Benign tumors of soft tissues and bones of head at children. Classification, etiology. Diagnostics, differential diagnostics, treatment and rehabilitation of children.

> Pediatric Surgical Dentistry Lector – Popelo julia 066-460-33-03 (Viber)

Plan of lecture and their organizational structure.

N⊵	The main stages of the lecture and their content	Type of lecture. Means of activating students. Methodical support materials	time distribution
1.	Preparatory stage. definitionrelevance of the topic, learning objectives of the lecture and motivation	look p 1 and 2	5 %
2.	 The main stage. Teaching lecture material according to the plan: 1. 1 Frequency of malignant processes of SHLD in children. 2. Phases of carcinogenesis 3. Signs of benign and malignant process. 4. Methods of diagnosis of SHLD tumors. 5. The structure of malignant pathology of the thyroid gland in children. 6. Clinical and morphological features of malignant tumors of the thyroid gland in children. 7. Basic principles of rehabilitation of children with oncological pathology. 8. Stages of formation of bone regenerate. 9. Clinical case. 	Clinical lecture.	85 % -90%
1. 2. 3.	The final stage Lecture summary. General conclusions.	Answers to possible questions.	5 %

Classification of benign neoplasm

Type of tissue	Type of neoplasm	
Pavement epithelium	Squamous cell papilloma	
Secretory (glandular) epithelium	Adenoma	
Connective	Fibroma	
Adipose	Lipoma	
Smooth muscle	Leiomyoma	
Osseous	Osteoma	
Cartilaginous	Chondroma	
Lymphoid	Lymphoma	
Transversal striated muscle	Rhabdomioma	

Evaluation



'congenital vs. acquired requires systematic approach *important historical points:* 1 - age at onset 2 - functional impairment 3 - rapidity of growth and extension 4 - fluctuation in size 5 - appearance - inflammation - ulceration or bleeding - color change 6 - pain, paresthesias, or anesthesia

Evaluation



IBcation Mze, color, and texture Thobility Hender/nontender Manch with pressure Bruits Munctional interference - swallowing (drooling) speech - respiration

Evaluation



Classification

Oral Soft Tissue Tumors

Benign

- A. hemangioma
- **B.** lymphatic malformation
- C. fibrous tumors
- D. epithelial tumors:
 - 1) papilloma 2) dermoid 3) white sponge nevus
- E. cystic tumors:
- 1) ranula 2) thyroglossal duct cyst 3) branchial cysts
- **F**. hamartomas and choristomas
- G. miscellaneous and salivary gland tumors

Classification of vascular lesions in infants and children

These conditions are categorized into two families: a family of self-involuting tumors, growing lesions that eventually disappear, and another family of malformations, enlarged or abnormal vessels present at birth and essentially permanent. The importance of this distinction is that it makes it possible for early-in-life differentiation between lesions that will resolve versus those that are permanent. Examples of permanent malformations include port-wine stains (capillary vascular malformation) and masses of abnormal swollen veins (venous malformations)

Hemangiomas:

- Proliferating phase
- Involuting phase
- **B.** Malformations:
- Capillary
- Cavernous
- Arterial
- Limphatic
- Fistulae

The terminology used to define, describe and categorize vascular anomalies, abnormal lumps made up of blood vessels, has changed. The term hemangioma was originally used to describe any vascular tumor-like structure, whether it was present at or around birth or appeared later in life

Incidence:

- 2,6% all newborns
- Up to 22% preterm infants<1000 gm
- 12% at 1 year
- 1/3 life-threating site-oral cavity
- Lip, cheek, tongue



Differential diagnosis: capillary hemangiomas



Differential diagnosis cavernous hemangiomas



Differential diagnosis infantile hemangioma and Strawberry hemangioma



Congenital hemangioma. also known as strawberry nevus, usually presents around the time of birth but may not be apparent until childhood

Pathophysiology/Histology

- arrested development in mesenchymal vascular primordia
- 3 stages of development:
- capillary stage
 early=superficial
 late=deeper, subcutaneous
 retiform stage
- mature, end stage



