

**Chapter 20: Carotid Body Tumor, Hemangioma, Lymphangioma,**

**Melanoma, Cysts, and Tumors of the Jaws**

**Carotid Body Tumor**

The normal carotid body, situated at the carotid bifurcation, is a chemoreceptor similar to the aortic body. It responds to arterial changes in pH, temperature, oxygen, and carbon dioxide tension. The carotid body is different from the carotid sinus, which is a pressoreceptor.

The carotid body tumor is a nonchromaffin paraganglioma associated with a network of chemoreceptors. It is believed to arise from neurocrest cells. These cells are derived from sympathoblasts rather than from chromaffinoblasts. Other than at the bifurcation of the carotid, such tumors can be found in other chemoreceptors of the head and neck, middle ear, jugular bulb, carotid bulb, base of the skull, lateral pterygoid, vagal, and aortic regions. Unlike the carotid body, most tumors have no demonstrable chemoreceptive or hormonal function. These tumors occur generally in equal frequency in men and women, most commonly in the third and fourth decades of life. The carotid body tumor usually presents as a firm, rubbery, painless, slow-growing mass. When the tumor is large, it may cause a mild pain. Syncopal episodes may occur when the tumor compromises the cerebral blood flow. A bruit is often heard over the tumor. Carotid angiography that shows an "egg shell-like" mass displacing the internal carotid artery laterally and widening the crotch is pathognomonic of this tumor. The major blood supply of this tumor may be from the vertebral artery, the thyrocervical trunk, as well as the carotid arteries.

A familial tendency has been reported by Rush and Chase. Bilateral carotid body tumors have been reported by Rush and Katz.

Tumors of the carotid body are considered radioresistant. Therefore, a symptomatic tumor should be resected. However, an asymptomatic tumor in an elderly patient is best left alone. Carotid body tumor is said to originate from the adventitia of the artery. Consequently, one should be able to free this tumor from the carotid artery. Conley described this tumor as a mass developing in the adventitia of the bifurcation, pushing the internal and external carotid arteries apart. It may eventually encircle both arteries. The weakest point of this encirclement has been determined to be in the posterolateral aspect of the internal carotid artery. Consequently, it is the safest place to begin resection of the tumor. When the internal carotid artery needs to be sacrificed, it would be wise to apply a vascular graft. Sacrificing the internal carotid artery without applying a graft carries a 30-50% mortality with another 40% incidence of neurologic deficits. Nelson in 1962 stated that 5-10% of these tumors are malignant. Regional lymph nodes and distant metastases have been reported. However, Conley indicated that multicentricity of paraganglionic foci in the head and area is not uncommon. It is, therefore, difficult to differentiate metastases from de novo foci.

## **Hemangiomas**

Hemangiomas are congenital vascular abnormalities rather than true neoplasms. The most common sites are the face and neck. Approximately, 63% of hemangiomas are cutaneous, 15% subcutaneous, and 22% mixed. The most common site of deep hemangioma in the head and neck is within the masseter muscles. They generally are more prevalent in females in a frequency of 3:1 except for subglottic hemangiomas where the sex ratio is about equal. They may be classified pathologically into three types: capillary, cavernous, and mixed. Pathologic classification is probably of little value, compared to clinical classification. Approximately 75% of hemangiomas are present at birth, while 85% will have manifested themselves by the first year of life. Approximately 3% of the patients have a positive family history.

### ***Hemangiomas of the Skin and Subcutaneous Tissue***

#### **Port-Wine Hemangioma**

This reddish blue lesion is composed of capillaries with adult endothelium and persists with little change in life. The important features clinically are: (1) the lesion grows only in proportion to body growth and will always cover the same percentage of body surface area; and (2) the lesion is not raised above the surrounding skin. This lesion does not respond to radiation and should be treated with surgical excision, tattooing, or cosmetic coverage. Unless absolutely necessary, treatment is not recommended before the age of 2. Hemangiomas that grow rapidly during the first few months of life are also the ones that involute subsequently.

