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Review Article

Risks and Complications in Bone Diseases

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Abstract

Bone conditions can be common in Maxillofacial Surgery consultations or in any of the stomatological specialties. Given that there are various diagnoses, and likewise the triggering causes of these processes, on many occasions it becomes complex for the professional to reach an accurate and timely recognition, to establish the appropriate therapy. In this chapter, together with each pathological lesion, the different risks and complications will be presented according to the evolution of the conditions. A bibliographic review was carried out with the aim of delving into the main complications and risks of injuries present in bone tissue. Inflammatory, congenital and tumor-type lesions can occur, among other variants, also divided into benign and malignant.

Keywords: Bone; Osteitis; Osteoma; Osteogenic Tumors

Introduction

Bone density Bone density (BO) is recognized as the measure of the amount of minerals contained in a given volume of bone, usually taking into account calcium and phosphorus. These OD measurements are used to diagnose conditions such as osteoporosis, determine whether osteoporosis treatments are effective, and estimate the likelihood of bones breaking. In dentistry and maxillofacial surgery, the density of the maxillary bones is essential to carry out multiple procedures, mainly surgical. Such is the case of implantology, since depending on the classification that the bone tissue presents according to certain characteristics, this will be the behavior to be followed by the surgeon. Low bone density can occur in patients treated for cancer, or with systemic conditions, etc [1,2].

Objective

Describe the main complications and risks of bone tissue diseases and injuries.

Reference Search Methods

The scientific information was compiled through a search using the following descriptors in English: The Medical Subject Headings (MeSH): "bone tissue, osteopathies, osteoma, bone tumor

Analysis strategy

The search was based solely on bone conditions of the oromaxillofacial complex.

Developing

A bone densitometry shows if there is a disorder that makes bones more fragile and more likely to break. The analysis uses x-rays to estimate how many grams of calcium and other bone minerals are in a segment of bone. The bones most frequently analyzed are those of the spine, the hip, and in dentistry the mandible and upper jaw. Panoramic radiography offers reliable data with evaluation indices to detect decreased OD, beginnings or presence of osteopenia or osteoporosis [1,2].

Many patients with osteoporosis do not present symptoms before having a fracture and panoramic radiography, being an examination that is requested relatively frequently by dentists, would be considered an ideal means of diagnosis. There are the following evaluation indices: 1. Panoramic mandibular index (MPI) 2. The width of the mandibular cortex (MCA) or mental index (MI). These are related to the amount of bone or BMD. 3. Mandibular cortical index (MCI), which is related to bone quality through the shape of the jaw. 4. Gonial index (GI). 5. Antegonial index (IA), which measures the posterior and anterior thickness of the mandibular ramus (they are not stable) 6. Mandibular-mandibular relationship (M/M), which is the relationship between the total height of the mandibular body and the height from the lower edge of the mandible to the lower edge of the mental foramen, considered the least significant [1,3].

The ICM, the ACM or MI and the IMP would be the most significant and stable for the prediction and early detection of osteo-

porosis in postmenopausal women with low BMD. To evaluate the shape of the mandibular cortex, the ICM is used where the changes produced from the mandibular cortex to the distal mental foramen are observed bilaterally, according to the following scale according to Klemetti., *et al*.: Normal cortex (C1): The endosteal cortical margin is uniform and sharp on both sides. Mildly to moderately eroded cortex (C2): The endosteal cortical margin shows resorption cavities or lacunar resorption, with 1 to 2 layers of endosteal cortical residues on one or both sides. Severely eroded cortex (C3): The endosteal cortical margin presents well-marked porous cortical residues [1,4].

Osteoporosis of the jaws Osteoporosis is a common skeletal disorder characterized by low bone mass and deterioration of the microarchitecture of bone tissue leading to increased bone fragility and, consequently, increased risk of fractures. It is the most common metabolic bone disease. Significant risk factors for the development of osteoporosis include female sex, advanced age, and early menopause. The dentist can screen patients with undetected osteoporosis using the information available in the consultation. You can then refer patients so that systemic treatment can be performed. Systemic treatment of osteoporosis aims to increase bone mass and includes hormone replacement therapy, bisphosphonates and calcitonin. It is not clear (yet) whether osteoporosis contributes to periodontal attachment loss, tooth loss, alveolar bone loss, and loss of residual ridge height. An indication of the quantity and quality of bone in the jaws can be useful when planning reconstructive interventions and subsequent implant treatment [1,2].

Various investigations have reported that the loss of bone substance in the jaws is a reflection that the same thing is happening in other bones of the body. This limits the possibility of effective rehabilitation of oral function. Likewise, the alveolar ridge constitutes a sensitive indicator of bone metabolism, warning of the existence of systemic bone diseases in humans [5].

Among the main conditions that are evident in these patients are

- Reduction of the alveolar ridge
- Decrease in bone mass and maxillary bone density
- Edentulism
- Decrease in cortical bone thickness
- Periodontal Alterations

To avoid the effects of osteoporosis for as long as possible, it is advisable to maintain a healthy diet, eliminate toxic habits, and exercise periodically. The treatment of osteoporosis must be comprehensive, including lifestyle measures, physical exercise, adequate calcium and vitamin D intake, and, when indicated, pharmacological treatment [6].

There are different therapeutic options at the present time, whose effectiveness and safety are well proven, especially in osteoporosis in postmenopausal women. Oral bisphosphonates (alendronate, risedronate) are the treatment of choice in most cases, but there are other options (teriparatide, denosumab, intravenous zoledronate) that must be applied in a very individualized manner, depending on factors such as age, presence or not of fragility fractures and comorbidities. Osteonecrosis of the jaws Osteonecrosis is an injury in which there is an interruption of blood circulation in the bone tissues, causing the bone to become devitalized. In the oromaxillofacial complex it is more common in the jaw.

It can also occur due to excessive use of steroids, abuse of toxic substances such as alcohol, joint conditions and a variety of diseases, such as cancer, collagen diseases, sickle cell anemia, coagulation disorders, HIV or anti-HIV medications. at some time after cancer treatment that includes radiation therapy or chemotherapy (methotrexate, bisphosphonates, or corticosteroids). Also called aseptic necrosis, avascular necrosis, and ischemic necrosis (Table 1).

Box of risk factors according to the consensus of the American Association of Oral and Maxillofacial Surgeons	
Medication related	**Local factors**
Potency of the bisphosphonate: the more powerful, the greater the risk of causing osteonecrosis of the jaws.	Alveolantary surgery:
Duration of treatment: the longer the treatment, the more likely it is to cause osteonecrosis.	Dental extractions.
The association with corticosteroids.	Implant placement.
Concomitant oral diseases: history of inflammatory pathology, especially periodontal.	Periapical surgery.
Demographic and systemic factors: advanced age is more associated with this pathology; The association with other chemotherapeutic agents is debated and smoking appears to increase the risk.	Periodontal surgery that affects the bone.
Genetic factors: some genetic alterations appear to be associated with an increased risk of osteonecrosis of the jaws in patients with multiple myeloma treated with bisphosphonates.	Local anatomy
	Mandible: lingual torus and mylohyoid line.
	Maxilla: palatine torus.

Table 1

Osteonecrosis of the jaws Osteonecrosis of the jaws (ONJ) does not have a unanimously accepted definition or etiology, although it is considered an oral lesion that mainly affects the mandibular bone. ONJ is a chronic osteomyelitis with slow and torpid evolution, which does not tend to cure. It can occur spontaneously or after a surgical procedure on bone tissue, trauma, treatment with bisphosphonates or high doses of Denosumab. Osteonecrosis of

the jaw may be refractory osteomyelitis rather than true osteonecrosis, especially when it occurs after the use of bisphosphonates. It is diagnosed when exposed necrotic bone is identified in the maxilla or mandible for at least 8 weeks.

