Developmental Disorders of Oral Cavity

Prof. Shaleen Chandra
• Developmental disturbances
  • Jaws
  • Lips and palate
  • Gingiva
  • Oral mucosa
  • Tongue
  • Salivary gland
  • Tooth size
  • Tooth shape
  • Tooth structure
DEVELOPMENTAL DISTURBANCES OF JAWS

1. Agnathia
2. Micrognathia
3. Macrognathia
4. Facial Hemihypertrophy
5. Facial Hemiatrophy
AGNATHIA (OTOCEREBRALIA)

• Hypoplasia / absent mandible
• Autosomal recessive
• Unilateral missing jaw
• Ramus → ear deformities
• Etiology
  • Failure of migration of Neural crest cells into maxilary prominence in 4-5th week gestation
MICROGNATHIA

• Small jaw
• DD ➔ Abnormal positioning
• Classification
  • Congenital
    • Congenital heart disease
    • Pierre Robin syndrome
    • Maxillary micrognathia ➔ mouth breathing
  • Acquired
    • TMJ ➔ trauma, infection, ankylosis
• Congenital conditions
  • Catel-Manzke syndrome
  • Cerebrocostomandibular syndrome
  • Cornelia de Lange syndrome
  • Femoral hypoplasia - unusual facies syndrome
  • Fetal aminopterin-like syndrome
  • Miller-Dieker syndrome
  • Nager acrofacial dysostosis
  • Pierre Robin syndrome
  • Schwartz-Jampel-Aberfeld syndrome
  • van Bogaert-Hozay syndrome

• Intrauterine acquired conditions
  • Syphilis, congenital

• Chromosomal abnormalities
  • 49,XXXX syndrome
  • Chromosome 18 trisomy syndrome
  • Chromosome 8 recombinant syndrome
  • Chromosome 8 trisomy syndrome
  • Cri du chat syndrome 5p-
  • Turner's syndrome
  • Wolf-Hirschhorn syndrome

• Mendelian inherited conditions
  • CODAS (cerebral, ocular, dental, auricular, skeletal) syndrome
  • Diamond-Blackfan anemia
  • Noonan's syndrome
  • Opitz-Frias syndrome
• Autosomal dominant conditions
  • Camptomelic dysplasia
  • Cardiofaciocutaneous syndrome
  • CHARGE syndrome
  • DiGeorge's syndrome
  • Loeys-Dietz syndrome
  • Marfan syndrome
  • Micrognathia with peromelia
  • Pallister-Hall syndrome
  • Treacher Collins-Franceschetti syndrome
  • Trichorhinophalangeal syndrome type 1
  • Trichorhinophalangeal syndrome type 3
  • Wagner vitreoretinal degeneration syndrome
  • Weissenbacher-Zweymuller syndrome

• Autosomal recessive conditions
  • Bowen-Conradi syndrome
  • Carey-Fineman-Ziter syndrome
  • Cerebrohepatorenal syndrome
  • Cohen syndrome
  • Craniodiaphyseal dysplasia
  • De la Chapelle dysplasia
  • Dubowitz syndrome
  • Fetal akinesia-hypokinesia sequence
  • Hurst's microtia-absent patellae-micrognathia syndrome
  • Kyphomelic dysplasia
  • Lathosterolosis
  • Lethal congenital contracture syndrome
  • Lethal restrictive dermopathy

• X-linked inherited conditions
  • Atkin-Flaitz-Patil syndrome
  • Coffin-Lowry syndrome
  • Lujan-Fryns syndrome
  • Otopalatodigital syndrome type 2
  • Scott craniodigital syndrome

• Autoimmune conditions
  • Juvenile chronic arthritis
MACROGNATHIA

- Large jaws
  - Gigantism
  - Pagets disease
  - Acromegaly
  - Leontiasis ossea

- DD $\Rightarrow$ prognathism
  - Increased ramus height
  - Increased mandibular body length
  - Decreased maxillary length
HEMIFACIAL HYPERTROPHY