#### **Strawberry Hemangioma**

This lesion is raised above the surrounding skin, blanches somewhat on pressure, and has a strawberry red color. It is the most common type, accounting for 90% of all infant hemangiomas. Approximately 90% of these hemangiomas are capillary in type and likewise 80-90% involute by the fifth year.

Excision should not be performed before age 5, unless serious problems ensue (bleeding, blockage of an important orifice, ulceration, thrombocytopenia, etc). Steroid treatment seems promising, and sclerosing agents are occasionally useful.

The embryonal vascular endothelium of strawberry hemangioma is sensitive to irradiation, but even small doses may inhibit facial bone growth with severe sequelae.

#### **Hemangioma of the Parotid Gland**

It is the most common tumor of the parotid in infancy. Goldman advocates prompt surgical excision as soon as definite growth is recognized, while others suggest waiting until age 5. Nussbaum et al reported cavernous hemangioma of the salivary gland in five adults. Infantile hemangiomas of the salivary gland are usually of the capillary type.

## **Hemangioma of Bone**

This lesion is found in the vertebral bodies, frontal and parietal bones, mandible, or maxilla; in females usually during the fourth decade. Slow progressive swelling is seen clinically, and a characteristic "honeycomb" or "sunburst" appearance is present radiographically. Massive hemorrhage may occur following tooth extraction in a mandible involve with a hemangioma. Surgical excision is the treatment of choice.

## ***Hemangioma of the Larynx***

### **Subglottic Hemangioma of Infancy**

The symptoms produced are of a croup-like syndrome with varying degrees of stridor, absence of hoarseness when crying, weight loss, and marked persistent cyanosis. The cyanosis may be worse when the patient is excited or cries. It is usually of the cavernous type and usually located anteriorly. Ninety percent of patients develop symptoms before the age of 3 months, but only 50% have associated subcutaneous hemangiomas.

The diagnosis is made by laryngoscopy and tracheoscopy. Biopsy is generally contraindicated because of the possibility of severe hemorrhage. The treatment is tracheostomy to relieve the airway obstruction. Gradual involution of the hemangioma in 12-18 months is the usual course. Steroids occasionally seem to promote involution. Radiation is not recommended because of (1) absence of proof that in small doses of 300-600 R any significant histologic effect occurs, (2) possible effects of radiation on the growth and development of the larynx, and (3) the possible cause of thyroid carcinoma years later. Healy advocates CO<sub>2</sub> laser to destroy the lesion because of the properties of laser (minimal edema, scarring, and limited bleeding).

### **Adult Laryngeal Hemangioma**

The location is usually supraglottic or glottic and often polypoid or pedunculated in appearance. Adult hemangiomas of the larynx rarely cause respiratory embarrassment and generally should be left untreated.

## **Hemangioma of Nasal Bone**

This is a rare tumor. To date only 15 cases have been reported. This is a benign, slow-growing, painless vascular tumor within the nasal bone. The overlying skin or mucosa can be telangiectatic. Some patients present with epistaxis while others have no bleeding tendency. The radiographic appearance of "soap bubble" texture is pathognomonic. Feeding vessels are usually the facial and internal maxillary arteries. Treatment involves excision with a margin of normal bone.

## **Hemangiopericytoma**

Stout and Murray were the first to accurately describe and name this entity. The capillary pericyte of Zimmermann was the cell of origin of this tumor. It is about equally distributed in both sexes. It shows a propensity for the fourth, fifth, and sixth decades.

Clinically, it presents as a slowly expanding asymptomatic mass. In bony cavities, pressure pain may be noted. With those in the nose and paranasal sinuses, epistaxis is a frequent complaint. Histologically, it features sheets or a random distribution of ovoid or spindle-shaped cells with indistinct cytoplasm, large nuclei, and rare mitoses. Silver reticulin stain is essential to establish the diagnosis. It is usually considered a malignant lesion with metastatic rates ranging from 35 to 57%. This tumor is radioresistant. Wide excision is the treatment of choice.