Pathophysiology/Histology 2 phases of growth 1) proliferating phase -endothelial hyperplasia with thymidine incorporation -rapid growth during infancy -hormone response-estradiol-17 beta receptors 2) involutional phase -fibrosis and fat deposition -low to absent thymidine incorporation -rapid growth with regression

Evaluation

1) physical exam: a) superficial lesions -blue or reddish hue -compressible -nontender -partially submerged b) deep lesions -similar to superficial -firm -less circumscribed

- Lymphangiomas and cystic hygromas are rare benign hamartomatous lesions of the lymphatic system. Determining the true incidence of these lesions is difficult because uniformity in classification and nomenclature is lacking.
- Of lymphatic malformations, 50% are present at birth, and 90% are diagnosed by the time the individual is aged 2 years. However, the time of diagnosis can range from 19 weeks' gestation to the individual's second decade of life.
- Of lymphatic malformations in the oral cavity, 40-50% involve the tongue, which is the preferred site of intraoral involvement. The buccal mucosa is the second most common. These distinctions can be somewhat artificial because lesions that involve the oral cavity may extend from the orbits to the upper mediastinum and axillae.
- Lymphatic malformations are typically classified as simple microcystic, simple macrocystic, or mixed microcystic and macrocystic lesions, according to their predominant histologic features.

- A neoplasm is classically defined as an abnormal mass of tissue. Its growth exceeds and is uncoordinated with that of healthy tissues and persists in an excessive manner after the inciting stimuli is removed. In contrast, lymphatic malformations and/or lymphangiomas tend to grow commensurately with the child's growth and rarely regress spontaneously. Rapid enlargement of the lesions (out of proportion with the surrounding tissues) is observed only in conjunction with infections of the upper respiratory tract or the lesion itself or with trauma and hemorrhage into the malformation. In addition, the lesions have a typical endothelial cell cycle.
- For these reasons, lymphangiomas are considered to be malformations rather than neoplasms. In a teleologic sense, this determination remains rather unsatisfying because the predisposing event (ie, the sequestration of embryonic anlagen) occurs long before these lesions develop. As previously stated, only 50% of the lesions are diagnosed at birth, and a few reports of lesions developing in early adulthood exist.
- Moreover, at prenatal ultrasonography, fetal lymphatic malformations are observed to develop and occasionally resolve in utero. Some unrecognized event must be superimposed on these earlier events resulting in the development of a clinically apparent lesion. These events are apparently reversible because the lesions occasionally spontaneously regress in fetuses and children. The inciting event must then be occurring during the maturation of the formed lymphatic systems.

History

- Although most lesions appear in the individual's first 2 years of life, lesions occasionally appear in early adulthood.
- Typical histories include annoying and repetitive surface bleeding, paroxysms of lesion expansion, usually in association with upper respiratory tract infections, intralesional hemorrhage, or repeated infections.

Physical

Superficial lymphatic malformations have a pebbly surface and are clear or bluish. The bluish color may be caused by either a venous component or an intralesional hemorrhage.



Superficial lymphatic malformations

Imaging Studies

Approximately 40% of lesions are diagnosed on the basis of their clinical appearance alone; however, this observation does not obviate further imaging. Imaging plays several roles in the evaluation and treatment of oral lymphatic malformations. Imaging helps in determining the extent of the lesion and its proximity to vital structures, in determining whether the lesion contains a vascular component, and is used to assess recurrence in treated lesions.

Ultrasonography

- Prenatal ultrasonography can be used to identify fetal cystic hygromas. A diagnosis of fetal cystic hygroma has important ramifications for the fetus because this lesion is often associated with major chromosomal abnormalities. However, caution is warranted with such a diagnosis because these lesions are known to regress in utero. In addition, the prenatal diagnosis of fetal cystic hygroma has an error rate as high as 70%, depending on the time of diagnosis.
- Ultrasonography may be an invaluable tool for monitoring a lesion for regression or recurrence. Lesions will appear cystic on ultrasound, with little evidence of flow upon Doppler interrogation.