• Hyperplasia rather than hypertropy

• Syndromes associated
  • Beckwith Wiedmann syndrome
  • Neurofibromatosis
  • McCune Albright syndrome
  • Mafucci’s syndrome

• Classification (hoyme et al 1998)
  • Complex hemihyperplasia ➔ half of body
  • Simple Hemihyperplasia ➔ single limb
  • Hemifacial hyperplasia ➔ One half of face

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CLINICAL FEATURES

• F> M
• Macroglossia
• Premature development and eruption of teeth
  • Rowe et al
    • Crown size
    • Root size and shape
    • Rate of development

Histologically ➔ NO MUSCULAR HYPERTROPY
FACIAL HEMIATROPY

- Parry Romberg syndrome
  - Progressive atrophy of soft tissues
  - Confined to one half of face
- Etiology
  - Cerebral disturbance
    - Unregulated activity of sympathetic NS
  - Local trauma
    - Extraction of teeth
    - Infection
  - Genetic factors
CLINICAL FEATURES

- Painless cleft
- Coup de sabre (mid line of face)
- Bluish hue
  - Atropic fat
- Dental malformations
  - Incomplete root formation
  - Delayed eruption
  - Severe facial asymmetry
DEVELOPMENTAL DISTURBANCES OF LIPS AND PALATE
CONGENITAL LIP PITS AND COMMISURAL PITS

• Etiology
  • Notching of lip (early stage) \(\rightarrow\) fixation of tissue at the base of the notch
  • Failure of complete union of embryonic lateral sulci of lip
  • Commisural pits
    • Defective development of embryonic fissure

• Clinical features
  • Unilateral / bilatera
  • LL > UL
VAN DER WOUDE’S SYNDROME

- Autosomal dominant
- Deletion of chr 1q32 and alteration in chr 17p11
- Features
  - Cleft lip + palate
  - Pits of lower lip
  - Maxillary hypodontia
  - Syngnathia
  - Ankyloglosia
CHELITIS GLANDULARIS
(ACTINIC CHELITIS)

• Progressive enlargement and eversion of lower labial mucosa
• Exposure
• Erosion + ulceration + crusting
- Basophillic collagen degeneration
- Ductal ectasia, atrophy
- Hyperkeratosis and fibrosis

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CLASSIFICATION

• Simple type
  • Multiple painless, papules with central depression

• Superficial (suppurative) type
  • Baelz disease
  • Painless indurated swelling of lip with shallow ulceration

• Deep suppurative type
  • Deep seated abscess + sinus
CHEILITIS GRANULOMATOSA

• Melkersson Rosenthal syndrome
  • Granulomatous inflammation
  • Cheilitis
  • Facial nerve palsy
  • Plicated tongue

• Etiology
  • Genetic → siblings affected
CLINICAL FEATURES

• Chelitis and ulceration
  • Episodic
  • Nontender swelling
  • Cracked fissured lips
  • Red to brown discolouration
  • Fissured tongue → 20-40% cases
  • Facial nerve palsy → 30%
HISTOPATHOLOGY

- Tuberculoid granuloma
- Chronic inflammatory cell infiltrate
- Focal noncaseating granuloma
- Epitheloid cells
- Langhans cells
• Diagnosis
  • Serum ACE test
  • Chest radiograph
  • Gallium or positron emission tomography

• Rule out sarcoidosis
Orofacial clefts

• A developmental defect characterized by the failure of fusion of facial processes.
• 6\textsuperscript{th} and 7\textsuperscript{th} week $\rightarrow$ upper lip
• 8\textsuperscript{th} week $\rightarrow$ palate
  • Anterior to posterior
• Median nasal process vs maxillary process $\rightarrow$ cleft lip
• Maxillary process $\rightarrow$ cleft palate
ETIOLOGY

- **Heredity**
  - Single mutant gene
    - Syndromic (high risk)
  - Polygenic → low risk