### **Lymphangioma**

Lymphangioma is a congenital, benign, unilocular or multilocular, endothelium-lined, fluid-containing swelling of lymphatic origin. In 80% of the cases, the lesion is located in the neck. This condition is present at birth in 65% of the cases and would have manifested itself by age 2 in 90%. When symptomatic, they should be resected to prevent stridor and dysphagia in the infant. Resection should be performed carefully to spare all the vital structures. Lymphangioma has been said to be the most common tumor of the parotid gland in children.

### **Melanomas of the Head and Neck**

Melanocytes are believed to be derived from the neural crest cells that have migrated peripherally to the integument by the twelfth week of gestation. The melanocyte forms the pigment which is then transmitted to the malpighian cells of the basal layer of the skin.

Approximately 20-35% of melanomas occur in the head and neck region. Less than 1% occur intraorally. Conley reports the scalp to be the most frequent site, followed by the face, neck, and ear. Melanoma occurs slightly more frequently in men, except for superficial melanoma of the face which is more common in women. Less than 2% of melanomas occur before puberty. Predisposing etiologic factors are solar exposure, chronic infection, irradiation, friction irritation, thermal burns, and endocrine changes of puberty and pregnancy.

Nevi may be classified into three basic types depending upon the location of the melanocytes.

#### **Junctional Nevus**

Melanocytes are present at the dermoepidermal junction. Grossly, it is flat, light to dark brown or black, and non-hairy. Melanoma may arise from junctional nevi, but very rarely before puberty.

#### **Compound Nevus**

Melanocytes are present in both the epidermis and dermis. Grossly, it combines the features of a junctional and an intradermal nevus.

## **Intradermal Nevus**

Melanocytes are exclusively in the dermis. Grossly, this is the common adult mole and may be papillary, pedunculated, or flat and is usually hairy.

Other varieties of nevi are the (1) Spitz nevus: a dome-shaped benign nevus, generally pink or red, primarily in children, usually measuring less than 1 cm in diameter. (2) Halo nevus: a central brown papule surrounded by a pale white circle of depigmentation. The regularity of the circle of depigmentation and benign-looking nevus in the center distinguishes it from melanoma. (3) Blue nevus: appears in infancy or childhood as a small black or dark blue, round dome-shaped hard papule with a smooth surface. These tend to appear on the face and on top of the hands and feet and persist unchanged through life. It is benign. The junctional nevus of childhood gradually matures into a compound and then an intradermal nevus. In pregnancy, new moles often appear and pre-existing one become darker.

Only 25% of melanomas seem to arise from previously benign nevi, almost all junctional. Signs of possible malignant change include: deepening pigmentation, spread of pigment beyond the gross confines of the lesion, ulceration, rapid growth, appearance of flat areas of depigmentation in a black mole, inflammation, satellite nodules, bleeding, and the presence of itching.

Melanoma may be classified into three basic types:

### **Superficial Spreading Melanoma**

The surface is elevated, the margins are palpable, and the color variable. The prognosis is intermediate between lentigo maligna melanoma and nodular melanoma.

### **Lentigo Maligna Melanoma (Melanoma in a Hutchinson's Melanotic Freckle)**

Hutchinson's freckle typically occurs on the cheek of elderly patients as a flat, slow-growing brown lesion. Malignant melanoma frequently develops in Hutchinson's melanotic freckle and is characterized by thickening and development of black or amelanotic tumor nodules. It infrequently metastasizes and the prognosis is good.

### **Nodular Melanoma**

A palpable nodule with rapid growth is present in this variety with the poorest prognosis. A lateral flat component is not seen clinically or microscopically.

The differential diagnosis also includes seborrheic keratosis, senile hemangioma, sclerosing hemangioma, pyogenic granuloma, and pigmented basal cell carcinoma. Eight percent of melanomas in Conley's series were nonpigmented.

## **Biopsy**

An excisional biopsy is performed if the lesion is small. When the location or size makes this impractical, a careful preoperative incisional biopsy is justified. Epstein found there was no evidence to indicate that incomplete removal of malignant melanoma followed by definitive surgery, even 1 week later, decreases the probability of survival. A radical resection should obviously be made on only definite histologic proof. Frozen sections also are highly diagnostic.

