DIVING INTO THE WORLD OF DENTAL ANOMALIES

Clinical Reference Handout | @ThePathoRDH | Andreina Sucre RDH, MSc | ThePathoRDH.com

01 THE FRAMEWORK: HOW TO THINK THROUGH DENTAL ANOMALIES

"Instead of memorizing anomalies one by one — organize them through two lenses: NUMBER and STRUCTURE. When you think in patterns, you recognize them in real patients."

NUMBER ANOMALIES

- ↓ Less teeth

Hypodontia · Oligodontia · Anodontia

- ↑ More teeth

Hyperdontia (Mesiodens · Paramolar · Distomolar)

- Retained primary teeth

STRUCTURE ANOMALIES

- Size: Microdontia · Macrodontia
- Enamel: Hypoplasia · AI
- Dentin: DI · Regional Odontodysplasia
- R&C; Morphology: Fusion · Gemination · Taurodontism · Dilaceration

EMBRYOLOGICAL STAGE → WHEN DID IT GO WRONG?

Developmental Stage	Anomalies Associated
Initiation Stage (wk 6–8)	Agenesia / Hyperdontia
Bud Stage (wk 8)	Macrodontia / Microdontia
Cap Stage (wk 9–10)	Fusion · Gemination
Apposition Stage	Enamel Hypoplasia (quantitative defect)
Maturation Stage	Hypomineralization / Hypocalcification (qualitative defects)
Post-Bell / Root Stage	Taurodontism · Dilaceration · Concrescence · Supernumerary roots
Any disrupted stage	Amelogenesis Imperfecta · Dentinogenesis Imperfecta

02 NUMBER ANOMALIES: AGENESIA SPECTRUM

TYPE	DEFINITION	PREVALENCE	MOST AFFECTED TEETH / NOTES
HYPODONTIA	Up to 5 missing teeth (excl. 3rd molars)	1.6 – 6.9%	Upper lateral incisors, 2nd premolars
OLIGODONTIA	6+ missing teeth (excl. 3rd molars)	0.1 – 0.3% (1:1,000)	Upper laterals, 2nd premolars lower (88.2%) & upper (87%)
ANODONTIA	Complete absence of ALL teeth (primary + permanent)	1:10,000	Associated: Ectodermal Dysplasia Gene mutations: EDA, EDAR, EDARADD, PAX9, MSX1

"Missing teeth are not empty spaces — they are missing developmental events in time."

03 HYPERDONTIA: SUPERNUMERARY TEETH

DISTOMOLARS vs. PARAMOLARS

FEATURE	DISTOMOLARS (4th Molars)	PARAMOLARS
LOCATION	Behind 3rd molars (distal)	Adjacent to 1st or 2nd molars
MORPHOLOGY	Resembles molar or rudimentary	Often peg-shaped, conical, or tuberculated
FREQUENCY	Less common	More common
COMPLICATIONS	Impaction, crowding, cyst formation	Occlusal interference, food impaction, perio issues

SYNDROME	GENE	KEY DENTAL + SYSTEMIC FEATURES
CLEIDOCRANIAL DYSPLASIA	RUNX2 gene	Multiple impacted supernumeraries, clavicular hypoplasia, delayed skull sutures
GARDNER SYNDROME	APC gene	Multiple supernumeraries, odontomas, jaw osteomas — COLON CANCER RISK → refer
FABRY DISEASE	GLA gene (X-linked)	Supernumerary teeth, enamel defects, delayed eruption
EHLERS-DANLOS	Collagen synthesis mutations	Supernumerary teeth, fragile oral mucosa, hypermobile joints

04 SIZE ANOMALIES: MACRODONTIA & MICRODONTIA

TYPE	MACRODONTIA	MICRODONTIA
TRUE GENERALIZED	All teeth genuinely oversized	All teeth proportionally smaller
RELATIVE GENERALIZED	Normal teeth in a smaller jaw (space problem, not tooth problem)	Small teeth in a larger jaw (proportional mismatch)
SINGLE / LOCALIZED	One enlarged tooth — rule out gemination May coexist with Turner's hypoplasia	Most common form Peg lateral incisor / 3rd molar most affected

"When you see a single tooth looking enlarged — slow down. Macrodontia can coexist with Turner's Hypoplasia on the same tooth — two events, two different stages of development. You're not looking at one problem. You're looking at a timeline."

