidence of tumor recurrence. No sensory deficit of the lower lip or unaesthetic appearance of the scar was noted.

To conclude, considering the local aggressiveness of the lesion and the youth of the patient, the marginal resection with inferior alveolar nerve preservation and 1-phase reconstruction revealed to be a valid surgical option.

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Pathological Fracture of Zygomatic Arch Secondary to a Large Mandibular Ameloblastoma

To the Editor: Ameloblastoma is the second most common true neoplasm of odontogenic origin that accounts for 11% to 13% of all odontogenic tumors.[1] It is known to occur over a broad age range from 10 to 90 years. Almost 85% of cases are known to affect the mandible especially the molar ramus region.[2] The ramus of the mandible provides attachment to masseter muscle insertion, whereas the origin of masseter is from zygomatic arch and maxillary process of the zygomatic bone. This arrangement of the fibers tends to distract the zygomatic arch inferiorly and medially following a Trikala/podfracture because of its pull/action.[3] A 74 years old woman with clinical, radiological, and histopathological diagnosis of mandibular ameloblastoma was referred to our center for further management. History revealed 20 years old presentation with no medical intervention/consultation sought till date because of socio-economic reasons. A huge extra oral swelling was seen on the left side of the face below the zygomatic arch region up to the inferior border of the mandible not crossing the midline (Fig. 1a). The swelling was obliterating the buccal sulcus and limiting the mouth opening. The computed tomography scan was suggestive of a large well defined exophytic, expansile, lobulated lesion involving the body, coronoid process, and ramus of left mandible sparing the condyle measuring $7.4 \times 7.5 \times 10.0$ cm (AP × TR × CC) in size. Cortical expansion with thinning of cortices, extension up to condylar region and infratemporal fossa, loss of fat planes, displacement of parotid, submandibular gland, IJV, EJV, CCA, homogenously enhancing lymph nodes at level II and III on both sides were some usual salient features noticed. An unusual pathological fracture of the zygomatic arch was evident at the zygomatico temporal suture region (Fig. 1b). A wide block excision with continuity defect was done without reconstruction. Postoperative histopathology report was suggestive of Plexiform ameloblastoma with negative margins. No intervention was carried out for the pathological fracture of the zygomatic arch. Keeping this background in mind, the authors arrived at a conclusion of an unusual complication, ie, fracture of zygomatic arch in a large mandibular ameloblastoma who gave negative history of trauma in her entire life span. The pull of masseter muscle is known to cause displacement and communition along the axis parallel it its fibers.[4] Hence, it is hypothesized that the excessive growth of the lesion might have caused overstretching of the masseter muscle which may have led to preoperative pathological fracture of the zygomatic arch. In massive pathologies of the mandible one should not only concentrate primarily on the mandible for pathological fractures, but also on the adjacent bones from where the muscles responsible for the movement of mandible are arising or inserting. To the best of the knowledge of the authors, the present case is the first case of this rarest complication in the existing literature.

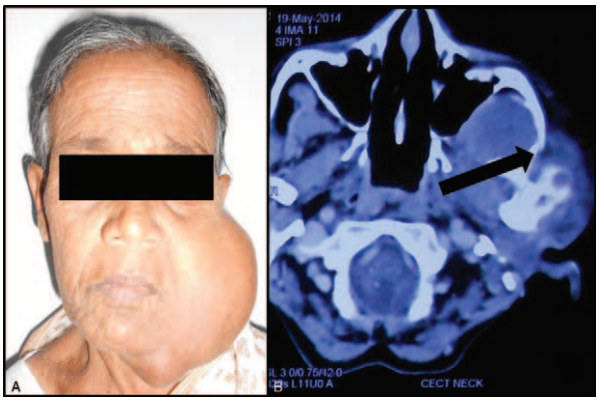

FIGURE 1. A, Clinical presentation of the ameloblastoma. B, Pathological fracture of the zygomatic arch on axial section of the CT scan.

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A Long-Term Follow-Up of Rare and Aggressive Odontogenic Tumor in a 5-Year-Old Child

To the Editor: Ameloblastic fibro-odontoma (AFO) is a rare odontogenic tumor located within the oral cavity, frequently leading to associated functional impairment and esthetic involvement once it assumes large proportions.[1,2] Early detection through imaging examinations may avoid misdiagnosis of this condition with others that often lead to marked intraoral swelling. Therefore, the aim of the present report was to describe the diagnosis, treatment, and long-term follow-up of a large and locally aggressive AFO occurring in an uncommon location, in a 5-year-old boy, which was initially misdiagnosed as an odontogenic infection.

A 5-year-old boy was referred by a general practitioner to the School of Dentistry of the Federal University of Ceará due to a large slow-growing swelling in the left posterior upper region of the maxilla, which led to dysphagia, nasal obstruction, and weight loss. The lesion was noticed to 3 years of age. An increase in volume followed by nasal obstruction led to the diagnosis of a "dental infection." The patient was treated with oral amoxicillin during 21 days followed by cefalexicin for 14 days, not showing clinical improvements. The clinical examination showed a firm large mass in the left maxilla covered by a normal mucosa without signs of infection was observed, and deciduous teeth present within this anatomical site with small caries lesions (Fig. 1). The panoramic radiograph showed a well-circumscribed unilocular radiolucency with a large amount of radiopaque bodies occupying entirely the left maxilla. In addition, the lesion was in proximity with canine and premolar tooth buds. The computed tomography scan showed a huge predominantly hyperdense lesion, which extended to the midline and displaced the nasal cavity (Fig. 1).

The lesion was easily and entirely removed from the surgical site under general, and the specimen was sent for histopathological analysis. Microscopically, the excised tissue expressed island and cords of odontogenic epithelum showing peripheral palisading, dentinoid material, sheets of enamel matrix, mineralized component, cell-rich ectomesenchyme resembling the dental papilla, and tooth-like structures (Fig. 1). Thus, the final diagnosis was AFO. A 9-year follow-up showed no recurrence of the lesion and a normal bone healing with complete eruption of the first and second left premolars (Fig. 1).

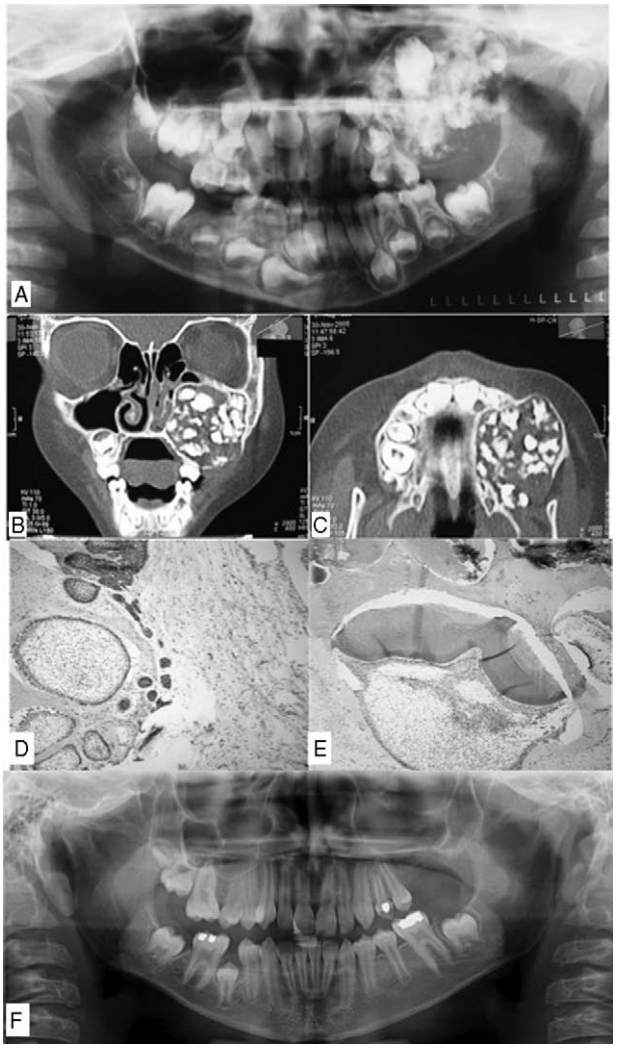

FIGURE 1. (A–C) Panoramic radiographic and computed tomographic examination revealing a large hyperdense lesion, with a significant amount of radiopaque bodies, containing unerupted teeth and displacing the nasal cavity; (D, E) Photomicrography (HE, ×40) showing island and cords of odontogenic epithelum with peripheral palisading, dentinoid material, enamel matrix, mineralized component, cell-rich ectomesenchyme. (F) Panoramic radiograph showing a 9-year postoperative follow-up with normal bone healing and complete eruption of the upper left canine and premolars.