MRI

- Contrast-enhanced MRI is the most useful imaging modality for the assessment of lymphatic malformations.
- The superior soft-tissue definition is critical in planning surgery and determining the extent of the lesion.
- MRI is particularly important in distinguishing vascular malformations or mixed lesions from lymphatic malformations. Both vascular and lymphatic variants have a low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. However, in contrast to vascular malformations, lymphatic variants fail to enhance after the administration of contrast material.
- Fetal MRI is an excellent diagnostic tool for further evaluation of oral and anterior cervical lesions detected on prenatal ultrasound. It can be invaluable in determining the potential for airway obstruction and the need for ex utero intrapartum treatment (EXIT procedure) at delivery.

■ CT scanning

- CT scans are superior to MRIs in delineating bony deformation.
- CT scans are superior to other modalities in detecting phleboliths, which were once considered pathognomonic of venous malformations. Phleboliths observed in lymphatic malformations are most likely caused by a previous intralesional hemorrhage.
- Three-dimensional CT scans may be obtained to permit the fabrication of stereolithographic models with which to plan orthognathic surgical correction of the maxillofacial skeletal deformity at maturity.
- Plain radiography: Posteroanterior and lateral cephalometric radiographs are invaluable in diagnosing maxillofacial skeletal deformities secondary to lymphatic malformations.

Staging

- Lymphatic malformations in the oral cavity can be classified as superficial lesions and deep lesions on the basis of their histologic and clinical features.
 - Superficial lesions include lymphangioma simplex, lymphangioma circumscriptum, and capillary hemangioma.
 - Deep lesions are further divided into microcystic variants (eg, cavernous lymphangioma) and macrocystic variants (eg, lymphangioma cystoides, cystic lymphangioma, cystic hygroma).
- In the oral cavity, superficial and microcystic lesions are most common.
- Below the mandible and above the maxilla, macrocystic lesions predominate.
- Any lesion may comprise any or all of the histomorphologic variants.

- A staging system for cervical lymphatic malformations by de Serres is based on the extent of anatomical involvement. It has been validated in terms of its ability to predict the incidence of preoperative complications, postoperative complications, longterm morbidity, and risk of recurrence, which increase with the stage of the lesion. The system is perhaps better termed a classification because staging implies a progressive neoplastic behavior.
- Stage/class I Unilateral infrahyoid lesions
- Stage/class II Unilateral suprahyoid lesions
- Stage/class III Unilateral suprahyoid and infrahyoid lesions
- Stage/class IV Bilateral suprahyoid lesions
- Stage/class V Bilateral suprahyoid and infrahyoid lesions
- Stage/class VI Bilateral infrahyoid lesions

Surgical Care

- Treatment of lymphatic malformations continues to evolve. Although surgical removal is overwhelmingly the most common modality used, sclerotherapy is being increasingly used to treat macrocystic lesions throughout the head and neck. Often the 2 modalities are combined for optimal outcomes. Determine the timing of intervention on a case-bycase basis. Lesions that impinge on the airway usually require the earliest intervention.
- Isolated superficial malformations are often more of a nuisance than a debilitating condition. Given the fact that these are malformations and not true neoplasms and that they are entirely devoid of malignant potential, treatment should be aimed at maximizing the patient's function. In addition, treatment that results in a loss of function should not be tolerated. Early involvement of consultants can be helpful in meeting the goal of maximizing the patient's function and determining an optimal treatment plan.

Incision and drainage

- Limit incision and drainage to emergent decompression of a lesion.
- This procedure does not eliminate the lesion but decreases the risk of infection.
 Surgical excision
- Surgical excision remains the standard for treatment of lymphatic malformations, although many lesions are especially difficult to remove entirely because of their involvement with important neural and muscular structures; thus, these lesions are associated with the highest risk of recurrence and complications.
- Recurrence rates of 20-40% are typically reported after surgical excision when the surgeon believes that the lesion is removed in its entirety.6
- Complications of surgical excision of suprahyoid and oral lesions are also common. These operations can be difficult undertakings because of the diffuse infiltrative nature of the lesions and the difficulty in determining normal tissue from abnormal tissue.
- Complication rates typically are 20-30%. Complications include airway obstruction, seromas and hematomas, infections, and cranial nerve palsies. Moreover, the reported operative mortality rates are 2.5-11.4%.
- Surgical debulking, as part of a staged intervention plan, may be useful with large lingual malformations. Surgical debulking may obviate long-term tracheostomy, facilitate feeding, improve speech, and simplify further treatment, regardless of the modality used.
- Surgical excision of macrocystic lesions may be facilitated by instilling fibrin glue into the cysts after partial aspiration of cystic contents.