## **Prognostic Factors**

The two most important prognostic factors are the type of melanoma and depth of invasion. McGovern et al have classified depth of invasion as:

Level 1: tumor confined to the epidermis

Level 2: tumor invading the papillary dermis (80-90% 5-year survival)

Level 3: tumor filling the papillary dermis (50% 5-year survival)

Level 4: tumor invading the reticular dermis (30% 5-year survival)

Level 5: tumor invading the subcutaneous tissue (less than 20% 5-year survival).

Tumors less than 2 cm in size have a better prognosis than larger ones. Ulceration is associated with a poorer prognosis. Flat lesions have a better prognosis than pedunculated or polypoid lesions. Women of the premenopausal age have a better prognosis than men.

Metastasis to regional lymph nodes varies greatly according to location of the primary tumor (76% for melanoma of the scalp to 19% for primary superficial melanomas of the cheek). Diffuse hematogenous spread may occur to any organ but has a propensity for the brain, liver, and abdominal viscera. Spontaneous regression rarely occurs.

## **Staging**

Stage I: local disease

Stage II: regional metastasis

Stage III: distant spread.

## **TNM classification of Melanoma**

T1: smaller than 2 cm in diameter, superficial, no satellite nodules

T2: 2-5 cm or with minimal infiltration of dermis regardless of size, no satellite nodules.

T3: larger than 5 cm, or with deep infiltration of dermis, or with satellite nodules.

N0: no palpable nodes.

N1: movable homolateral nodes.

N2: movable bilateral or contralateral nodes.

N3: fixed nodes.

M0: no distant metastasis.

M1: distant metastasis or satellite nodule farther than 5 cm from the primary tumor.

### **Treatment**

Conley divides surgical treatment into two basic categories:

1. Superficial melanomas of the cheek in females that arise in the lentigo nevus have little capacity for metastasis and should be treated with adequate local excision. Likewise, superficial melanomas of the helix may be treated by wedge excision only because of the low rate of metastasis.

2. Nodular melanomas or melanomas with significant invasion should be treated with wide resection of the primary lesion in continuity with the regional lymphatic drainage. Bilateral neck dissection is not justified and produces no cures for gross metastasis.

Melanoma is radioresistant although an occasional patient may be palliated but rarely cured. Chemotherapeutic agents have produced only minimal short-term improvement. Treatment with a combination of autologous tumor cells, BCG vaccine, and cytarabine was reported by Eldstrom.

### **Results**

The 5-year determinate cure rate in Conley's series of 200 patients with melanoma of the head and neck was 35%. Local excision alone produced a 5-year cure rate of 62%, which rose to 76% with an elective neck dissection if no evidence of metastasis was found in the regional nodes. The cure rate abruptly drops to 25% if occult metastases were found in the lymph nodes. Composite resection when clinically palpable nodes were present reduced the five-year cure rate to 14%. Spontaneous regression, has been reported by Whicker.

### **Melanoma of the Mucous Membrane**

Melanoma of the mucous membrane comprises approximately 10% of the melanomas of the head and neck. The oral cavity is the site of approximately 50% of cases, the nasal and sinus cavities 35%, and the pharynx and larynx 15%. The most common sites are the palate and the inferior alveolus.

Mucosal melanomas are usually not related to junctional nevi and rarely occur in the olfactory area of the superior nasal recess where pigmentation is abundant. Mucosal melanomas are extremely rare in blacks and there is no apparent relationship to local irritation, chronic infection, or allergy.

Nasal or paranasal sinus melanomas presented with epistaxis or nasal obstruction in 88% of patients. The nasal septum and maxillary sinus are the most common sites of origin.

