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Bone Pathologies Of The Jaw: A Comprehensive Review

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ABSTRACT

Bone pathologies of the jaw are a diverse group of conditions that can significantly impact oral health and quality of life. This review provides an overview of the etiology, diagnosis, and management of various bone pathologies affecting the jaw, including osteogenesis imperfecta, cleidocranial dysplasia, and other rare conditions. We discuss the clinical and radiological features of each condition, as well as current treatment options and future directions for research. This comprehensive review aims to provide clinicians and researchers with a thorough understanding of bone pathologies of the jaw, facilitating accurate diagnosis and effective management of these complex conditions.

Keywords : Bone Pathologies, Clinical features, Radiographic features, Histopathological features, Treatment.

INTRODUCTION

The jawbones, comprising the mandible and maxilla, form the foundation of the facial structure and play a vital role in oral functions such as mastication, speech, and respiration. However, various bone pathologies can affect the jaws, leading to alterations in their shape, size, and function. These conditions can be congenital or acquired, and their severity can range from mild to severe, impacting not only oral health but also overall quality of life.

Bone pathologies of the jaws can be categorized into several groups, including genetic disorders, inflammatory conditions, benign and malignant tumors, and metabolic bone diseases. Accurate diagnosis and timely management of these conditions are crucial to prevent complications, ensure optimal oral function, and improve patient outcomes. This chapter aims to provide a comprehensive overview of the various bone pathologies affecting the jaws, their clinical and radiological features, and current treatment options.

BONE PATHOLOGIES

OSTEOGENESIS IMPERFECTA

Osteogenesis imperfecta (OI) is a rare genetic condition characterized by fragile bones and skeletal abnormalities.Osteogenesis imperfecta (OI) encompasses a diverse group of inherited disorders, primarily autosomal dominant conditions caused by mutations in the COL1A1 and COL1A2 genes. These genes are responsible for producing type I collagen, a crucial component of connective tissues, particularly in bones, teeth, and skin. Mutations in these genes can lead to either reduced collagen production or structurally flawed collagen, resulting in a more severe skeletal phenotype[1].

Clinical features

Defects in type 1 collagen secretion impede osteoid production, disrupting both endochondral and intramembranous bone formation. As a result, individuals with Osteogenesis Imperfecta (OI) primarily experience bone fragility, leading to:Delayed growth, Recurrent fractures throughout life, Joint laxity, Blue-tinged sclera, Hearing loss.Additional complications may include heart valve problems and aneurysms.Notably, OI does not affect cognitive function. Therefore, treatment and care should be tailored to the individual's age, rather than their height.[1]

Oral manifestations

Abnormal bone growth in individuals with Osteogenesis Imperfecta (OI) can lead to:Underdeveloped upper jaw (maxillary hypoplasia)Bite problems, such as:Angle Class III malocclusion, Anterior crossbite.Additionally, dental development is often affected, resulting in Dentinogenesis imperfecta (DI): abnormal dentin structure,Variations in tooth Number, Shape, Position.These dental alterations are common features of OI, further complicating the oral health of affected individuals.

Radiographic features

In Osteogenesis Imperfecta (OI) patients, the primary dental alteration visible on radiographic examination is dentinogenesis imperfecta, characterized by Partial or complete closure of the pulp chamber and root canals, Shortened and narrowed roots. These distinctive features are hallmarks of dentinogenesis imperfecta in OI patients[2].

Histopathological features

The severity of Osteogenesis Imperfecta (OI) can be characterized by distinct bone tissue features. Severe forms are seen Disorganized trabecular pattern,Crowded osteocytes due to impaired collagen production, extensive areas of woven bone. Less severe forms like Crowded osteocytes, Thin lamellar bone are seen. These histological features reflect the varying degrees of bone tissue disruption in OI.

Treatment

Currently, there is no known treatment for osteogenesis imperfecta. Management of the condition is primarily focused on addressing complications, such as infections, as they arise. The outlook for individuals with osteogenesis imperfecta varies widely, ranging from relatively favorable to very poor[2].

OSTEOPETROSIS

Osteopetrosis (OP), also known as Albers-Schonberg disease or marble bone disease, is a rare inherited disorder characterized by Impaired osteoclast function, Reduced bone resorption, Decreased bone turnover, Accumulation of immature bone tissue. The incidence of osteopetrosis is estimated to be 1 in 100,000 to 500,000 individuals.

Clinical features

Osteopetrosis (OP) presents with varied and severe clinical manifestations, depending on the form and stage of the disease. The autosomal recessive form, also known as malignant infantile OP, is a life-threatening condition that typically begins within the first few months of life.Characteristics of this severe form include Anemia, Hepatosplenomegaly, Increased susceptibility to Fractures. Craniofacial skeletal changes, leading to Frontal bossing, Cranial nerve damage due to narrowed foramina, causing Deafness, Visual disturbances,Facial palsy, Malformed paranasal sinuses, leading to respiratory issues and stuffy nose[3].

Oral manifestations

Osteopetrosis (OP) can lead to several dental complications, including Enamel hypoplasia, malformed tooth crowns and short roots, Embedded teeth, Poor oral hygiene, Increased risk of dental caries. Dental radiographs typically reveal Constriction of the inferior alveolar canal and dental pulp canal, Thickening of the lamina dura, Reduced visibility of bone marrow cavities and dental pulp chambers due to increased bone density[3].

Treatment

Due to compromised blood circulation, surgical interventions like sequestrectomy, tooth extraction, and free bone grafting should be approached cautiously. Instead, oral rehabilitation using obturator prostheses in the maxilla is the preferred method for managing the defect[4,5]

CLEIDOCRANIAL DYSPLASIA

Cleidocranial dysplasia (CCD) is a rare genetic disorder that affects approximately 1 in 1 million live births. It is characterized by distinct physical features that are often diagnostically distinctive, making it easily identifiable. As an autosomal dominant condition, CCD can be inherited from a single parent carrying the mutated gene [3].