AFO accounts for 1–3% of a broad spectrum of odontogenic tumors commonly observed in children and adolescents,[1] consists of a rare neoplasm with histologic features of an ameloblastic fibroma associated with the presence of enamel and dentin,[3,4] and occurs mainly in the posterior mandible of young individuals up to the second decade of life.[5] Usually, it occurs as an asymptomatic slow growing swelling, which can be early recognized by the presence of missing teeth. Radiographically, this neoplasm shows a mixed appearance containing radiopaque tooth-like structures expressing a broad level of mineralization, with the presence of unerupted teeth.[4–8] In the present case report, this tumor occurred in the posterior maxilla, which is considered a rare event. Once a patient presents with an extensive tumor as observed, differential diagnosis should be based mainly on imaging features, and must

include lesions such as complex odontoma, adenomatoid odontogenic tumor, calcifying cystic odontogenic tumor, and calcifying epithelial odontogenic tumor.[7]

Due to the benign characteristic and nonaggressive biologic behavior of AFO, a conservative approach should be considered. Nevertheless, an en-bloc resection with disease-free margins has been recommended to minimize postoperative recurrence.[8] Presently, a nonaggressive treatment was performed, with total excision of the lesion without bone resection and maintenance of the surrounding unerupted left premolar and canine tooth buds. According to many authors,[9,10] the preservation of adjacent erupted and unerupted teeth is controversial, because lesion recurrence remains a possibility. However, some authors have reported no recurrence after lesion excision without extraction of the surrounding teeth.[1,10]

Regardless of the rare occurrence of these lesions in the posterior maxilla, complete surgical excision with preservation of the adjacent teeth and tooth buds showed a successful clinical and radiographic outcome after a 9-year follow-up.

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Antibacterial Effects of Platelet-Rich Plasma in Promoting Facial Scars Healing in Combination With Adipose-Derived Stromal Vascular Fraction Cells

To the Editor: We have read with great interest the paper of Gentile et al (2014)[1] published in this journal, concerning the role of platelet-rich plasma (PRP) in promoting facial scars healing. The authors highlighted the role of PRP and stromal vascular fraction in promoting facial scars healing. We were impressed by the project of the investigation. The face scars can influence human health, and also affect human psychology and emotion. Therefore, it is very important to investigate the methods to improve facial appearance and correct the psychological barrier and stabilize the mood of patients. The authors, however, did not clarify the antibacterial effects on facial scars healing. Recently, researchers showed the antibacterial effects of PRP in ulcers.[2] Therefore, we write the manuscript to comment on the antibacterial effects and molecular mechanism of PRP in promoting facial scar healing with the authors and other readers.

Platelet-rich plasma, as a new biological material, released a lot more growth factors and cytokines while activation to induce wound healing and bone regeneration. Meanwhile, PRP is activated by thrombin and calcium or by endogenous platelet activation, which converted soluble fibrin into insoluble fibrin, to form platelet-rich gel (PRG) to maintain an optimal microenvironment for the growth of adipocytes, and also protect the wound via preventing bacterial invasion into wound area. These substances released a lot more growth factors and cytokines while activated.[3] After activation, the platelet degranulation released growth factors, antimicrobial peptides, and other active substances to display antimicrobial properties. The growth factors induced the early angiogenesis and enhanced the effect of ulcer healing.[4,5] Platelets also released large amounts of active substances, such as fibronectin, osteonectin, catecholamines, serotonin, proaccelerin, von Willebrand factor, and other substances to provide the optimum microenvironment to display its biological role.[6] Platelet-rich plasma contains substantial amount of leukocytes in the process of preparation, including neutrophils and lymphocytes; the former can kill bacteria directly and defend against pathogens to prevent infections and destroy microbes, and the latter inhibited bacterial growth by an antigen-specific immune response.[7] It showed that the leukocytes were relevant to the antimicrobial role and tissue remodeling. Macrophage is

conducive to remove of debris to initiate tissue repair.[8] Therefore, the white blood cells in PRP displayed a key role in wound healing through immune-modulating effects.

Facial scar repair is a complex series of events. The main process involves re-epithelialization, formation of granulation tissue, and contraction of underlying connective tissues. This effect is first regulated by neutrophils and macrophages, and then mast cells emigrated from nearby tissues and the blood circulation. Macrophages promote the recruitment and proliferation of fibroblasts, and express of the growth factors to stimulate angiogenesis in wound healing.[9] The PRP resist the infection via following 3 plasma proteins, including vitamin D binding protein, a1-macroglobulin, and a2-microglobulin. The activating macrophages induced the expression of tumor necrosis factor a, vascular endothelial growth factor, interleukin-1β, interleukin-6, and so forth through toll-like receptor-4 to present antibacterial effects.[10]

Platelet-rich plasma is an effective topical antimicrobial biomaterial, and its antimicrobial performance is enhanced by the antibiotics in plasma, whereas compared with PRP, the antibacterial effect of PRG is serious. PRG is formed through PRP extra added thrombin and calcium, therefore, the PRG containing extra thrombin and calcium. Researcher showed that the antibacterial effects against *Staphylococcus aureus* mediated by platelet activation in diabetic dermal ulcer, and does not present obvious antibacterial effects on *Escherichia coli* or *Pseudomonas aeruginosa*. Combination PRG with antibiotics presented synergistic antibacterial effects. Platelet-rich gel can inhibit the growth of *Streptococcus oralis*, *Enterococcus faecalis*, *Streptococcus agalactiae*, and *S. aureus* and enjoyed a powerful activity against methicillin-sensitive *S. aureus* and inhibited the growth of methicillin-resistant *E. coli and S. aureus*. It, however, cannot inhibit actively the growth of *Klebsiella pneumonia*, *E. faecalis*, and *P. aeruginosa*. Moreover, it induces the growth of *P. aeruginosa* in vitro.[11] Platelet-rich gel inhibited the growth of methicillin-sensitive *S. aureus* with a mean zone diameter of 19.8 mm. *Escherichia coli, S. aureus*, and *P. aeruginosa* are the common bacterial species resident in chronic ulcers and wound infections. In particular, the ulcers infected with bacteria displayed a significant larger and serious than those not infected with bacteria. The presence of *P. aeruginosa* in venous leg ulcers induced enlargement of the ulcer and delayed healing.[11] Numerous investigators have isolated platelet-specific antimicrobial molecules from human and animal platelets. Yeaman[12] isolated and purified rabbit platelet antimicrobial polypeptides in vitro from thrombin to induce biomaterials released.

The antibacterial mechanisms of PRP are not clear. It is mainly associated with the antimicrobial peptides secreted by platelets attributed to host multiple functional antimicrobial defenses. Activated platelets could secrete platelet microbicidal proteins, which contain a series of biomaterials presenting antibacterial activity. Tang isolated and characterized 7 antimicrobial peptides from human platelets after thrombin activation to release cytokines. There are 7 antimicrobial peptides secreted while thrombin activation PRP. They included connective tissue-activating peptide-3, platelet factor-4, fibrinopeptide A, fibrinopeptide B and so on. The author studied the antimicrobial activities of these peptides against *E. coli, S. aureus*, *Candida albicans,* and *Cryptococcus neoformans,* and found that the peptides play a powerful role against bacteria, and the antimicrobial effects were dose dependent. These findings suggested a direct relationship between the antimicrobial effect and platelet antimicrobial peptides.[13] Platelet microbicidal proteins in contact with the bacterial membrane change the membrane permeability, enter into the cell, and inhibit the synthesis of a series of big molecules and other functions.[14] The study proved that the antibacterial effect of PRP derived from diabetic dermal ulcers in vitro against *S. aureus* was due to the activation of platelets to release platelet microbicidal proteins.[2]

The antibacterial mechanism of PRP is also associated with platelets displaying antimicrobial host defense. These include navigation to the inflammatory chemoattractant, expression of immunoglobulin-G Fc receptors and for C3a/C5a complement receptors fragments, and antimicrobial oxygen metabolites, including superoxide, hydrogen peroxide, and hydroxyl free radicals. The platelets interact directly with microorganisms, contribute to the clearance of bacteria in bloodstream, and actively participate in antibody-dependent cytotoxicity inhibiting microbial pathogens.[12] The platelets of PRP can decrease the pathogen in bloodstream by inhibiting the growth of microorganisms.[15] Platelet-rich plasma can be used to cover wounds in open fractures because its gelatinous mass forms a barrier against microbes. It demonstrated effective inhibition of *E. coli, Bacterium megaterium, P. aeruginosa, E. faecalis, and Proteus mirabilis*. A possible molecular mechanism of PRP displays a potential antimicrobial activity to inhibit *E. coli* and *P. mirabilis* and associated with the platelets to release the antimicrobial peptide, such as human beta-defensin 2.[16]

A novel molecular mechanism: Platelet-rich plasma can enhance the mRNA level of the cytokines IL-1β and TGF-β1 to induce an increased expression at mRNA and protein levels of myogenic regulatory factors of MyoD1, Myf5, Pax7, and the muscular isoform of IGF-1. Platelet-rich plasma also regulated the expression of miR-133a and target serum response factor to enhance the phosphorylation of aB-cristallin, with an improvement in apoptotic factors of caspase-3 and NF-kB-p65 leading to cell survival. Moreover, it can modulate the molecular mediators of the inflammatory and myogenic pathways to control the pathways modulated by heat shock proteins and myomiRNAs to improve tissue regeneration effectively.[17] The detailed mechanisms of PRP need to be further researched.