*The total genetic liability of an individual reaches a certain minimum level.*

- Nutritional disturbances
- Physiological, emotional and traumatic stress
- Defective vascular supply
- Mechanical disturbances
- Infections
- Lack of inherent developmental force

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CATEGORIES

• CL +- CP ➔ same etiology

• CP ➔ separate etiology
MEDIAN CLEFT FACE SYNDROME

- Hyper telorism
- Median cleft of premaxilla and palate

Etiology
- Precocious limitation of growth of primary ossification centers on either side of mid line
- Failure to fuse
TREATMENT

• Multispeciality
  • Rule of 10
    • 10 weeks
    • 10 lbs
    • 10 mg / dl Hb
    • 10000 WBC count
  • Surgery, orthodontics, speech therapy
DEVELOPMENTAL DISTURBANCES
OF THE ORAL MUCOSA
FORDYCE’S GRANULES

• Heterotrophic collections of sebaceous glands
• Usually symmetrical
• Appear at puberty
  • Not all cases (*mile*)
    • Sebaceous nevi
FOCAL EPITHELIAL HYPERPLASIA

- HPV 13, 32
- Epithelium 8-10 times thicker
DEVELOPMENTAL DISTURBANCES OF GINGIVA
HEREDITARY GINGIVAL FIBROMATOSIS

• Benign → idiopathic
• Autosomal dominant
• Nodular form
• Clinical features
  • Dense, diffuse, growth
  • Crown may be hidden
  • No inflammation
  • Normal / pale colour
RETROCUSPID PAPILLA

- Hirshfield 1933
- Soft well circumscribed
- Between
  - Free gingival margin and
  - Mucogingival junction
- Elevated mucosal tag
  - Hyper orthokeratosis
  - Highly vascular CT
  - Large stellate fibroblasts
DEVELOPMENTAL ANOMALIES OF SALIVARY GLANDS

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ABERRANT SALIVARY GLANDS

- Location
  - Cervical region near parotid
  - Body of mandible
  - Region of brachial clefts and bronchial cleft cysts
  - Tongue

- Histology similar to the normal salivary gland
APLASIA AND HYPOPLASIA

• Along with congenital anomalies
  • Cleft palate
  • Mandibulofacial dysostosis

• Symptoms
  • Xerostomia
  • Dentinal caries
  • Melkerson Rosenthal syndrome
ACCESSORY DUCTS

• Common > 50% cases
• Superior and anterior to the normal stensons duct
• Rauch and Gorlin → 450 cases
DIVERTICULI

- Small pouches or out pocketings of the ductal system
- Recurrent acute parotitis
- Sialogram
POLYCYSTIC (DYSGENETIC) DISEASE OF PAROTID GLANDS

Least common
Developmental malformation of the duct

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CLINICAL FEATURES

- Female (7/8)
- Recurrent painless swelling of the involved gland
- Swelling is due to the anomaly of the gland
GROSS

- Exaggerated lobularity of the subcapsular surface
- Cut surface
  - Mottled yellow ivory nodules
  - With fine spongy consistancy
HISTOLOGY

- Lobules markedly distended
- Cysts → honey combed or lattice like appearance
- Squamous cuboidal or Columnar cells have abundant eosinophilic cytoplasm
- Lumen contain eosinophilic material
- Spheroliths and microliths
• The lobular architecture is preserved, but variably sized cysts have replaced the normal lobular-ductal units.