Currently it is fundamentally associated with the use of Antiresorptives: it includes Bisphosphonates and RANKL Inhibitors known as monoclonal antibodies, among which are: • Actonel (generic name: risedronate) • Aredia (generic name: pamidronate disodium) • Bonefos (generic name: clodronate) • Boniva (generic name: ibadronate) • Fosamax (generic name: alendronate sodium) • Zometa (generic name: zoledronic acid) • Xgeva (generic name: denosumab) Monoclonal Antibody The symptoms are varied but generally those affected come to consultation with the following symptoms: [7].

ONM semiology Pain Inflammation
Erythematous areas Infection in the protective periodontium
Cavities that do not heal after dental treatments
Tooth mobility
Paresthesia or dysesthesia in the mandibular region
Fistula in exposed bone
Patient who reports received or is receiving treatment with bisphosphonates.
Presence of one or more ulcerated lesions in the mucosa of the alveolar processes, with exposure of the maxillary or mandibular bone.
The exposed bone has a necrotic appearance.
The lesion occurs spontaneously or, more frequently, after a history of dento-alveolar surgery (especially extractions).
Absence of healing for a period of at least 6 weeks.

Table 2

It is important that all the clinical characteristics mentioned above have no relationship with radiotherapy. The American Association of Oral and Maxillofacial Surgeons proposes 4 stages, from 0 to 3. Later, other authors subdivide stage 2 into 2a and 2b. (Ruggiero., *et al.* 2009, Bagan., *et al.* 2009). The different clinical stages would look like this

- Stage 0: patients who have no clinical evidence of necrotic bone but who present with non-specific clinical or radiographic symptoms or findings (dentalgia without dental explanation; dull pain in the jaw that can radiate to the temporomandibular joint; sinus pain; alteration of neurosensory function ; loss of teeth without periodontal explanation; fistula without pulp necrosis due to caries; loss or resorption of alveolar bone not attributable to periodontics; dense spongy bone; persistence of unremodeled bone in the alveoli after extraction; thickening of the lamina dura and decreased space of the periodontal ligament; narrowing of the mandibular canal.
- Stage 1: bone exposure with necrotic bone or a small ulceration of the oral mucosa without exposure of necrotic bone. Both would be asymptomatic.
- Stage 2a: bone exposure with necrotic bone or a small ulceration of the oral mucosa without exposure of necrotic bone, but with symptoms. Soft tissue/bone pain and infection. It is controlled with conservative treatments and does not progress.
- Stage 2b: bone exposure with necrotic bone or a small ulceration of the oral mucosa without exposure of necrotic bone, but with symptoms. Soft tissue/bone pain and infection. It is not controlled with conservative treatments and necrosis or the infectious signs derived from it progress.
- Stage 3: bone exposure. Necrotic bone. Pain, infection, and one or more of these signs: pathologic fracture, extraoral fistula, or osteolysis extending to the lower border.

The characteristics of the jaws are different from other structures of the body; they contain a complex ecosystem, where the dental structures and their adjacent tissues are exposed to the external oral environment. Over the course of an individual's life, it is common to suffer from dissimilar conditions in the oral cavity that are accompanied by infectious and inflammatory processes in the periodontal tissues, periapical abscesses, root pulp treatments, among other injuries that increase bimaxillary bone cell turnover. . It is important that the vascularization of the jaw is mainly through terminal arteries, which explains the appearance of necrotic processes more frequently in this location. In the oral cavity, the maxilla and mandible are subject to constant stress from masticatory forces. For this reason, microfractures occur in the mouth every day. Theoretically, in patients taking bisphosphonates these alterations are not repaired, laying the foundation for osteonecrosis to occur. This need for bone repair and remodeling increases greatly when there is an infection in the jaws, and/or when a tooth extraction is performed [5-8].

Treatment Modalities for ONJ

- Hyperbaric oxygen in combination with antiseptic rinses, antibiotics and surgery.
- Bone surgery guided by autofluorescence.
- Conventional bone surgery.
- Autofluorescence-guided sequestrectomy.
- Fluorescence guided sequestrectomy with tetracycline.
- Growth factors and autologous platelet concentrates.
- Platelet-rich fibrin after bone surgery.
- Bone Morphogenetic Protein 2 along with platelet-rich fibrin.
- Concentrated growth factor and closure of the wound by first intention.
- Pharmacological treatment with Teriparatide.
- Low level laser therapy.
- Topical ozone therapy

Role of stomatologists. The role in identifying patients at risk of OM is fundamental. mental. Studies have shown that the risk of developing the condition can be substantially reduced if patients are evaluated by a dental professional and preventative measures are taken (Nicolatou-Galitis., *et al*., 2019). Before starting antiresorptive therapy

Clinical-radiographic study

- Extraction of non-curable teeth or those with a poor prognosis.
- Rehabilitation of areas that need it.
- Eliminate cavities.
- Perform periodontal treatment.
- Educational talks for the patient and family members. Stages and proposed treatment
- **Stage 0:** Rinse with 0.12% chlorhexidine and control.
- **Stage 1:** NSAIDs and antibiotics, rinse with chlorhexidine 0.12% .
- **Stage 2:** NSAIDs and antibiotics, rinse with 0.12% chlorhexidine, debride.
- **Stage 3:** NSAIDs and antibiotics, 0.12% chlorhexidine rinse and resection.

This is a topic in which much remains to be investigated and clarified, so we recommend that readers continue to update themselves and look for literature that goes deeper into these important issues.

Osteomyelitis Osteomyelitis (OM) is an infectious inflammatory disease caused by pyogenic germs, mainly staphylococcus and sometimes streptococci, pneumococci and enterobacteria. Based on bacterial cultures, Staphylococcus aureus is the main causative agent of chronic osteomyelitis.

As a secondary cause, it can occur due to post-surgical complications such as dental extractions, maxillofacial trauma to the lower third with inadequate management of a fracture that subsequently further compromises the jaw. The typical age of presentation is between the fifth and seventh decade of life, with greater frequency of appearance in men. The most common site is the posterior body of the mandible. The incidence, outside of those immunocompromised patients who received head and neck irradiation, increases in patients who have poor oral hygiene and are alcohol or tobacco consumers. Osteomyelitis of the jaw is more common, since the blood supply to the jaw is much more extensive.

To diagnose it, clinical examination and imaging tests are essential. Bone scintigraphy was very useful for the present case as it confirmed the bone resorption activity present in the medullary cavity of the mandible. Diagnostic confirmation and susceptibility through the antibiogram were key tests to define the appropriate treatment. The varied pathogenesis of osteomyelitis requires specific treatment and strategies that focus on the eradication of the infection, along with the preservation of the integrity and function of the involved bone [1,2].

