Pediatric Dentistry

5th Year

Lec. 20

ACQUIRED AND DEVELOPMENTAL DISTURBANCES OF THE TEETH AND ASSOCIATED ORAL STRUCTURES (2)



Assistant Professor Zainab Juma Jafar



EARLY EXFOLIATION OF TEETH

A variation of as much as 18 months in the exfoliation time of primary teeth may be considered normal. However, this pattern must be consistent with other aspects of dental development. Exfoliation of teeth in the absence of trauma in children younger than 5 years of age merits special attention because it can be related to local or systemic pathology. Early exfoliation may be caused by the followings:

HYPOPHOSPHATASIA

Hypophosphatasia is characterized by improper mineralization of bone caused by deficient tissue-nonspecific alkaline phosphatase activity in serum, liver, bone, and kidney. The earlier the appearance of the disease, the greater is its severity.

The dental findings for the diagnosis of hypophosphatasia in children is premature exfoliation of the anterior primary teeth associated with deficient cementum. The loss of alveolar bone may be limited to the anterior region.

CHERUBISM (FAMILIAL FIBROUS DYSPLASIA)

Cherubism is a childhood disease affecting jaw development. The children have "chubby" faces, symmetric enlargement of the jaws and upturned eyes, giving the condition its name. Although disease progression is expected to stabilize or even regress after puberty, a few very aggressive cases, sometimes producing morbid results, have been reported.

Radiographically the condition presents multilocular areas of bone destruction (soap-bubble lesions) and thinning of the cortical plate. Teeth in the involved area may exfoliate prematurely because of the loss of support or root resorption or, in permanent teeth, because of interference in the development of roots. Developing permanent teeth are usually ectopic, being displaced by the growing lesion.

ACRODYNIA

Acrodynia or pink disease results from the exposure of young children to minute amounts of mercury; ointments and medications or playing repeatedly with a broken sphygmomanometer. Dental amalgam restorations do not cause acrodynia.



The clinical features of the disease include fever, anorexia, desquamation of the soles and palms (causing them to be pink), sweating, tachycardia, gastrointestinal disturbance, and hypotonia. **The oral findings** include inflammation and ulceration of the mucous membrane, excessive salivation, loss of alveolar bone, and premature exfoliation of teeth.

HYPOPHOSPHATEMIA (RICKETS)

Clinical features become evident in the second year of life and include short stature and bowing of the lower extremities in affected boys.

The dental manifestations often include premature teeth exfoliation, periapical radiolucencies, abscesses associated with pulp exposures. Pulpal necrosis is caused by abrasion of the thin, hypomineralized enamel, exposing the pulp horns that extend to the dentinoenamel junction or even to the external surface of the tooth. **Dental radiographs** show rickety bone trabeculations and absent or abnormal lamina dura. **Treatment** is by pulp therapy and stainless steel crown.

CYCLIC NEUTROPENIA

Cyclic neutropenia affected individuals are at risk for opportunistic infection during intervals of neutropenia cycle. The patients have fever, malaise, sore throat, stomatitis, and regional lymphadenopathy as well as headache, cutaneous infection, and conjunctivitis.

Orally Children exhibit severe gingivitis with ulceration. When the neutrophil count returns to normal, the gingiva may return to a nearly normal clinical appearance. Children experiencing repeated insults from the condition have a considerable loss of supporting bone around the teeth leading to early exfoliation.

INHERITED DEFECTS OF DENTIN

DENTINOGENESIS IMPERFECTA

It is inherited as autosomal-dominant trait. This anomaly may be seen with osteogenesis imperfecta.



Clinically: The teeth are a characteristic reddish-brown to blue-gray opalescent color.Soon after the primary dentition is complete, enamel is worn and often breaks away. The exposed soft dentin abrades rapidly, occasionally to the extent that the smooth, polished dentin surface is continuous with the gingival tissue.

Radiographs show slender roots and bulbous crowns. The pulp chamber is large initially and undergoes obliteration.

The treatment is difficult. The placement of stainless steel crowns on primary posterior teeth may be considered as a means of preventing gross abrasion of the tooth structure. Bonded veneer restorations on anterior teeth have also been used successfully for aesthetic improvement.

DENTIN DYSPLASIA

Dentin dysplasia is a rare disturbance of dentin formation that is categorized into two types: radicular dentin dysplasia (type I) which affects the roots, and coronal dentin dysplasia (type II) which affects the crowns. Both primary and secondary dentitions are affected.

INHERITED DEFECTS OF ENAMEL

AMELOGENESIS IMPERFECTA

Amelogenesis imperfecta is a developmental defect with a heterogeneous etiology.

