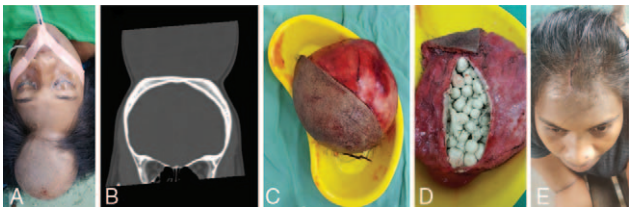

FIGURE 1. (A) A 21-year-old lady presenting with an anterior scalp lesion, with computed tomography scan (B) revealing an underlying deformational plagiocephy. The lesion was excised (C), revealing a fluid filled cyst containing (D) dermal hair follicles. One month postoperative image of the patient (E).

pathogenesis of this condition and the optimum timing for surgical intervention to avoid the deformity.

Key Words: Dermoid cyst, Kiribati, plagiocephaly

CLINICAL REPORT

We present a report of a 21-year-old female from Kiribati, with a lesion of her anterior scalp (Fig. 1A) present after birth with progressive enlargement. She had undergone computed tomography (CT) imaging, revealing a cystic lesion (Fig. 1B) with no intracranial involvement, interestingly the lesion had resulted in underlying deformational plagiocephaly of her cranial bones.

The patient underwent surgical excision of her lesion, showing a fluid filled cyst (Fig. 1C) with the presence of dermal hair follicles evident (Fig. 1D). The patient had an unremarkable postoperative recovery (Fig. 1E).

DISCUSSION

Dermal cysts (DCs) are commonly located in the midline of the scalp, occupying the subgaleal space,[1] and usually result from anomalous isolation of ectoderma and mesoderm tissues during fusion with the cranial suture lines.[2]

Due to their close proximity and potential extensions into the intracranial space, imaging in the form of CT or magnetic resonance imaging is recommended prior to surgical excision.[3]

Adjacent bony involvement with DCs is relatively common, with a recent review reporting a high association, with bony erosions or defects being the most commonly observed abnormality.[2]

To the best of our knowledge, this is the 1st report of a DC in a Pacific Islander and 1 with consequent deformational plagiocephaly, the latter we infer is the result of the long standing presence of the DC, which for the most part is rarely seen, due to early surgical intervention. Given the age of the resection in our patient, it is unlikely that the plagiocephaly will correct itself. We therefore recommend early excision prior to 12 months of age, consistent with the literature, to allow for correction.[4]

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Surgical Management of Primary Chronic Osteomyelitis of the Jaws: The Use of Computer-Aided-Design/ Computer-Aided Manufacturing Technology for Segmental Mandibular Resection

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Abstract: The term osteomyelitis of the jaws identifies different pathological patterns characterized by the involvement of the cortical bone and bone marrow in osteoarticular infections.

At the head and neck level, the segment most affected by osteomyelitis is the mandible and in most of the cases the cause of the infection is bacterial, as a result of pulp or periodontal infections, post-extraction alveolitis, foreign bodies and fractures. The mandibular PCO often presents with an insidious onset, without a striking acute phase, and it is characterized by recurrent episodes of pain, swelling, lockjaw, latero-cervical lymphadenopathy, without signs of suppuration.

Three patients have been collected and recorded for the study.

The authors believe that in the more advanced cases of PCO in adult patients, in which the mandibular bone appears almost entirely sclerotic and deformed, and that are not responsive to pharmacological therapy or to conservative surgical therapies such as decortication, it is necessary to perform a complete removal of the portion affected by osteomyelitis, with lower alveolar nerve preservation and contextual reconstruction with free microvascular bone flap.

Our review aims to describe the clinico-pathological features of a rare pathological entity, propose a surgical treatment algorithm using computer-aided-design/computer-aided manufacturing technology and review the existing literature.

Key Words: Bone tumor, maxillary surgery, osteoarticular infections

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Received June 29, 2019.
Accepted for publication September 30, 2019.

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The authors report no conflicts of interest.
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).

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000006121

The term osteomyelitis of the jaws identifies different pathological patterns characterized by the involvement of the cortical bone and bone marrow in osteoarticular infections.

