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Endodontic Assessment

Mucocele

Learning Objectives:
Gain understanding of detection of developmental anomalies of the dentition.

Be able to identify radiographically the following anomalies: enamel hypoplasia, amelogenesis imperfecta, dentinogenesis imperfecta, radicular dentin dysplasia, coronal dentin dysplasia and odontodysplasia.

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Panoramic Radiologic Appraisal of Anomalies of the Dentition: Chapter 4 – Tooth Structure

By Dr. Allan G. Farman

Developmental anomalies in tooth structure can involve dental enamel, dentin, pulp, cementum, or a combination of these tissues. While relatively precise typing of some of these anomalies is now possible using techniques in molecular biology, radiography remains important in the assessment of phenotypic manifestations and is often essential in treatment-planning for esthetics and function of dental restorations. The panoramic radiograph provides a useful overview of the dentition both for dental anomalies affecting all teeth and for those that are localized to a specific region. The panoramic radiograph may need to be supplemented by selected periapical images when restorative procedures are to be planned.

Enamel Hypoplasia

Enamel hypoplasia is either an inherited imperfect enamel formation (amelogenesis imperfecta), or "environmental hypoplasia" acquired during development due to local or systemic influences [1]. Systemic conditions will affect the portion of the crown being formed during the influence of the condition. Systemic conditions associated with enamel hypoplasia include birth-related trauma (Fig. 1D), certain chemicals (e.g. excess fluoride – Fig. 1C, tetracycline, thalidomide), infections (e.g. chicken pox, measles, rubella, syphilis – Fig. 1B), malnutrition, and metabolic disorders [1-4], (See Figure 1). Aine *et al.* (2000) determined that the prevalence of enamel defects in children born prematurely was significantly higher compared with controls in both the primary (78 % versus 20 %, $p < 0.001$) and permanent (83 % versus 36 %, $p < 0.001$) dentitions [5]. Low birth weight is also associated with a significantly increased rate of enamel hypoplasia. Ninety-six percent of low birth weight versus 45 % of the normal control children had at least

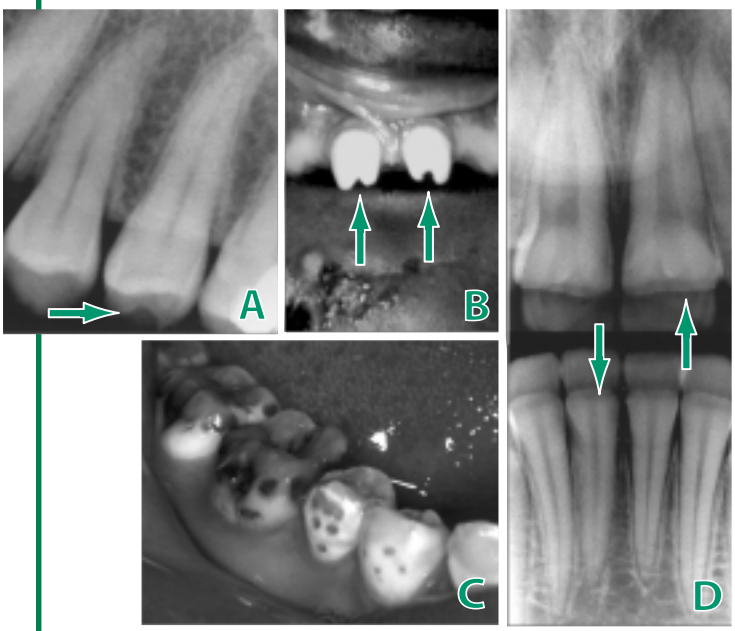


Fig. 1: Enamel Hypoplasia: **(A)** The second premolar is a Turner's tooth with hypoplasia (arrow) likely due to local infection from an abscessed primary second molar. **(B)** Hutchinson's incisors due to congenital syphilis showing typical notching of the incisor edges (arrow). **(C)** Enamel hypoplasia due to dental fluorosis. **(D)** Enamel hypoplasia in a band (arrows) representing the degree of crown formation of the permanent teeth at the time of birth trauma.

" Local causes of enamel hypoplasia include inflammatory disease from a primary tooth with the underlying permanent tooth becoming a Turner's tooth, local infections, local mechanical or electrical trauma, or childhood radiotherapy."

one tooth with enamel defects (a mean of 8 teeth were affected per low birth weight child versus a mean of one affected tooth per control child; $p < 0.001$)[6]. Nunn *et al*. (2000) found 22 % of children with renal disease to have enamel hypoplasia [7]. Local causes of enamel hypoplasia include inflammatory disease from a primary tooth with the underlying permanent tooth becoming a Turner's tooth (Fig. 1A), local infections, local mechanical or electrical trauma, or childhood radiotherapy [1].