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A Meta-Analysis of Association Between Pesticides Exposure and Glioma Risk in Adults

To the Editor: Glioma is one of the most common cancers of brain, no matter in the developed or in the developing countries. Although glioma is one of the most lethal diseases, its cause is poorly understood. Pesticides exposure is associated with an increased risk of several cancers including leukemia, prostate cancer, breast cancer, and even children and young adults brain tumors. Several studies have been conducted to assess the potential association between pesticides exposure and adult glioma risk over the last several decades, but the results are inconsistent. Therefore, we performed a meta-analysis of all of the available studies to clarify the effect of pesticide exposure on glioma risk to assess the evidence currently available.

We conducted an extensive literature search for relevant studies on pesticide exposure and glioma risk in PubMed, EMBASE, and the Cochrane Library from inception to June 1, 2014. Reference lists of the relevant articles were also reviewed to identify other potential studies. The following criteria were used to select the studies:

- a case-control study;
- research the relationship between pesticides exposure and glioma risk;
- medically confirmed of glioma;
- odds ratio (OR) with its 95% confidence interval (95% CI) were reported, or could be calculated; and
- including adults only.

If the same populations were reported twice, only the largest sample size or the most recent publication was included. The following data were extracted from all of the included studies: first author's last name, publication year; study country and period; cases age; number of cases and controls; diagnostic criteria; control type; research instrument; and pesticides exposure variables such as ORs with its 95% CIs. The quality of all of the included studies was assessed based on the Newcastle-Ottawa Scale by 2 authors independently. Scores ranged from 0 to 9 stars, and the high-quality study was defined as a study with $\geq$7 stars. All of the studies were evaluated by 2 authors independently. Disagreements were resolved through discussion and consensus.

Owing to glioma is a rare disease, relative risk (RR) was used as the common measure of association across studies, OR was deemed equivalent to RR. We conducted the meta-analysis using STATA 12.0 (Stata Corporation, College Station, TX). Generic inverse variance data were combined to obtain a summary of RR and 95% CI. The random-effects model, a conservative method to estimate the pooled effect, was used in our meta-analysis. Statistical heterogeneity between studies was evaluated by Cochran's Q statistic, and measured with the I^2 statistic. Heterogeneity was considered significant, when $I^2 > 50\%$ and $P < 0.1$. In addition, subgroup analyses were performed based on study design: control type (population-based and hospital-based); research instrument (interview, mail, and phone); sex (men and women). To investigate the influence of single studies on the overall risk estimate, we also conducted a sensitivity analysis by omitting each study in each turn. Begg funnel plots and Egger regression test were used to assess the potential publication bias. All of the analyses were 2 sided with a $P \leq 0.05$ considered statistically significant.

Finally, 11 eligible articles including 6393 cases and 15,258 controls were included in our meta-analysis.[1–11] Of the studies, 7 were population-based, 3 were hospital-based, and 1 was reported for both hospital- and population-based. Our study did not detect any significant association between pesticides exposure and the risk of glioma, for overall pesticides exposure RR = 1.15 (95% CI: 0.96–1.37; $I^2 = 64.7\%$, $P_{\text{heterogeneity}} = 0.002$); for insecticides exposure RR = 0.96 (95% CI: 0.76–1.22; $I^2 = 60.4\%$, $P_{\text{heterogeneity}} = 0.039$); for herbicides exposure RR = 1.07 (95% CI: 0.87–1.32; $I^2 = 41.9\%$, $P_{\text{heterogeneity}} = 0.129$). In subgroup analyses by control type, research instrument, and sex, the significant association was also not shown between any pesticides exposure and glioma risk. The Begg funnel plot and Egger regression test were used to assess potential publication bias in the included studies. Although limited by the small number of individual studies, we did not detect any bias ($P = 0.436$ and $P = 0.132$) affecting the pooled risk estimates RR.

The utilization of pesticides has contributed to unprecedented growth in agricultural production and productivity; however, the

negative effect of environmental and health risks associated with pesticides exposure has increased simultaneously.[12] Previous reviews have concluded that there was a weak-positive association between brain cancers and farming.[13] In 2013, a meta-analysis was conducted that supports an association between parental occupational exposure to pesticides and brain tumors in children and young adults[14]; however, our study indicated that pesticides exposure and the risk of adult glioma have no association. The reasons for the phenomenon are still controversial. By the current study, no significant correlation between pesticides exposure and glioma risk was found, but we should avoid prolonged exposure to pesticides in our daily life.

In summary, our study is the first comprehensive meta-analysis to assess the associations between pesticides exposure and adult glioma risk. The current study did not support an association between both. Owing to the heterogeneity and the limited number of studies, the results of our study were not robust. Therefore, further research in well designed cohort or intervention studies are warranted to validate this finding from this meta-analysis. Although no significant correlation was detected, we still should stay away from pesticides.

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A Clinical Analysis of Impacted Teeth in Edentulous People

To the Editor: Prophylactic removal of impacted teeth free of any pathology still remains controversial today. Third molar removals are most frequently implemented at 20 to 23 years of age;[1] however, 17.3% of patients requiring 3rd molar extraction are over 40-years old, and 19.5% of the patients over 40 years are older than 60 years.[2] The elderly patient requiring surgical removal of an impacted 3rd molar presents a major dental and medical challenge nowadays. Not only are the aging patients generally associated with systemic diseases, but also they have more horizontally positioned 3rd molars fully covered by bone,[3] which increases risks and difficulties of their extraction. Meanwhile, increased age leads to more clinical problems involving an increased incidence of localized osteitis and delayed wound healing after surgery. The clinical symptoms of the edentulous patients are atypical, frequently confused with denture stomatitis caused by poor denturehygiene, continual and nighttime wearing of removable dentures, accumulation of denture plaque, bacterial and yeast contamination of denture surface, and poor-fitting dentures increasing mucosal trauma, leading to presence of inflammation or swelling of mucosal tissues.[4] The purpose of our study was to analyze the characters of impacted teeth in the edentulous people, and to investigate the etiology, diagnosis, and cautions in the treatment of impacted teeth.

Thirty seven patients (45 impacted teeth) were referred to our department from 2010 to 2014, including 17 male and 20 female who were 58- to 82-years old. Among them 51.4% were with hypertension, 32.4% were with heart diseases, 13.5% were with diabetes mellitus, and 24.3% were with osteoporosis. Twenty two of them were edentulous and 14 were partially edentulous, all with complaint of painful and swollen gums, except for 1 complaining pain and swelling in parotideomasseteric region. After radiologic examination and evaluation of systemic condition, all the patients underwent extraction of impacted teeth with the utilization high-speed handpiece with a fissure bur or piezosurgery. We recorded the position and impacted type of each tooth, as well as associated lesions.

Of all the 45 impacted teeth, mandibular 3rd molars accounted for 80% (36/45), including 16 invertedly impacted, 6 horizontally impacted, and 1 vertically impacted. Maxillary 3rd molars accounted for 13.3% (6/45), with 2 distoangularly impacted, 2 mesioangularly impacted, and 2 vertically impacted. Supernumerary teeth accounted for 6.7% (3/45), consisting of 1 mesiodens and 2 embedded in horizontal plate of palatine bone. Dentigerous cysts found in 7 and 5 teeth were accompanied by local osteomyelitis. Duration of pain was 3–5 days, and wound healing was uneventful. No patients developed severe complications.