At the head and neck level, the segment most affected by osteomyelitis is the mandible and in most of the cases the cause of the infection is bacterial, as a result of pulp or periodontal infections, post-extraction alveolitis, foreign bodies and fractures.

Osteomyelitis may also be caused autoimmune diseases or vascular injuries (endarteritis), but in most of the cases the etiology is unknown. Marx and Mercuri defined as acute an osteomyelitis lasting less than 4 weeks, and as chronic one that exceeds 1 month.[1,2] Chronic osteomyelitis can be of 2 different types: suppurative, also called secondary chronic osteomyelitis (SCO), characterized by suppuration, abscess, fistula and sequester formation, and supported by a specific bacterial etiological agent, such as Staphylococcus aureus or Staphylococcus epidermidis; non-suppurative, without a well-defined etiological cause and characterized by a common intense sclerotic periosteal reaction. The latter form of osteomyelitis is called primary chronic osteomyelitis (PCO).[3–5] This form was initially called Garre's sclerosing osteitis when it affected children or adolescents, or diffuse sclerosing osteomyelitis for its radiographic and histopathological characteristics. The mandibular PCO can be isolated or a chronic multifocal systemic osteomyelitis CRMO (chronic recurrent multifocal osteomyeliti)[6–13] or of other heterogeneous systemic syndromes such as SAPHO Syndrome (synovitis, acne, pustolosis, hyperostosis, osteitis).[14–19]

The mandibular PCO often presents with an insidious onset, without a striking acute phase, and it is characterized by recurrent episodes of pain, swelling, lockjaw, latero-cervical lymphadenopathy, without signs of suppuration.

Radiographically, there is characteristically patchy radiopacity-radiolucency. Frequently subperiosteal bone formation (onion-skin appearance) of the jawbone is highlighted by CT-scan. These features can be localized, diffuse, mono or bilateral (Supplemental Digital Content, Table 1, http://links.lww.com/SCS/B52).

PCO prevails in female patients and has two incidence peaks, which are before age 20 and after 50 to 60 years of age,[20,24,25,26] although pediatric cases (childhood or adolescent years)[22,25,27] have been described.

From an immunological point of view, it is characterized by a moderate increase in C-reactive protein, VES, and leukocyte count.

From a microbiological point of view, since there is no suppurative process, in the PCO the culture examination on bone samples is often negative or contaminated by commensal flora of the oral cavity. For this reason, antibiotic therapy is often not effective.

According to the literature existing, the treatment choice for PCO can be variable. In fact, it can range from conservative approach using medications (antibiotics and steroids), or mini-invasive surgery (extraction/bony curettage) combined with antibiotics, to more aggressive surgical procedures such as marginal or segmental resection of the mandible. However, conservative management often leads to multiple recurrences of the disease, and aggressive management leads to significant comorbidity needing for reconstructive surgery.

Nowadays, computer-assisted technologies in maxillo-facial diagnosis and treatment allow us to improve the percentage of success in terms of disease care and reconstructive outcomes for the most challenging PCO cases.

PURPOSE

Our review aims to describe the clinico-pathological features of a rare pathological entity, propose a surgical treatment algorithm using computer-aided-design/computer-aided manufacturing technology and review the existing literature.

Clinical Report

In this article 3 cases of PCO of the jaw, treated at Maxillo Facial Unit of Policlinco S.Orsola di Bologna Hospital, have been recorded and presented.

Patient 1

A 25-year-old female patient, who arrived to our unit of Maxillofacial Surgery in July 2017 after a long history of pain and swelling of the right jaw that started 7 years earlier was initially considered as a form of odontogenic pain. In January 2011, the patient was submitted to endodontic treatment of element 4.5, that was considered responsible for the clinical presentation. In April 2011 and November 2011, due to the persistence of pain and periosteal reaction of the mandibular cortical bone with sclerotic reaction of bone marrow evident at the CT Dental Scan, surgical curettage and 2 bone biopsies were performed, with a result compatible with fibrous dysplasia. Total body PET was negative for systemic disease. In 2012 2 lower right alveolar nerve decompressions were carried out together with end to terminal neurorrhaphy between the left and right lower alveolar nerves, in order to reduce the pain. Because of the latter, the patient had undergone prolonged cortisone therapy for which she developed an exogenous cushing syndrome. The follow-up radiographic images showed a progressive involvement of the entire jaw by the sclerotic and periosteal reactions. After having thoroughly analyzed the clinical history, the X-ray images and the biopsies, we made a diagnosis of PCO at our Hospital and therefore decided to perform a subtotal mandibular resection, from right subcondylar region to left mandibular angle, and reconstruction with a CAD/CAM plate and a free fibular flap (Fig. 1 A–M).