Amelogenesis Imperfecta

Amelogenesis imperfecta (AI) includes a variety of developmental alterations in enamel structure unrelated to systemic disease. The reported prevalence of AI varies from 1:700 to 1:8,000 depending on the population studied [1]. Both primary and permanent dentitions are affected. According to Witkop [8] there are at least 14 different varieties of AI, which can be divided according to the enamel development stage affected, namely: elaboration of organic matrix (hypoplastic), mineralization of the matrix (hypocalcified), and maturation of enamel (hypomaturation). AI has seen much recent publication of findings regarding genetic causations [e.g. 9-12]. This review will, however, keep to phenotypic signs as is appropriate to a paper concerning radiologic signs.

Hypoplastic AI: These conditions are caused by inadequate enamel matrix deposition. Generalized pitted AI (Type IA) is autosomal dominant with affected teeth displaying horizontal rows of pits or linear depressions. Localized pitted AI can be dominant (Type IB) or recessive (Type IC), the latter typically being the more widespread and severe of these localized varieties.

Smooth hypoplastic AI (Fig. 2) results in smooth, glossy enamel of less than regular thickness. The tooth

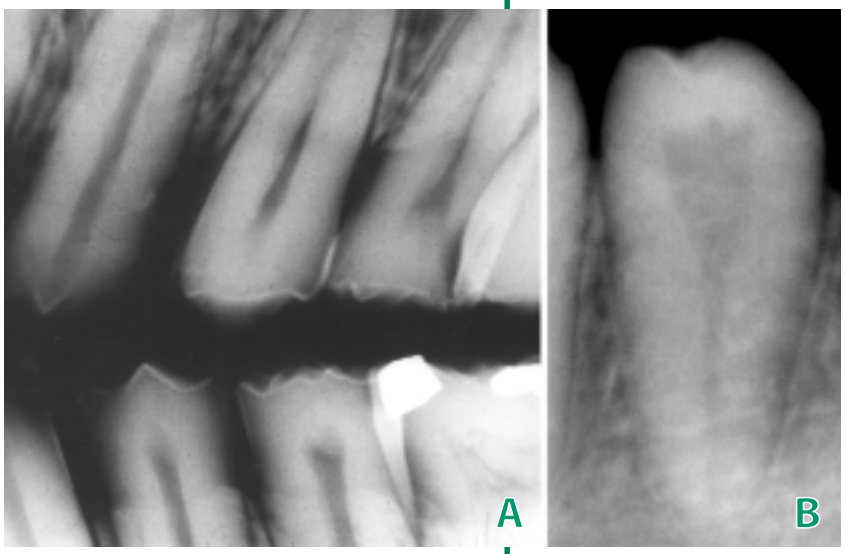


Fig. 2: Smooth hypoplastic Amelogenesis Imperfecta. Two cases **(A & B)** where the enamel is much thinner than normal but smooth in surface configuration.