• The cysts are formed by dilatation of the ducts and are lined by attenuated epithelial cells.
DIFFERENTIAL DIAGNOSIS

- Mucoepidermoid carcinoma
- Acinic cell adenocarcinoma
- Cystadenocarcinoma
- Differentiation
  - Wide spread involvement
  - Variable epithelial lining
  - Presence of spherolitheis and microliths
  - Lack of inflammation
DEVELOPMENTAL DISTURBANCES OF TONGUE

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AGLOSSIA / MICROGLOSSIA SYNDROME

• Extremely rare

• Associated with
  • Anomalies of hand and feet
  • Cleft palate
  • Dental agenesis

• Microglossia
  • Lack of muscle stimulus
    • Mandible fails to grow forward
MACROGLOSSIA

• Papyrus Ebers 1550 BC
• True macroglossia
  • Congenital
  • Acquired
• pseudo macroglossia
  • Relative small jaw
  • Atonia
  • Vitamin deficiencies
  • Neoplasms displacing tongue
• Congenital
  • Muscle hypertrophy
  • Gland hyperplasia
  • Downs syndrome
  • Beckwith’s weidmann’s
  • Lymphangioma
  • Gargoylism

• Acquired
  • Hypothyroid
  • Syphilis
  • Candidiasis
  • Acromegaly
  • Amyloidosis
  • Sarcoidosis
ANKYLOGLOSSIA

• Short lingual frenum
• Speech problem

• Frenectomy
CLEFT TONGUE

• Deep groove in midline of the dorsal tongue
• Associated with
  • Orofacial digital syndrome
FISSURED TONGUE (SCROTAL TONGUE)

- Grooves of varying depth
- Melkersson Rosenthal syndrome
  - Facial palsy
  - Chelitis granulomatosa
  - Fissured tongue
- Downs syndrome

Histology: loss of filiform papillae and neutrophillic microabscesses
MEDIAN RHOMBOID GLOSSITIS

- Dorsal surface of the tongue along the midline, just anterior to the foramen cecum
- rhomboid or oval, well-demarcated shape
- red, flat or slightly multilobulated smooth, depapillated surface
- 1 to 3 cm
- usually asymptomatic
• Re-termed as POSTERIOR MIDLINE ATROPHIC CANDIDIASIS
• Atrophic stratified squamous epithelium
• Moderately fibrous CT
• Chronic candidal infection
• Always antifungal therapy prior to biopsy
BENIGN MIGRATORY GLOSSITIS

- Psorasiform mucositis
- Multiple sensitive irregularly shaped erythematous patches on the tongue
- Arcuate white rims that enlarge and change

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• Associations with human leukocyte antigen DR5 (HLA-DR5), DRW6 (HLA-DRW6), and Cw6 (HLA-Cw6)

• Similar to psoriasis

• Histopathology
  • Neutrophillic exocytosis
  • Monro’s abscess
  • Thin long rete ridges
  • Small epithelium over the papillae
HAIRY TONGUE

- Defective desquamation of filiform papillae
- Black – brown to white

Etiology
- Hypertrophy of filiform
- Lack of mechanical stimulation
- Tobacco
- Coffee
CLINICAL FEATURES

- M > F
- 1 – 15 mm papillae
- Tickling soft palate
- Asymptomatic
  - Candida → glossopyrosis
- Halitosis
HISTOLOGY

• Mild elongated papillae
• Mild hyper keratosis
• Occasional inflammatory cells
• Accumulated debris
LINGUAL VARICES

• Varix ➔ Dilated, tortuous Vein
  • Increased hydrostatic pressure
  • Poorly supported by surrounding tissue

• Lingual Ranine veins
  • Red to purple shot like cluster of vessels
  • Ventral and lateral surfaces
• No direct association between varicosities and organic diseases

• Kleinman
  • Aging process
  • < 50 years if present
    • Premature aging
DEVELOPMENTAL DISTURBANCES INVOLVING THE TOOTH SIZE
MICRODONTIA

- Teeth smaller than normal

Types

- True generalised
  - All teeth smaller than normal
  - Pitutary dwarfism
- Relative generalized
  - Normal or slightly smaller
  - Jaws larger
- Microdontia of single tooth
  - Maxillary lateral (peg lateral)
  - Third molar

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MACRODONTIA

- True generalised macrodontia
  - Pitutary gigantism
- Relative generalised
  - Hereditary
  - Relative larger size
- Macrodontia of single teeth
  - Hemihypertrophy
    - One side larger
DEVELOPMENTAL DISTURBANCES OF SHAPE OF TEETH