In the early days, dentists believed that if 3rd molars did not give people problems by age 40, they probably would never develop symptoms.[2] But from our view, such a statement is biased. We

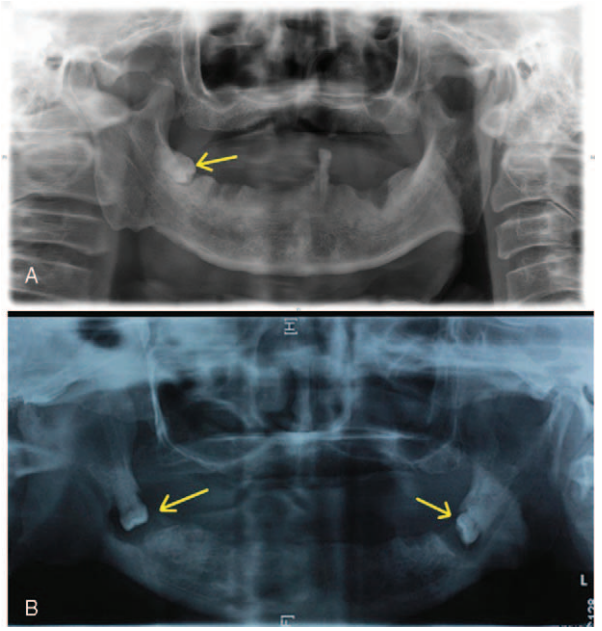

FIGURE 1. (A) Horizontally impacted right mandibular 3rd molar. (B) Impacted 3rd molars associated with dentigerous cysts.

consider that 2 reasons account for fully impacted teeth appearing clinical symptoms in the elderly. For one thing, fully impacted teeth may turn into partially impacted due to alveolar bone loss and become susceptible to external stimulus. Resorption was found to be significantly higher in the molar region than in the anterior region,[5] which may explain why most symptomatic impacted teeth of the aging people were mandibular 3rd molars according to our study (Fig. 1A). The other reason might be pessimal stimulation from removable dentures further aggravating pathologic changes of impacted teeth (Fig. 1B), which may predispose patients to the onset of inflammatory or reactive hyperplasia owing to bacterial plaque adhering to the base of the denture, inadequate design of the prosthesis, continual and nighttime wearing of removable dentures, and poor denture hygiene.[4]

The asymptomatic impacted teeth can have pathological changes, and prophylactic removal of impacted teeth has had major success in reducing pericoronitis, dentigerous cyst formation, and adjacent teeth impairments.[2] According to our findings, aging people have more horizontally and invertedly positioned 3rd molars covered by bone, which needs more bone removal and adds more difficulty to the surgery due to increased bone density[6] and decreased tolerance in the elderly. It was reported that the total amount of alveolar bone loss would be at least 1.9 mm by the age of 80.[7] So in our opinion, impacted teeth at depths less than 2 mm should be extracted preventively. However, it still needs a long-term study to decide the indications of removal of asymptomatic impacted teeth.

The symptoms of pericoronitis in the elderly are not typical, and the patient complaining pain in parotideomasseteric region was even diagnosed parotid tumor by other doctor. Also it is easily misdiagnosed as denture stomatitis because of red and swollen gums. Consequently, we suggest panoramic radiographic examination when denture wearers, especially edentulous patients present with pain and swelling in gums or parotideomasseteric region to check if it is because of impacted teeth.

Piezosurgery and high-speed handpiece with a fissure bur permits precise and safe cutting of mineralized tissue, low-bleeding tendency, less vibration for patients, optimal view, and excellent wound healing with fewer complications postoperatively.[8]

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Conventional Panoramic Radiograph Cannot Identify the Bifid Mandibular Canal

To the Editor: Bifid mandibular canal (BMC) is an anatomical variation of the mandibular canal with 2 offshoots. Previous documents have reported the incidence of BMC as ranging from 0.08 to 65.0%.[1] Since each canal of the BMC might contain a mandibular nerve and a vessel bundle, the presence of the extra mandibular canal can lead to inadequate anesthesia or paresthesia from surgical manipulations. Although the key to identify the BMC is to employ three-dimensional (3D) imaging such as cone beam computed tomography (CBCT), many dentists unfortunately utilize only panoramic images for second or third molar extraction. The authors identified the case of BMC reported here on CBCT images, but the BMC was not apparent in the panoramic view. In this letter, we attempt to delineate the case and warn of potential danger with using only the panoramic view for identification of the mandibular canal in surgical procedures.

CLINICAL REPORT

A 39-year-old man visited for intermittent pain on the right mandibular retromolar area. Intraoral examination of the patient revealed pericoronitis around the partially erupted right mandibular

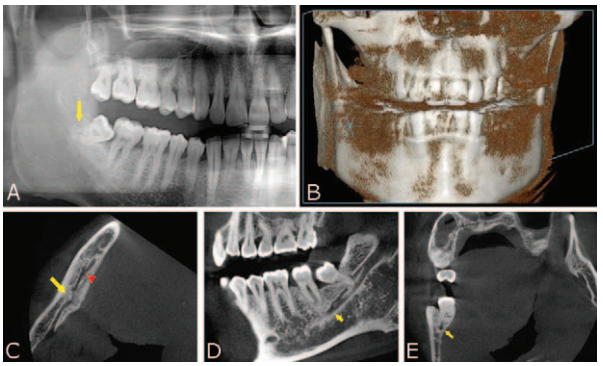

FIGURE 1. The panoramic (A) and CBCT (B–E) radiographic images of the patient. A, The panoramic radiograph of the patient. The arrow indicates the apex of the involved tooth (mesially tilted right third mandibular molar). B, The 3D reconstructive image of the cone beam computed tomography (CBCT). C–E, Sectional CBCT image of the involved teeth and bifid mandibular canal. In C (axial section), yellow arrow indicates the root apex of the involved tooth and red arrowhead indicates the bifurcation of the mandibular canal. In D (sagittal section), the arrow indicates the bifurcation of the mandibular canal. In E (coronal section), the arrow indicates section of the partially separated shape of the mandibular canal.

third molar. Panoramic radiologic imaging showed the tooth in a mesially tilted position and decay was observed on the distal side of an adjacent second molar. Extraction of the right mandibular third molar followed by operative treatment on the second molar was planned.

In the panoramic view, the canal was deemed to be single with a clear upper border (Fig. 1A). A vague positional relationship between the radicular apex of the involved tooth and the mandibular canal was observed, and an aberrant shape of the apex was suspected. Thus, additional topography information was obtained with CBCT equipment (POINT 3D COMBI 400s, Pointnix, South Korea) for safe surgical extraction (Fig. 1B).

On a cross sectioned CBCT image at the apical tip of the involved tooth, the mandibular canal was located between the buccal and lingual roots (Fig. 1C). The mandibular canal was observed to bifurcate into 2 canals in the ramus area. On a sagittal sectioned image through the buccal root position of the involved tooth, the mandibular canal bifurcated into 2 canals at the interdental position of the involved third molar and the adjacent second molar (Fig. 1D). The upper offshoot of the mandibular canal appeared to be juxtaposed to the apex of the involved third molar. On a coronal sectioned image at the apical tip of the buccal root of the involved third molar, partially separated canals were observed (Fig. 1F).

The patient was informed that the mandibular neurovascular bundle was at risk during surgical extraction and the involved third molar was surgically extracted. Postoperative complications associated with the mandibular nerve did not occur.

DISCUSSION

The incidence of BMC reported in previous studies varies. The incidence of BMC was reported as very rare (0.08–0.09%) in a study using conventional panoramic radiograph.[2] However, studies using CBCT have reported a high incidence rate (15.6–64.8%) for BMC. Of course, bifid mandibular canals have been observed on not only CBCT but also on conventional panoramic radiograph in some patients.[1] Based on the significant difference between the incidence of BMC using CBCT and conventional panoramic radiograph, a deceptive appearance of the mandibular canal lacking the bifurcation of BMC is presumed to be very common on conventional panoramic radiograph.

In the present patient, although a single mandibular canal was expected based on the results of the panoramic view, distinct bifid canals were identified in subsequent CBCT images. The conventional panoramic view is not suitable for determining the exact position and dimensions of the mandibular canal or for identifying the presence of an accessory offshoot of the mandibular canal, due to ghost imagery resulting from overlap with an opposing semimandible and imperfect information regarding the bucco-lingual position.[1–3]

Unfortunately, conventional panoramic tomography is broadly employed to determine the topographic relationship between the radicular apex and the mandibular canal. Many surgeons think that a clear upper border in the mandibular canal guarantees the absence of additional offshoots from the canal. However, the incidence of BMC is higher than previously believed.[2,3] The BMC contains separate neurovascular bundles and impingement of its accessory offshoot can lead to various complications such as paresthesia or bleeding during the surgical intervention.[4]

The present patient demonstrates that clear findings indicating the lack of bifurcation on panoramic radiograph cannot guarantee the absence of BMC. The patient calls attention to the necessity of routine application of CBCT for safe surgical intervention during third molar extraction. Taking diagnostic CBCT image would be also helpful for following treatment after sequelae such as sensory disorder after the extraction of mandibular molars. Furthermore, ambiguous topographic relationship of mandibular third molar root with not only BMC but also main mandibular canal has been commonly observed in the panoramic view. We suggest that the clinician obtain 3D information by means of CBCT for surgical manipulations including extraction and dental implant placement.

