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Management of amelogenesis imperfecta and Dentinogenesis imperfecta: A comprehensive review

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Abstract

The formation of teeth is a complicated process. It's the outcome of a complicated interaction between several genes and enzymes. The ectomesenchyme and epithelium are both involved. Any mutations in these genes can result in an abnormal odontogenesis process. The major goal in disorders like dentinogenesis imperfecta and amelogenesis imperfecta is to preserve as much dental structure as possible and retain the correct vertical dimension at an early stage so that a stable foundation for a more optimal treatment may be established later. Therefore, an interdisciplinary approach is required including prosthodontic, restorative and orthodontic treatment. This article presents the various treatment approaches that can be done in cases with amelogenesis imperfecta and dentinogenesis imperfecta.

Keywords: amelogenesis imperfecta dental anomalies; dentinogenesis imperfecta;

Introduction

The formation of teeth is a complicated process. It's the outcome of a complicated interaction between several genes and enzymes. The ectomesenchyme and epithelium are both involved. Any mutations in these genes can result in an abnormal odontogenesis process. Amelogenesis imperfecta and dentinogenesis imperfecta are two examples of such abnormalities. The cosmetic and functional restoration of children and adult patients with these problems is a huge task for a dentist. The distinctions in the characteristics and treatment possibilities of amelogenesis imperfecta and dentinogenesis imperfecta are the subject of this research review.

Methods of collecting data: PubMed/Medline was used to conduct a systematic search, and publications from 2001 to 2021 were examined. The OR Boolean operator was used with the keywords amelogenesis imperfecta dentinogenesis imperfecta. There were 137 case studies, reviews, and systematic reviews found. Tables 1 and 2 summarise some of the case reports.

Results

Dentinogenesis Imperfecta: Barret was the first to discover dentinogenesis imperfecta in 1892. In the year 1892, Finn and Hodge did research on it. The term "hereditary opalescent dentin" was invented by Skiller.[17] There are three different forms of Dentinogenesis imperfecta.

- DI with osteogenesis imperfecta (Type I)
- DI without osteogenesis imperfecta (Type II)
- Brandywine isolate (type III)

It is almost often inherited as an autosomal dominant characteristic. This illness is inherited in an autosomal dominant fashion, which means that one copy of the mutated gene in each cell is enough to produce it. Some researchers feel that dentinogenesis imperfecta types II and III, as well as dentin dysplasia type II, are all symptoms of the same disease. Dentin dysplasia type II has indications and symptoms that are remarkably similar to dentinogenesis imperfecta. Dentin dysplasia type II, on the other hand, affects primary teeth more than permanent teeth.[18]

Table 1: Case reports on dentinogenesis imperfecta.

Author/chief complaint	Oral examination and radiographic features	Treatment plan
P Subramaniam etal (2008)[1] 16-year-old complained of brown-coloured teeth.	Gingival recession with respect to 31, 41 Yellowish brown small teeth Generalised attrition Pulp exposure in maxillary anterior Unilocular radio lucencies with respect to 34, 46 Open apices with respect to premolars Missing with respect to 18,27,28	Cyst enucleation with respect to 34, 46 Extraction of 11, 12, 16, 21, 64, 33, 34, 36, 43, 44 coma 46 Root canal treatment with respect to 13, 23, 22 Apexification with respect to 14 Stainless steel crowns placed on erupted molars, polycarbonate crowns on premolars end pfm crowns in all other teeth Removable prosthesis given in upper and lower jaw for remaining edentulous area
Anil Goud (2011)[2] 35-year-old complaints of occasional chipping of teeth and dislodged FPD	Missing with respect to 36, 46, 16, 27, 47 Fpd with respect to 14, 15, 16, 17 ,18, 33, 34, 35, 43, 44, 45, 46, 47, 48 Total obliteration off pulp chambers with respect to 33, 34, 35, 43, 44, 45, 48	Full crowns and fpd for maxillary teeth Overdenture reinforced with glass fibers for lower jaw
Sedat Guven (2015)[3] 20-year-old complaints of aesthetic and chewing problems	Loss off many teeth No enamel on remaining teeth Impacted teeth, teeth with short roots and large pulp chambers	Extraction of impacted teeth Endodontic treatment on 13, 14, 15, 24,45 Periodontal treatment and oral hygiene instructions were given