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GEMINATION

- Attempted division of single tooth germ
- Complete or incompletely separated crowns
  - Single root and root canal
- DD
  - Fusion b/n normal teeth and supernumerary tooth
TWINNING

- Schizodontia

- Complete cleavage of tooth bud
- Extra tooth formation
  - One normal and one supernumerary
FUSION (SYNODONTIA)

- Union of two normally separated tooth germs
  - Complete / Incomplete
  - Before calcification
    - Complete fusion
  - Only roots
- Pathogenesis
  - Physical force / pressure
- Deciduous > permanent

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CONCRESCEENCE

• Form of fusion
  • After root completion
  • United by cementum

• Cause
  • Trauma
  • Crowding

• Types
  • True → union during dev
  • Acquired → after root completion → hypercementosis

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DILACERATION

• Angulation
  • Sharp bend or curve
  • Root / crown of tooth

• Etiology
  • Trauma
  • Deciduous injures the permanent bud

• Radiograph always needed prior to extraction
TALON’S CUSP

- Cingulum areas
  - Maxillary or mandibular incisors
  - Deep developmental grooves
  - Normal enamel and dentine
  - Normal pulp horn

- Rubinstein Taybi syndrome
  - Developmental retardation
  - Broad thumb’s and great toes
  - Incomplete decent of testes

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DENS IN DENTE (DENSI
INVAGINATUS)

• Etiology
  • Invagination in the surface of tooth crown before calcification
  • Growth retardation
  • Trauma → localised external pressure
  • Focal growth stimulation

• Maxillary lateral incisors
  • Accentuation of lingual pit
CLASSIFICATION

• Oehler’s

• Hallet’s 1953
  • Type 1:
    • Definite cleft parallel
    • No expansion
  • Type 2
    • Extends towards pulp chamber
  • Type 3
    • Deep into pulp chamber + dialated
  • Type 4
    • Occludes coronal pulp chamber
    • Beyond CEJ

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DENS EVAGINATUS

• Leong’s premolar
• Pathogenesis
  • Proliferation and evagination
  • Odontogenic mesenchyme
• Clinical features
  • Mongoloid ancestry
  • Accessary cusp
  • Globule of enamel between cusps
  • Extra cusp → displacement of teeth, pulp exposure
TAURODONTISM

- Sir Arthur Keith 1913
- Bull like teeth
- Body of the teeth expanded at the expense of root.
- Shaw classification
  - Hypotaurodont (mildest)
  - Mesotaurodont
  - Hypertaurodont (at apex)
CAUSES OF TAURODONTISM

- Mendelian recessive trait
- Atavistic feature
- Mutation resulting from odontoblastic deficiency
- Failure of hertwigs root sheath to invaginate at proper horizontal level
• may occur in patients with
  • amelogenesis imperfecta,
  • Down syndrome, and
  • Klinefelter syndrome
    • Due to extra X
    • Male patients with taurodontism must have chromosome analysis performed
SUPERNUMERARY ROOTS

• Common
• Single root ➔
  • mandibular bicuspid & cuspids
• Molars most commonly affected
• Significant in exodontia
DEVELOPMENTAL DISTURBANCES IN NUMBER OF TEETH

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ANODONTIA

• True anodontia
  • Total
    • All the teeth are missing
    • May involve deciduous and permanent dentition
    • Hereditary ectodermal dysplasia
  • Partial
    • Hypo/oligodontia
    • $3^{rd}$ molar > max lateral > second molar
• Pseudo anodontia
  • Total extraction
ANODONTIA : ETIOLOGY

- Familial tendency
- Point mutations
- Autosomal dominant

- X-ray irradiation
  - Single quadrant teeth missing
SUPERNUMERARY TEETH

• Etiology
  • Extra tooth bud
  • Splitting of tooth bud
  • Hyperactivity theory
    • Local independent, conditioned hyperactivity of dental lamina