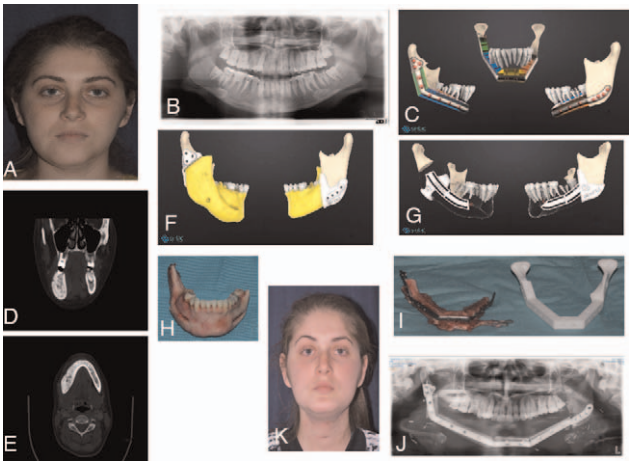

FIGURE 1. (A) Preoperative clinical image. (B) OPG at onset of disease in 2011 shows diffuse osteosclerosis of the body of the right mandible with marked thickening of the cortical bone and periosteum. (C) Preoperative CT scan axial shows an extensive osteosclerotic process with a ground glass aspect, involving the entire body and ramus of mandible on the right, excluding the right condyle. (D) Computed tomography scan (coronal) corresponding to the orthopantomograph shown in A shows an extensive osteosclerotic process with a ground glass aspect, involving the body of the mandible (E) Virtual reconstructive 3D planning using four bone fibular segments. (F) Virtual surgical resection from the right subcondylar level to the left mandibular angle. (G) Virtual planning of template for alveolar nerve lateralization and preservation. (H) Virtual planning of mandibular cutting guide. (I) Surgical bone specimen. (J) Intra-operative image showing right mandibular reconstruction using a CAD-CAM plate to fix the fibula segments. (K) The 3D printed model of the reconstruction and the real one. (L) Postoperative OPT shows the reconstruction of the mandible with CAD/CAM plate and fibular free flap. (M) Postoperative clinical result.

Currently the patient no longer has mandibular pain and the CT scan does not show signs of PCO recurrence.

Patient 2

A 24-year-old male patient with no history of granulomas, caries or odontogenic infections. In May 2016 the extraction of the element 3.8 was performed because it was affected by dysodontiasis. After 5 months of well-being, edema, swelling and pain lockjaw at the level of the left mandibular branch began, with recurrent acutisations every 3 weeks and good response to antibiotic therapy (Spiramycin); a CT scan was requested and it showed the presence of osteolytic and osteoaddensing areas at the level of the left mandibular branch. In February 2017, a bone biopsy was carried out at another hospital, resulting in a fibro-osseous lesion, without a precise diagnosis. In June 2017, the CT scan of the mandible and bone scintigraphy showed hyperaccumulation of the contrast medium at the level of the left jaw; the same results were obtained in October 2017, in the absence of systemic inflammatory-infectious processes. In July 2017 we performed a new bone biopsy and surgical curettage in our unit of Maxillofacial Surgery, with a result compatible with mandibular PCO. The microbiological culture test was positive for *Streptococcus oralis*, which was considered as a contaminating bacteria (Fig. 2 A–F).

Patient 3

A 24-year-old female patient with a history of extraction of element 3.8 due to dysodontiasis in May 2016. In November of the same year, there was the onset of pain and swelling on the left hemimandible. The patient began intravenous antibiotic therapy with resolution of the symptomatology. Because of the recurrent episodes of pain and swelling, she was addressed to our unit of Maxillofacial Surgery where a mandibular bone biopsy, surgical curettage and a microbiological examination were performed, which showed presence of *Actinomyceso dontolyticus* and *S. oralis*. After curettage and hyperbaric oxygen treatment without satisfying results in terms of reduction of pain and swelling, we decided to perform a left mandibular resection with disarticulation and reconstruction with a CAD/CAM plate and a free fibular flap (Fig. 3 A–K).