There is no universally accepted classification for osteomyelitis and this is due to the multifaceted presentation of the disease. The two best-known classification systems are Cierny-Mader and Waldvogel. Since they were introduced, there have been considerable advances in treatment strategies and options for osteomyelitis. In recent years, new systems of classification. However, they are not widely used due to the complexity and lack of evidence to support its clinical effectiveness. (See boxe 2, 3, 4, 5, 6)

Boxe 2. Depending on the duration of symptoms
Acute (7 and 14 days) • Acute hematogenous • Acute due to puncture wound
Subacute (several weeks or months)
Chronic (several months) It is associated with an epicenter of bone necrosis called sequestrum, which is usually surrounded by vascular reactive bone called involucrum, and the cloaca (connection between bone and periosteum) or sinus formation (connection between bone and periosteum). and skin).
Boxe 3.Etiological classification (Lew and Waldvogel)
Hematogenous osteomyelitis
Osteomyelitis of contiguous focus (due to trauma, surgery, prosthetic material or soft tissue dissemination)
Osteomyelitis due to vascular insufficiency (often related to diabetes mellitus)
Boxe 4. Classification according to anatomy and comorbidities (Cierny Mader)
Host condition • To healthy • Bs altered by systemic factors • B1 altered by local factors • B1s altered by local and systemic factors • treatment worse than the disease
Boxe 5. Functional impairment caused by the disease
Anatomical extent of infection • Spinal cord only (acute hematogenous) • Superficial cortex (contiguous spread of trauma of soft tissues) • Diffuse (cortical and spinal cord, mechanical instability)
Boxe 6. Degree of bone necrosis
Pediatric acute osteomyelitis
Osteomyelitis secondary to a contiguous focus of infection
Osteomyelitis secondary to hematogenous spread of infection
Osteomyelitis secondary to a contiguous focus of infection associated with vascular insufficiency

Table 3

As a result of an odontogenic infection, osteomyelitis can occur in the maxilla or mandible directly or indirectly. • Direct route: tooth and periodontal tissues. • Indirect route: hematic and lymphatic. [7,8,9] There are multiple etiological factors in osteomyelitis of the jaws. (See box7)

Certain systemic states can predispose to the appearance and negative evolution of this infection, for example, malnutrition, diabetes, leukemia and various types of anemia, as well as certain diseases that are characterized by the formation of avascular bone, which prevent an effective defensive response. Among these, Osteopetrosis is mentioned. (See Figure 1 and 2)

Risks and complications of OM

- Severe pain Regional lymphadenopathy
- Tooth mobility Vincent sign (anesthesia of the corresponding lip)
- Swelling or redness after the infection has penetrated and invaded the periosteum

Endogenous local factors	Exogenous local factors.
Dental: spread through the apices, mainly molars, from pulp lesions due to caries, granulomas, periodontitis, as well as traumatic extractions.	Physical: (Radiotherapy, electro fulguration, electric currents)
Periodontal diseases	Chemicals
Affected mucous membranes	Mummified pulp pasta
Facial Cellulite	Occupational hazard

Table 4

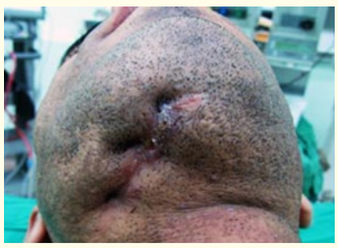

Figure 1: Male patient diagnosed with osteomyelitis resulting from osteopetrosis. Fistulas in the submental and submandibular region are shown. Courtesy of Dr. Juan Guiyermo Sánchez Acuña.

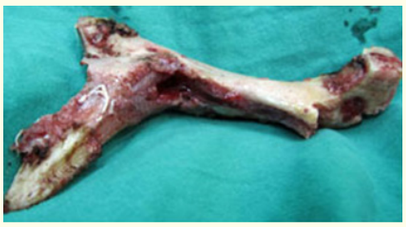

Figure 2: Male patient diagnosed with osteomyelitis resulting from osteopetrosis. The surgical piece is shown. Courtesy of Dr. Juan Guiyermo Sánchez Acuña.

- Leukocytosis
- Hyperthermia
- Limitation of mouth opening invasion of the aponeurotic spaces of the neck
- Rapid destruction of the cortices
- Exudate invading the subperiosteal space
- Local necrosis Cortical resorption
- Purulent exudate due to fistulization
- Sclerosis and bone hyperplasia facial cellulite
- Pathological fractures
- In children, the clinical picture of OM is manifested by symptoms septicemic type (high fever, loss of weight and appetite, weakness and alterations of sleep, which are frequently associated with digestive disorders such as vomiting and diarrhea) Then the local facial and oral characteristics become evident.

Fistulas towards: orbit, nasal fossa and alveolar ridge

Drug treatment is mainly based on prolonged antibiotic therapy, penicillin or amoxicillin as the first choice, although culture and antibiogram are recommended. Duration not less than three months nor more than six months. Along with analgesics, hyperbaric oxygen (inhalation of oxygen under pressure of 2 atmospheres) to increase tissue oxygenation. two and act as a bactericidal [10].

Surgical treatment includes: • Incision and drainage (fistulectomy and sequestrectomy). • Extraction of the causal tooth and removal of granulation tissue. • Establish drainage of the area when performing the extraction or by incising the mucosa or skin at the fluctuating point. • When there are fistulas or swellings at the skin level, incisions are made with the placement of drainage tubes, to facilitate washing with physiological saline. • Sequestrectomy or saucerization (marsupialization).

Paget's disease

Paget's disease of bone (PDB) is the second most common metabolic bone disease, predominantly affecting adult men. Various environmental factors have been described in its etiology and it is strongly conditioned by genetic variants such as sequestoma (SQSTM1). The bone undergoes accelerated, disordered and aberrant turnover. It can manifest in a monoostotic or polyostotic manner. This involvement gives rise to a deformity of the bones with an increase in their size with deformity that can produce bone pain, arthralgia and nerve compression syndromes at the level of cranial nerves, spinal stenosis or compression of the cord. It also produces an increased risk of fracture of the affected long bones [11].

We must not forget that pagetic tissue can undergo neoplastic transformation with a higher incidence of sarcomas, especially in polyostotic cases, which develops in 0.3-1% of cases. In 50-75% of cases it is asymptomatic, and the doctor is alerted when typical deformities appear (enlargement of the skull or bowing of the tibia), or when an elevated alkaline phosphatase is detected in a routine analysis, or a radiological finding in an examination for another reason. The diagnosis of PDB is made in many cases after complications have occurred, and if Paget's is active, bone turnover markers are elevated. Among bone turnover markers, the most useful seem to be the amino-terminal telopeptide of type I collagen, bone-specific alkaline phosphatase, and the amino-terminal propeptide of type I procollagen; However, taking into account its easy determination and low cost, the determination of alkaline phosphatase concentrations is still a valid alternative [11].

The etiology of Paget's bone disease remains unknown, although there are two hypotheses: viral etiology and genetic influence. Since the 1970s, studies have been published suggesting that paramyxoviruses may play an etiopathogenic role. Studies have also been published demonstrating familial clustering of Paget's disease, suggesting a genetic component to this disease [12].

Main risks and complications

- Bone pain bone deformity
- Osteoarthritis Cotyloid protrusion
- Fractures Spinal canal stenosis Hearing loss (the most common neurological condition)
- Cranial nerve deficiencies basilar impression
- Hydrocephalus Spinal canal stenosis
- Congestive heart failure Increased cardiac output
- Aortic stenosis Generalized atherosclerosis
- Endocardial calcification
- Hypercalciuria due to immobilization
- Hypercalcemia
- Hyperuricemia
- Nephrolithiasis
- Sarcoma (osteosarcoma, chondrosarcoma, fibrosarcoma)
- Giant cell tumor

The minimal evaluation of a patient with Paget's disease should include an x-ray of the affected bones and at least one biochemical marker of bone metabolic activity. In most patients, changes in serum total alkaline phosphatase are sufficient to measure disease activity, but the serum alkaline phosphatase level in any patient is a reflection of the total bone surface area affected by the disease and the degree of disease activity in these locations. A bone scan is necessary to define the total extent of the disease and identify the lesions, even asymptomatic, located in the "risk" areas. Specific antipagetic treatment consists of administering a drug capable of inhibiting osteoclasts, it is based on the use of antiresorptives, mainly if the patient presents sensory alterations in dissimilar regions of the body, or neurological syndromes.