Clinical features

A general physical examination revealed a range of distinctive characteristics. The individual had short stature, a thin and lean build, and exhibited slurred speech and drooping shoulders that could be easily brought forward. The skull was notably large and globular in shape, with prominent frontal, parietal, and occipital bossing, characteristic of an "Arnold head." Additional facial features included a depressed nasal bone, widely spaced eyes (hypertelorism), underdeveloped mid-face, and a protruding lower jaw (mandibular prognathism) [6].

Oral manifestations

The dental manifestations of this condition are numerous and varied. They include an underdeveloped maxilla, relative mandibular prognathism, and a range of tooth-related issues such as retained primary dentition, multiple impacted permanent teeth, delayed eruption of permanent teeth, and supernumerary teeth. Additionally, abnormalities in tooth crown and root formation are common, and crypt formation often occurs around impacted teeth. A high-arched palate is also a characteristic feature [6].

Radiographic features

Radiological examination reveals several characteristic features. The cranium shows a widely patent anterior fontanel and sutures, accompanied by wormian bones. The clavicles are typically fragmented, with the middle portion often deficient, and these changes can be asymmetric. Additionally, there is a notable delay in the ossification of pelvic bones, particularly the pubic and ischial bones. Spina bifida occulta is also commonly observed in the cervical and upper thoracic regions. Furthermore, the hands and feet exhibit various anomalies, including shortening and broadening of the carpal, metacarpal, tarsal, and metatarsal bones [3].

Treatment

There is no specific treatment for cleidocranial dysplasia. However, managing oral health is crucial. Retained deciduous teeth should be restored if they decay, as extracting them does not guarantee the eruption of permanent teeth.Life expentency is normal [3].

IDIOPATHIC OSTEOSCLEROSIS

Idiopathic Osteosclerosis is a condition also referred to by several other names, including Dense bone islands, Enostoses, Bone scar, Bone whorl, Focal periapical osteopetrosis.However, Idiopathic Osteosclerosis is the preferred term as it accurately describes the condition: a benign, focal growth of compact bone characterized by increased radiodensity within the cancellous bone, without cortical expansion.

Clinical features

Idiopathic osteosclerosis is usually asymptomatic, discovered as an incidental finding on OPG or cone-beam or dental CT.

Etiology and Nature

Idiopathic osteosclerosis has an unknown cause and is not associated with any infection or systemic disease. It is considered a developmental variation of normal bone architecture, potentially resulting from Altered bone turnover or maturation, Excessive osseous deposition, Retained primary root fragments, Unusual occlusal forces. Idiopathic osteosclerosis is most commonly found (90%) in the mandible, specifically near the First molar, Premolar region. The lesion can be Periapical (80%): associated with the roots of teeth and Remote (20%): separated from teeth by normal trabecular bone. In all cases, the periodontal space and lamina dura remain normal [7].

Radiographic features

Idiopathic osteosclerosis is characterized by a well-defined, homogeneous, radiopaque lesion of dense bone in the mandible. Its key radiographic features include Solitary or multiple lesions, Variable size: ranging from 2-3 mm to 1-2 cm, Diverse shape: round, elliptical, or irregular, Sharp margins: typically without a radiolucent

rim, Occasional peripheral spiculation.Overall, idiopathic osteosclerosis presents as a distinct, dense bone lesion with clear boundaries, making it identifiable on radiographs [7].

Treatment

Idiopathic osteosclerosis is generally a benign condition with no significant clinical implications. However, in rare cases, it may lead to Tooth displacement,External root resorption, Impaired tooth eruption, Interference with orthodontic tooth movement (e.g., inclination of teeth).Due to its benign nature, it is essential to Confirm the diagnosis through follow-up evaluations to differentiate it from other pathologies caused by inflammatory processes or systemic diseases.Avoid unnecessary biopsies by ensuring an accurate clinical diagnosis [3].

GORHAM – STOUT SYNDROME

Gorham's disease, also known as "Phantom bone disease" or "Disappearing bone disease," is a rare condition characterized by the spontaneous and progressive resorption of bone, leading to its complete disappearance. Typically affecting children and young adults, this condition may be linked to a type of bone hemangioma. The affected bones undergo dissolution, often accompanied by an abnormal proliferation of blood vessels. Although the condition is self-limiting, its progression is unpredictable, and it is not inherited genetically.

Clinical Features

Massive osteolysis typically affects older children, young adults, and middle-aged individuals, with equal distribution between males and females. Interestingly, about half of the patients recall experiencing a traumatic event, often minor, before diagnosis. While a single bone is usually affected, some cases involve multiple bones (polyostotic). The most commonly affected bones include Clavicle, Scapula. The disease often begins suddenly, progressing rapidly, and may or may not be painful. As the condition advances, the affected bone is replaced by a thin layer of fibrous tissue surrounding a cavity. Notably, laboratory test results are typically normal [3].

Oral Manifestations:

Gorham's disease can affect the jawbone (mandible) and other facial bones. Review of reported cases by Ellis and Adams, as well as Murphy and colleagues, reveals only two cases resulted in complete destruction of the mandible.At least three cases involved concurrent damage to the maxilla (upper jawbone).Common symptoms include pain and facial asymmetry.Notably, facial asymmetry is a consistent finding in patients with Gorham's disease affecting the oral and facial bones [3].

Histopathological features

Histopathologically, the disease is characterized by a benign growth of endothelial channels within the bone, resulting in extreme thinning of bony trabeculae. This process triggers osteoclast-mediated resorption, leading to the replacement of bone with fibrous tissue. According to Gorham and Stout's theory, the proliferation of hemangiomatosis causes hyperemia, which in turn leads to excessive bone destruction [8].

Pathogenesis:

The exact cause of Gorham's disease remains unclear. While osteoclasts (bone-resorbing cells) are often present, most experts agree that increased osteoclastic activity is not the primary driver of the disease. Notably, osteoclasts are frequently absent in areas of active bone resorption.