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Impure Blow-in Orbital Fracture With Severe Proptosis

To the Editor: A reverse blow-out fracture has been identified as a "blow-in" fracture. It is a rare injury, in which the fractured floor/wall of the orbit is elevated into the orbit.[1] Impure blow-in fractures are more common, where in the orbital rim is also disrupted along

with the orbital wall which is displaced inwards into the orbit thereby reducing the orbital volume and resulting in proptosis of the globe.

CLINICAL REPORT

A young man with alleged history of assault with hammer to his right side of face presented to us with proptosis of the globe, orbital rim, and lateral orbital wall blow-in fracture, along with fracture zygoma and bilateral Le fort II fractures as seen in Figure 1. He had vision of finger counting and diplopia in his right eye, with no extraocular movements. He was treated with reposition of the globe, orbital rim fixation, zygoma fixation, and fixation of the Le fort II fractures with titanium plate and screws and 24 g stainless steel wires which were used to stabilize minor fracture fragments. Cornea was protected by lid opposing sutures as shown in Figure 1. Orbital floor was not repaired in this setting due to fear of increasing the orbital pressures resulting in raised intraocular tension. Patient received postoperative systemic steroids and intravenous and topical antibiotics. His intraocular pressures, visual acuity, fundoscopic monitoring was performed postoperatively at routine intervals. He made remarkable recovery with vision of 6/16 in the right eye. He was offered orbital floor reconstruction at 3 months interval but the patient refused any further treatment.

DISCUSSION

Pure blow-in fractures are characterized by the presence of an isolated, inwardly displaced fracture of the orbital roof, floor, or walls, whereas the circumferential bony rim remains intact.[2] Dingman and Nativig[3] reported the first patient of pure blow-in orbital fractures. Pure blow-in fractures are rare entity. Blow-in orbital injuries were classified as impure fractures when the orbital rim itself was disrupted. In all patients, early decompression of the orbit and open reduction of fracture fixation was necessary. In regard to mechanisms of trauma, several mechanisms have been suggested. First, a blunt trauma to adjacent facial bones, for example, the frontal wall of the maxillary sinus, may cause a sudden increase in pressure in the maxillary sinus. As a consequence, fragments of the orbital floor are forced into the orbit.[4] Second, blow-in fractures are caused by a buckling of the orbital floor secondary to severe compression of the orbital rim. Third mechanism could be the impact of a force on the superolateral part of the zygoma.[5] This is very similar to our patient, as it was due to assault by a hammer resulting in an inward force onto the orbital rim, lateral orbital wall, and zygoma being displaced medially, with resultant outward displacement of the globe. The impact was so strong that despite there being a fracture of the orbital floor the globe was forced out of the orbit. This is a very rare presentation of orbital blow-in fracture associated with orbital floor fracture. This paradoxical phenomenon makes this case unique as one would have expected orbital bow out to occur with orbital floor fractures, which naturally decompresses the orbit and protects the globe. The authors think the most likely explanation to this paradox is the severity of impact with marked medial displacement of the zygoma, forcing the globe to pop out despite their being an orbital floor fracture.

Prompt early recognition and treatment of these injuries is of utmost importance as failing to do so will result in loss of vision and eventually the entire globe. It is very crucial to closely monitor these patients in the early postoperative period as there is an increasing risk of loss of vision. Also the danger of endophthalmitis and sympathetic ophthalmitis setting in and affecting the opposite normal eye would require an emergency enucleation. Postoperative topical and systemic antibiotics are crucial in prevent these threatening complications. Also the use of systemic steroids in the form of high-dose methylprednisolone for the first 3 days in the postoperative period and thereafter tapering it is important in reducing optic nerve edema and also edema of soft tissue envelope of the globe. Judicious monitoring of intraocular tension, visual acuity, fundoscopy in postoperative period is crucial and can only be reemphasized.

FIGURE 1. Patient with compound orbital blow on fracture with proptotic globe (top right). Three-dimensional computed tomography reconstruction showing impure orbital fracture with inward (medial) displacement of zygoma (top left). Open reduction and internal fixation with titanium plates and screws and stainless steel wires used for minor fragments (bottom right). Early postoperative period showing the frost sutures (bottom left).

CONCLUSIONS

Blow-in orbital fractures though a rare entity must be considered in patients of orbital proptosis resulting due to trauma. Prompt recognition, early orbital decompression, and fracture fixation form the main stay of treatment.

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OPEN

Comment on "S267P Mutation in FGFR2: First Report in a Patient With Crouzon Syndrome"

To the Editor: Ke et al[1] recently reported on a patient clinically diagnosed with Crouzon syndrome showing missense point mutation S267P in fibroblast growth factor receptor-2 (*FGFR2*). Ke and his colleagues analyzed the pathogenic gene (*FGFR2*) and found the new mutation, which is impactful for the Crouzon syndrome researchers. Ke et al found a heterozygous missense mutation with direct DNA (deoxyribo-nucleic acid) sequencing in exon 8 of *FGFR2* which resulted in the replacement of serine (TCC) by proline (CCC). They identified it as "a new missense mutation in *FGFR2* gene" that provides "further evidence for *FGFR2* mutations as a main cause of Crouzon syndrome."

According to HGMD (The Human Gene Mutation Database), Oldridge et al[2] reported this exact missense mutation in a patient with clinically diagnosed Crouzon syndrome in 1995, which should be considered as the first known discovery of S267P mutation in *FGFR2* in a patient with Crouzon Syndrome.

Based on the above argument, it might be more appropriate for the authors to change the title of their paper to "S267P Mutation in *FGFR2*: First Report from China of a Patient with Crouzon Syndrome." In addition, we summarize the previous reports of *FGFR2* mutations in Crouzon Syndrome (see Supplemental Digital Content, Table E1, http://links.lww. com/SCS/A165).

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Supplemental digital contents are available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.jcraniofacialsurgery.com).

Malignant Transformation of Facial Epidermoid Cyst With Distant Metastasis

To the Editor: Most epidermoid cysts are histologically benign. Isolated cases of premalignant and malignant conditions, however, have been identified in their walls. These include Merkel cell carcinoma, basal cell carcinoma, bowenoid papulosis, Bowen disease, Paget disease, mycosis fungoides, and squamous cell carcinoma.[1] Instances of squamous cell carcinoma arising in the wall of an epidermal inclusion cyst of the skin are rare, particularly with distant metastasis. Here, we present an unusual case describing the malignant transformation of a facial epidermoid cyst in a 79-year-old man with a 2.6 × 1.7-cm lesion on his left cheek with distant metastasis to both lung and mediastinal lymph nodes.

CLINICAL REPORT

A 79-year-old man presented with a 1-year history of a cystic solitary nodule on his left cheek (Fig. 1). He reported having received occasional punctures at local clinics during this time. The clinical diagnosis was epidermoid cyst; however, the initial, small nodule gradually increased in size, and the lesion grew rapidly 3 months before visit. Gross examination revealed a fistulated firm, solitary lesion with swelling and erythema measuring 2.6 × 1.7 cm on his left cheek. We performed excision and biopsy revealed squamous cell carcinoma. A computed tomography (CT) scan revealed a poorly defined well-enhancing lesion in skin and superficial soft tissue of left zygomatic and cheek area and enhancement in left masseter, temporalis muscle, and left zygomaticus muscles (Fig. 2). Positron emission tomography-CT demonstrated fluorodeoxyglucose-avid lesions in both lung field and multiple mediastinal lymph nodes (Fig. 3). We planned a wide surgical excision and chemotherapy. The patient subsequently underwent wide excision of the primary lesion with clear margins. Microscopically, neoplastic cells were detected next to the epidermal inclusion cyst with the transitional zone from benign to malignant changes (Fig. 4). The neoplastic squamous cells contain round-to-oval hyperchromatic nuclei with distinctive nucleoli and abundant cytoplasm. In addition, these lesions demonstrate frequent mitosis, keratin pearls, dyskeratotic cells, and intercellular bridges, indicative of well-differentiated squamous cell carcinoma (Fig. 5).