In a patient in whom surgery for a pagetic lesion is scheduled, to reduce the risk of hypercalcemia due to prolonged immobilization or due to a very high serum alkaline phosphatase level. slow local progression and reduce the risk of future complications. fibrous dysplasia The term fibrous dysplasia refers to a set of benign bone lesions that are characterized by the replacement of normal bone tissue by connective tissue. The monostotic variant is the most common, accounting for 70% of all cases and in order of frequency it affects the ribs, femur, tibia, maxilla, mandible, skull and humerus. Its most common characteristic is that it develops slowly and increases in volume. In the lower jaw it causes expansion of the vestibular and lingual cortices, and even affects the basal portion. In the case of the upper jaw, the bottom of the vestibular sulcus and the hard palate are affected [12,13].

There are cases in which the maxillary sinus is included and with this, sometimes the floor of the orbit, causing ophthalmic injury. In its expansion, the malar bone, the ethmoid, sphenoid, auditory canals, skull base and frontal bone can be affected. The lesions are duropetreal, and radiographically teeth can be seen displaced by the roots. The polyostotic variant accounts for approximately 30% of cases and its onset is classically described at a younger age than the monostotic variant. In 50% of cases the craniofacial region is affected. More frequently, and in decreasing order: maxilla, mandible, frontal bone, sphenoid bone, ethmoid, parietal and occipital bone, causing expansion and deformity of the affected regions.

It is rare, some authors name it as Jaffe type and in addition, there is talk of another variant that is accompanied by metabolic endocrine disorders known as Mccune Albright Syndrome, patients with this last condition may present brown spots on the skin with milk.

The clinical characteristics of fibrous dysplasia lesions depend mainly on the location and size of the lesion. Therefore, extensive asymptomatic conditions can be evident, and others of small diameter that, because they are generally located in the craniofacial region, can produce functional alterations in the ophthalmic or otorhinolaryngological regions and at the same time affect the aesthetics of the individuals. From a radiographic point of view, fibrous dysplasia lesions are characterized by a ground-glass appearance due to the mixture of bony and fibrous elements. The radiographic density of the lesion will depend on the relative proportion of these elements. Thus they can adopt a sclerotic, cystic (lytic) or mixed pattern [11].

The sclerotic variant constitutes 35% of the cases described and tends to be located at the base of the skull. The mixed variant is the most common (40% of cases), and the cystic pattern is the least common. It should be kept in mind that these lesions should be operated on surgically only in cases with specific indications such as great deformity, or involvement of important structures, to reduce any compression, but the risk of recurrence, necrosis with formation of bone sequestrations. Radiation treatment is not indicated due to the risk of malignant transformation.

Imperfect osteogenesis

Osteogenesis imperfecta (OI) (brittle bone disease, bone fragility, brittle bones) is a congenital disorder in which bones can fracture easily and for no apparent reason. It can also cause weak muscles, brittle teeth, a deviated spine, and hearing loss. OI has a hereditary component. The way the body produces collagen is affected and is evidenced by irregular bone formations, showing up as a variety of osteoporosis. OI is the most common bone fragility disorder with an incidence of approximately 1 in 15,000 [13-15].

The disease affects both men and women. Classically, the pathology was divided into two forms, congenital osteogenesis imperfecta (also known as Vrolik disease) and late osteogenesis imperfecta (also known as Ekman-Lobstein disease). Its original classification is distributed like this: • Type I- mild • Type II-lethal • Type III-progressive deformation • Type IV-lethal This classification system has been generally accepted throughout the world. In 2000, F.H. Glorieux presented a classification of osteogenesis imperfecta, in which, in addition to the known types, he added four more types of OI (V, VI, VII, VIII). In 2016 the INCDS Committee

(International committee of nomenclature of constitutional disorders of the skeleton) reduced the classification to 5 forms, retaining 4 types, which were originally described by Silence and adding a fifth type [16].

This condition can bring with it multiple consequences for those who suffer from it, so professionals must be oriented regarding its evolution. (See table 5)

Severe forms	Mild and moderate forms
Multiple fractures in the fetus and newborn	Bone fragility
Early death due to intracerebral hemorrhages	Osteoporosis
Respiratory infections	Frequent fractures
	Deformities secondary to trauma
	Scoliosis and the basilar impression
	Blue sclerae.
	Dentinogenesis imperfecta
	Recurrent pneumonia
	Heart failure
	Anomalies at the craniocervical junction
	Conductive or sensorineural deafness due to compression of the auditory nerve.

Table 5

The dental professional and related specialties must know these generalities when providing care to these patients, due to the risk of mandibular fracture, dental dislocations, necrosis, infections, among other specific procedures. Furthermore, always keep in mind that on many occasions these individuals are treated with bisphosphonates, so appropriate measures must be taken to avoid any complications added to the disease. Osteopetrosis Osteopetrosis is a heterogeneous group of hereditary entities that consist of an increase in bone mass and density ("bone within bone"), defined as early as 1904 in the description of the German radiologist Albers-Schönberg [16].

The pathogenesis is a failure of bone resorption and an imbalance between osteoclastic and osteoblastic activity that triggers an alteration of remodeling, which generates a lower activity of the macrophage colony stimulating factor (MCSF), interleukin 1 and interleukin 6, which in physiological conditions are necessary for the differentiation of osteoclast precursor cells2; all on a basis of CLCN, CAII and TCIRG gene mutations. Ostropetrotic conditions vary greatly in their presentation and severity, ranging from a neonatal form of onset, with life-threatening complications such as bone marrow failure, to accidental findings of osteopetrosis on radiographs. It is characterized by fractures, short stature, compressive neuropathies, hypocalcemia with tetanic seizures, and life-threatening pancytopenia [16].

The presence of primary neurodegeneration, intellectual deficit, involvement of the immune and skin system or renal tubular acidosis may indicate rarer variants of osteopetrosis, in addition, fundamentally skeletal manifestations such as fractures and osteomyelitis may be evident in childhood or adolescence. The diagnosis is based largely on clinical and radiographic evaluations (See Figure) and must be confirmed by genetic tests when relevant. Once the diagnosis of the primary osteopetrotic condition has been made, it is important to distinguish between the two subtypes. A correct diagnosis is essential to predict and understand the natural history of the disease, providing specific treatments when available, and offering adapted advice on the risk of recurrence and prenatal diagnosis of severe forms [16,17].

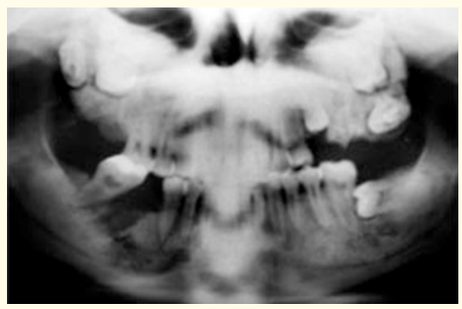

Figure 3: Male patient diagnosed with osteomyelitis resulting from osteopetrosis. Courtesy of Dr. Juan Guiyermo Sánchez Acuña.

It is classified into congenital, juvenile and adult osteopetrosis.