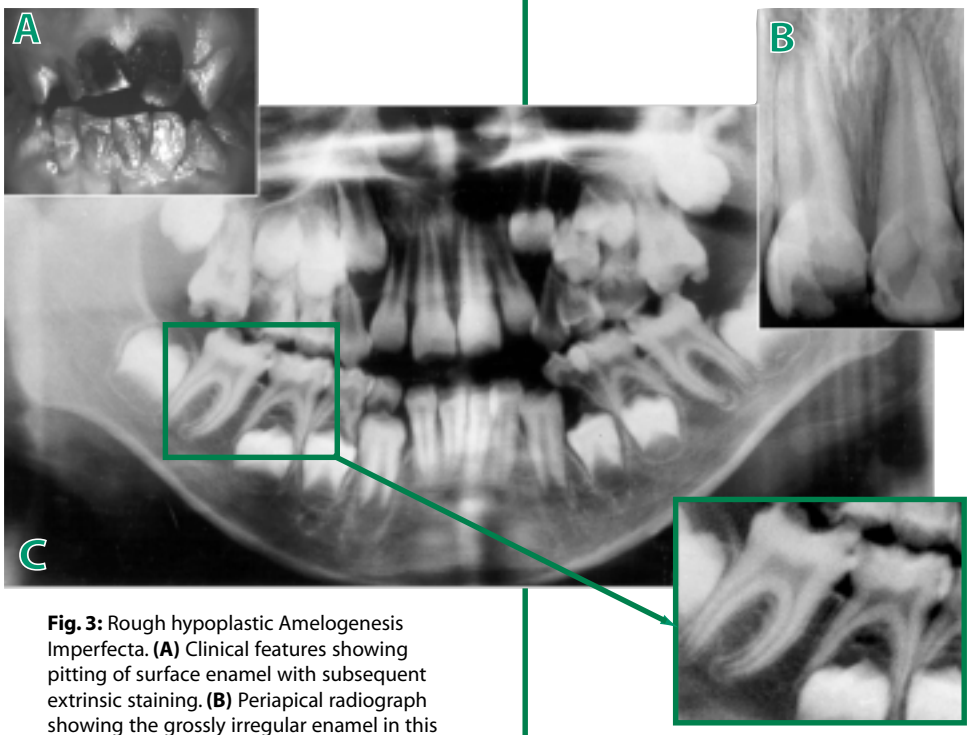


Fig. 3: Rough hypoplastic Amelogenesis Imperfecta. **(A)** Clinical features showing pitting of surface enamel with subsequent extrinsic staining. **(B)** Periapical radiograph showing the grossly irregular enamel in this condition. **(C)** Panoramic radiograph showing widespread involvement of teeth from both the primary and permanent dentitions.

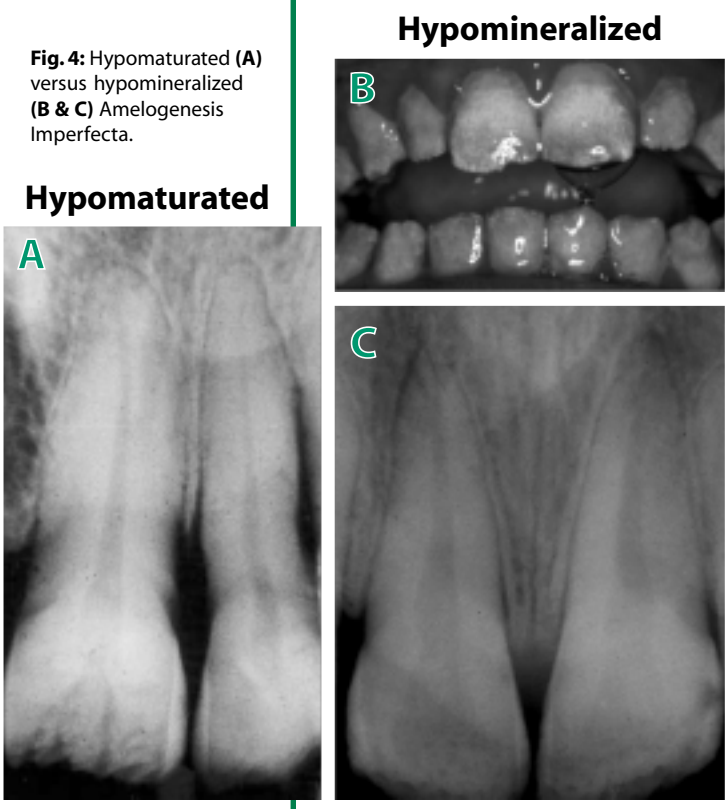


Fig. 4: Hypomaturated **(A)** versus hypomineralized **(B & C)** Amelogenesis Imperfecta.

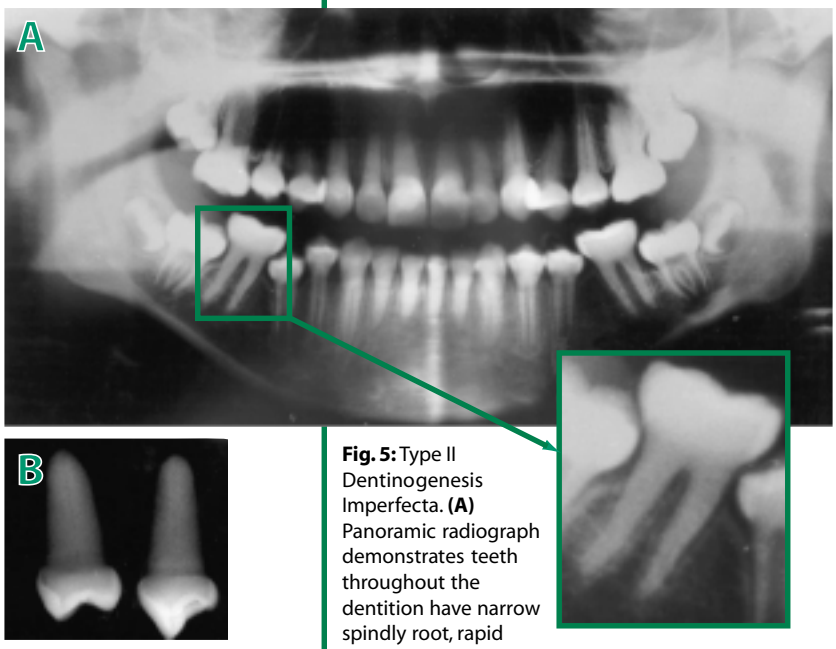


Fig. 5: Type II Dentinogenesis Imperfecta. **(A)** Panoramic radiograph demonstrates teeth throughout the dentition have narrow spindly root, rapid sclerosis of the pulp chamber and root canals, and bulbous crowns. **(B)** Radiograph of extracted teeth showing detail of early pulpal sclerosis.