		Overdentures for upper and pfm restorations for mandibular teeth were planned.
Sompop Bencharit (2014)[4] 33-year-old complaints of soft teeth that easily break and had to get restorations done frequently	Composite resin restorations in anterior teeth Multiple missing posterior teeth Remaining posterior teeth had fractured amalgam or composite resin restorations Poorly mineralised roots	Extraction with respect to #17, #32 Dental implants with respect to #18, #30, #31, #12 followed by implant supported crowns Increase of vertical dimension by 2 mm with the help of a splint Pfm crowns with respect to #3, #4, #5, #14 All ceramic crowns with respect to #6, #7, #8, #9, #10, #11, #20, #21, #28, #29
Fan Fan (2019)[5] 20-year-old complaints of unesthetic appearance and severe wear of anterior teeth	Amber crowns with severe attrition Gingival hyperplasia Class i malocclusion with excessive vertical and horizontal overlap	Orthodontic treatment to correct the malocclusion Periodontal treatment to lengthen maxillary and mandibular anterior crowns Smile designing to improve aesthetics
Shi Shi (2020)[6] 19-year-old complaint of dis coloured teeth	Narrow short and bulbous crowns Constricted cemen to enamel junction Obliterated pulse chambers Thinner and shorter roots	Digital smile designing with full coverage crowns
Hamas at Ghedaff (2011)[7]	Defective and decayed maxillary and mandibular teeth Maxillary overdentures on remaining teeth Class 2 division 2 malocclusion Decreased vertical dimension Gummy smile	Extraction of #2, #7, #10, #15 Implants placed followed by implant supported crowns

Table 2: Case reports on amelogenesis imperfecta.

Author	Oral examination and radio graphical features	Treatment plan
Mehnaz Arshad (2018)[8] 27-year-old complained of unattractive smile and difficult mastication.	Edematous gingiva Dental caries Brown pitted teeth with no proximal contact Open bite Loss of vertical dimension Missing upper left lateral	Orthognathic surgery to correct the open bite Implant placed with respect to 22 All other teeth were prepared to receive crowns

Adilson Luiz (2011)[9] 15-year-old complained of discoloration of his teeth.	Anterior open bite Discoloration of teeth Narrow maxilla	Orthodontic treatment Laminates for anterior teeth to improve aesthetics Composite restorations on all posterior teeth
K malik (2012)[10] 20-year-old female complained of crooked gums and teeth.	Reduced clinical crown height High smile line with an aesthetic appearance Loss of vertical dimensions	Increase in vertical dimension by 3 mm using a splint followed by permanent crowns at increased vertical dimension
Ian Brignell (2011)[11] A 24-year-old complained of poor dental esthetics.	Pitting and unaesthetic appearance in anterior teeth	Smile analysis followed by wax up and finally composite veneers
Emin Murat (2010)[12] 26-year-old female patient presented with a chief complaint of discolored teeth.	Discoloured teeth Hyperemic edematous gingiva Retained deciduous teeth Short clinical crowns Pulpal classifications Worn out enamel Loss of vertical dimension	Periodontally therapy to reduce gingival edema Increase of vertical dimension by 3 mm followed by placement of permanent crowns
Muhammad Rizwan Nazeer (2020)[13] A 20-year-old complained of unaesthetic smile, generalized sensitivity and difficulty in chewing	Loss of vertical dimension Grossly carious posterior teeth Anterior deep bite Reduced enamel thickness	Extraction of 3rd molars Crown lengthening Restoration of lost vertical dimension Cementation of permanent crowns
Salima Visram (2006)[14] 15-year-old complained of appearance of his front teeth and sensitivity.	Class 2 Division I malocclusion Anterior open bite 4-5 mhm over jet Generalised pitting and discoloration	Veneers from canine to canine Composite restorations on molars to increase vertical dimension
Neil S Nathwani (2010)[15] 20-year-old complained of missing maxillary lateral incisors, sensitivity, poor colour and shape of the teeth and the appearance of the gums.	Gingival overgrowth Yellow discoloration of teeth with white flecking Sensitivity	Gingivectomy to improve appearance Night guard vital bleaching Replacement of upper laterals with adhesive bridge from canine to canine.
Vaibhav D. kamble[16] 20-year-old complained of sensitivity in his teeth.	Gingivitis Thin enamel layer Hypersensitive exposed dentin Loss of vertical dimension and increased freeway space	Increase off vertical dimension by 4 mm with the help of a splint for 4 months followed by PFM crowns

Clinical features

- Discoloration that ranges from a dark blue to a brownish colour.
- Localised mesodermal dysplasia affects both primary and permanent dentitions.
- Enamel is hypoplastic or hypocalcified, which causes it to fracture away from the dentin.
- The radiographic appearance of shell teeth is characterised by bulbous crowns, short roots, and a large pulp chamber.