Congenital and juvenile osteopetrosis

- It is considered malignant
- Affects any sex Starts in childhood
- Short stature enlarged
- skull Spontaneous fractures
- Hearing loss and deafness
- Blindness
- Hematological alterations
- Anemia
- leukopenia
- thrombocytopenia
- Delayed eruption
- hepatosplenomegaly
- malocclusion
- Risk of maxillary fracture during surgical procedure bone infections
- Formation of bone sequestrations
- Delayed tooth eruption,
- malformed crowns and roots, decreased alveolar bone growth, impacted primary and permanent teeth in both the maxilla and mandible.

Adult osteopetrosis

- It is more benign It can cause jaw fractures and osteomyelitis (See Figure)
- Flattening of the midface
- Not very prominent zygomatic arches
- Mandibular prognathism resulting in a Class III Malocclusion
- Compressed and underdeveloped maxillary arch
- Abnormal thickening of the periodontal membrane

Cherubism

Cherubism is a skeletal dysplasia characterized by bilateral and symmetrical fibro-osseous lesions limited to the mandible and maxilla that, in most cases, have dominant mutations of the SH3BP2 gene located on chromosome 4p16.3. Bone lesions by clinical and radiological behavior are quiescent, non-aggressive and aggressive. There are multiple conditions that this disease can cause.

- Multilocular radiolucent lesions in the mandible or maxilla between 2 and 7 years.
- Submandibular and cervical lymphadenomas.
- Affect the eruption of permanent molars
- Zygomatic arch and condyles affected.
- Eyeball condition Extracranial injuries (rare in ribs)
- Craniosynostosis and deviation of the fingers
- Retrobulbar lesions with proptosis and optic nerve lesions
- Vision loss, macular striae, optic neuropathy, and scarring tongue displacement
- Airway obstruction Obstructive sleep apnea Disruption with absence of molars
- Dental malformations
- Bilateral and symmetrical volume increase in the cheeks above the mandibular angle
- Buccolingual widening of the alveolar ridge
- Tuberosity deformity
- May be associated with Noonan syndrome (mental retardation, skeletal disorders, cardiovascular conditions, and craniofacial deformities)
- Dental retention or oligodontia
- V-shaped palate [16,18,19].

Moderate forms of cherubism without facial deformity, dental and ocular anomalies do not require treatment and return spontaneously after 7 years of age. Clinical-radiographic follow-up should be performed 2-5 years after quiescence. In some cases they last up to 20 years, and interventions can be performed for aesthetic purposes, but explaining to the affected person and their family the possibility of worsening the situation instead of improving it. When there is airway obstruction, severe ophthalmological or facial involvement, surgical intervention is indicated, which includes partial or contour resection, curettage or a combination of both. These procedures must be done after quiescence. Only anesthetic or functional complications justify intervention before adolescence. The development of the dentition must be closely monitored due to the danger of lytic lesions in the eruption of the secondary dentition. Autotransplantation of teeth that erupt ectopic can be performed. In grade II, surgical curettage has been chosen, which results in interruption of the growth of any abnormal tissue that remains [16].

Radiation therapy is contraindicated as it produces osteoradionecrosis and increases the incidence of malignancy (osteosarcoma). Treatment with salmon calcitonin nasal spray daily for 15 months has been suggested with regression of the lesions, although there is a report of a case with exacerbation of the lesions. These cases have also been treated with interferon in combination with resection in the proliferation phase, but requires further investigation, as do proinflammatory cytokines and bisphosphonates. Because genetic mutations occur in this disease, it is expected that genetic medicine will play an important role in the treatment of these patients. Marfan syndrome Marfan syndrome is a genetic disease with an autosomal dominant inheritance pattern, which compromises the connective tissue systemically, and with a high degree of clinical variability [19,20].

Typical manifestations are those involving the ocular, skeletal, and cardiovascular systems. The skeletal system is characterized by bone overgrowth and joint hypermobility. the limbs are disproportionately long with respect to the size of the trunk (dolichostenomelia). Accelerated rib growth can lead to pectus excavatum or pectus carinatum. Scoliosis is common and can range from mild to severe and progressive. The highest degree of morbidity and early mortality is given by cardiovascular disease.

Manifestations include: dilation of the aorta at the level of the sinuses of valsalva, with a predisposition to dissection and rupture, mitral valve prolapse with or without regurgitation; tricuspid valve prolapse and proximal pulmonary artery enlargement. It affects the buccomaxillofacial complex mainly in the temporomandibular joint, causing laxity and exaggerated mobility. In addition, multiple patients have been seen with high-arched palates, which sometimes require placement of palatal expanders.

Malocclusion associated with posterior crossbite, cleft palate and bifid uvula, and mandibular prognathism have been diagnosed. Patients with Marfan Syndrome have medical conditions that can make dental treatment difficult. These conditions create a predisposition to dental caries, periodontal disease and malocclusions. Orofacial disorders must be treated early to prevent future complications.

Achondroplasia

Achondroplasia is the most common non-lethal skeletal dysplasia. Its general incidence varies between 1 in 10,000 and 1 in 30,000. live births. Achondroplasia is caused by mutations in the FGFR3 gene. This gene provides instructions for making a protein that is involved in the development and maintenance of bone and brain tissue. Two specific mutations in the FGFR3 gene are responsible for almost all cases of achondroplasia. Researchers believe that these mutations cause the FGFR3 protein to be overly active, which interferes with skeletal development and results in the alterations in bone growth seen in achondroplasia.

Clinical features

- Short stature: Those affected with achondroplasia have short stature.
- The average height of adult men is 131 ± 5.6 cm and of adult women is 124 ± 5.9 cm.
- Disproportionately large head
- Facial dimorphism with depressed "saddle" nose and prominent forehead
- Abnormally accentuated curvatures of the spine such as kyphosis and lumbar lordosis
- Normal-sized trunk with short, arched arms and legs
- Small hands with trident-shaped fingers
- Characteristic alterations seen on radiography as abnormalities of the vertebral bodies and long bones.
- Discrete hypotonia
- Motor development a little slow at the beginning but then normalizes
- Usually normal intelligence
- Glucose intolerance and hydrocephalus secondary to narrowing of the opening where the spinal cord enters the head (foramen magnum)
- Spinal complications
- Brachiocephalic skull
- Mandibular prognathism relative to retruded maxilla
- Malocclusion and oligodontia

Currently, the morbidity due to achondroplasia is well known, but the mortality associated with this entity is still somewhat confusing. Mortality during the first year of life is high. A higher risk of neonatal death has also been detected in achondroplastics, although it is not clear whether this is due to a high incidence of homozygous achondroplastic fetuses or other complications during childbirth. Infantile cortical hyperostosis Infantile cortical hyperostosis, also called Caffey disease, Caffey-Silverman, familial infantile cortical hyperostosis or sporadic infantile cortical hyperostosis. It has a low prevalence, probably due to its underdiagnosis. It has no sex predilection and can affect any race. Its incidence is of 48/100,000. It is a self-limiting disease, almost exclusive to infants, although it has been described in older children and can follow a relapsing course that ends before 3 years of age [20-22].

The average age of onset is between one month and 2 months of age, although neonatal and even intrauterine cases have been described, as well as later cases, but almost always onset before 5-6 months of age. age. It usually begins abruptly as a painful swelling of the affected bone or bones, which extends to adjacent tissues, accompanied by crying, irritability, moderate or high fever, refusal of food or change in character. It is a rare bone disease characterized by hyperostosis of the cortex of the affected bone, usually the jaw, clavicle or ulna. There may be hematological alterations such as anemia, accelerated erythrocyte sedimentation rate, and leukocytosis. There is no treatment for this disease; depending on the case, antibiotics, steroids or NSAIDs can be used.