Clinical appearances, with three broad categories: the hypocalcified type, the hypomaturation type, and the hypoplastic type. The defective tooth structure is limited to the enamel.

On radiographic examination, the pulpal outline appears to be normal, and the root morphology is that of normal teeth. The difference in the appearance and quality of the enamel is thought to be attributable to the state of enamel development at the time the defect occurs.

The treatment the teeth can be prepared for full-coverage restorations or bonded veneer restorations depending on the severity of the condition and the demands for aesthetic improvement.



ENAMEL AND DENTIN APLASIA

Teeth with characteristics of both dentinogenesis imperfecta and amelogenesis imperfecta have been reported and referred as (odontogenesis imperfecta).

The primary teeth were essentially devoid of enamel, and the smooth, severely abraded dentin was reddish-brown. The permanent teeth are partially covered with a thin, gray, poorly coalesced coating of enamel. Brown dentin could be seen.

Radiographs showed normal alveolar bone around the roots of the teeth. The pulp chambers and canals in the primary teeth are extremely large, with no evidence of becoming obliterated.

Treatment: by stainless steel crown restorations which should be placed even before complete eruption to protect the teeth from continued abrasion.

INTRINSIC DISCOLORATION OF TEETH

DISCOLORATION IN HYPERBILIRUBINEMIA

In several conditions, excessive levels of bilirubin are released into the circulating blood. If the teeth are developing during periods of hyperbilirubinemia, they may become intrinsically stained. If an infant had severe, persistent jaundice during the neonatal period, the primary teeth may have a characteristic blue-green or brown color.

DISCOLORATION IN PORPHYRIA

The porphyrias are inherited and acquired disorders in which abnormally elevated levels of porphyrins and/or their precursors are produced, which accumulate in tissues and are excreted.

Children with congenital erythropoietic porphyria have red-colored urine, are hypersensitive to light, and develop subepidermal bullous lesions when their skin is exposed to sunlight. Their primary teeth are purplish-brown as a result of the deposition of porphyrin in the developing structures. The permanent teeth also show evidence of intrinsic staining but to a lesser degree.



DISCOLORATION IN TETRACYCLINE THERAPY

Children who received tetracycline therapy during the period of calcification of the primary or permanent teeth show a degree of pigmentation of the clinical crowns of the teeth because the tetracyclines chelate calcium salts, are incorporated into bones and teeth during calcification. The crowns of affected teeth are discolored, ranging from yellow to brown and from gray to black. The larger the dose of drug relative to body weight, the deeper the pigmentation.

BLEACHING OF INTRINSIC TOOTH DISCOLORATION

Vital bleaching of intrinsically discolored teeth became a popular dental cosmetic procedure during the late twentieth. The accepted procedures incorporate the use of a peroxide compound placed on the tooth surface that may be accelerated by heat, light, or laser radiation that bleaches the intrinsic tooth pigments to a lighter hue.

MICROGNATHIA

Micrognathia is usually congenital, but it may be acquired in later life. The mandible is most often affected. The etiology of **congenital micrognathia** is related to deficient nutrition of the mother and intrauterine injury (pressure or trauma). In addition, it may be a part of the Robin sequence. **Acquired micrognathia** may develop gradually and is usually related to ankylosis of the jaw caused by a birth injury or trauma or infection in the temporomandibular joint.

Infants with mandibular micrognathia have difficulty in breathing and experience episodes of cyanosis; they must be kept in a ventral position as much as possible.

Mandibular advancement by orthopedic force is sometimes recommended, as well as surgical mandibular reconstruction. In cases of true ankylosis of the mandible, arthroplasty should be recommended.



ANOMALIES OF THE TONGUE

MACROGLOSSIA

Macroglossia refers to a larger-than-normal tongue size and may be either congenital or acquired. Congenital macroglossia, which is caused by an overdevelopment of the lingual musculature or vascular tissues, becomes increasingly apparent as the child develops. An abnormally large tongue is commonly observed in hypothyroidism, type 2 glycogen-storage disease, neurofibromatosis type 1, and Down syndrome.

Both allergic reaction and injury can cause such severe enlargement of the tongue that a tracheotomy is necessary to maintain a patent airway.

A disproportionately large tongue may cause both an abnormal growth pattern of the jaw and malocclusion. Flaring of the lower anterior teeth and an Angle Class III malocclusion .

The treatment of macroglossia depends on its cause and severity. Surgical reduction of a portion of the tongue is occasionally necessary.