color varies from white opaque to brown. It can be inherited as autosomal dominant (Type ID) or X-linked recessive (Type IE). The dominant variety is generalized, whereas the X-linked variety is generalized for males but can show a mosaic for females where there may be one affected and one normal X chromosome present. In such females there can be alternating areas of normal and abnormal enamel.

Rough hypoplastic AI (Type IF) is autosomal dominant. The enamel is thin, hard, rough-surfaced, and can readily become stained (Fig. 3). Enamel agenesis (AI Type IG) has teeth that are the color and shape of dentin with crowns tapering towards the rough, occlusal surface.

Hypomaturation AI (Fig 4A): In hypomaturation varieties, the enamel matrix is formed normally and initiates mineralization, but the enamel crystals do not mature normally. Radiographically, the affected enamel has a radiodensity approximating that of dentin. Hypomaturation AI can be autosomal recessive and pigmented (Type IIA), X-linked recessive (Type IIB), or X-linked with “Snow-Capped cusps” (Type IIC). There may also exist an autosomal dominant variety that is present with “Snow-Capped cusps.” (Type IID).

Hypocalcified AI (Fig. 4B & 4C): In these types, the enamel matrix is formed normally, but calcification is slight. The teeth are of normal appearance on eruption but the enamel is very soft and rapidly abrades. Radiographically, the radiodensities of the enamel and dentin are similar. Autosomal dominant (Type IIIA) and autosomal recessive (Type IIIB) types exist. Combined hypomaturation/hypoplastic AI with taurodontism is another type of this condition; Type IVA shows predominantly hypomaturation whereas Type IVB shows predominantly hypoplasia. Type IV AI is autosomal dominant. AI with

taurodontism could represent a partial expression of tricho-dento-osseous syndrome [1].

AI types ID, IE, IG, IIA, IIIA and IVB have enamel that rapidly abrades if left untreated. These types require full crown coverage as soon as possible. If treatment is delayed there may be a need to resort to overdentures. Other forms of AI are mainly a cosmetic problem and therefore require either anterior crowns or veneers.

Dentinogenesis Imperfecta

The most widely-accepted classification of dentinogenesis imperfecta (DI) is that of Shields *et al* [13]. Shields Type I DI is associated with a systemic hereditary bone disorder, osteogenesis imperfecta. Osteogenesis imperfecta (OI) is a group of closely-related inherited diseases characterized by abnormal bone fragility. Present clinical classification delineates six types, one of which (Type II) is so severe that mortality is 100%, either intrauterine or perinatal [14]. Malmgren and Norgren (2002) studied the dental aberrations in a group of non-related individuals with various forms of osteogenesis imperfecta (OI), ages 0.3 to 20 years, with the aid of panoramic radiographs in most instances. DI Type I was present in 27 of 65 patients. The presence or absence of DI showed almost complete accordance in affected parents and children and in affected siblings.

Type II DI is autosomal dominant hereditary opalescent dentin unassociated with osteogenesis imperfecta (Fig. 5). It is found in approximately one in 8,000 US Caucasians. Both DI Type I and Type II result in teeth that are similar clinically, radiographically, and histopathologically to those encountered in individuals having DI Type I. The teeth of both dentitions of affected individuals are translucent with a blue to brown hue. High-resolution synchrotron radiation-computed tomography and small-angle x-ray scattering on normal and DI Type II (DI-II) teeth showed that the mineral concentration was 33% lower on average in DI-II dentin than in normal dentin [16]. Radiographically, the teeth have bulbous crowns, cervical construction, narrow roots and early obliteration of the pulp chamber and canals. The enamel is poorly supported by the underlying abnormal dentin, and readily chips away. The enamel-dentin junction when viewed histologically is not normally scalloped. DI shows 100% penetrance but