Laser therapy

- Surface laser photocoagulation is used as an adjunct for controlling the size of the tongue, treating superficial lesions, and controlling bleeding.
- Good results are reported. With the exception of isolated superficial lesions, surface laser photocoagulation does not lead to a cure.

Orthognathic surgery

- Maxillofacial skeletal deformities and malocclusions should be treated with combined orthognathic and osteoplastic surgical procedures. Lesions of the upper part of the neck and, particularly, the floor of the mouth tend to be associated with bony overgrowth leading to class III malocclusions and open bite deformities. These lesions usually require combined surgical and orthodontic correction.
- Orthognathic surgery is usually delayed until growth is complete, except in patients in whom the severity of the deformity necessitates earlier intervention.

Sclerotherapy

- Myriad agents are used as sclerosing agents in attempts to avoid surgery and its attendant problems or to treat remnant lesions after partial excision.
- Repeated injections of sclerosing agents are often required, and resolution usually occurs over 1-4 months.
- Macrocystic lesions are most amenable to sclerotherapy, followed by microcystic lesions and then superficial lesions.
- Sclerotherapy does not represent an advantage compared with surgical ablation, with respect to success and ease of treatment. Success rates vary considerably depending on the agent used. Generally, boiling water, sodium morrhuate, tetracycline, 50% dextrose, and steroids are not effective in treating lymphatic malformations.
- The encouraging results reported with sclerotherapy in conjunction with its low complication rate favor its adoption into the treatment protocols for lymphatic malformation, either as a stand-alone modality or as an adjunct to surgery.

Fibroma

- The fibroma, also referred to as irritation fibroma, is by far the most common of the oral fibrous tumorlike growths. While the terminology implies a benign neoplasm, most if not all fibromas represent reactive focal fibrous hyperplasia due to trauma or local irritation. Although the term focal fibrous hyperplasia more accurately describes the clinical appearance and pathogenesis of this entity, it is not commonly used.
- A fibroma may occur at any oral site, but it is seen most often on the buccal mucosa along the plane of occlusion of the maxillary and mandibular teeth as depicted below. It is a round-to-ovoid, asymptomatic, smooth-surfaced, and firm sessile or pedunculated mass. The diameter may vary from 1 mm to 2 cm. The surface may be hyperkeratotic or ulcerated, owing to repeated trauma.
- Fibromas are most often observed in adults, but they may occur in individuals of any age and either sex.

Fibroma

Histologically, a fibroma is an unencapsulated, solid, nodular mass of dense and sometimes hyalinized fibrous connective tissue that is often arranged in haphazard fascicles. A mild chronic inflammatory infiltrate may be present. The surface epithelium may be hyperkeratotic, either hyperplastic or atrophic, and it may be ulcerated. Conservative excisional biopsy is curative, and its findings are diagnostic.

Peripheral Cemento-ossifying Fibroma

A peripheral cemento-ossifying fibroma is also known as a peripheral ossifying fibroma, a calcifying fibrous epulis, or a peripheral fibroma with calcification. It is a reactive gingival lesion that is believed to arise from cells of the periodontal ligament or periosteum. Most often, it is located in the gingival papilla between adjacent teeth. A peripheral cemento-ossifying fibroma manifests as a sessile or pedunculated mass, which is often ulcerated and generally has a diameter of less than 2 cm as shown below. Peripheral cementoossifying fibromas may occur in persons of any age but are most often seen in persons aged 10-20 years.