Currently the patient no longer has mandibular pain and the CT scan does not show signs of PCO recurrence.

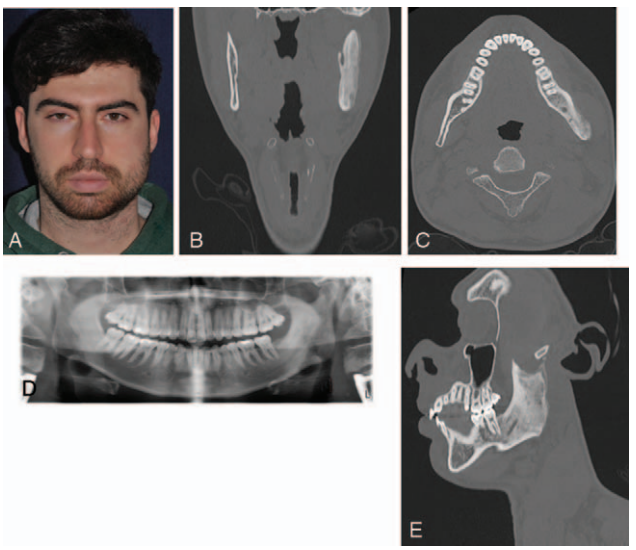

FIGURE 2. (A) Preoperative clinical image with evident swelling of the left mandible. (B) OPG at onset of disease shows a diffuse osteolytic and osteosclerotic process of the left mandible ramus, excluding the condyle. (C) Computed tomography scan coronal corresponding to the orthopantomograph shown in A; it shows a mixed pattern with sclerotic and lytic zones of the left mandibular ramus, a subperiosteal bone formation with thickening of the left mandibular angle and the dissolution of the cortical boarder. (D). Computed tomography scan axial corresponding to the orthopantomograph shown in A; it shows a mixed pattern with sclerotic and lytic zones of the mandibular ramus, a subperiosteal bone formation with thickening of the left mandibular angle and the dissolution of the cortical boarder. (E) Computed tomography scan axial corresponding to the orthopantomograph shown in A; it shows a mixed pattern with sclerotic and lytic zones, a subperiosteal bone formation with thickening of the left mandibular angle and the dissolution of the cortical boarder. (F) Computed tomography scan sagittal corresponding to the orthopantomograph shown in A; it shows a mixed pattern with sclerotic and lytic zones, a subperiosteal bone formation with thickening of the left mandibular ramus and the dissolution of the cortical boarder.

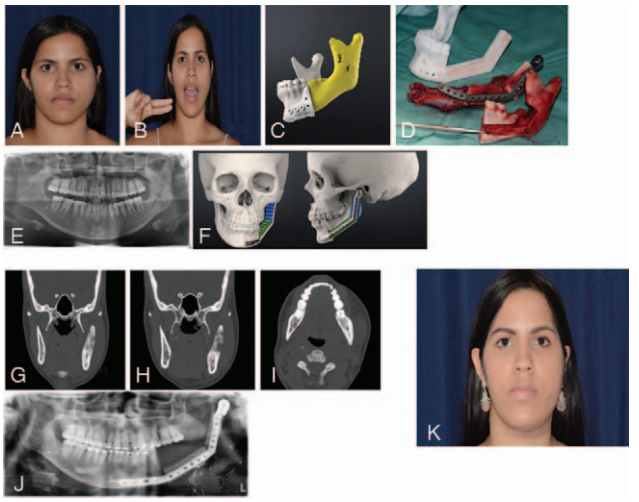

FIGURE 3. (A) Preoperative clinical image with swelling of the left mandible. (B) Preoperative clinical image with lockjaw and reduced mouth opening. (C) OPG at onset of disease shows a diffuse osteolytic and osteosclerotic process of the left mandible ramus including the left condyle. (D) Computed tomography scan (axial) corresponding to the orthopantomograph shown in C; it shows a diffuse osteolytic and osteosclerotic process of the left angle of the mandible with thickening of the cortical bone and periosteum. (E-F) Computed tomography scan (coronal) corresponding to the orthopantomograph shown in C; it shows diffuse osteolytic and osteosclerotic process of the left mandible ramus including the left condyle and thickening of the cortical bone and periosteum is evident. (G) Virtual reconstructive 3D planning using two bone fibular segments. (H) Virtual planning of mandibular cutting guide. (I) The 3D printed model of the reconstruction, the surgical bone specimen and the two bone segments of a fibula free flap supported by a custom-made titanium reconstructive plate including condyle. (J) Postoperative OPT shows the reconstruction of the mandible with CAD/CAM plate and fibular free flap. (K) Postoperative clinical result.