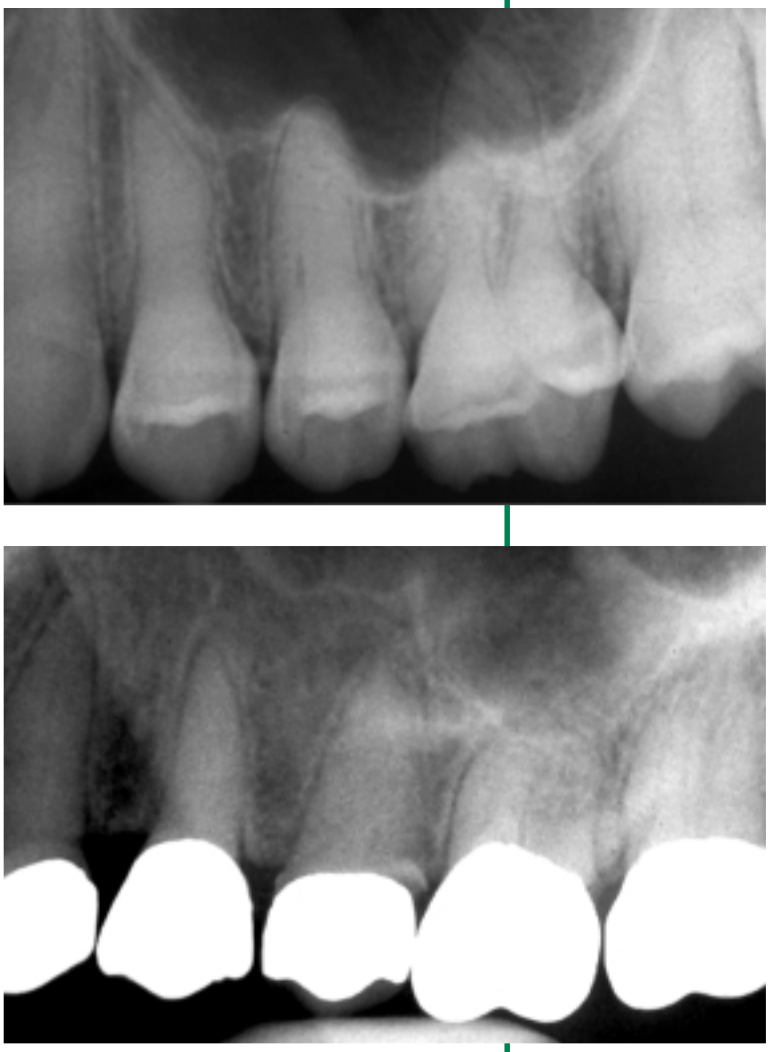


Fig. 6: Type II Dentinogenesis Imperfecta: Before and after prosthodontic treatment. Full crowning of all teeth is recommended to prevent rapid attrition of poorly attached enamel and underlying dentin down to gum level. This is especially necessary as pulpal sclerosis will likely make endodontic therapy unachievable.

" Treatment of choice for DI is full crowning of the teeth before the enamel is lost and the dentin abrades down to gum level."

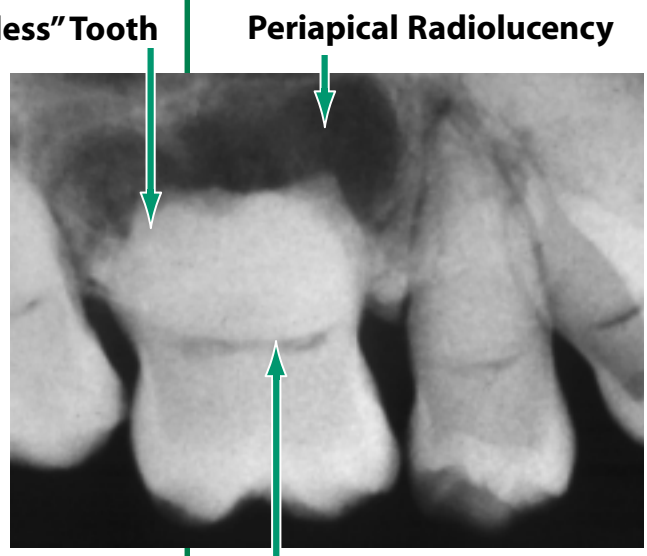


Fig. 7: Radicular (Type I) dentin dysplasia: Note typical features of short blunt roots without noticeable pulp canals. Periapical radiolucencies are frequent. Maintenance of such teeth is usually not feasible.