The highest incidence occurs between ages 50 and 70. Grossly, the lesion often appears brownish grey with a smooth, flat lacy pattern that appears deceptively benign. Nasal melanomas present usually as dark fleshy tumors that bleed easily. Conley advises the removal of all pigmented lesions of the mucous membranes in white patients, for diagnostic purposes, as well as prophylaxis. Powell and Cummings cited the differential diagnosis of oral pigmentation:

1. Endogenous causes: racial, Addison's, Peutz-Jeghers syndrome (intestinal polyposis), polyostotic fibrous dysplasia, jaundice, Cooley's anemia (beta-thalassemia or thalassemia major), sickle cell anemia, thrombocytopenic purpura, hemochromatosis (bronze diabetes), antimalarial therapy, pregnancy, chlorpromazine (Thorazine) therapy, oral contraceptives, neurofibromatosis, hyperpituitarism, hyperthyroidism, infectious mononucleosis, nevi, oral melanosis, melanomas.

2. Exogenous causes: heavy metals (bismuth, lead, mercury, silver, gold, arsenic, copper, chrome, cadmium, zinc, brass), amalgam tattoo, charcoal, intraoral trauma.

3. Endocrine and metabolic causes: nicotinic acid deficiency (pellagra), folic acid deficiency secondary to malabsorption syndrome (sprue), vitamin A or C deficiency, hyperthyroidism, cutaneous inflammatory diseases.

Data by Conley showed metastasis to regional lymph nodes from mucosal melanoma had a lower incidence than melanoma of the skin. Local recurrence after excision varied from 25% in the pharynx to 40% in the nasal cavity and sinuses.

### **Treatment**

1. Local resection: A wide local resection (i.e. maxillectomy) is favored because of the relatively low incidence of regional metastasis and the discontinuity in the regional lymphatic system.

2. Electrodesiccation which gives localized control without extreme ablation is used for superficial melanomas of the palate.

3. Composite resection of the primary melanoma and the regional lymphatics is applied in continuity when the surgical anatomy permits. For melanomas of the inferior alveolus, lateral pharynx, or floor of the mouth, it is the treatment of choice.

4. Irradiation is almost universally ineffective against melanoma and should never be used as primary treatment. However, some authors use it preoperatively or for palliation,



especially in nasal or sinus lesions.

### **Prognosis**

Conley reports a 15% 5-year determinate cure rate; 11% living with melanoma beyond 5 years; and 74% dead within 5 years.

Melanoma of the nasal septum has a better prognosis than melanoma of the turbinates or sinuses.

## **Cysts and Tumors of the Maxilla and Maxilla**

### **Odontogenic Tumors**

#### **Ameloblastoma**

Ameloblastoma (adamantinoma, adamantine epithelioma, soft odontoma, adamantoblastoma, epithelial odontoma) is a very uncommon neoplasm that arises from odontogenic epithelium or the enamel organ and comprises about 1% of tumors and cysts found in and around the maxilla and mandible. Rarely, ameloblastoma may arise from a dentigerous cyst. Eighty percent of these tumors are found in the mandible, especially in the molar-ramus region. In the maxilla, its predominant site is in the cuspid and periantral areas. The average age in reported series is 34-38 years old. The usual symptom is painless swelling with occasional pathologic fracture. The radiographic picture is quite pathognomonic in that it presents multiple radiolucent compartments with a honeycomb arrangement. However, it could present as a unilocular cystic lesion. These are benign tumors though locally invasive, and are radioresistant. Wide local surgical excision is the preferred treatment to prevent recurrence. Inadequate removal may cause wide local spread at a future date. A small number of cases have been reported with pulmonary metastasis. The most frequent extraoral primary site is the pituitary gland.

Grossly, the tumor is seen as a cylindrical or fusiform swelling that expands the bone. Histologically, it has the follicular and the plexiform patterns. The follicular type consists of discrete islands or follicles of epithelial cells in connective tissue stroma. The island of epithelium resembles enamel. In the plexiform type, islands of epithelium form continuous strands.

#### **Adenomatoid Odontogenic Tumor**

The other name for this tumor is adenameloblastoma which is unfortunate because it bears no relationship to ameloblastoma. It usually is located in the anterior teeth in the maxilla. It usually is related to unerupted teeth. The treatment of choice is locally shelling it out. Recurrence is rare.