RESULTS

All 3 patients showed the appearance of the symptoms during the second and third decade of life (in 1 case, symptoms occurred at the age of 18, in the remaining 2 cases at the age of 22). The jaw is always involved and a dental extraction at the level of the inferior arch, usually involving molars or premolars, is present in the anamnesis. Symptoms are always represented by pain, swelling and difficulty in mouth opening, they are responsive to the pharmacological treatment with anti-inflammatories (NSAID) and antibiotics, but they also show a chronic and recurrent pattern. The facial CT shows a progressive dimensional increase of the mandibular bone at the level of the body in the affected side, with aspects of diffuse osteosclerosis associated with osteolytic areas and more or less marked thickening of the cortical bone and of the periosteum (Fig. 3G).

The histopathology was always compatible with PCO, showing the presence of a bone which presented features of c.c. pagetoid pattern in the osteoid trabeculae, with multiple areas of osteolysis and osteosclerosis together with thickening and irregular distribution of the bone trabeculae, with periosteal reaction and minimal plasma cell infiltration.

MICROBIOLOGY FEATURES

From a microbiological point of view, in 2 out of 3 cases *S.oralis* was isolated, alone or together with *A. odontolyticus*.

Laboratory tests showed no significant alteration in terms of increase in inflammatory indices. In the second case, further blood and urinary tests were carried out, such as calcium, parathormone, alkaline phosphatase, osteocalcin, and hydroxyproline, which excluded Paget disease as a possible differential diagnosis.

In first and third cases, surgical curettage was performed. Since this conservative approach failed for both the patients a subsequent segmental mandibular resection associated with free microvascular flap was performed.

The second case underwent surgical curettage followed by medical therapy based on periodic cycles of antibiotics. The patient refused segmental resection.

In the patients treated with demolitive surgery, a complete remission of the clinical symptomatology was observed; in the second case, instead, the signs and symptoms continued to occur on a monthly basis.

DISCUSSION

Eyrich et al proposed a classification for the osteomyelitis of the jaw which distinguishes 3 different groups: acute osteomyelitis, secondary chronic osteomyelitis (SCO) and primary chronic osteomyelitis (PCO).[28] Both chronic osteomyelitis occur for longer than 1 month after the onset of symptoms, differing on the duration of the symptoms, the radiologic findings and the treatments, as described by Julien Saint Amand M et al.[3] PCO is a rare and specific bone pathology of the maxillofacial district that affects almost exclusively the mandible. Baltensperger[20] and Van Markestyn[21] describe a case in which the PCO is localized to both the mandible and the zygomatic bone, Lygidakis[22] presents a PCO case of the maxilla and the zygomatic bone with involvement of the inferior orbital frame, Tsuchimochi[23] instead documents a case of PCO of the sternoclavicular joint. Probably, as Bevin describes, this prevalence of location is explained by the fact that at the mandibular level, since there is a terminal vascularization, the blood supply is reduced compared to the maxilla.[25]

PCO, once called sclerosing osteitis of Garrè or DSO (diffuse sclerosing osteomyelitis), has 2 incidence peaks, which are before age 20 and after 50 to 60 years of age,[20,24,25,26] although pediatric cases (childhood or adolescent years)[22,25,27] have been described. In our cases, the localization of PCO has been exclusively mandibular, without systemic involvement visible at the total body PET, thus excluding the CRMO hypothesis; all three patients of our study presented the osteomyelitis in their adolescent years.