ANKYLOGLOSSIA (TONGUE-TIE)

In ankyloglossia a short lingual frenum extending from the tip of the tongue to the floor of the mouth and onto the lingual gingival tissue limits movements of the tongue and causes speech difficulties.

Surgical reduction of the abnormal lingual frenum is indicated if it interferes with the infant's nursing (frenotomy).

In the older child, a reduction of the frenum by lingual frenectomy, or frenuloplasty should be recommended only if local conditions or speech problems warrant the treatment.

FISSURED TONGUE

A fissured tongue is seen in a small number of children and may be of no clinical significance, although it is sometimes associated with hypothyroidism and Down syndrome, and vitamin B–complex deficiency.



Treatment of the fissured tongue is usually by brushing of the tongue and improved oral hygiene aid in reducing the inflammation and soreness.

GEOGRAPHIC TONGUE (BENIGN MIGRATORY GLOSSITIS)

Geographic tongue is often detected during routine dental examination of pediatric patients who are unaware of the condition. Red, smooth areas devoid of filiform papillae appear on the dorsum of the tongue. The margins of the lesions are well developed and slightly raised. The involved areas enlarge and migrate by extension of the desquamation of the papillae at one margin of the lesion and regeneration at the other. Every few days, a change can be noted in the pattern of the lesions. The condition is self-limiting.

COATED TONGUE

A white coating of the tongue is usually associated with local factors. The amount of coating on the tongue varies with the time of day and is related to oral hygiene and the character of the diet. The coating consists of food debris, microorganisms, and keratinized epithelium found on and around the filiform papillae.

Children who have a congenital or acquired deficiency in salivary flow, systemic disease with associated fever and dehydration may have a white coated tongue which may become stained with foods or drugs.

Increased ingestion of liquid is appropriate to alleviate this situation. Brushing the tongue with a toothbrush and dentifrice reduces the coating.

WHITE STRAWBERRY TONGUE

An enlargement of the fungiform papillae extending above the level of the white desquamating filiform papillae gives the appearance of an unripe strawberry. The condition has been observed in cases of scarlet fever and Kawasaki disease in young children. The tongue returns to normal after recovery from the systemic condition.

BLACK HAIRY TONGUE

Black hairy tongue is rarely seen in children but occurs in young adults and has been related to the oral and systemic intake of antibiotics, smoking, and excessive



ingestion of dark drinks such as coffee and tea.

MEDIAN RHOMBOID GLOSSITIS

It is an oval, rhomboid, or diamond- shaped reddish patch on the dorsal surface of the tongue immediately anterior to the circumvallate papillae.

It stands out distinctly from the rest of the tongue because it has no filiform papillae.

This atrophic area is usually asymptomatic. It had been believed to be a developmental anomaly, the condition is now recognized almost exclusively to represent a chronic, localized, and mild candidal infection.

Treatment with topical antifungal agents is appropriate.

TRAUMA TO THE TONGUE & TONGUE PIERCING

A child may bite his or her tongue as a result of a traumatic blow or fall. The dentist may inadvertently traumatize the tongue with a cutting instrument during operative procedures.

Deep laceration of the tongue requires suturing to minimize scarring and to aid in hemorrhage control.

Tongue piercing, a deliberate trauma, is one of the popular types of body piercing occurring in all parts of the world today, especially among teenagers and young adults.

Fractured teeth, dental abrasion, and gingival recession, brain abscess, cephalic tetanus, endocarditis, Ludwig's angina, and upper airway compromise are reported to be common complications following piercing procedures. Patients should be advised to remove them during athletic activities in which the risk of injury is high.

ABNORMAL LABIAL FRENUM

A maxillary midline diastema is frequently seen in preschool children and in those in the mixed-dentition stage. It is important to determine whether the diastema is normal (ugly duckling) or is related to an abnormal maxillary labial frenum. Many dentists delay considering an abnormal labial frenum as the cause of a diastema until all the



maxillary permanent anterior teeth, including the canines, have erupted.

The abnormal labial frenum, in addition to causing a midline diastema, can produce other undesirable clinical conditions such as the difficulty to place the brush at the proper level in the vestibule to brush in the conventional manner, stretching of the lip during mastication and speech may cause stripping of the tissue from the neck of the tooth, also it may cause the accumulation of food particles and eventual pocket formation, and it may restrict movements of the lip, interfere with speech, and may produce an undesirable cosmetic result.

يا بنيَ ...

أحكِم سفينتك فإن بحرك عميق، وخفّف حملك فإن العقبة كؤود، وأكثر الزاد فإن السفر بعيد، وأخلص العمل فإن الناقد بصير

يا بنيَ ...

كن أميناً تعش غنياً