DISCUSSION

The nature of malignant transformation of epidermoid cyst is uncertain. It, however, has been suggested that chronic irritation or inflammation of the lesion induces dysplastic changes and malignant transformation.[2,3] Rupture of an epidermal cyst releasing its contents into the dermis causes a considerable foreign body reaction and leads to disintegration of the cyst wall and subsequent keratin granuloma formation.[4] It can also result in pseudocarcinomatous proliferation in the remnants of the cyst wall, which in turn,

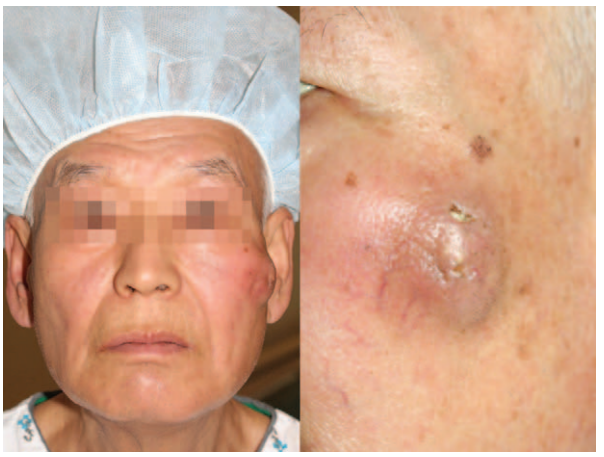

FIGURE 1. A 2.6 × 1.7-cm cystic, fistulated nodule with swelling and erythema on the left cheek.

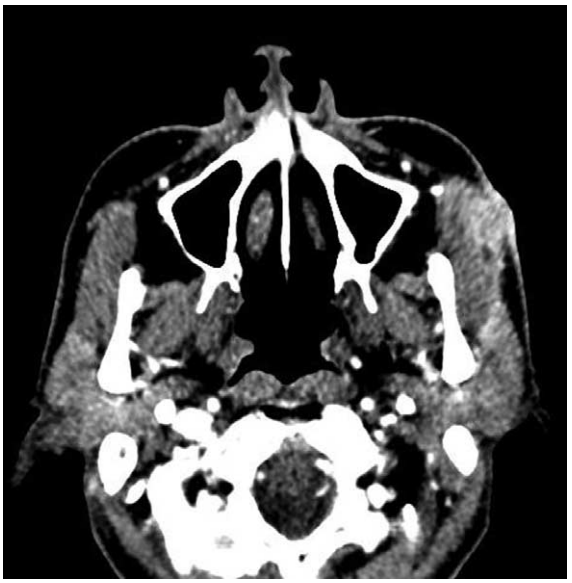

FIGURE 2. A computed tomography scan revealed a poorly defined, well-enhancing lesion in the skin and superficial soft tissue of the left cheek area and enhancement in the left masseter, temporalis, and zygomaticus muscles.

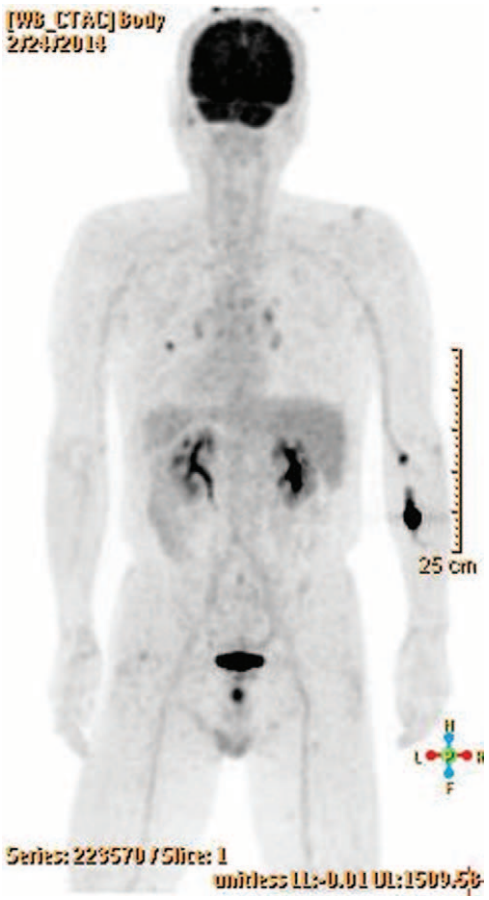

FIGURE 3. A positron emission tomography-computed tomography image demonstrated fluorodeoxyglucose-avid lesions in both lung field with multiple mediastinal lymph nodes. Suggested numbers indicate maximum standardized uptake values. Left cheek (3.5), right upper lobe nodule and left lower lobe nodule (3.9), superior mediastinal node (2.7), right hilar lymph node (3.3), right interlobar lymph node (2.5), and left interlobar lymph node (2.6).

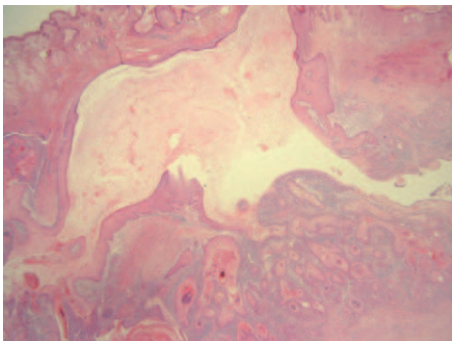

FIGURE 4. Specimen shows squamous cell carcinoma originating in the cyst wall. The covering epidermis of the skin and the underlying lesion of epidermal inclusion cyst are demonstrated (×40, hematoxylin–eosin stain).

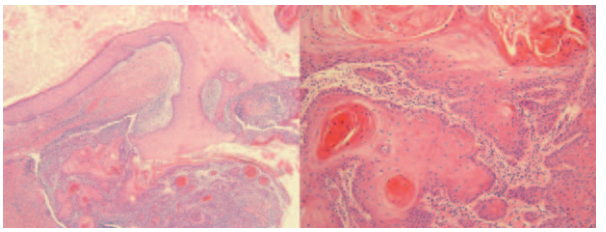

FIGURE 5. Left: The transitional zone from benign to malignant changes can be identified (×100, H&E). Right: Lesions demonstrate frequent mitosis, keratin pearls, dyskeratotic cells, and intercellular bridges, indicating a well-differentiated squamous cell carcinoma (×200, H&E). H&E, hematoxylin–eosin stain.

stimulate squamous cell carcinoma.[5] Moreover, ultraviolet radiation and human papillomavirus may contribute to the malignant transformation of epidermoid cysts.[3,6,7]

There have been only 2 well-documented cases of malignant transformation of epidermoid cyst with distant metastasis.[2,8] Lesions in the abdomen and gluteal region were 7.6 and 5 cm, respectively, whereas the durations of the lesions were 10 and 28 years, respectively. Both patients presented no evidence of metastasis on a systemic CT scan at the time of surgical excision. Follow-up examinations after 4 months, however, revealed lung metastasis in both cases. In our patient, the size of the lesion was 2.6 cm and had persisted for 1 year. Compared with the previously published cases, this size is relatively small and the morbidity is relatively short term. Therefore, this is a fairly rare case report of squamous cell carcinoma from an epidermoid cyst with aggressive metastasis, particularly of short term within 1 year. Despite the rarity of malignancy in an epidermoid cyst, there is general agreement that, malignant transformation should be suspected in case of skin lesions that are large, rapidly changing in size, inflamed, ulcerated, fistulated, and do not respond to medical treatment, and an associated microscopic study of the entire lesion should be performed to exclude malignancy. In addition, we recommend the histopathologic investigation of the entire lesion, even in those that have presented for a short term and have a small sized. Surgeon should be mindful of this risk for distant metastasis.

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Age as a Risk Factor for Flap Failure in Free Tissue Transfer

To the Editor: It is with genuine interest and excitement that the authors read ''Risk Assessment for Free Tissue Transfers: Is Old Age a Determining Factor?'' by Saçak et al,[1] which explored the association between age and complications following free tissue transfer. This study reports a single operator's experience with free tissue transfer in both young and old patients, and concludes that age is not an independent risk factor for various adverse outcomes. Although certainly a very interesting article, a number of concerns come to mind.