Massive osteolysis

Massive osteolysis (evanescent bone disease, phantom bone disease or Gorham-Stout syndrome) is a rare disorder of uncertain pathogenesis associated with angiomatous vascular proliferation, uncertain prognosis and whose management includes several modalities. Due to its rarity, early diagnosis is difficult and is sometimes suspected when a pathological fracture occurs and bone healing is delayed or does not occur. There is a progressive and spontaneous bone resorption of one or several adjacent bones around a focus, without respecting the joint limits. It can affect any part of the skeleton, in the orofacial complex it mainly affects the jaw. There is no associated family history and the age of presentation is usually in adolescents and young adults. Bone regeneration does not occur, even if osteolytic progression ceases. Multiple simultaneous non-contiguous foci or distant metastases have not been described. The pathology is considered benign and bone lysis usually stops after a few years. The diagnosis is one of exclusion based on the clinical-radiological evolution and compatible histological findings [16,23-25].

The evolutionary course is unpredictable, with up to 16% fatality. Radiographically, radiolucency can be observed at the beginning and limited areas with imprecise borders and a sclerotic area. As the condition develops, the absence of bone tissue becomes evident in all the internal and external layers of the bone, surrounded by bone condensation instead of periosteum. There is no specific or effective treatment for this noxa; radiation, surgical techniques with bone grafts, among other procedures, have been applied.

Ossifying fibroma Ossifying fibroma (osteofibroma, fibrosteoma) is a rare type of tumor characterized by the proliferation of fibrous tissue with the formation of hard tissue similar to cementum or bone. They are benign neoplasms characterized by the replacement of normal bone by a fibrous cellular stroma containing foci of mineralized bone trabeculae and cement-like material that varies in quantity and appearance. They show progressive proliferative capabilities with bone expansion and well-defined radiological margins. It is a rare, well-defined monostotic tumor. The etiology of this type of fibroma is unknown, but odontogenic, developmental, and traumatic origins have been suggested. It develops from the multipotent mesenchymal cells of the periodontal ligament that can form both bone and cementum. Recent genetic studies have revealed a mutation in the tumor suppressor gene HRPT2, a protein product known as parafibronin that leads to tumor formation. They occur most frequently in the posterior region of the mandible and can also occur in the maxilla, commonly in the region of the canine fossa and in the area of the zygomatic arch. They are more common in women, and have a higher incidence in the third and fourth decades of life. Occasionally it can cause deformities and shifting of the teeth [26].

Most are not painful and the lesion is generally asymptomatic until growth produces pain, paresthesia, and facial asymmetry.

Figure 4: Female patient diagnosed with ossifying fibroma. Courtesy of Dr. Arelis Rabelo Castillo. Kitui Country Referral Hospital, Kenya.

When the extension of the tumor mass reaches the ramus of the mandible and affects the lower border, it can cause paresthesia of the inferior alveolar nerve. In the maxilla it causes cortical expansion with obliteration of the gingivobuccal sulcus, extension towards the nasal cavity and orbital floor leading to epistaxis and epiphora. (View Figure 5)

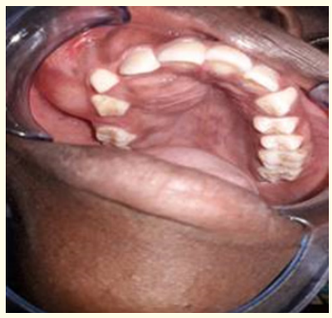

Figure 5: Female patient diagnosed with ossifying fibroma. Courtesy of Dr. Arelis Rabelo Castillo. Kitui Country Referral Hospital, Kenya.

Conventional x-rays and imaging techniques such as computed axial tomography help differentiate this lesion from other fibro-osseous lesions. (View Figure 6).

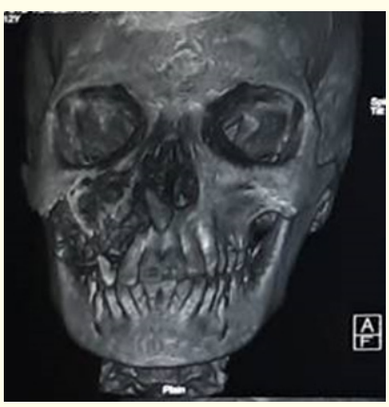

Figure 6: Computed axial tomography with expansive and destructive Figure of the tumor, in addition to the tooth germ belonging to the intraosseous upper right canine. Courtesy of Dr. Arelis Rabelo Castillo. Kitui Country Referral Hospital, Kenya.

Lesions can be unilocular or multilocular. In the early stages, it appears as a radiolucent lesion. With increasing maturity of the tumor, radiopaque masses appear. Radiologically, the lesion presents different stages of development with a centrifugal growth pattern. The premolar and molar regions of the mandible are the most common sites. Small lesions are asymptomatic and, as they grow and expand, cause painless swelling, despite significant facial asymmetry [26].

Pain and paresthesia are rarely associated with ossifying fibromas. Mobility and root resorption of the teeth involved are common findings and root divergence can be found in 17% of cases. Treatment involves performing bloc resections and grafts in some cases. Many of these lesions present recurrences (one in five), so surgical removal must be complete and include safety margins, rather than conservative curettage. Central fibroma of the jaws Central ossifying fibroma (FOC) is a benign bone tumor, which is included within the so-called fibro-osseous lesions, it is rarer than soft tissue fibroma [26].

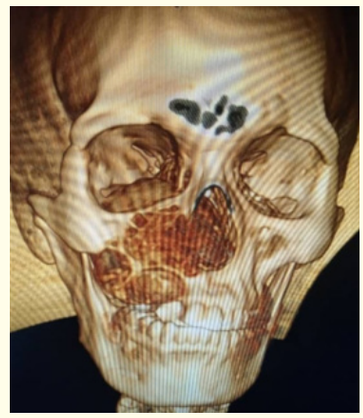

Figure 7: 3D computed axial tomography, showing a patient with a diagnosis of central ossifying fibroma of the maxilla that extends to the antral, nasal and zygomatic-orbital regions. Courtesy of Dr. Carlos Juan Puig González.

It does not have osteogenic potential, it occurs mainly in children and young people. It is generally asymptomatic and invades the mandible and maxilla in areas close to the maxillary antrum. Treatment of FOC includes surgical removal and curettage of the bone bed with an extent that depends on the size and location of the lesion. The prognosis is generally favorable, with almost no evidence of malignant transformation. Juvenile ossifying fibroma Juvenile ossifying fibroma (aggressive ossifying fibroma, psamamoid ossifying fibroma) is a benign neoplasm of a slightly fibroosseous nature. common that occurs in the facial bones of the oral complex. Although it is called a juvenile fibroma due to its frequency in that age period, it is not exclusive of them. There are two variants due to the histopathological findings: trabecular and psammomatoid [26-28].

The largest percentage presents rapid and aggressive growth that produces erosion and destruction of bone structures. They

predominate in the maxilla and mandible, with a tendency to invade other regions such as ethmoid, nasal bones, ophthalmic-orbital region, and skull base. (View Figure 8).

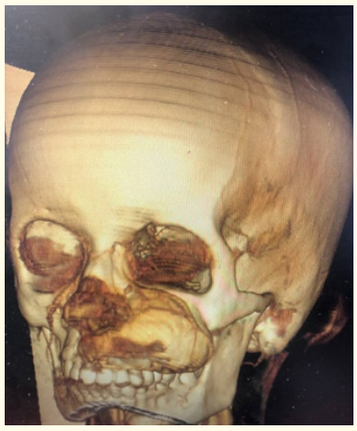

Figure 8: 3D computed axial tomography, showing a patient diagnosed with juvenile ossifying fibroma in the maxillary region that extends to the nasal and zygomatic-orbital regions. Courtesy of Dr. Carlos Juan Puig González.