• Associated with
  • Cleft lip and palate
  • Cleidocranial dysplasia
  • Gardner syndrome
CLASSIFICATION: SUPERNUMERARY TEETH

- Morphology and location
  - Conical
  - Tuberculate
  - Supplemental
  - Odontome

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TYPES

• Conical
  • This small peg-shaped conical tooth
  • Commonly found in the permanent dentition
  • It develops with root formation ahead of or at an equivalent stage to that of permanent incisors and usually presents as a mesiodens.
    • inverted into the palate
    • horizontal position.
    • result in rotation or displacement of the permanent incisor, but rarely delays eruption.
• Tuberculate
  • More than one cusp or tubercle.
  • barrel-shaped and may be invaginated
  • Root formation is delayed compared to that of the permanent incisors.
  • Often paired
  • Commonly located on the palatal aspect of the central incisors.
• Supplemental
  • Duplication of teeth in the normal series and is found at the end of a tooth series
  • The most common: permanent maxillary lateral incisor,
  • Majority supernumeraries found in the primary dentition are of the supplemental type
ODONTOMA (HOWARD)

• Category is not universally accepted
• Hamartomatous malformation rather than a neoplasm.
• Two types
  • complex composite odontoma
    • the diffuse mass of dental tissue which is totally disorganized
  • compound composite odontoma.
    • the malformation which bears some superficial anatomical similarity to a normal tooth
GARDNER’S SYNDROME

- Desmoid tumours
- Osteomas
- Polyposis of large intestine
- Sebaceous cysts
- Impacted supernumerary teeth

**Cause**
- Pleiotropic gene
- Autosomal dominant
- Complete penetrance

- Fader and Duncan

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PREDECIDUOUS DENTITION

• Hornified epithelial structures
• Over the crest of ridge on the gingiva

• At birth → natal teeth
• < 28 days eruption → neonatal teeth
DEVELOPMENTAL DISTURBANCES IN STRUCTURE OF TEETH
ENAMEL HYPOPLASIA

• Incomplete or defective formation of organic enamel matrix

• Types
  • Hereditary
    • Amelogenesis imperfecta
  • Environmental
    • Nutritional deficiency (Vit A, C, D)
    • Exanthematous diseases
    • Congenital syphilis
    • Birth injury
    • Ingestion of chemicals
    • Idiopathic causes
AMELOGENESIS IMPERFECTA

- Autosomal dominant
- Autosomal recessive
- X-linked

Types
- Hypoplastic (60-73%)
- Hypocalcified (7%)
- Hypomature (20-40%)
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ETIOLOGY

• Alterations in genes involved in formation and maturation of enamel

• DXS 85 at Xp22
  • Localization of amelogenin (AMELX and AMELY)

• Other genes involved
  • AMBN → ameloblastin
  • Enamelin → Multiple mutations ENAM gene mutations are associated with different autosomal inherited AI types
• Enamelysin:
  • *MMP20* gene located on chromosome 11
  • proteinase that cleaves amelogenin for processing the enamel matrix proteins
  • Enamelysin knockout mouse has a reduced enamel thickness, poorly mineralized enamel and the enamel lacks a prismatic structure.

• Kalikryn 4:
  • *KLK4* gene located on chromosome 19
  • Proteinase that is secreted predominantly during the maturation stage
  • Mutation of *KLK4* is associated with autosomal recessive hypomaturation AI that is characterized by poorly mineralized enamel.