The study states that ''age is still considered as an independent risk factor among many reconstructive surgeons,'' and that the ''results of this study demonstrate that age is not an independent predictor of the surgical or medical complications in free tissue transfers.'' This conclusion may be premature, because of study design and inadequate power. To identify independent risk factors, multivariate analysis must be performed. Bivariate analysis can identify correlations, but without controlling for confounding variables only limited conclusions may be drawn. The underpowering of the study may also account for the statistical insignificance without acknowledging potential clinical significance. For example, having 20% hematoma rate in the $\geq$60-year age-group versus 6% in the $<$60-year age-group may be clinically relevant, but was not statistically significant in this study of 70 patients. Similarly questions arise with a 15% rate of seroma in the older group compared with a 2% rate in the younger group.[1]

Saçak et al report only 3 total flap failures in the sample population, making it difficult to identify a statistically significant association between a rare outcome and a given risk factor. In addition, no statistical difference is identified regarding patient backgrounds, including the existence of comorbid disease and American Society of Anesthesiologists (ASA) score. The authors note this limitation, reporting that only 1 patient had an ASA class of 3 or above.

TABLE 1. The Multivariate Odds Ratio of Age as a Risk Factor for Various Adverse Outcomes

	Multivariate Odds Ratio of Age $\geq$60	95% Confidence Interval	*P* Value
Flap Failure	5.191	2.995–8.998	<0.001
Return to OR	2.272	1.637–3.155	<0.001
Wound Complications	1.949	1.391–2.732	<0.001
Medical Complications	1.232	0.961–1.682	0.141

OR, odds ratio.

The 2005–2013 American College of Surgeons National Quality Improvement Program (ACS NSQIP) database were used to identify 798 free tissue transfers similar to those described in the study by Saçak et al (CPT codes 15756). Using this data, and performing multivariate analysis to control for confounding variables of diabetes, obesity, smoking status, and an ASA class of 3 or greater, the impact of age on outcomes of free tissue transfer can be better described (Table 1).

Many other surgeons have had excellent success with free tissue transfer reconstruction in the elderly,[2–4] but we must not disregard the impact that aging has on our healing and regenerative capacity. Analyzing ACS NSQIP data has also shown that increased age is an independent risk factor for flap failure, return to the odds ratio (OR), and wound complications, but may not be an independent risk factor for medical complications.

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The Prolonged Double Vision Is not Only Caused by Adhesion of Orbital Contents After Blowout Fractures: Important Role of the Orbital Proprioception

To the Editor: The representative symptoms of orbital blowout fractures are double vision and enophthalmos. In general, the former gradually improves and the latter progressively worsens over time. Enophthalmos results from expansion of the orbit and atrophy of the orbital content. Diplopia results from deformity of the orbit (causing positional changes of the muscles), adhesive impingement of the content (disturbing ocular excursion), and impairment of the contracting muscle (resulting from neurogenic or myogenic palsy).[1] Residual diplopia is really rare but must be predictable as one of the worst possible sequelae even after a faultless surgery (Fig. 1). In this time, a prolonged diplopia in downward gaze was observed in a noticeable patient who displayed a good downward excursion with monocular vision but restricted eye movement with binocular vison.

A 39-year-old man accidentally suffered a bruise on his left eye, and complained severe diplopia with the eyelid swelling and partial numbness of the perceptual domain of the left infraorbital nerve. The preoperative computed tomography (CT) revealed blowout fracture of the left orbital floor as a punched-out type (Fig. 2, upper).[2] Twelve days after injury, the broken orbit was repaired with the calvarial bone graft (Fig. 2, lower). The forced duction test was negative during operation. Aftercare was applied to expand the binocular focused field in addition to monocular rehabilitation with a monoculus on the unaffected eye. Diplopia was getting improved but the double vision was prolonged in the down gaze especially in binocular vision (Fig. 3). Numbness also still continued in the left nasolabial area

To regulate an adequate alignment of the either eyes, the orbital proprioception must play an important role as well as optical inputs. Based on both afferent signals, brain tactfully controls extraocular muscles for binocular vision.[3–4] Most of residual diplopia results from orbital adhesion and neurogenic or myogenic palsy of extraocular muscles. These patients must show the same restriction of the downward gaze, both with monocular and binocular visions (Fig. 1). In this patient, the down gaze restriction with binocular vision, however, was more serious than that with monocular vision. The eye movement was so sufficient in monocular vision that no mechanical impedance, such as fibrous adhesion and muscle palsy, existed in the affected side. Adjustment of ocular motion may acquire more proprioceptive sense than visual perception, because the patient complained prolonged diplopia with good vision in both eyes. In elder blowout fractures, in spite of minor finding of CT, less ability for sensory recover may cause less retrieval of eye movements.[5] In

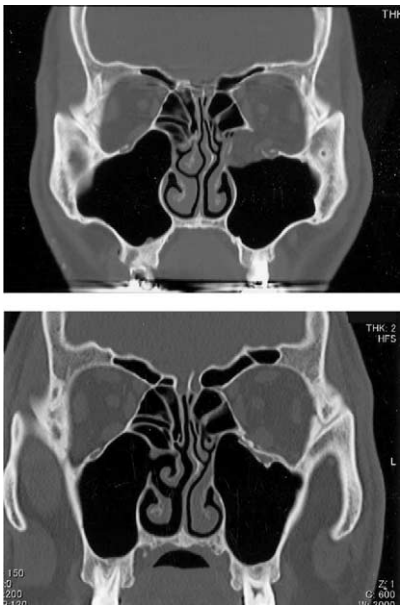

FIGURE 2. A CT image of a 39-year-old man before operation (upper). The left orbital floor was punched-out and the orbital content with the inferior rectus muscle was herniated to the maxillary sinus. A CT image 8 months after operation (lower). The floor was reconstructed with the calvarial bone graft. CT, computed tomography.

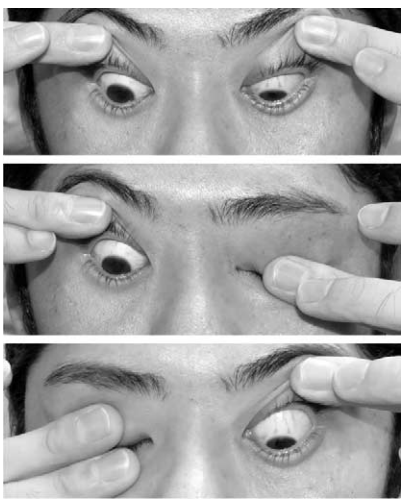

FIGURE 1. Photos of common right eye-movement restriction in downward gaze after blowout fracture. The restriction is the same with binocular and monocular visions.

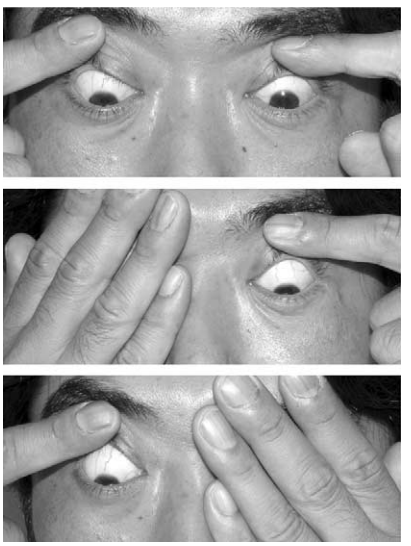

FIGURE 3. The down gaze images of the left orbital floor fracture. Downward restriction was seen with binocular vision. A good excursion was observed in the left eye as well as the right with monocular vision.

addition, the paradoxical eye movement after blowout surgery[2] may be attributable to impairment of orbital proprioceptions.

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Hede: Postoperative Low-Flow Cerebrospinal Fluid Leak of Endoscopic Endonasal Transsphenoidal Surgery for Pituitary Adenoma: Wait and See, or Lumbar Drain?

To the Editor: I am writing in reference to the article titled ''Postoperative Low-Flow Cerebrospinal Fluid Leak of Endoscopic Endonasal Transsphenoidal Surgery for Pituitary Adenoma: Wait and See, or Lumbar Drain?'' by Zhan et al.[1] The article is well written; however, the study raises some questions, which I would like to highlight through your esteemed journal.

The author's conclusion is in agreement to previous conducted studies,[2] on the same topic. Since this, however, is a retrospective study and the same surgeon has operated all the patients, how were the patients divided into lumbar drain and conservative groups? What were the criteria used for deciding conservative management in some patients and lumbar drain in the others?

Another question which I would like to ask is how long did it take for the cerebrospinal fluid leak to stop in each group? If the cerebrospinal fluid leak stopped earlier in the lumbar drain group, then the duration of hospital stay would be reduced in those patients, thereby reducing the cost. This will make the insertion of lumbar drain cost effective. There have been previous studies, which have proven the efficacy of lumbar drain.[3] In our experience also placing the lumbar drain is effective and it helps stop the leak in 3 to 4 days, this way the duration of hospital stay of our patients is reduced.