Radiographically, 50% of cases show radiolucent lesions that contain mineralized areas, but can also be mixed or radiopaque in Depending on the degree of calcification, it can also sometimes show an aggressive pattern with bone destruction reminiscent of a malignant neoplasm. The treatment is surgical with safety margins, without performing curettage or prescribing radiotherapy. Osteoma Osteomas are benign neoplasms, made up of adult compact bone (eburnous) and spongy bone (trabecular or reticular), and characterized by slow, continuous and asymptomatic growth. They are classified into three forms: central, peripheral and extra skeletal. Most osteomas are detected in young adults (15-50 years) and are generally asymptomatic solitary lesions until they cause asymmetry or compression of adjacent structures. There is little information on whether there is any gender predilection. Osteomas may arise from bone graft sites or may be multiple in patients with Gardner syndrome. The most typical thing is that the tumor presents as a painless lump that expands slowly and although its growth potential is irregular [28,29].

The osteoma is generally observed radiographically well circumscribed and radiopaque, a mass with a density similar to that of normal bone is evident, always depending on the characteristics of the bone tissue, whether it is compact or spongy. It shows a well-defined oval shape with a wide base. The etiology is not yet fully clarified; the criteria focus on whether it is a true neoplasia or a reactive lesion. It is considered that various factors could be involved in the pathogenesis, a link with trauma and the muscle traction force towards the periosteum is proposed, which induces osteogenic reactions; reactions to infectious processes are also not ruled out.

The compact osteoma is more evident in the female sex and the compact one in the male. It has been diagnosed mainly in the area of the ascending ramus of the mandible, near the angle, although these criteria vary somewhat among researchers. In general, peripheral osteomas of the craniofacial region are discovered accidentally by routine radiographic examinations due to the fact that they are asymptomatic. Occasionally, the size of the tumor can cause facial deformity due to an increase in volume or cause some detrimental effect on the patient's functional area. It occurs most frequently in the paranasal sinuses. Other locations include external auditory canal, orbit, temporal bone, and pterygoid processes. It is more common in the mandible than in the maxilla. Cases related to the mandible have a predilection for the region of the mandibular angle on its lower border or condyle, followed by the molar areas of the body on its lingual aspect and mandibular ascending ramus [26-29].

Intraosseous is more complex to identify due to the long period that must pass before it causes asymmetry; it is also detected most of the time in a routine radiographic examination. The treatment is surgical with a favorable prognosis. Osteoblastoma Osteoblastoma (OB) is a benign bone tumor rare, accounting for 1% of all primary bone tumors; It commonly occurs in the spine and long bones. Approximately 15% of osteoblastomas occur in the maxillofacial skeleton and are most common in the mandibular region. The size of the lesion is between two and four centimeters, sometimes painful and not relieved with analgesics (aspirin), as happens with osteoid osteoma. It is observed radiographically as a well-contoured radiolucent lesion with sclerotic areas that tend to delimit calcifications [26-30].

The treatment is surgical with a safety margin to avoid recurrence, thus giving a favorable prognosis. Osteoid osteoma Osteoid osteoma is a very painful, benign bone tumor that consists of the proliferation of immature osteoblasts. It does not have great growth potential and its size rarely exceeds 1 cm. It occurs more frequently in long bones, especially in the femur, with a frequency of 25%. It is uncommon in the maxillofacial region, but it can occur in the jaw, far from the dental structures. When it is close to the periphery, the skin presents painful symptoms. The treatment is surgical and tumor recurrence is rare when the resection is complete with a favorable prognosis. Many authors describe it as a synonym for osteoblastoma. In the following box we show how the differential diagnosis can be made [26, 28-30].

Characteristics	Osteoblastoma	Osteoid osteoma
Age of diagnosis	90% before the age of 30	5 and 25 years
Sex	Female	Male
Size	2 to 4 centimeters	Less than 1cm in diameter
Pain	Not relieved by pain relievers (aspirin)	It is relieved with NSAIDs (aspirin) More intense during the night
Origin	Arises from the medullary bone	Arises from cortical bone

Table 6

Bone exostoses of the jaws Torus and exostosis Toruses are abnormal, benign proliferations of mature bone tissue that appear in both the maxilla (torus palatine) and the mandible (torus mandibularis), producing a protuberance on these surfaces. They have been able to be classified according to their size, location and number. The small torus reach sizes no larger than 3mm, while the medium ones range from 3 to 5 mm. The large ones, for their part, will be characterized by reaching sizes greater than 5 mm (Seah, 1995). Palatine torus The palatine torus constitute slowly growing bony protuberances, whose base is flat. They can be observed in the midline of the hard palate, rising at the margins of the palatine process at the level of the middle suture of the palate, compromising both sides of said suture (Shafer and Levi, 1987).

Its cause is still undetermined, although some authors attribute a genetic origin to it. Although it is not life-threatening, some cases require surgical intervention. They can be flat; fusiform, nodular and lobular. They are characterized by being lobulated, multilobed, flat, lobular, nodular or use-shaped.

The palatine torus can be demonstrated on an occlusal radiograph, in which film an oval-shaped opacity located in the midline is observed. Among the difficulties that it can bring are: • Difficulty phonation • Repeated chewing trauma • Ulcer that does not heal in the mucosa that covers it • They can be used as a bone graft in another area that needs it • Affect aesthetics • Related to infectious processes such as osteomyelitis • Difficult for prosthetic rehabilitation (Figure 9).

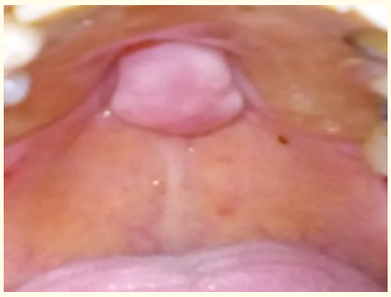

Figure 9: Patient whose torus palatine did not allow the prosthesis to adjust properly and frequently suffered from traumatic ulcers. Courtesy of Dr. Otto Alemán Miranda.

With respect to the surgical procedure, the main risks are those of anesthesia techniques, and in the intraoperative period the main ones are the creation of an oral-nasal or sinus communication, due to uncontrolled movement, injury to adjacent structures, tears, injury to the arteries. palatine, fall of a bone fragment into the airway, damage to the soft palate, injury to the posterior wall of the oropharynx, section of the salivary gland ducts. In addition, postsurgical infection must be prevented. mandibular torus Mandibular tori are observed on the lingual surface of the mandible in the area of the premolars (Shafer and Levi, 1986) and multiple exostoses are observed on the buccal surface of the maxilla and mandible below the mucobuccal fold in the molar region [26-30].

Like its palatine counterpart, it constitutes a non-neoplastic bony protuberance, formed by mature bone tissue. They can be planes that appear as a smooth symmetrical convexity and broad base; The fusiform ones are more pronounced and sometimes with a groove in the midline, the nodular ones have several protuberances with individual bases and the lobar ones have a broad and common base for the different lobes.