Peripheral Cemento-ossifying Fibroma

The maxillary gingiva is involved more often than the mandibular gingiva; usually, the anterior region is affected. Mobility and/or migration of adjacent teeth is occasionally observed as depicted in the image below.



Peripheral Odontogenic Fibroma

A peripheral odontogenic fibroma is a rather uncommon neoplasm that is believed to arise from odontogenic epithelial rests in the periodontal ligament or the attached gingiva itself. The entity, formerly confused with peripheral cemento-ossifying fibroma, is considered to be the extraosseous counterpart of the central odontogenic fibroma of the World Health Organization type.14 A peripheral odontogenic fibroma manifests as a firm, slowly growing, sessile, and nodular growth of the gingiva, most often on the mandibular buccal or labial aspect as depicted below. It occurs in persons of a wide age range and affects both sexes equally.



Fibromatoses (aggressive juvenile)

- The fibromatoses represent a group of infiltrating fibrous proliferations with a biologic behavior and microscopic appearance intermediate between those of benign fibrous lesions and fibrosarcomas. In the head and neck region, they are sometimes referred to as juvenile or aggressive juvenile fibromatoses. Patients of any age may be affected, but three quarters of all cases are diagnosed when the patient is younger than 10 years. No significant sex predilection is apparent.
- The most frequent site of occurrence is the soft tissues adjacent to the mandible. Intraoral presentations are rare, but they most often involve the tongue or buccal mucosa. Lesions appear as firm, painless, poorly demarcated masses with a variable growth rate. They are locally aggressive and often cause resorption of the underlying bone when present. A desmoplastic fibroma occurring within the medullary cavity of bone is considered to be the intraosseous counterpart of the soft tissue fibromatosis.

Neurofibromatosis



Papilloma

Papilloma refers to a benign epithelial tumor growing exophytically (outwardly projecting) in finger-like fronds. In this context papilla refers to the projection created by the tumor, not a tumor on an already existing papilla (such as the nipple). When used without context, it frequently refers to infections caused by human papillomavirus (HPV), such as warts. There are, however, a number of other conditions that cause papilloma, such as choroid plexus papilloma (CPP) and pearly penile papules. Papillary and verruciform epithelial proliferations are quite common in the oral and paraoral region. Of the several types of papillomas, the one occurring in the mouth and oropharynx is almost always the squamous papilloma.

Squamous papilloma in oral cavity

The typical lesion is a soft, pedunculated mass with numerous finger-like surface projections (papilla = "nipple-shaped projection"). Projections may be pointed and the surface may be covered with a considerable amount of keratin, producing a white surface change. The heavily keratinized lesion with short rounded projections is cauliflower-like, while a similar but less keratinized lesion resembles a raspberry or mulberry with a pink or red coloration.

The squamous papilloma typically has a narrow stalk below a mass with numerous blunted and pointed surface projects, often characterized as finger-like



- Osteoid osteoma is a benign skeletal neoplasm of unknown etiology that is composed of osteoid and woven bone. The tumor is usually smaller than 1.5 cm in diameter. Osteoid osteoma can occur in any bone, but in approximately two thirds of patients, the appendicular skeleton is involved. The skull and facial bones are involved exceptionally.
- Most patients with <u>osteoid osteoma</u> are young. Rarely, an ossification center is affected. The classic presentation is that of focal bone pain at the site of the tumor. The pain worsens at night and increases with activity; it is dramatically relieved with small doses of aspirin. The lesion initially appears as a small sclerotic bone island within a circular lucent defect. This central nidus is seldom larger than 1.5 cm in diameter, and it may be associated with considerable overlying cortical and endosteal bone sclerosis. The tumors may regress spontaneously. The mechanism of this involution is not known, but tumor infarction is a possibility.

Osteoid osteoma is classified as cortical, cancellous, or subperiosteal.