The clinical symptomatology described is in most cases characterized by pain, lockjaw, swelling, loco-regional hardening, and in rare cases there can be hypoesthesia in the territories innervated by the inferior alveolar nerve or latero-cervical lymphadenopathy.[20,23,25,26,27]

As described in the literature, our patients never showed signs of secondary chronic osteomyelitis such as extra or intra-oral pus and fistula formation.

The etiology and pathogenesis of PCO still remains unclear, although many theories have been formulated in this regard: bacterial infections of dental origin, bacteremia, autoimmune processes able to determine endarteritis or vascular alterations, chronic periostitis, diabetes mellitus.[25,29]

Many authors have described low-grade dental infection as a possible cause of PCO,[1,14,28,30–32] however in many other studies patients presented with good dental health and therefore there could be no correlation between dental pathology and osteomyelitis.[20,21,24,25] However, in the totality of our cases, a dental extraction occurred about a year before or in conjunction with the onset of the symptomatology.

From a microbiological point of view, the bacteria isolated from the bone biopsy are mostly pathogenic commensals of the oral cavity, therefore, they could be the result of contamination of the surgical specimen, especially when the specimens are harvested transorally.[26,27,29] In our series of 3 patients, we isolated 2 microorganisms: *S. oralis* and *A. odontolyticus*.

The histological examination of PCO is characterized by paget-like bone tissue, represented by areas of fibrosis alternating with osteolytic areas, especially in the early stages of disease, while in the more advanced ones the bone is typically fibrotic. Furthermore, a sclerotic pattern is often accompanied by normalization of the cortical bone.

Also, from the radiological point of view it is possible to notice a mixed pattern of sclerosis and osteolysis with a different degree of periosteal reaction. In the early stages there is a tendency to increase of the osteolytic areas that correlates with a more marked symptomatology; in the more advanced and chronic phases of the pathology instead there is a gradually increasing degree of bone sclerosis with accompanied by an increase in the thickness of the mandibular cortex.

In our study, in 1 case there was a mixed pattern of osteosclerosis and osteolysis, while in the remaining 2 cases, having become chronic, the mandible was completely sclerotic with an increase in height and thickness of the same.

PCO enters in differential diagnosis with benign and malignant bone diseases[20,22,25,28,35,36]: the benign ones are represented by fibrous dysplasia, Paget disease, cementoma, ossifying and non-ossifying fibroma, infections of the salivary glands (mumps or chronic sialadenitis) or chronic lymphadenitis; malignant ones are represented by Ewing's sarcoma, osteosarcoma, chondrosarcoma, non-Hodgkin lymphoma and metastases.

Regarding the treatment of PCO we must distinguish two different ways: a non-surgical one, based on the use of antibiotics, NSAIDS, bisphosphonates,[35,37] pamidronate,[35] muscle relaxants[30] and hyperbaric oxygen therapy[30,38,35]; and a surgical one, based on bone decortication, alone[38,39] or associated with bone grafting,[40] or on partial or segmental resection of the pathological segment.[41,46,47]

The contribution of antibiotic therapy is, for many authors, still controversial, mainly because this pathology is not directly related to an infection caused by a specific pathogen.[26,42]

In most cases, surgical mandibular decortication represents the most adopted therapeutic choice for the forms of PCO not responsive to pharmacological treatment: it consists in removing the affected or necrotic cortical bone while preserving the healthy bone at the periphery of the lesion in order to allow a correct subsequent healing. This procedure can be performed intraorally,[24] as described for the first time by Obwegeser,[43] or with an extraoral submandibular approach,[44] also removing the involved dental elements.[24,44]

In the case series described by Eyrich, Bevin and Lygidakis[22,25,28] extensive decortication was performed on all patients, it was often repeated more than once, with a good resolution of the pathological picture in the subsequent follow-up. Bevin also combines decortication with the placement on the surgical site of a gauze soaked in antibiotic.

For the more resistant and more advanced cases, Suei proposes to perform a marginal or segmental resection of the mandible.[41]

In agreement with Suei, we also preferred to perform a demolitive surgical therapy based on the resection of the affected mandibular tract, since both the pharmacological therapy and conservative surgery had no positive effects. In the 2 cases surgically treated, we performed a mandibular breech with a piezo surgery tool using CAD/CAM cutting guides and an external alveolar nerve dime. The surgical defect was then reconstructed in both cases using a microvascular free flap of fibula modelled and fixed to a reconstructive CAD/CAM plate.