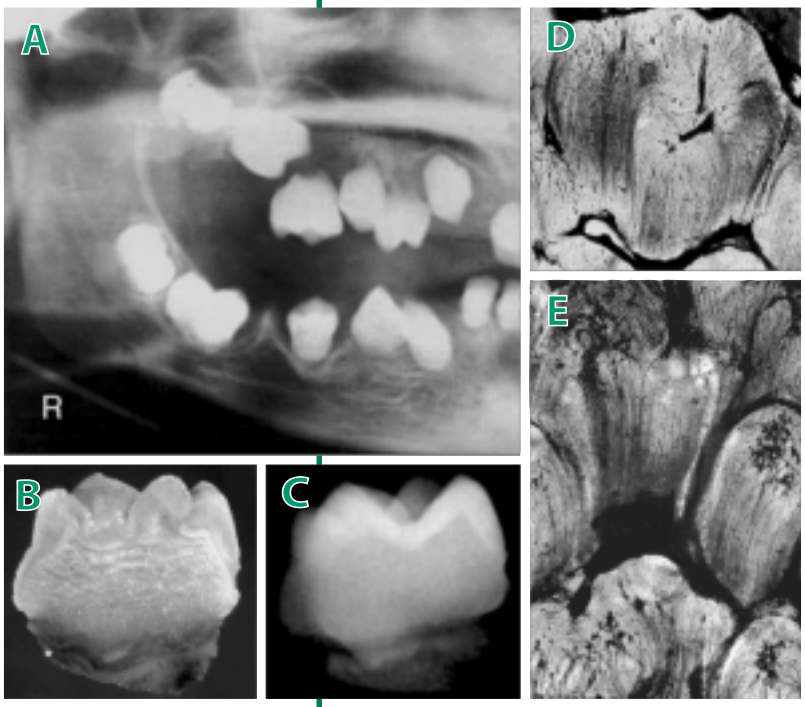


Fig. 8: Radicular (Type I) dentin dysplasia: **(A)** Panoramic radiograph showing generalized nature of this condition. Note the "pulpless""rootless" teeth throughout. **(B)** Hemisected tooth from same case showing normal enamel over wavelike abnormal dentin. **(C)** Radiograph of extracted tooth. **(D)** and **(E)** Undemineralized histological sections showing the dentin to resemble the osteodentin found in certain fish species.

variable expressivity. While pulpal obliteration is a common feature, in some cases the dentin is thin with a large pulp and normal enamel thickness. Such teeth are termed "shell teeth." A third type of DI (Type III) has been described, but this might be nothing more than variable expressivity of DI Type II [1].

Genetic linkage studies have identified the critical loci for DI Types II and III on human chromosome 4q21 [17]. Treatment of choice for DI is full crowning of the teeth before the enamel is lost and the dentin abrades down to gum level (Fig. 6). Alternative treatments are overdentures, full dentures or dental implants [18].

Radicular Dentin Dysplasia

Radicular (Type I) dentin dysplasia (Fig. 7 & 8) is an autosomal dominant condition affecting both dentitions in which the enamel and coronal dentin are normal in appearance, but the root dentin is disorganized and the tooth roots are shortened, sometimes resulting in apparently rootless teeth [19, 20]. Periapical pathoses are frequently encountered. More severely affected teeth may appear to have no pulp chamber or canal. Less severely affected teeth have a crescent-shaped pulp chamber that resembles a finger-nail crimp to an analog film radiograph. Mildly-affected teeth may have roots of normal length with a dilated pulp chamber containing a large pulp stone. While dentin dysplasia is not related to systemic disease, dentin dysplasia-like anomalies are sometimes reported in association with calcinosis universalis, tumoral calcinosis, and certain rheumatoid or skeletal abnormalities.

Coronal Dentin Dysplasia

Coronal (Type II) dentin dysplasia (Fig. 9) is a rare autosomal dominant condition [21]. The chromosomal defect causing this condition is on the

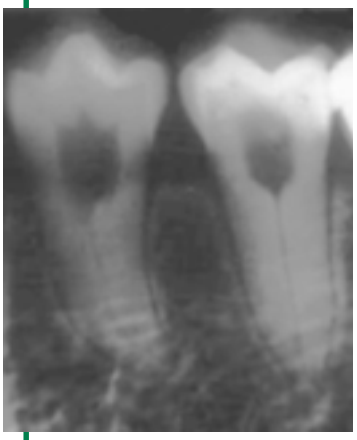
" Regional odontodysplasia is an uncommon non-inherited developmental anomaly that can affect both the primary and the permanent dentition."

same chromosome as that found in DI Type II [22]. The primary teeth closely resemble DI; however, the permanent teeth are normal in color and radiographically demonstrate apical extension of the pulp chamber, producing a thistle-tube or flame shape [21]. Pulpal calcifications can be numerous. Teeth have normal root length. This can be differentiated from pulpal dysplasia in that for the latter, thistle-tube shaped pulps are found in both primary and permanent dentitions.