- Cortical tumors are the most common. The radiolucent nidus is within the cortical bone, where it is surrounded by a fusiform cortical thickening or solid or laminated periosteal new bone formation.
- Cancellous osteoid osteoma has an intramedullary location. Intraarticular osteoid osteomas are difficult to identify, and a delay of 4 months to 5 years before diagnosis is not unusual. The most common sites affected by cancellous osteoid osteomas include the juxta-articular region of the femoral neck, the posterior elements of the spine, and the small bones of the hands and feet. Usually, little sclerosis occurs around the nidus. Intra-articular tumors are associated with joint-space widening as a result of joint effusion or synovitis.
- Subperiosteal osteoid osteoma is a rare form of the disease that usually presents as a rounded soft-tissue mass adjacent to a bony cortex, which it excavates. Surrounding reactive changes are usually absent. The common sites involved include juxta- or intra-articular regions of the medial aspect of the femoral neck and the hands and feet, in particular, the neck of the talus.

 The classic presentation includes focal <u>skeletal</u> <u>bone pain</u>, which worsens at night and is frequently relieved with a small dose of aspirin.





- Osteoblastoma is an uncommon osteoid tissue-forming primary <u>neoplasm</u> of the bone.
- It has <u>clinical</u> and <u>histologic</u> manifestations similar to those of osteoid osteoma; therefore, some consider the two <u>tumors</u> to be variants of the same disease, with osteoblastoma representing a giant <u>osteoid osteoma</u>. However, an aggressive type of osteoblastoma has been recognized, making the relationship less clear.
- Although similar to osteoid osteoma, it is larger (between 2 and 6 cm).

The etiology of osteoblastoma is unknown. Histologically, osteoblastomas are similar to osteoid osteomas, producing both osteoid and primitive woven bone amidst fibrovascular connective tissue. Although the tumor is usually considered benign, a controversial aggressive variant has been described in the literature, with histologic features similar to those of malignant tumors such as an <u>osteosarcoma</u>.

- Patients with osteoblastoma usually present with pain of several months' duration. In contrast to the pain associated with osteoid osteoma, the pain of osteoblastoma usually is less intense, usually not worse at night, and not relieved readily with <u>salicylates</u> (<u>aspirin</u> and related compounds). If the lesion is superficial, the patient may have localized swelling and tenderness.
- When diagnosing osteoblastoma, the preliminary radiologic workup should consist of radiography of the site of the patient's pain. However, computed tomography (CT) is often necessary to support clinical and plain radiographic findings suggestive of osteoblastoma and to better define the margins of the lesion for potential surgery. CT scans are best used for the further characterization of the lesion with regard to the presence of a nidus and matrix mineralization. MRI aids in detection of nonspecific reactive marrow and soft tissue edema, and MRI best defines soft tissue extension, although this finding is not typical of osteoblastoma. Bone scintigraphy (bone scan) demonstrates abnormal radiotracer accumulation at the affected site, substantiating clinical suspicion, but this finding is not specific for osteoblastoma. In many patients, biopsy is necessary for confirmation.





- The first route of treatment in Osteoblastoma is via medical means. Although necessary, radiation therapy (or chemotherapy) is controversial in the treatment of osteoblastoma. Cases of postirradiation <u>sarcoma</u> have been reported after use of these modalities. However, it is possible that the original histologic diagnosis was incorrect and the initial lesion was an osteosarcoma, since histologic differentiation of these two entities can be very difficult.
- The alternative means of treatment consists of surgical therapy. The treatment goal is complete surgical excision of the lesion. The type of excision depends on the location of the tumor.
- The clinical course of osteoblastoma often makes it difficult to diagnose. The tumor may have a slow indolent course or display characteristics that are confused with malignancy. Other diagnoses that share similar clinical, radiographic, and histologic features with conventional osteoblastoma include osteoid osteoma, giant cell tumor, and fibrous dysplasia. Osteoblastomas may also have features that mimic malignant tumors such as osteosarcoma.



Ameloblastoma

Ameloblastoma is an entirely epithelial tumor arising from the dental lamina, Hertwig sheath, the enamel organ, or the lining of dental follicles/dentigerous cysts. Ameloblastoma is the most common epithelial odontogenic tumor. Ameloblastomas usually occur in individuals aged 20-40 years; however, the unicystic variant (see Surgical considerations, below) most often occurs in adolescents. This lesion occurs in both the maxilla and mandible, but the posterior mandible is the most common location; only 20% of lesions are found in the maxilla. The lesion is distributed equally between males and females.