Treatment and Prognosis:

There is no specific treatment for Gorham's disease. However Radiation therapy has shown benefits in some cases.Surgical resection has halted disease progression in others. Without treatment, the disease often leads to complete destruction of the affected bone [3].

PAGET'S DISEASE

Paget's disease is a prevalent condition characterized by abnormal and excessive bone remodeling, typically affecting middle-aged and elderly individuals. This disorder leads to the formation of bones that aremExtensively vascularized,Weak, Enlarged, Deformed. Consequently, Paget's disease can cause various complications.

Etiology of Paget's Disease

The exact cause of Paget's disease remains unknown, but research suggests two possible factors.Genetic Link: Studies have found a significant increase (7-10 fold) in the incidence of Paget's disease among relatives of affected patients. This pattern suggests an autosomal dominant inheritance, indicating a potential genetic component.Viral Infection: Some evidence points to a possible link between Paget's disease and viral infections, although further research is needed to confirm this association. [9].

Clinical features

The prevalence of Paget’s disease increases with age. Paget’s disease is recognized most commonly after age 50 years and is rarely diagnosed in people younger than 20 years. Paget's disease often progresses without noticeable symptoms, and many individuals are unaware they have the condition. When symptoms do occur, they can vary greatly depending on the affected bones.The disease is frequently diagnosed incidentally during Routine radiographic exams (e.g., X-rays)and Biochemical investigations (e.g., blood tests).In severe cases, Paget's disease can manifest with significant Musculoskeletal impairments, Neurological complications and Cardiovascular complications.

The most common presenting complaint is pain. The bone pain is perceived as a dull constant aching pain deep below the soft tissues. It may persist or exacerbate during the night. The involved bones become warm to the touch because of the increased vascularity. Other typical findings and complaints of patients with Paget’s disease may include the following: pathologic fractures commonly result from weakened pagetic bone, nonspecific headaches, impaired hearing, and tinnitus are common symptoms of Paget’s disease with skull involvement. Facial bone involvement is a rare occurrence in Paget's disease. Historically, this manifestation was referred to as "leontiasis ossea," which describes a lion-like facial appearance [3].

Oral manifestations

Paget's disease, also known as osteitis deformans, frequently affects the jaws, making jaw involvement a relatively common oral manifestation of the disease.Here's a rephrased version:

Dental and facial chamges

As Paget's disease progresses, the following changes may occur:Maxillary enlargement: The upper jawbone (maxilla) expands, causing the alveolar ridge to widen and the palate to flatten.Tooth changes: Teeth may become loose, migrate, and spacing may increase.Mandibular involvement: The lower jawbone (mandible) may also be affected, but typically less severely than the maxilla.As the disease advances,The mouth may remain open due to the enlarged jaw, exposing the teeth.Edentulous patients (those without natural teeth) may

experience Difficulty wearing dentures due to increasing jaw size an Need for frequent denture remakes to accommodate jaw expansion [3].

Histopathological features

Paget's disease is characterized by distinct bone architecture changes, which unfold in three phases :Osteolytic phase: Marked by increased bone resorption due to abnormal osteoclasts with multiple nuclei. Mixed phase: Overlapping of osteolytic and osteoblastic activities. Osteosclerotic phase: Dominant osteoblastic activity leading to excessive, fibrous, and coarse bone formation. Jigsaw-puzzle piece appearance: Irregularly shaped bone particles, characteristic of Paget's disease. Disorganized bone development: Fragmented and irregular bone formation.

Vascularized fibrous tissue: Marrow space filled with fibrous tissue, causing warmth and fever. Absence of centralized blood vessels and Haversian systems: Unique feature of Paget's disease-affected bones.Poorly mineralized new bone: Lacking structural integrity, resulting from the osteoblastic phase [9].

Treatment

Some patients diagnosed with Paget disease may not require any treatment.Various treatment regimens can help prevent bone breakdown and subsequent abnormal bone formation. Common drug therapies include:First-Line Treatment Bisphosphonates: Approved as the primary treatment option due to their ability to regulate bone remodeling.Alternative Treatments includes Calcitonin: Typically used as a second-line treatment, offering analgesic effects and assisting in bone absorption, Denosumab: Used off-label for patients intolerant or contraindicated to bisphosphonates, with promising results. Supportive Therapies includes Calcium and Vitamin D supplements: May provide symptomatic benefits.Pain management is achieved through NSAIDs or acetaminophen.Surgery is only considered for patients with Paget's disease that has progressed to osteosarcoma [9].

CENTRAL GIANT CELL GRANULOMA

Giant cell granuloma and its related jaw lesions are categorized together, but they exhibit diverse clinical behaviors, ranging from:Simple reactive lesions, Neoplastic growths, Aggressive malignant tumors

The presence of giant cells in various unrelated bone lesions complicates differential diagnosis. Additionally, secondary reactive changes within the lesion can mimic malignancy, making it challenging to determine the lesion's true nature. Accurate diagnosis requires specialized expertise [10].

Clinical features

Central giant cell granuloma can occur at any age, but it is more prevalent in young individuals, particularly those under 30 years old and slightly more common in females than males.Often asymptomatic, discovered incidentallyMay cause Cortical expansion and perforation, Tooth mobility, displacement, and root resorption, Lesion borders may be regular or diffuse.Classification includes Central giant cell granuloma can be categorized into two types based on clinical and radiographic features. They are Non-Aggressive Type - Slow-growing, No root resorption or cortical perforation and Often exhibits new bone formation. Aggressive Type- Rapid growth, Associated with pain, Cortical perforation and root resorption occurs [3].

Treatment

Curettage and Surgical Excision. Prognosis is Lesions typically heal with new bone formation after treatment.Recurrence is possible, but rare.In most cases, recurrence does not necessitate more aggressive. X-ray radiation is contraindicated, as it is not an effective or safe treatment option for central giant cell granuloma [10].