Odontodysplasia

Regional odontodysplasia Fig. 10 is an uncommon non-inherited developmental anomaly that can affect both the primary and the permanent dentition [23]. Although it is generally recognized as a localized disorder of dental tissue, its etiology has not yet been well explained [24]. Affected teeth usually are found only in an isolated segment of the dentition in one arch. The involved teeth show anomalous hypoplastic and hypomineralized enamel, dentin, pulp, and cementum resulting in a "ghost-like" appearance radiographically, with correspondingly enlarged pulp chambers and canals. The affected teeth frequently do not erupt. Follicular tissue surrounding the unerupted crown may be thickened and can contain discrete calcifications [25,26]. Rarely the condition may affect more than one dental segment [27] and very occasionally the condition is generalized [28,29]. Treatment usually involves leaving the unerupted teeth in place to maintain the alveolar ridge. The missing teeth are replaced by a fixed or removable prosthesis. Should the teeth erupt, they are hypoplastic and are often mobile [30]. When affected teeth do erupt, a dentin-bonded porcelain bridge can minimize destruction to the hypoplastic tooth tissue if affected teeth are used as abutments [31].

Fig. 9: Coronal (Type II) dentin dysplasia: The permanent teeth radiographically demonstrate apical extension of the pulp chamber producing a thistle-tube or flame shape.



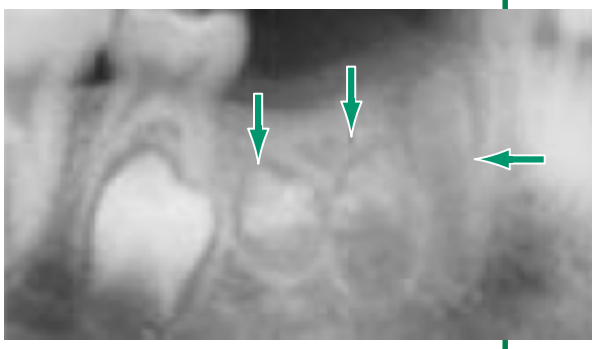


Fig. 10: Regional odontodysplasia (right mandibular canine and premolar region).

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In The Recent Literature:

Minor oral surgery: Complications could have been prevented if a panoramic radiograph had been appropriately evaluated. Ordering and reading appropriate radiographs prior to surgery should be considered the normal standard of care.

Mozaffari E, Mupparapu M, Otis L. Undiagnosed multiple myeloma causing extensive dental bleeding: report of a case and review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94:448-453. [From the Department of Oral Medicine, University of Pennsylvania School of Dental Medicine, Philadelphia, USA.]

Radiology plays an important role in the detection of bone changes associated with undiagnosed multiple myeloma. Extensive bleeding that occurred during a minor dental surgical procedure could have been prevented if the panoramic radiograph had been evaluated carefully before initiation of the treatment. Etiologic factors responsible for the formation of such abnormalities in multiple myeloma are reviewed and the value of panoramic radiology used in diagnostic assessment of the disease is presented.

Osteogenesis imperfecta: Panoramic radiography revealed a high prevalence of dentinogenesis imperfecta.

Malmgren B, Norgren S. Dental aberrations in children and adolescents with osteogenesis imperfecta. Acta Odontol Scand 2002;60:65-71. [From the Department of Pediatrics, Huddinge University Hospital, Karolinska Institute, Stockholm, Sweden.]

The investigators studied dental aberrations in a large sample of unrelated patients having different types and forms of osteogenesis imperfecta (OI). Sixty-eight non-related patients aged 0.3 to 20 years (mean is 10 years) were examined clinically and panoramic radiographs from 49 patients were analyzed. Dentinogenesis imperfecta (DI) Type I was found in 27 of 65 patients and was significantly more common in OI Type III than in Types I and IV. The presence of DI was almost completely in accordance with affected parents, siblings and children. The percentage of patients with no apparent dental aberrations was approximately the same in patients with OI Types I and III and in patients with mild and more severe forms of OI. The high prevalence of dental aberrations in OI shows the importance of clinical and radiographic dental examinations in the OI population. In patients with mild forms of the disease, in whom the medical diagnosis is uncertain, demonstration of disturbances in dental development can be crucial for establishing the OI diagnosis.

Osteogenesis imperfecta: Mortality rates depend on the type of osteogenesis imperfecta involved.

Singer RB, Ogston SA, Paterson CR. Mortality in various types of osteogenesis imperfecta. J Insur Med 2001;33:216-220. [From the Department of Epidemiology and Public Health, Ninewells Hospital and Medical School, University of Dundee, Dundee DD1 9SY, Scotland.]