Other authors classify it as unilobed, multilobed, flat, lobular and in the form of use. Radiographically, it is seen as a radiopaque area near the dental roots. The professional can rely on an occlusal or periapical x-ray. Surgical treatment is indicated when: • Difficulty phonation • Repeated chewing trauma • Ulcer that does not heal in the mucosa that covers it • They can be used as a bone graft in another area that needs it • Affect aesthetics • Related to infectious processes such as osteomyelitis • Difficult for prosthetic rehabilitation. With respect to the surgical procedure, the main risks are those of anesthesia techniques, and in the intraoperative period the main ones are injury to adjacent dental structures, tears, injury to the lingual arteries, fall of a bone fragment into the airway, damage to the soft palate, injury to the posterior wall of the oropharynx, section of the salivary gland ducts, affection of the dental and lingual nerve, injury to the floor of the mouth or tongue. In addition, postsurgical infection must be prevented.

bone exostoses They are common in the jaws, mainly in males, and should not be confused with post-surgical bone irregularities. Depending on the location, they may cause difficulties with phonation, chewing, aesthetic problems, and inability to place the dental prosthesis. They are uncomfortable for the patient, they generally occur in the vestibular or tuberosity (See Figures 10-20)

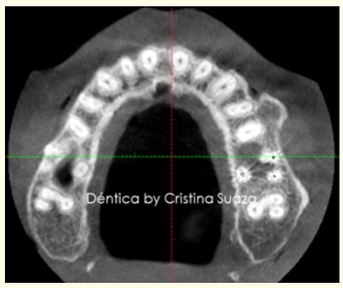

Figure 10: Computed axial tomography showing bone exostoses on the buccal side of the upper jaw and towards the tuberosity. Courtesy of Dr. Eliana Cristina Suaza Ortiz.

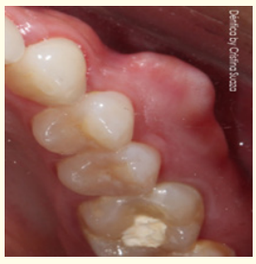

Figure 11: Patient with buccal bone exostoses of the upper jaw. Courtesy of Dr. Eliana Cristina Suaza Ortiz.

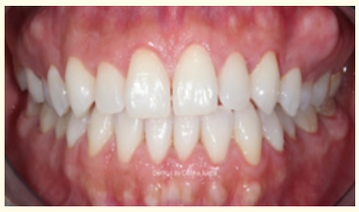

Figure 12: Patient with multiple bone exostoses in the jaws. Courtesy of Dr. Eliana Cristina Suaza Ortiz.

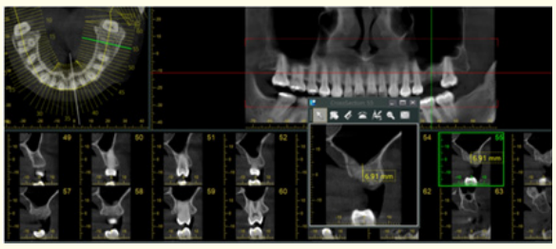

Figure 13: Computed axial tomography showing bone exostoses towards the tuberosity. Courtesy of Dr. Eliana Cristina Suaza Ortiz. Malignant lesions in bone tissues. These constitute a high group of injuries that cause major deformities and sequelae to affected patients. In the case of head and neck neoplasms, the risks and complications vary by region and were explained in the chapter on oral cancer. Here we show an Figure of a patient with osteosarcoma, the most common malignant neoplasm in the jaw bones after squamous cell carcinoma.

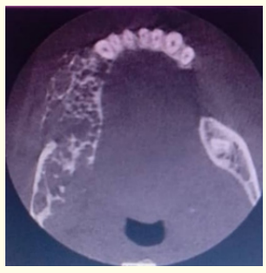

Figure 14: Computed axial tomography. Shows an Figure of a jaw osteosarcoma. Courtesy of Raquel Jacqueline Sánchez.

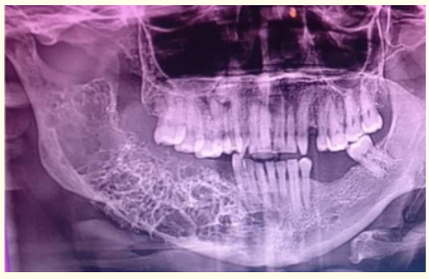

Figure 15: Panoramic radiography. Shows an Figure of a jaw osteosarcoma. Courtesy of Raquel Jacqueline Sánchez.

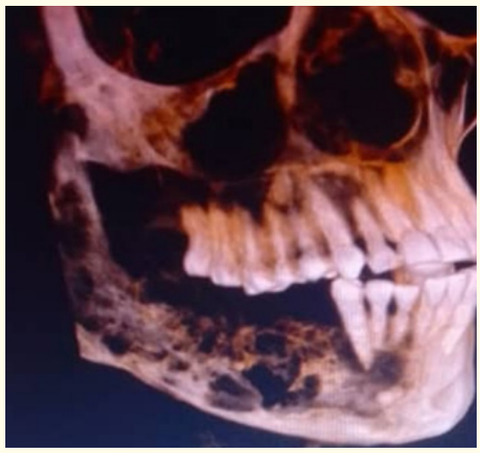

Figure 16: Computed axial tomography. Shows 3D Figure of a jaw osteosarcoma. Courtesy of Raquel Jacqueline Sánchez.

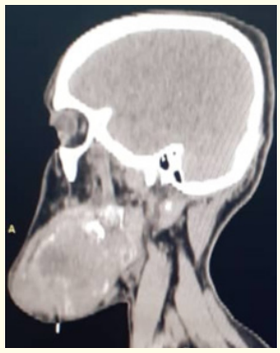

Figure 17: Tomography of a patient with a diagnosis of Osteosarcoma in the mandibular region. Courtesy of Dr. Carlos Juan Puig González.

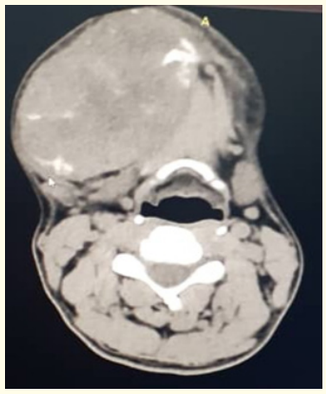

Figure 18: Tomography of a patient with a diagnosis of Osteosarcoma in the mandibular region. Courtesy of Dr. Carlos Juan Puig González.

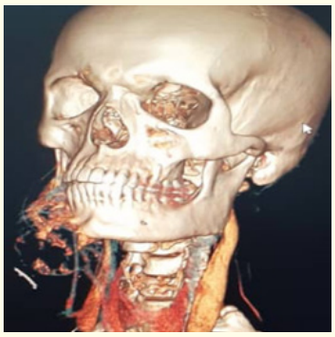

Figure 19: 3D computed axial tomography of a patient diagnosed with Osteosarcoma in the mandibular region. Courtesy of Dr. Carlos Juan Puig González.

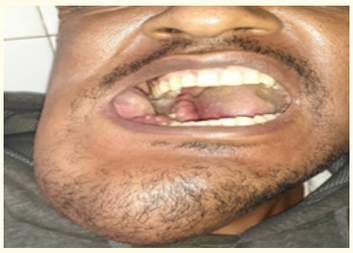

Figure 20: The patient with a diagnosis of Osteosarcoma in the mandibular region is shown clinically. Courtesy of Dr. Carlos Juan Puig González.

Conclusion

The main complications and risks of bone conditions in the oromaxillofacial complex, as well as their sequelae, were described. Based on an exhaustive review of the literature, as well as the author's previous experience. These injuries have a high international morbidity rate and cause multiple physical, mental and social conditions.

Acknowledgements

To my wife for all her unconditional professional and personal support.

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