CHERUBISM

Cherubism is a rare genetic disorder characterized by Autosomal dominant inheritance pattern which is Painless, often symmetrical enlargement of the jaws, replacement of bone tissue with fibrous tissue, leading to jaw deformity

Key Features:

Typically affects children, with symptoms appearing between 2-5 years old and may cause facial disfigurement and dental problems and often resolves or improves with age, but may require surgical intervention in severe cases.

Clinical features

Males are more commonly affected than females, with a 2:1 ratio.Typical presentation includes, Bilateral enlargement of the jaws, resulting in a swollen face, Bone consistency of the lesion, Intact mucosa, Dental malocclusion, Upward gaze (due to maxillary involvement), Absence of pain, Additional Features includes, Cervical and/or submandibular lymphadenopathy may be present and Initial signs of the disease typically appear around 2 years of age, Accelerated growth occurs between 8-9 years of age and Spontaneous interruption of the disease usually occurs after puberty [11].

Radiographic features

Radiographic examination reveals Multilocular radiolucent lesions mainly affecting the mandibular body and ascending ramus and coronoid process, maxilla, particularly the maxillary tuberosity and Bilateral involvement showing radiolucencies often occur on both sides. Orbital involvement is seen and the lesions may extend to the lower portion and fundus of the orbit, with thin but intact cortical bone. Dental complications includes displaced and impacted teeth, Root resorption and mandibular canal displacement are often observed due to lesion expansion [11].

Treatment

Conservative management is Waiting for stabilization and spontaneous remission of the disease.Tooth extraction that is Removing teeth in areas with significant fibrous alterations.Cosmetic osteoplasty: Reshaping the affected jaws for aesthetic purposes, typically after disease activity has regressed. Surgical intervention: Curettage of lesions, often performed in cases of functional impairment.Medical treatment: Administration of calcitonin to manage disease activity.Treatment planning should be individualized, taking into account the patient's specific needs and disease severity [11].

TRAUMATIC BONE CYST

Traumatic bone cysts are typically marked by a cavity or void within the bone, absence of an epithelial lining, usually asymptomatic, with no associated pain or discomfort.

Clinical Features

Traumatic bone cysts are predominantly found in the mandible, commonly affecting the body and ramus of the mandible, Symphysis.Less Common Locations are Maxilla.Most cases of maxillofacial traumatic bone cysts are asymptomatic and do not cause expansion of the cortical area – these cysts are diagnosed as accidental findings in routine radiographs [12].

Radiographic features

Traumatic bone cysts typically appear on radiographs as Unilocular radiolucent areas, Single, well-defined radiolucent lesions.It is commonly found in the posterior region of the mandible. Irregular borders that curve around dental roots. Multilocular appearance, may present as multiple radiolucent areas.Association with unerupted/impacted teeth. Multiple cysts in the same patient: More than one traumatic bone cyst may be present in the same individual [12].

Histopathological features

Microscopically, traumatic bone cysts are characterized by Connective tissue membrane, a thin layer of connective tissue lining the cavity, typical of pseudocysts.Absence of epithelial lining: Unlike true cysts, traumatic bone cysts lack an epithelial lining.Presence of various features like Cholesterol crystals, Hemorrhagic foci (areas of bleeding), Osteoclasts (bone-resorbing cells) [12].

Treatment

Surgical curettage: Scraping the bone walls to remove the cystic tissue. High success rate and Short-term healing is typically achieved. Recurrences are rare, usually occurring within 3 months of surgery.

ANEURYSMAL BONE CYST

An aneurysmal bone cyst (ABC) is a rare, benign tumor that affects the bone. It's characterized by a blood-filled cystic lesion that can occur in any bone, but is most commonly found in the long bones of the arms and legs, as well as the spine and pelvis.

Clinical features

Aneurysmal bone cysts (ABCs) can present themselves in a wide range of ways, making them clinically variable. In some cases, they may appear as small, slow-growing, and asymptomatic lesions. However, they can also develop rapidly into large, destructive lesions that expand and cause significant symptoms. These symptoms may include pain, swelling, deformity, neurological issues, and even fractures or perforation of the bone cortex [13].

Radiographic features

The radiological features of Aneurysmal Bone Cysts (ABCs) in the jaws can be quite varied and conflicting. Typically, the bone appears expanded and cystic, with a characteristic honeycomb or soap bubble-like texture, and is often eccentrically ballooned. In some cases, the cortex may be destroyed or perforated, and a

periosteal reaction, indicating new bone formation, may be visible. Additionally, the radiographic appearance of ABCs in the jaws can vary, appearing radiolucent, radiopaque, or mixed [13, 14].

Histopathological features

Histologically, ABC consists of many sinusoidal blood-filled spaces set in a fibrous stroma, with multinucleated giant cells and osteoid. Hemosiderin is present in variable amounts and there is evidence of osteoid and bone formation. This description is characteristic of the "classic or vascular" form [13, 15].

Treatment

Treatment of ABC is usually directed toward complete removal of the lesion. Various treatment modalities are available for managing Aneurysmal Bone Cysts (ABCs). These include percutaneous sclerotherapy, diagnostic and therapeutic embolization, curettage, block resection and reconstruction, radiotherapy, and systemic calcitonin therapy. Interestingly, some cases of ABCs have also been reported to self-heal over time, as observed during long-term follow-up [13].

FIBROUS DYSPLASIA

Fibrous dysplasia is a non-hereditary skeletal disorder that affects bone development, resulting from a defect in osteoblastic differentiation and maturation. Any bone in the body can be impacted, and the exact cause remains unknown. Although the condition's etiology is unclear, research suggests that a mutation in the GNAS1 gene is typically responsible for its development.

Clinical features

Fibrous dysplasia is classified into three main forms: monostotic, polyostotic, and craniofacial. Typically, the first signs of the condition appear in individuals between the ages of 3 and 15. Notably, two-thirds of patients with polyostotic disease remain symptom-free until around age 10, while those with monostotic disease may not exhibit symptoms until their 20s or 30s. The condition affects males and females equally, with no racial predisposition. A worsening of pain and an enlarging soft tissue mass are indicative of potential malignant transformation [3].