Osteogenesis imperfecta (OI) comprises a group of closely related inherited diseases characterized by abnormal bone fragility. Six clinical types are recognized, one of which (Type II) is so severe that intrauterine or perinatal mortality is 100%. Types are differentiated by clinical groups, severity, and by the presence or absence of features such as blue sclerae and dentinogenesis imperfecta. From a registry created in association with the Brittle Bone Society, 743 patients with OI in England and Wales were observed from 1980 through 1993. Patients were classified into 3 groups (Type IA, Type III, and Types IB, IVA, and IVB combined). Average OI annual mortality rates were determined and compared with 1981 rates in the general population of England and Wales matched by sex and age. In Type IA (52 % of the OI cases), there was no significant excess mortality

(mortality ratio 108 %, based on 15 deaths). In Type III, on the other hand, excess mortality was very high in children, adolescents and young adults. In the combined group of Types IB, IVA, and IVB, the mortality ratio was 157 % in patients aged 45 and up (not significant at the 95% confidence level); however, higher ratios at younger ages were statistically significant, even though based on a total of only 5 deaths.

Amelogenesis imperfecta: Molecular biology makes inroads into explaining the causation of varieties of this group of phenotypes.

Kida M, Ariga T, Shirakawa T, Oguchi H, Sakiyama Y. Autosomal-dominant hypoplastic form of amelogenesis imperfecta caused by an enamelin gene mutation at the exon-intron boundary. J Dent Res 2002 ;81:738-742. [From the Research Group of Human Gene Therapy, Hokkaido University Graduate School of Medicine, N-15, W-7, Kita-ku, Sapporo, 060-8638, Hokkaido, Japan.]

Amelogenesis imperfecta (AI) is currently classified into 14 distinct subtypes based on various phenotypic criteria; however, the gene responsible for each phenotype has not been defined. Previous studies have mapped an autosomal-dominant human AI locus to chromosome 4q11-q21, where two candidate genes, ameloblastin and enamelin, are located. The authors performed molecular genetic studies on a Japanese family with a possible autosomal-dominant form of AI. They studied AI patients in this family, focusing on the ameloblastin and enamelin genes, and found a mutation in the enamelin gene. The mutation detected was a heterozygous, single-G deletion within a series of 7 G residues at the exon 9-intron 9 boundary of the enamelin gene. The mutation was detected only in the AI patients and was not detected in unaffected family members or control individuals. The male proband and his brother showed hypoplastic enamel in both primary and permanent dentitions, and their father showed local hypoplastic defects in the enamel of his permanent teeth. The findings are consistent with heterogeneous mutations in the enamelin gene being responsible for an autosomal dominant hypoplastic AI.

Endodontic assessment: Panoramic radiographs can be used to evaluate the treatment outcomes for endodontic restorations.

Lupi-Pegurier L, Bertrand MF, Muller-Bolla M, Rocca JP, Bolla M. Periapical status, prevalence and quality of endodontic treatment in an adult French population. Int Endod J 2002;35:690-697. [From the Department of Public Health, University of Nice, Sophia, Antipolis, France.]

This study used panoramic radiographs to determine the periapical status and the quality of root-canal treatment amongst an adult population attending a dental school. Patients who attended the dental school in Nice, France for the first time during 1998 were included. The survey involved 344 patients: 180 females and 164 males. Panoramic radiographs, taken by a trained radiology assistant, were used in this study. The periapical areas of all teeth with the exception of third molars, were examined and the technical quality of root fillings were evaluated for both apical extension and density. Statistical analyses were conducted using ANOVA, Chi-square, Fisher's PLSD and Cohen's Kappa tests. Males had significantly fewer natural remaining teeth than females ($p < 0.03$). Similarly, the average number of root-filled teeth was lower for males ($p < 0.01$). Nonroot-filled teeth (n = 6126) had significantly fewer signs of periapical pathology than root-filled teeth (n = 1429) (1.7 % vs. 31.5 %, $p < 0.0001$). Many root-canal treatments were technically unsatisfactory in terms of quality and treatment outcome. There was a significant correlation between the presence of periapical pathology and inadequate root-canal fillings ($p < 0.001$).

Mucocele: A lesion close to sensitive structures in the skull was an important incidental finding from panoramic radiography.

Patinen P, Hietanen J, Peltola J. Sphenoid sinus mucocele: case report of an appearance on a panoramic radiograph. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93:747-750. [From the Institute of Dentistry, University of Helsinki and Department of Oral and Maxillofacial Surgery, Helsinki University Central Hospital, Finland.]

Mucoceles of the sphenoid bone are significant as they are located deep in the skull close to such sensitive structures as the optic chiasm and the upper six cranial nerves. A case of an incidental finding of a sphenoid sinus mucocele on a dental panoramic radiograph is described in a totally symptom-free, 22-year-old woman. Thorough knowledge of the manifestations of oral and paranasal disease plays a vital role in early diagnosis of a variety of diseases of the head and neck region. This requires systematic evaluation of the whole panoramic radiograph.

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