05 FUSION VS. GEMINATION

FEATURE	FUSION (SYNODONTIA)	GEMINATION (TWINNING)
WHAT IS IT?	TWO separate tooth buds fuse together forming ONE large tooth	ONE tooth bud attempts to divide but fails — results in a bifid crown with one root
TOOTH COUNT	REDUCED (one tooth where two should be)	NORMAL (extra crown, normal count)
CLINICAL	Single large tooth with multiple cusps; may have >1 root canal opening	Two separate crowns on a single root; crowns may be normal or slightly smaller
RADIOGRAPHIC	Large single image, complex internal structure, possible multiple pulp chambers	Two separate crowns with a single root structure
FREQUENCY	Less common	More common than fusion
THE HINT	Two adjacent teeth MERGED → fewer teeth than expected	ONE tooth with BIFID crown → normal number of teeth

06 ENAMEL DEFECTS

AMELOGENESIS IMPERFECTA (AI) — CLASSIFICATION

TYPE	CLINICAL FEATURES	STAGE AFFECTED	GENE
TYPE 1 — HYPOPLASTIC	Thin, pitted enamel; hard surface	Apposition	ENAM, AMELX mutations
TYPE 2 — HYPOCALCIFIED	Normal thickness but soft, chalky, yellow-brown, porous	Maturation	FAM83H mutations
TYPE 3 — HYPOMATURE	Normal thickness, soft mottled enamel "snow-capped" appearance; rapid wear	Late Maturation	MMP20, KLK4 mutations
TYPE 4 — MIXED + TAURODONTISM	Enamel defects + enlarged pulp chambers; taurodontic molars	Mixed stages	AMELX (X-linked)

AMELOGENESIS IMPERFECTA vs. ENAMEL HYPOPLASIA

FEATURE	AMELOGENESIS IMPERFECTA	ENAMEL HYPOPLASIA
CAUSE	Genetic mutations	Genetic OR environmental (nutritional deficiencies, childhood illness)
INHERITANCE	Follows specific inheritance patterns	May occur without family history
DISTRIBUTION	Majority of teeth, relatively uniform	Can be localized (Turner's) or generalized
OTHER ISSUES	May have dentin problems + sensitivity	May not cause issues beyond enamel defect

QUANTITY vs. QUALITY — ENAMEL PROBLEMS AT A GLANCE

DEFECT TYPE	Enamel Hypoplasia	Hypomineralization / Hypocalcification	Decalcification
	Apposition	Maturation	Post-eruptive
	Missing / reduced (pits, grooves)	Present but soft / porous	Present — minerals lost after eruption
	Before eruption	Before eruption	After eruption
	Pits, grooves, missing surface	Soft, chalky, mottled	White patches → can progress to cavity

"One was never built correctly… the other is being broken down."

07 DENTIN DEFECTS: DENTINOGENESIS IMPERFECTA

FEATURE	DI TYPE I	DI TYPE II (most common)	DI TYPE III
SYSTEMIC ASSOC.	Osteogenesis Imperfecta	None	None
GENETIC MUTATION	COL1A1, COL1A2	DSPP	DSPP (variant)
TOOTH COLOR	Opalescent (gray-blue-brown)	Opalescent (amber-gray-brown)	Less intense discoloration
PULP CHAMBERS	Obliterated early	Obliterated early	"Shell teeth" — large chambers, thin dentin
ROOT SHAPE	Short, constricted	Short, constricted	Normal to thin roots
ENAMEL DEFECTS	Enamel fractures	Enamel fractures	Enamel fractures

DI TYPE II vs. OSTEOGENESIS IMPERFECTA (WITH DI TYPE I)

FEATURE	DI TYPE II	OI (WITH DI TYPE I)
GENETIC MUTATION	DSPP gene	COL1A1 or COL1A2 gene
SYSTEMIC INVOLVEMENT	None	Systemic connective tissue disorder
BONE FRAGILITY	Absent	Present: frequent fractures, skeletal deformities
TOOTH COLOR	Amber, gray, or brown opalescent	Opalescent (blue-gray or brown)