Radiographic features

Fibrous dysplasia typically appears as a lucent lesion in the diaphysis or metaphysis of long and short tubular bones, characterized by endosteal scalloping. The lesion may cause bone expansion and is often surrounded by a thick sclerotic border, known as the "rind sign". Notably, there is usually no periosteal reaction, which helps distinguish fibrous dysplasia from other bone conditions.

In the maxillary and mandibular bones, fibrous dysplasia typically presents with a mixed radiographic pattern, featuring both radiolucent and radiopaque areas. This can lead to dental displacement and distortion of the nasal cavities, resulting in altered facial architecture [3].

Histopathological features

The lesions characteristic of fibrous dysplasia consist of fibrillar connective tissue interspersed with numerous trabeculae of immature, woven bone. These irregularly shaped trabeculae are evenly spaced and lack any correlation with functional patterns. Notably, the osteocytes are enlarged, and collagen fibers from the trabeculae often extend into the surrounding fibrous tissue. Bone formation is mediated by stellate osteoblasts,

although the typical osteoblastic rimming, characterized by rows of cuboidal osteoblasts lining the trabecular surfaces, is absent [3].

Treatment

Treatment for fibrous dysplasia is typically conservative, focusing on preventing deformity and addressing any underlying endocrine issues. In many cases, particularly those affecting the upper extremities, nonsurgical management is effective, with over 80% of patients responding well. While there is no specific medical treatment for the bone disease, early evidence suggests that vitamin D and bisphosphonates (after epiphyseal closure) may help alleviate pain and potentially promote normal bone growth. Surgical intervention, involving curettage and bone grafting, is often unsuccessful due to graft resorption at the surgical site.Osteosarcoma and fibrosarcoma are the most common tumors. Chondrosarcomas occur less frequently [3].

OSSIFYING FIBROMA

The term "peripheral ossifying fibroma" refers to a relatively common gingival lesion that is highly cellular and typically exhibits bone formation. However, in some cases, this lesion may instead contain cementum-like material or, more rarely, dystrophic calcification.

Clinical features

The peripheral ossifying fibroma can affect individuals of any age, but it tends to occur more frequently in younger populations, particularly children and young adults.The peripheral ossifying fibroma presents as a well-defined, localized mass on the gingiva, typically arising from an interdental papilla. It has a sessile or pedunculated base and is usually the same color as the surrounding mucosa or slightly reddened. The surface of the lesion may be intact or ulcerated, and while its appearance is characteristic, it is not exclusively diagnostic [3].

Radiographic features

Radiographic examination typically reveals no underlying bone involvement associated with the peripheral ossifying fibroma. However, in rare instances, superficial erosion of the adjacent bone may be visible on the radiograph [3].

Histopathological features

The peripheral ossifying fibroma is primarily composed of a highly cellular connective tissue mass. This mass consists of numerous plump, proliferating fibroblasts that are interspersed throughout a delicate, fibrillar stroma, giving the lesion its characteristic cellular appearance.Calcification may be in the form of single or multiple interconnecting trabeculae of bone or osteoid [3].

Treatment

Surgical excision of the lesions is necessary, followed by microscopic examination to confirm the diagnosis. Removing adjacent teeth is rarely required or justified. However, recurrence of the lesions is relatively common, and repeated recurrences can also occur [3].

OSTEOMA

An osteoma is a type of non-cancerous growth that involves an abnormal proliferation of bone tissue. This benign tumor typically develops in the compact or cancellous bone, usually on the surface or within the bone.

Clinical features

The lesion of periosteal origin manifests itself as a circumscribed swelling on the jaw producing obvious asymmetry.Osteomas are slow-growing tumors that often go unnoticed by patients. Those that develop within the bone, known as endosteal osteomas, may take a long time to show symptoms as they need to grow significantly before causing any noticeable expansion. Typically, osteomas do not cause pain. In some cases, multiple osteomas in the jaws, long bones, or skull can be a characteristic sign of Gardner syndrome [3].

Radiographic features

A central osteoma typically appears as a well-defined, radiopaque mass within the jawbone, which can be difficult to distinguish from scar tissue. In some cases, the osteoma may present as a diffuse lesion, requiring differentiation from chronic sclerosing osteomyelitis. Similarly, the periosteal form of the disease also manifests as a sclerotic mass.

Histopathological features

An osteoma is composed of either extremely dense compact bone or coarse cancellous bone, with normally formed bone in any given area. Typically, the lesion is well-defined but not encapsulated. In some cases, osteomas may also contain foci of cartilage, leading to the term "osteochondroma," or rarely, intermingled myxomatous tissue [3].

Treatment

Treatment for osteoma typically involves surgical removal, especially when the lesion is causing difficulties or interfering with the placement of a prosthetic appliance. This is often the case when the tumor is located near the surface of the alveolar bone. Fortunately, osteomas do not recur after surgical removal.

OSTEOBLASTOMA

Benign osteoblastoma is a rare, non-cancerous bone tumor that primarily affects the vertebrae and long bones. This unique tumor is characterized by the active deposition of osteoid and bone tissue, and is distinguished by an abundance of osteoblasts, the cells responsible for bone formation.Osteoblastoma is an uncommon osteoblastic tumor that rarely involves facial bones.

Clinical Features

Benign osteoblastoma predominantly affects young individuals, with approximately 75% of cases occurring in people under 20 years of age and 90% in those under 30. Although it is most common in youth, this bone tumor can also occur in older adults.Benign osteoblastoma typically presents with symptoms of pain and swelling at the tumor site. The duration of these symptoms can vary significantly, ranging from just a few weeks to a year or even longer.It occurs both in maxilla and mandible [3].