#### **Ameloblastic Fibroma**

This tumor occurs mainly in the premolar-molar region of the mandible. Radiographically, it appears as a unilocular cyst. It usually appears before age 20. The

treatment of choice is simple curettage which gives a low recurrence rate. An ameloblastoma with formation of dentin has been referred to as "dentinoma".

### **Odontomas**

Odontogenic tumors may be composed of one or all tissues that make up the normal tooth. They are the most common tumor of the maxilla and mandible. These tumors can arise from cells during any stage of embryogenic development. The most common site in the maxilla and mandible is in the third molar region. It is impossible to make a clinical separation of odontogenic tumors from odontogenic cysts, since they are quite similar and contain the same substances.

X-rays generally show recognizable, though distorted, tooth elements, with or without cystic cavities.

On histologic sectioning (after decalcification), such structures as enamel, dentin, or cementum will be recognized in varying proportions.

These tumors are usually asymptomatic and identified at routine dental x-rays. Conservative removal is the treatment of choice.

### **Odontogenic Myxoma**

Myxomas are tumors composed of young connective tissue similar to the umbilical cord. These are usually central in origin, arising in both the maxilla and the mandible, growing rapidly, and producing marked distortion of the face. The tumor readily expands the bone and loosens the teeth. X-rays show bone expansion, destruction, and trabeculation. Myxomatous lesions in the mandible and maxilla are considered to be benign, whereas those arising in other parts of the body are considered malignant, thus giving rise to the idea that their origin may be different. Though benign, it is locally invasive. Hence, wide total local excision is essential. Radiographically, it is unilocular or multilocular cyst. It is radioresistant.

### **Odontogenic Fibroma**

Unlike odontogenic myxoma, this is not locally invasive and can be shelled out uneventfully.

### **Cementoma**

This is periapical cemental dysplasia. It is asymptomatic and is a routine dental film finding.

### **Osteomas**

### **Torus Mandibularis**

Torus mandibularis is usually a bilateral osteoma occurring lingual to the lower canine or first premolar. This should be left alone unless symptomatic.

### **Torus Palatinus**

This osteoma occurs in the midline of the palate. It also should be left alone unless symptomatic. Both torus mandibularis and palatinus appear after puberty.

### **Arteriovenous Aneurysm**

There are two varieties: (1) congenital and (2) traumatic. It usually presents as swelling, discoloration, pulsation, and a murmur or thrill. Radiographically, a lytic lesion is noted and it is difficult to differentiate it from a benign cyst. Treatment is surgical. Preoperative angiography may be helpful.

### **Fibro-Osteoma (Ossifying Fibroma)**

Fibro-osteomas grow slowly and destroy the normal spongy bone. This fibrous tissue growth shows evidence of osteogenesis. The tumor produces asymmetry of the face - usually one side, either in the maxilla or mandible. In the maxilla, the teeth are spread out, the canine fossa usually obliterated, and the palatal arch asymmetric. In the mandible, there is distortion of the mandibular arch and the bite. The disease appears less often and less extensively in the mandible. In the maxilla, the whole face occasionally is distorted and the eyes displaced upward.

Normal landmarks are obliterated in x-rays. The bone cavity may be completely eroded with no evidence of osteogenesis and the margins of the bore are irregular. In large masses, however, there is usually evidence of both bone destruction and bone formation.

### **Benign Giant Cell Tumor**

This tumor occurs as a solitary lesion. Its origin and classification are disputed by a variety of authors. It is most commonly seen in the symphysis and bicuspid tooth areas of the mandible. In the maxilla, the canine fossa area is the most frequent site. The lesion causes expansion and destruction of the bone. Some authors attribute the cause to trauma. Regardless of the cause, a metabolic workup is indicated when such a lesion is found to rule out systemic disease.