Accordingly to the presented data it seems that in the more advanced cases of PCO in adult patients, in which the mandibular bone appears almost entirely sclerotic and deformed, and that are not responsive to pharmacological therapy or to conservative surgical therapies such as decortication, it is necessary to perform a complete removal of the portion affected by osteomyelitis, with lower alveolar nerve preservation and contextual reconstruction with free microvascular bone flap. For this specific aim, the CAD/CAM technology represents a reliable and reproducible technology that allows a correct and prosthetic-guided reconstruction of the mandible.[45]

However, in agreement with other authors, it seems that the most correct option in the mixed phase (patchy osteosclerotic/osteolytic) of the PCO and in pediatric or adolescent patients remains drug therapy and decortication.

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Pulsatile Tinnitus Caused by Internal Jugular Phlebectasia in an Adult

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Abstract: Internal jugular phlebectasia is a rare condition. Children with internal jugular phlebectasia are often discovered by their parents when they notice a soft mass in the neck that appears when the child cries, coughs, or breathes deeply. Most internal jugular vein dilatations occur unilaterally on the right side according to the literature reports. To our knowledge, no other internal jugular phlebectasia patients reported pulsatile tinnitus as the major complaint without a soft mass in the neck. The authors reported a female adult patient with left-side internal jugular phlebectasia with pulsatile tinnitus as the major complaint without a soft mass in the neck. Internal jugular phlebectasia was diagnosed by color ultrasound of the internal jugular vein. Pure-tone audiometry, tympanometry, hemoglobin, thyroid function, and magnetic resonance imaging were made to differentiate other diseases that can cause the pulsatile tinnitus. Conservative treatment is recommended in this report. The possibility of internal jugular vein dilatation should be considered when differentiate the possible diseases that caused pulsatile tinnitus.

Key Words: Adult, internal jugular phlebectasia, pulsatile tinnitus

Internal jugular phlebectasia, 1st described by Zukschwerdt in 1929, refers to the appearance of saccular or fusiform dilatations in the internal jugular vein,[1–3] with 1 study applying a definition of an internal jugular vein diameter >2.0 cm during breathing.[4] The pathogenesis of internal jugular phlebectasia is unclear, but it is probably due to congenital developmental defects in the venous wall,[5] which reduces the flexibility of elastic fibers and mass of smooth muscle, leading to wall dilatation and thinning.[6,7]

The prevalence of internal jugular phlebectasia is unknown, likely because it is rare. There are only 50 patients in the recent literature most of which are children according to the recent report.[8] It is often considered a congenital disease that usually occurs in childhood, and it has even been associated with childhood asthma.[9] However, the condition can occur at nearly any age.[5] One study of 26 patients from 1984 to 1997 involved individuals aged 5 to 67 years.[4] Children with internal jugular phlebectasia are often discovered by their parents when they notice a soft mass in the neck that appears when the child cries, coughs, or breathes deeply, and disappears when the child is calm.[10] A similar phenomenon occurs in adults with internal jugular phlebectasia.[11]

Here we describe a woman with internal jugular phlebectasia who was diagnosed after presenting at our hospital with pulsatile tinnitus in the left ear and without a soft mass in the neck during normal breathing and the Valsalva maneuver. To our knowledge, this is the 1st report of someone with this condition presenting with pulsatile tinnitus as the major complaint.

CLINICAL REPORT

A 32-year-old woman presented at West China hospital of Sichuan University with a complaint of left ear pulsatile tinnitus that followed the rhythm of her heart beat and that had persisted more than 3 years, since the 3rd trimester of her pregnancy. At 1 month after delivery, pulsatile tinnitus disappeared from the right ear but persisted in the left ear. The left pulsatile tinnitus temporarily disappeared when the left upper clavicle was pressed or when the head was turned to the left. The pulsatile tinnitus was more pronounced when she stood than when she lay down.

The patient had no noticeable hearing loss or history of hypertension, diabetes, hyperlipidemia, anemia, hyperthyroidism, or asthma. The tympanic membrane was intact, with no sign of hemotympanum, and the membrane did not change color when

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Received August 2, 2019.
Accepted for publication September 30, 2019.
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The authors report no conflicts of interest.

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000006123