Radiographic features

Osteoblastomas are typically characterized as well-defined, solitary, and expansive lesions that grow outward, often causing the surrounding bone to expand.Osteoblastomas often present with a thin rim of expanded cortical bone surrounding the lesion, or in some cases, the cortex may appear absent, with a soft tissue mass protruding outward. The margins of the lesion may appear somewhat indistinct due to the lack of reactive sclerosis in the mandible. Radiographically, the central radiopaque areas indicate new bone formation, but unlike osteoid osteoma, osteoblastomas typically do not provoke significant bony sclerosis. Additionally, adjacent teeth may be affected by root resorption, displacement, or tipping [16].

Histologic features

Under microscopic examination, osteoblastomas are characterized by the presence of long, irregularly shaped trabeculae of osteoid or immature woven bone. These trabeculae are interconnected and rimmed by active osteoblasts, all of which are embedded within a rich fibrovascular stroma [16].

Treatment

The treatment options for osteoblastoma typically involve surgical interventions, which may include conservative surgical excision, excision accompanied by vigorous curettage, bur ablation, and copious irrigation, or in some cases, en bloc resection [16].

CEMENTOBLASTOMA

Benign cementoblastoma is a rare and distinctive odontogenic tumor that is characterized by the formation of a cementum or cementum-like tissue mass, typically attached to the roots of a tooth [3].

Clinical features

Benign cementoblastoma primarily affects adults, with a mean age of 20.7 years, and exhibits a higher predilection for males, with a male-to-female ratio of 2.1:1. The mandible is more commonly involved than the maxilla, particularly in association with the roots of mandibular molars and premolars. Additionally, the tumor can be linked to multiple teeth, impacted molars, deciduous teeth, and occasionally, vital teeth. Notably, cementoblastomas are typically asymptomatic.Although typically asymptomatic, benign cementoblastoma can also exhibit locally aggressive behavior, leading to complications such as bony expansion, root resorption, displacement of adjacent teeth, and jaw deformity [17].

Radiographic features

Radiographically, benign cementoblastoma appears as a well-defined, round radiopaque mass that is fused to the root of a vital tooth, typically surrounded by a thin radiolucent periphery [17, 18].

Histopathological features

Histologically, benign cementoblastoma is characterized by the presence of cementum-like tissue, which exhibits numerous reversal lines, a distinctive feature of this tumor.Benign cementoblastoma exhibits prominent basophilic reversal lines, which can impart a pagetoid appearance to the lesion. The intervening fibrovascular stroma may contain multinucleated osteoclast-type giant cells and plump cementoblasts. Additionally, a band of connective tissue resembling a capsule may be observed at the periphery of the lesion [17, 19, 20].

Treatment

The recommended treatment for benign cementoblastoma involves surgical removal of the tumor along with the affected tooth, followed by curettage or peripheral ostectomy. This approach helps prevent recurrence and continued growth, which can occur if any lesional tissue is left behind after the initial surgery [17].

CHONDROMA

Chondroma, a benign tumor consisting of mature cartilage, is a well-established entity in various parts of the skeletal system. However, it is relatively rare in the maxilla and mandible, the bones that form the upper and lower jaw [3].

Clinical features

Chondromas can occur at any age and affect both genders equally. They typically present as a painless, slow-growing swelling in the jaw, which may cause tooth loosening. The overlying mucosa is rarely ulcerated. The most common location for chondromas is the anterior portion of the maxilla.In the mandible, chondromas most frequently occur in the area posterior to the cuspid tooth. This location often involves the body of the mandible or may extend to the coronoid or condylar processes [3].

Radiographic features

Radiographically, chondromas appear as predominantly radiolucent, irregular masses, often containing scattered calcified foci. These calcifications can vary in appearance, ranging from a powdery texture to dense aggregates [21, 22]

Histopathological features

Microscopically, chondromas are characterized by normal-appearing chondrocytes residing in well-formed lacunar spaces, surrounded by an abundant hyaline chondroid matrix. Notably, mitotic figures are scarce, indicating a low proliferative activity [21, 23]

Treatment

The recommended treatment for chondroma is surgical excision, as the tumor is resistant to radiation therapy. Considering the possibility of sarcomatous transformation, a treatment approach more aggressive than simple enucleation is advised. However, radical resection is only justified for unusually large tumors, and a balanced approach should be taken to ensure complete removal while minimizing unnecessary tissue loss [3].

OSTEOSARCOMA

Osteosarcomas are rare and highly malignant bone tumors, characterized by the presence of malignant mesenchymal cells that produce osteoid or immature bone. When occurring in the jaws, osteosarcomas are extremely rare, accounting for approximately 7% of all osteosarcomas and a mere 1% of all head and neck malignancies [24].

Clinical Features

Jaw osteosarcoma (JOS) affects males slightly more often than females, with a median age of 30-40 years. Both the maxilla and mandible are equally involved, with a similar prognosis. Symptoms typically persist for 3-6 months before diagnosis, with the most common complaints being swelling (almost universal) and local pain (reported by 70% of patients). Other symptoms may include numbness, facial dysesthesia, tooth loosening, trismus, and nasal obstruction or bleeding. Systemic symptoms like fever, asthenia, or weight loss are rare. Physical examination may reveal a painless, firm mass fixed to the underlying bone, with lymph node involvement being unusual [3].

Radiographic features

The radiographic appearance of jaw osteosarcoma is variable and dependent on the amount of tumor bone produced by malignant osteoblasts. Tumors with minimal tumor bone formation appear radiolucent, while those with significant bone formation appear radiodense. Lesions with a mix of both radiolucent and radiodense areas indicate an intermediate degree of tumor bone formation. Additionally, characteristic "cumulus cloud" densities may form within the intramedullary and soft tissue components due to mineralizing tumor osteoid.In osteosarcoma-affected long bones, the periosteum is lifted by the expanding tumor mass, forming a tent-like structure. At the point where the periosteum meets the bone, a characteristic acute angle is created, known as Codman's triangle. This radiographic feature is highly indicative of osteosarcoma [3].