• Tuftelin
CLINICAL FEATURES

- teeth vary in color from white opaque to yellow to brown
- all teeth are affected, smaller and pitted
- normal pulps and dentin but reduced enamel
• Few small grooves
• Pits/ fissures
• Severe deep rows of pits
• Portion of enamel missing
Hypocalcified type

Smooth type

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HISTOLOGY

• Hypoplastic type
  • Disturbance of differentiation and viability of ameloblasts

• Hypo calcified type
  • Defects of matrix structure and mineral deposition

• Hypomaturation
  • Alterations in enamel rod and rod sheath structures
NUTRITIONAL DEFICIENCY AND EXANTHEMATOUS DISEASES

• Ameloblasts most sensitive
• Usually pitting variety
• 1 year after birth
  • Central
  • Lateral
  • Cuspid and
  • 1st molars affected
CONGENITAL SYPHILLIS

- Hutchinsons teeth
- Moons molars
- Hutchinsons triad
HYPOCALCEMIA

- Pitting variety
- Ca++ less than 6-8 mg / 100 ml
- Tetany
  - Vitamin D deficiency
  - Parathyroid deficiency
HYPOPLASIA DUE TO BIRTH INJURIES

• Permanent maxillary incisors
• Maxillary / mandibular premolar
• Mild brownish discolouration → severe pitting

• TURNER’S TEETH / TURNER’S HYPOPLASIA
DENTINOGENESIS IMPERFECTA

- Autosomal dominant
  - chromosome #4
  - Dentin sialophosphoprotein

- Affects both deciduous and permanent teeth

- Gray to yellowish brown
  - Tulip shape
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PRESENT CLASSIFICATION

• Dentinogenesis imperfecta 1
  • Dentinogenesis without osteogenesis imperfecta

• Dentinogenesis imperfecta 2
  • Brandywine type
DENTINOGENESIS IMPERFECTA 1

- Mutation in DSPP gene chr 4q21.3
  - Encodes dentin phosphoprotein and sialoprotein
- Blue gray or amber brown opalescent
- Enamel may split readily
DENTINOGENESIS IMPERFECTA 2

- Brandywine triracial isolate in Maryland

- Clinical features
  - Rapid loss of enamel
  - Large pulp chambers
  - Shell teeth

- Dentin sialophosphoprotein + dentinmorphogenic protein + bone sialoprotein
HISTOPATHOLOGY: DI

- Enamel normal
- Irregular tubules
- Areas of complete absence of tubules
- Physical characters
  - Reduced
    - water content
    - X-ray absorption
    - density
DENTIN DYSPLASIA

- Normal enamel
- Atypical dentin + abnormal pulp morphology
- Classification (WITKOP)
  - Type 1: Radicular dentin dysplasia (rootless teeth)
  - Type 2: Coronal dentin dysplasia
RADICULAR DENTIN

- Autosomal dominant
- Both dentition affected
- Clinically → Appears normal
- Root is stunted
- Radiographically
  - Obliteration of pulp chamber
  - PA granuloma / cyst with out obvious reason

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- **Histology**
  - Obliterated pulp chamber
  - Tubular dentin
  - Fused denticles
  - Osteodentin
  - Appearance of lava flowing around boulders
CORONAL DENTIN

- Autosomal dominant
- Both dentition affected
- Deciduous teeth
  - Appear yellow brown to blue
  - Complete obliteration
- Permanent normal
  - Thistle tube
  - Pulp stone most characteristic

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HISTOLOGY

• Deciduous tooth
  • Coronal dentin normal
  • Radicular dentin $\rightarrow$ atubular dentin

• Permanent
  • Normal
  • Pulp stones
REGIONAL ODONTOGENIC DYSPLASIA

- Maxillary anterior region > mandible
- Etiology
  - Remnant viral infection
  - Vascular malformation (associated vascular nevi)
• **DELAYED ERUPTION**
  
  • Lack of calcification
  
  • Lack of density → **GHOST LIKE TEETH**
HISTOPATHOLOGY

• Pathology
  • Little amount of enamel and dentin
  • More predentin
  • MORE interglobular dentin
  • Follicular tissue around the crown is calcified
    • Enameloid conglomerates
CONCLUSION

• Developmental disorders
  • Variations in structure

• Oral manifestations may be a clue to many serious systemic unknown manifestations

• Less role of histopathology
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