Ameloblastoma



Beforeoperation and1 year after

Ameloblastoma



 Computed tomography scan of a cystic ameloblastoma of the mandible showing cortical penetration.

Complex Odontoma

Complex odontoma lesions represent further histologic differentiation of the odontogenic pluripotential epithelium. Findings from this lesion resemble those of ameloblastic fibroodontoma but extended one step further. The epithelium in this lesion has involuted, leaving disorganized dental hard tissues in place. These are common lesions, and they persist throughout life. They are usually detected in adolescence and have a predilection for the mandibular molar regions; however, they can be found in other areas of the jaws.

Radiographic findings

These lesions are generally described as sunburst radiopacities surrounded by a thin, uniform, radiolucent rim. Although this description may have some superficial resemblance to the radiographic description of osteosarcoma, the association with a tooth, the clear demarcation of the lesions' borders, and the lack of pain and/or swelling serve to delineate this very benign lesion from osteosarcoma.

Histologic characteristics

The histodifferentiation of this lesion is extended one step further than that of ameloblastic fibro-odontoma. In complex odontoma, a physiologic reduction of the ameloblastic epithelium is found. A mixed honeycomb presentation of enamel, cementum, dentin, and pulpal tissue is present. Histodifferentiation but no morphodifferentiation is observed in this lesion.

Treatment

Simple removal or radiographic observation is the method of treatment. These lesions do not recur.

Complex Odontoma

 The *complex* type is unrecognizable as dental tissues, usually presenting as a radioopaque area with varying densities. It usually appears in the posterior maxilla or in the <u>mandible</u>.



Compound Odontoma

This is the most common odontogenic tumor. It represents the product of both histodifferentiation and morphodifferentiation of odontogenic tissues, resulting in what appears as a **cluster of multiple abortive teeth**. It is most commonly found in the maxillary anterior alveolar bone but may be located anywhere within the tooth-bearing segments of the jaws. It is often responsible for preventing normal tooth eruption, thus it is usually discovered during adolescence.

Radiographic findings

Multiple tiny toothlike structures are contained within a fine radiolucent rim.

Histologic characteristics

Histology of compound odontomas approaches normal tooth structure. Gross clinical examination is usually sufficient for diagnosis.

Treatment

Simple removal is the method of treatment. These lesions do not recur.

Compound Odontoma



A compound odontoma still has the three separate dental tissues (enamel, dentin and cementum), but may present a lobulated appearance where there is no definitive demarcation of separate tissues between the individual "toothlets". It usually appears in the anterior <u>maxilla</u>.

Cementoblastoma

Cementoblastoma, as distinguished from cementoma, is a true neoplasm of cementum. This benign neoplasm is rare and is usually observed in patients younger than 25 years. It is most often found in association with the apex of the mandibular first molars (50% of lesions), and it is never found in association with the anterior dentition. The lesion is usually asymptomatic, although occasionally the associated tooth may be slightly sensitive to percussion.

Radiographic findings

A round opaque sunburst mass attached to the apex of a tooth that is welldemarcated and surrounded by a thin radiolucent rim is observed. The lesion obscures the lamina dura. Students sometimes confuse it with condensing osteitis, a common lesion resulting from low-grade periapical irritation that stimulates bone growth. Although the most usual location for the 2 lesions is the same, condensing osteitis does not obscure the periodontal ligament (PDL) space and tends to be more irregular in outline. The mature cementoma, also known as periapical cemental dysplasia, is another common lesion that students may confuse with cementoblastoma. However, cementoma is usually located in the mandibular anterior region and does not obscure the PDL space. Cementomas actually have 3 developmental stages: osteolytic (at which point the lesion appears as a radiolucency), cementoblastic (mixed radiolucent/radiopaque), and mature (radiopaque).

Treatment

Removal of attached tooth and tumor is the method of treatment. No recurrences are reported.

Cementoblastoma

 Periapical radiograph showing the mass delimited by a thin radiolucent halo and the lamina dura