Histopathological features

Histologically, osteosarcoma is categorized into central (intramedullary) and peripheral (surface) subtypes. The primary type of central osteosarcoma is conventional osteosarcoma, which exhibits a wide range of morphologies. Characteristic histological features include the production of osteoid and immature bone, anaplastic neoplastic cells with epithelioid, plasmacytoid, or spindle-shaped aspects, and a permeative growth pattern that fills the marrow space and erodes surrounding trabeculae. Conventional osteosarcoma is further classified into osteoblastic, chondroblastic, and fibroblastic subtypes based on the predominant type of extracellular matrix present.The well-differentiated osteosarcoma is characterized by a distinctive histological appearance. The peripheral osteosarcoma features a spindle cell stroma with minimal cellular atypia and rare mitotic figures. The tumor is composed of irregular trabeculae of woven bone, which are arranged in a parallel manner, separating the spindle cell stroma. [24].

Treatment

In cases where osteosarcoma affects the long bones, amputation is often necessary. For tumors located in other areas, radical resection is the preferred treatment approach. However, in sites such as the jaws, complete excision can be challenging. Radiation therapy is not effective as a primary treatment. Neoadjuvant chemotherapy has been shown to facilitate surgical removal by shrinking the tumor. Furthermore, adjuvant chemotherapy combined with surgery, including resection of pulmonary metastases, has emerged as a promising approach to improve survival rates for this disease.

CHONDROSARCOMA

Chondrosarcomas are rare malignant tumors of cartilaginous origin, infrequently found in the head and neck region. These tumors can exhibit aggressive behavior and are typically located near the anterior maxilla and base of the skull [25].

Clinical Features

Chondrosarcomas typically present as a painless swelling or a long-standing mass, which may eventually become painful. As the disease progresses, additional symptoms may arise, including paresthesia, trismus, and loosening of the teeth.Several rare variants of chondrosarcoma have been identified, each characterized by unique microscopic and clinical features. These variants include clear cell chondrosarcoma, mesenchymal chondrosarcoma, juxtacortical chondrosarcoma, extraskeletal myxoid chondrosarcoma, and dedifferentiated chondrosarcoma, occurring with frequencies of 1%, 2%, 2%, 5%, respectively [3].

Radiographic features

Radiographic findings for chondrosarcoma are often similar to those of benign chondromas, unless the lesion is long-standing and has caused significant bone destruction. In some cases, the tumor may appear as a radiopaque lesion due to calcification of the neoplastic cartilage [3].

Histopathological features

The diagnostic features proposed by Lichtenstein and Jaffe are invaluable in identifying chondrosarcoma, particularly when examining viable tissue sections. These features include cells with plump nuclei, occasional binucleated cells, and giant cartilage cells with large or multiple nuclei. Accurate diagnosis relies on the correct interpretation of these subtle qualitative characteristics. However, challenges may arise due to the presence of necrotic regions or areas with insufficient cytologic evidence. Typically, chondrosarcomas exhibit sheets of chondrocytes with a lobulated growth pattern, although some cases may display a cluttered appearance similar to synovial chondromatosis [3].

Treatment

The primary treatment for chondrosarcoma is radical resection, as it is considered a radioresistant tumor. However, for cases of locally recurrent or residual tumors, chemoradiation is typically recommended as an adjunctive treatment [25].

EWING'S SARCOMA

Ewing's sarcoma is a highly aggressive and lethal malignant tumor that falls under the category of non-epithelial, neuroectodermal neoplasms. It is the sixth most common small round cell tumor affecting bones and soft tissues, characterized by poor differentiation and aggressive biological behavior [26].

Clinical features

Ewing's sarcoma primarily affects children and young adults, with the majority of cases occurring between the ages of 5 and 25. The median age of diagnosis is 13 years, and approximately 80% of cases arise within the first two decades of life. Although less common, the disease can also occur in older patients.The initial clinical signs and symptoms of Ewing's sarcoma typically include intermittent pain and swelling of the affected bone. The long bones of the extremities are most commonly involved, but the tumor can also occur in other bones, such as the skull, clavicle, ribs, shoulder and pelvic girdles, as well as the maxilla and mandible.When Ewing's sarcoma involves the jaw, patients may experience facial neuralgia and lip paresthesia. The swelling of the jaw can progress rapidly, and the intraoral mass may become ulcerated. Additionally, patients may present with systemic symptoms such as low-grade fever and elevated white blood cell count, which can lead to an initial misdiagnosis of an infection.

Radiographic features

A distinctive radiographic feature of Ewing's sarcoma is the formation of layers of new subperiosteal bone, which creates an "onion skin" appearance on radiographs. This characteristic pattern is due to the thickening of the cortex, which is often infiltrated by the tumor.

Histopathological features

Microscopic examination revealed uniform small round cells arranged in a diffuse pattern, with indistinct cell outlines and scanty cytoplasm. The cells had well-defined nuclear borders, round-to-oval nuclei, and inconspicuous nucleoli. Additionally, mitotic figures were not a prominent feature [3].

Treatment

This neoplasm is responsive to radiation therapy, but historically, X-ray radiation alone has rarely led to a cure. Treatment approaches have included radical surgical excision, sometimes combined with X-ray radiation. However, metastases often develop in other bones and organs, such as the lungs and lymph nodes, within weeks or months. Fortunately, combining surgery with chemotherapy has significantly improved outcomes, with a five-year survival rate of 74% [3].

CONCLUSION

Jaw bone pathologies encompass a diverse range of conditions affecting the mandible and maxilla, including developmental disorders, infectious diseases, tumors and cysts, inflammatory conditions, and traumatic injuries. Accurate diagnosis through clinical examination, imaging studies, and biopsy is crucial for effective management. Treatment options vary depending on the condition, ranging from surgical intervention and medication to supportive care. Early detection and regular dental check-ups are vital for improved outcomes, emphasizing the importance of maintaining good oral hygiene and seeking prompt medical attention for symptoms or concerns related to jaw bone health.