DI vs. TETRACYCLINE STAINING

FEATURE	DENTINOGENESIS IMPERFECTA	TETRACYCLINE STAINING
LAYER AFFECTED	Dentin	Dentin (primary) — secondary enamel involvement
CAUSE	Gene mutations	Tetracycline during tooth development (in utero or childhood)
CLINICAL ASPECT	Discoloration (yellow-brown, gray-blue) Rapid wear, increased fracture risk	Discoloration (yellow-brown, gray-blue) Banding pattern in severe cases

FEATURE	DENTINOGENESIS IMPERFECTA	TETRACYCLINE STAINING
DENTITIONS AFFECTED	Both primary and permanent	Both — depends on timing of exposure
TRANSLUCENCY	Translucent opalescent sheen	Darker, less translucent in severe cases

08 ROOT MORPHOLOGY ANOMALIES

TAURODONTISM — SEVERITY CLASSIFICATION

FEATURE	MILD (Hypotaurodont)	MODERATE (Mesotaurodont)	SEVERE (Hypertaurodont)
PULP CHAMBER	Slightly enlarged	Moderately enlarged (>half root length)	Extends close to root apex
ROOT LENGTH	Slight shortening	Noticeably shortened	Severely shortened or almost absent
CEJ CONSTRICTION	Slight decrease	More prominent decrease or absent	Complete absence
RADIOGRAPHIC	Enlarged rectangular pulp chamber	More pronounced enlargement	Extreme enlargement + very short roots

ROOT DILACERATION — KEY FACTS

DEFINITION
Sharp angulation, bend, or curve in root structure — anywhere along root length

ETIOLOGY
Trauma to developing tooth (main cause) · Mechanical interference from cysts/tumors (dentigerous, OKC) · Idiopathic (most cases)

PREVALENCE
1–3% (higher in males)

CLINICAL IMPORTANCE
Complicates extractions & RCT — always assess root morphology before treatment

REGIONAL ODONTODYSPLASIA ("Ghost Teeth") — KEY FACTS

DEFINITION
Rare developmental anomaly affecting enamel, dentin, and pulp in a localized jaw segment

RADIOGRAPHIC SIGN
Teeth appear "ghost-like" — thin enamel + dentin, enlarged pulp chambers, poorly defined structures

ETIOLOGY
Vascular/ischemic theory (most accepted) · Neural crest cell dysfunction · Local trauma or infection

PREVALENCE
Extremely rare: <1 per million · More common in maxilla

09 CLINICAL DECISION FRAMEWORK: PATTERN RECOGNITION

"When you see an anomaly — ask these 3 questions: Is it a NUMBER or STRUCTURE anomaly? Is it LOCALIZED or GENERALIZED? Is it GENETIC or ENVIRONMENTAL? These 3 questions will guide your thinking every time."

WHEN TO REFER — CLINICAL RED FLAGS

CLINICAL FINDING	ACTION
Syndromic pattern suspected	Multiple supernumeraries → Rule out Cleidocranial Dysplasia, Gardner Syndrome
Generalized enamel/dentin defects	AI or DI — multidisciplinary treatment planning needed
Oligodontia in a child	Psychological support + early restorative/prosthetic planning
Regional Odontodysplasia	Extraction often required — consult oral surgeon
Dilaceration before extraction/RCT	CBCT imaging to assess root angulation and plan treatment approach

MISTAKE	HOW TO AVOID IT
MISLABELING AI vs. HYPOPLASIA	AI = genetic · Hypoplasia = developmental disruption. Check family history + distribution before labeling.
IGNORING SUPERNUMERARY COMPLICATIONS	Impacted supernumeraries → cysts, root resorption, crowding. Always refer for imaging if found.
MISSING SYNDROMIC PATTERNS	Multiple anomalies in one patient = possible syndrome. Think systemically — the mouth is a window.

10 A FINAL WORD

Dental anomalies are not about memorizing names. They are about understanding development.
Because when you understand development — you start seeing differently.

Recognition is step one. Clinically, the real question is: **Do I monitor · Refer · Educate?**

Most mistakes don't happen because we don't know. They happen because we don't pause and think.

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