X-rays show loss of bone. The margin of the bone loss is irregular in outline and the cavity unequally destroyed, producing a patchy outline on the x-ray. The teeth may be displaced or absorbed, depending on their relation to the cavity.

### **Endotheliomas**

Endotheliomas arise from the lining cells of the blood vessels and most often are seen on the hard or soft palate. These tumors tend to be small, round, and smooth, with an intact mucosa. When very large, the surface may become ulcerated. If lymph node enlargement occurs, it usually is due to infection of the ulcer. Voice change often occurs if the tumor is large.

## **Odontogenic Cyst**

The odontogenic cyst is derived from remnants of the dental lamina or from enamel organs. Symptomatically, it presents as a painless mass. It rarely causes any paresthesia or loosening of a tooth. Malignant degeneration of odontogenic cysts is rarely reported. Odontogenic cysts can be subclassified into:

### **Radicular Cyst (Periapical or Apical Periodontal Cyst)**

Radicular cysts are more common in the anterior teeth, and more common in permanent dentition than deciduous dentition. They develop from epithelial rests located in the membrane connecting the tooth root to the bone. They are true epithelial-lined cysts. The treatment is surgical removal of the cyst with preservation of the tooth.

### **Dentigerous (Follicular) Cyst**

Cystic odontoma is a form of dentigerous cyst that arises from the enamel or dental lamina of a developing or developed tooth. They are epithelial-lined sacs around the crown of an erupting tooth. Classically, they involve the crown of an unerupted tooth with the tooth inside the cyst wall; common sites include the third molar, cuspids, and bicuspid, in that order of frequency. Surgical removal of the cyst is advised with preservation of the tooth.

### **Eruption Cyst**

Eruption cyst is a dentigerous cyst associated with the erupting surface of a tooth, most commonly in third molars. They are above the tooth crown and cause delayed eruption.

### **Gingival and Palatal Cysts of Newborn**

The gingival cysts (Epstein's pearl, Bohn's nodules) are remnants of the dental lamina. The palatal cysts result from epithelial invagination during development.

### **Lateral Periodontal Cyst**

The lateral periodontal cyst is a cyst in bone along the root of a vital tooth.

### **Primordial Cyst**

Primordial cyst form from the enamel organ before any dental tissue develops. They usually occur in the mandibular third molar. Histologically, they have uniform epithelium covered with parakeratin and thick caseous debris.

### **Odontogenic Keratocysts**

These are more common in the mandible and in the third molar areas or ascending ramus. They resemble dentigerous cysts but are distinguished histologically by having a keratinized layer in the epithelial lining. These cysts have a tendency to recur and to become malignant.

### **Calcifying Odontogenic Cysts**

These are epithelial-lined cysts with calcification within the cavity.

### **Nonodontogenic Cysts**

Nonodontogenic cysts are derived from epithelial remnants trapped in the embryonic fusion lines during developmental stage.

#### **Globulomaxillary (Premaxilla-Maxillary) Cyst**

These are located in the bone. They are alveolar in location, being between the maxillary, lateral incisor, and cuspid teeth. They arise from epithelial invagination between the globular process of the frontonasal bone and the maxillary processes of the palatine bone. The cyst is lined with squamous or respiratory epithelium. It causes facial distortion with elevation of the ala and fullness of the canine fossa. The cyst is best shown on an occlusal intraoral views.

#### **Nasoalveolar (Nasolabial, Klestadt's) Cyst**

The nasoalveolar cyst arises from epithelial rests between the globular, lateral nasal, and maxillary processes. Unlike globulomaxillary cyst, it is on the bone with possible erosion of the bone. Ten percent of the patients with nasoalveolar cysts have them bilaterally. It is lined with respiratory epithelium (pseudostratified ciliated columnar) and occurs on the floor of the vestibule anterior to the inferior turbinate. It causes nasal obstruction and elevation of the ala. It can be palpated as a round mass high in the mucobuccal fold above the canine tooth.

Depending on the location, other cysts of the same type have been called nasopalatine, median anterior, maxillary, median posterior palatine, median mandibular, and anterior lingual.