REFERENCE

1.Prado, H. V., Soares, E. C. B., Carneiro, N. C. R., Vilar, I. C. O., Abreu, L. G., & Borges-Oliveira, A. C. (2023). Dental anomalies in individuals with osteogenesis imperfecta: a systematic review and meta-analysis of prevalence and comparative studies. Journal of applied oral science : revista FOB, 31, e20230040.

2.Ventura, L., Verdonk, S. J. E., Zhytnik, L., Ridwan-Pramana, A., Gilijamse, M., Schreuder, W. H., van Gelderen-Ziesemer, K. A., Schoenmaker, T., Micha, D., & Eekhoff, E. M. W. (2024). Dental Abnormalities in Osteogenesis Imperfecta: A Systematic Review. Calcified tissue international, 115(5), 461–479.

3.Shafer’s Textbook of Oral Pathology, R Rajendran, B Sivapathasundharam, 7th edition.

4.Celakil, T., Dogan, M., Rohlig, B. G., Evlioglu, G., & Keskin, H. (2016). Oral Rehabilitation of an Osteopetrosis Patient with Osteomyelitis. Case reports in dentistry, 2016, 6930567.

5. Krithika, C., Neelakandan, R. S., Sivapathasundaram, B., Koteeswaran, D., Rajaram, P. C., & Shetkar, G. S. (2009). Osteopetrosis-associated osteomyelitis of the jaws: a report of 4 cases. Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics, 108(3), e56–e65.

6. Bharti, K., & Goswami, M. (2016). Cleidocranial dysplasia: A report of two cases with brief review. Intractable & rare diseases research, 5(2), 117–120.

7. Knipe H, Merrett A, Moore C. (2020). Idiopathic osteosclerosis of the mandible. Reference article, Radiopaedia.org.

8. Foster, B. L., Ramnitz, M. S., Gafni, R. I., Burke, A. B., Boyce, A. M., Lee, J. S., Wright, J. T., Akintoye, S. O., Somerman, M. J., & Collins, M. T. (2014). Rare bone diseases and their dental, oral, and craniofacial manifestations. Journal of dental research, 93(7 Suppl), 7S–19S.

9. Shaker J. L. (2009). Paget's Disease of Bone: A Review of Epidemiology, Pathophysiology and Management. Therapeutic advances in musculoskeletal disease, 1(2), 107–125.

10. Ramesh V. (2020). "Central giant cell granuloma" - An update. Journal of oral and maxillofacial pathology : JOMFP, 24(3), 413–415.

11. Lima, G.deM., Almeida, J. D., & Cabral, L. A. (2010). Cherubism: clinicoradiographic features and treatment. Journal of oral & maxillofacial research, 1(2), e2.

12. Martins-Filho, P. R., Santos, T.deS., Araújo, V. L., Santos, J. S., Andrade, E. S., & Silva, L. C. (2012). Traumatic bone cyst of the mandible: a review of 26 cases. Brazilian journal of otorhinolaryngology, 78(2), 16–21.

13. Devi, P., Thimmarasa, V., Mehrotra, V., & Agarwal, M. (2011). Aneurysmal bone cyst of the mandible: A case report and review of literature. Journal of oral and maxillofacial pathology : JOMFP, 15(1), 105–108.

14. Kiattavorncharoen, S., Joos, U., Brinkschmidt, C., & Werkmeister, R. (2003). Aneurysmal bone cyst of the mandible: a case report. International journal of oral and maxillofacial surgery, 32(4), 419–422.

15. Capote-Moreno, A., Acero, J., García-Recuero, I., Ruiz, J., Serrano, R., & de Paz, V. (2009). Giant aneurysmal bone cyst of the mandible with unusual presentation. Medicina oral, patologia oral y cirugia bucal, 14(3), E137–E140.

16. Sahu, S., Padhiary, S., Banerjee, R., & Ghosh, S. (2019). Osteoblastoma of Mandible: A Unique Entity. Contemporary clinical dentistry, 10(2), 402–405.

17. Sankari, L. S., & Ramakrishnan, K. (2011). Benign cementoblastoma. Journal of oral and maxillofacial pathology : JOMFP, 15(3), 358–360.

18.Schafer, T. E., Singh, B., & Myers, D. R. (2001). Cementoblastoma associated with a primary tooth: a rare pediatric lesion. Pediatric dentistry, 23(4), 351–353.

19. Lemberg, K., Hagström, J., Rihtniemi, J., & Soikkonen, K. (2007). Benign cementoblastoma in a primary lower molar, a rarity. Dento maxillo facial radiology, 36(6), 364–366.

20. Huber, A. R., & Folk, G. S. (2009). Cementoblastoma. Head and neck pathology, 3(2), 133–135.

21. Dhirawani, R. B., Anand, K., Lalwani, G., Pathak, S., & Thakkar, B. (2014). True chondroma of the mandibular condyle: A rare case. Annals of maxillofacial surgery, 4(2), 220–223.

22. Fechner RE, Mills SE. (1993). Atlas of Tumor Pathology – Tumors of the Bones and Joints. Washington, DC: Armed Forces Institute of Pathology.

23.Lurie R. (1975). Solitary enchondroma of the mandibular condyle: a review and case report. The Journal of the Dental Association of South Africa = Die Tydskrif van die Tandheelkundige Vereniging van Suid-Afrika, 30(7), 589–593.

24. Ferrari, D., Moneghini, L., Allevi, F., Bulfamante, G., & Biglioli, F. (2017). Osteosarcoma of the Jaw: Classification, Diagnosis and Treatment. InTech.

25. Ram, H., Mohammad, S., Singh, G., & Singh, S. V. (2013). Chondrosarcoma of body of the mandible. National journal of maxillofacial surgery, 4(2), 242–244.

26. Ahuja, U. S., Puri, N., Gupta, D., Singh, S., & Kumar, G. (2019). Ewing's Sarcoma of Mandible: A Case Report with Review. International journal of clinical pediatric dentistry, 12(5), 470–474.