The foci of infection in patients who developed meningitis, in the lumbar drain group needs to be analyzed further, whether the infection actually occurred from the lumbar drain site or was it from the intranasal leak site?

I would recommend a prospective randomized trial with well-defined exclusion and inclusion criteria before a definitive conclusion regarding the placement of lumbar drains in low flow postoperative cerebrospinal fluid leaks can be made.

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Molluscum Contagiosum on the Lip

To the Editor: Recently, the authors encountered a 20-year-old Chinese woman who presented with a papule on the vermilion border of her lower lip. The lesion had appeared 8 months earlier, and it had not changed in size; the patient did not report pain or discomfort. She was otherwise healthy and had no history of allergy. Physical examination revealed a soft, gray, 2- to 3-mm papule, without nodules, on the vermilion border of the lower lip, with multiple cheese-like yellow points visible under the surface. No similar lesions were found on the oral mucosa or skin. To confirm the diagnosis, the lesion was completely excised and submitted for histopathologic analysis, which showed epidermal cells with eosinophilic cytoplasmic inclusion bodies and compressed nuclei. The lesion was diagnosed as molluscum contagiosum (MC) (Fig. 1). There was no recurrence during 1 year of follow-up after the excisional biopsy.

Molluscum contagiosum occurs worldwide and affects individuals of any age, but children are the most frequently affected.[1] A higher incidence of MC is also reported in sexually active and immunocompromised adults.[2] Molluscum contagiosum can involve any area of the skin but is most common on the trunk, arms, groin, and legs. The lower abdomen and genitals are commonly affected in sexually active and immunocompromised adults.[3]

Molluscum contagiosum is caused by the molluscum contagiosum virus (MCV).[4] Molluscum contagiosum virus is a DNA poxvirus that infects only humans.[2] Infection is by direct or indirect contact, including touching the affected skin or touching a surface with the virus on it, such as a towel or toys.[2,4] Clinically, after an incubation period of 2 weeks to 3 months, the virus induces a local response, which manifests at the infected skin as small (2–6 mm) flesh-colored papules with a dimpled center.[5] A diagnosis of MC is usually based on the appearance of the lesions, and can be confirmed on excision biopsy. Histologically, MC is characterized by molluscum bodies in the epidermis above the stratum basale, consisting of large cells with abundant granular

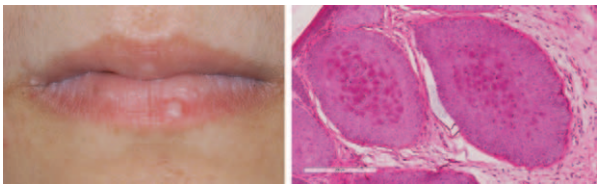

FIGURE 1. The patient had a 2 to 3-mm gray papule on the vermilion border of the lower lip. The excision biopsy showed epidermal cells with eosinophilic cytoplasmic inclusion bodies and compressed nuclei.

TABLE 1. Patients of Intraoral or Vermilion MC Reported in the Literature in the Past 30 Years

Reference	Year	Age (y)/Sex	Location	Treatment
Svirsky[11]	1985	32/M	Lower labial mucosa	Excisional biopsy + spontaneous involution
Fornatora[6]	2001	52/M	Maxillary gingiva	Excisional biopsy
Scherer[3]	2009	70/F	Retromolar region	Excisional biopsy
Cyntia[1]	2011	13/F	Lower labial mucosa	Excisional biopsy
Eri[12]	2013	63/M	Vermilion border of the upper lip	Excisional biopsy + discontinue topical
Present	2015	20M	Vermilion border of the lower lip	Steroid excisional biopsy

MC, Molluscum contagiosum.

eosinophilic cytoplasm, accumulated virions, and small peripheral nuclei.[4]

Although MCV could infect the mucous membranes occasionally,[2,4] molluscum contagiosum of the oral mucosa has rarely been reported. We searched PubMed for reports of MC patients that have affected only vermilion of the lip or the oral mucosa (Table 1) and found that only 5 patients have been reported during the past 30 years. A review of these patients reveals that the manifestations of oral MC are atypical. For example, in the patient reported by Fornatora,[6] MC of the oral mucosa was described as a sessile lesion with dome-shaped nodules.

The basic approaches for treating MC include destruction of the lesions, immune enhancement, and antiviral therapies.[7,8] There is no evidence to favor any single therapy, but physical destruction of the lesions may be favored.[9] First-line destructive therapies include cryotherapy and cantharidin. Cryotherapy is safe to use on the face and genitals. Cantharidin is a phosphodiesterase inhibitor. Its exact mechanism of eradication of the MC lesions is still unclear, but a number of researchers have found it to be a highly effective treatment.[7,8] Other therapies, such as imiquimod and cidofovir, are also reported. Imiquimod is a commonly used immune-enhancing therapy that has shown mixed results in terms of efficacy in treating MC. Cidofovir, a viral DNA polymerase inhibitor as a representative topical form of antiviral therapy, has been successfully used to treat severe and refractory MC in children infected with the human immunodeficiency virus.[10] According to our literature review, the preferred first-line treatment of oral MCs has been excisional biopsy, which was also performed in this patient.

Molluscum contagiosum occasionally occurs on the oral mucosa and with variable manifestations. To avoid misdiagnosis, the possibility of MC should be considered for papular lesions of the lip or oral mucosa. Referrals to specialist for further consultation or treatment recommendations may be beneficial for patients with oral MC.

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Mandibular Fracture After Inferior Alveolar Nerve Lateralization: A Rare and Misunderstood Complication

To the Editor: Jensen and Nock[1] in 1987 were the first to describe the technique of inferior alveolar nerve (IAN) lateralization. Since then, many complications have been described such as osteomyelitis, loss of implants, profuse hemorrhage, prolonged neurosensory disturbance, and the most infrequent mandibular fracture.

Recently, we have treated 3 cases of mandibular fracture after IAN lateralization. Three women, with ages between 60 and 70 years, were referred to our emergencies service by dental clinics for evaluation after IAN lateralization. None of the patients reported any previous traumatic record.

Only 4 cases[2–5] were found in our bibliographic survey, that, with the 3 cases we publish, add altogether to 7 cases of this complication. This complication is extremely rare, and possibly affected by publication bias.

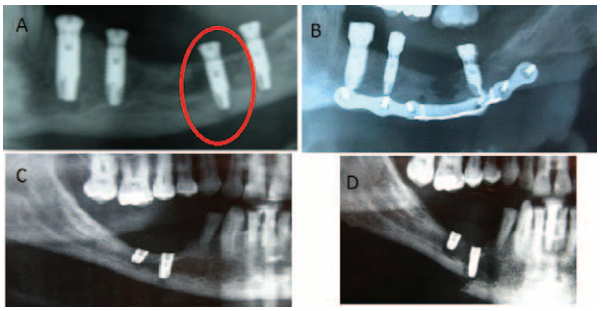

FIGURE 1. A and C, The placement of the implants transgressing the inferior cortical after IAN lateralization; B and D, the fracture in the place of that implants.

Five of these cases were treated with osteosynthesis without any kind of complication. The osteosynthesis used varied because there are not general recommendations for the treatment of this situation. From the 2 cases that were treated using a conservative approach, one was suffered a complication because of a nonunion and in the other one 60 days of liquid diet were needed. This could represent an inconvenience for the patient and does not guarantee a successful cure. Therefore, we believe that this complication should be treated by osteosynthesis.

Although the possible causes cannot be established with certainty, there are different hypotheses. The lingual position of the dental channel may help the production of a fracture as the amount of bone to be removed is larger.[4] Two of our patients showed this feature. In addition, these patients suffer a significant[5] reduction of the mandibular body vertical dimension, and even atrophy. The number of implants placed also helps the reduction of the bone stock that is capable of bearing forces. This explains why none of the cases published were associated with the previous trauma.

The positioning of the implant in these cases may facilitate the production of the fracture. Mason et al[6] recommend that the placement of the implant includes the lower cortical as this stabilizes the implant. The placement of successive implants covering the lower cortical, however, weakens the compression band of the mandible, and therefore it reduces the capability of bearing. Therefore, we agree with Karlis et al[3] that the stability of the implant should not be obtained by transgressing the lower cortical continuity, especially if multiple implants are placed. The transgression of the lower cortical was observer in our 3 patients.

Placing multiple implants transgressing the lower cortical and the lingual position of the alveolar canal are risk factors to develop such complication when associated with an IAN lateralization, especially in patients with moderate-to-severe mandibular atrophy (Fig. 1). These patients should be followed up closely. If the complication appears, it should be treated by osteosynthesis.

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