Features of histology: Histologically the dentino-enamel intersection has less scalloping. Reduced scalloping allows even prophylaxis instrumentation to easily remove dentin from the tooth structure. Atypical dentin with uncalcified matrix and irregular tubules is another histiological hallmark of dentinogenesis imperfecta. Normal scalloping is found in DI patients with OI. Enamel loss is caused by flaws in the dentine.[19]

Amelogenesis imperfecta:

- Amelogenesis imperfecta refers to a group of illnesses that generate qualitative and quantitative enamel abnormalities without generating systemic problems. Other names for these teeth include hereditary brown enamel, hereditary enamel dysplasia, and hereditary brown opalescent teeth. Amelogenesis imperfecta is also related to many syndromes like Jalili syndrome, Enamel renal syndrome, Mc Gibbbon syndrome and Kohlschutter tonz syndrome. [20-24]

Classification:

1. Weinmann et al: two types based on phenotype [25]

- Hypoplastic
- Hypocalcified

2. Darling etal: Five phenotypes based on clinical, microradiographic and histopathological findings.[20]

- Hypoplastic

➢ Group 1 – generalised pitting

➢ Group2 – vertical grooves (now known to be X-linked AI)

➢ Group 3 – Generalised hypoplasia

- Hypocalcified

➢ Type 4A – chalky, yellow, brown enamel

➢ Type 4B – marked enamel discolouration and softness with post-eruptive loss of enamel

➢ Type 5 – generalised or localised discolouration and chipping of enamel

3. Nusier et al[26]

- Hypoplastic
- Hypo maturation
- Hypocalcified
- Hypoplastic

4. Witkop et al.[27]

- Type I: hypoplastic
- Type IA: pitted autosomal dominant
- Type IB: Local autosomal dominant
- Type IC: Local autosomal recessive
- Type ID: smooth autosomal recessive
- Type IE: smooth X-linked dominant
- Type IF: rough autosomal dominant.
- Type IG: agenesis autosomal recessive

Genetic aetiology: febrile fever, vitamin deficiency

Local etiology: Fluoride intake, infection or damage on the local level

Clinical features

Hypoplastic: Enamel matrix thickness reduction with normal mineralization caused by failure during the secretory stage.

Enamel thickness is reduced.

Enamel appears normal and is less prone to wear.

The size of the teeth is reduced

Enamel that is irregular, rough, or pitted

Enamel and dentin appear normal on radiographs.[26]

Hypo maturation: Caused by inadequate protein removal from the enamel matrix during the maturation stage, resulting in enamel that is full thickness but fragile and fails prematurely.

Discoloration ranging from yellow brown to red brown, with a mottled look.

Enamel has the same radiodensity as dentin radiographically.[26]

Hypocalcified: Results from insufficient transport of calcium ions (Ca2+) into the forming enamel, resulting in a defect in the enamel mineralization process with normal matrix development.

Normal thickness with enamel translucency loss

Enamel that is hypo mineralized and has a mushy cheesy consistency

Enamel is quickly decomposable.

- Teeth appear darker.
- Enamel is less radiopaque than dentin on x-rays.[26]

Discussion:

Considerations for Treatment: Both amelogenesis imperfecta and dentinogenesis imperfecta have similar treatment goals.

- Getting rid of infection causes or getting rid of pain
- Bringing back the aesthetics
- Defending teeth against wear and caries
- Maintaining or enhancing performance
- Where necessary, improving malocclusions
- Wherever feasible, tooth tissue is preserved.

Many psychosocial issues have an impact on the patient, including variances in appearance, low self-esteem, poor academic performance, and speech disorders, all of which can contribute to social isolation. In such cases, strong family support and supportive peer interactions are essential. Dental and medical care has been found to improve a patient's social well-being and efficiency.[28,29]

Dentinogenesis imperfecta: Reduced vertical dimension, loss of functional occlusion, altered expression, tooth sensitivity, poor aesthetics, and excessive attrition are common clinical findings in cases of dentinogenesis imperfecta. In the temporomandibular joint, there is also pain and a grinding sensation.

Preventive treatment: Because teeth with DI have exposed dentin, which is more susceptible to caries, oral hygiene recommendations, fluoride application, and frequent dental exams should be followed to avoid cavities. They should be continuously examined after the permanent dentition emerges to keep an eye on tooth wear and intervene if necessary. Stainless steel crowns, pre-veneered stainless-steel crowns, or composite strip crowns can be used in the primary dentition.[30] If the child has lately presented, the only option is an overdenture, which must be evaluated and rebuilt as the infant grows. Implants and other costly treatments should be avoided during the important stages of growth and puberty. Adolescent patients should be treated more quickly. This gives the patient more immediate aesthetic, functional, and therapeutic help.

Prosthodontic treatment: Discoloration of teeth and bonding of various types of restorations are challenges that must be overcome while restoring such teeth. The enamel may debond from the dentine, making the bonding of the repair to the enamel uncertain. The hybrid layer that forms may also be harmed as a result of the dentine's changed nature. In these situations, resin modified Glass ionomer cement bonds better than traditional resin-based adhesives. This is due to the fact that they create genuine bonds with metal ions rather than the hybrid layer.[31] Prior to long-term treatment, provisional restorations with fixed or removable prosthesis should be offered. The patient will be motivated to complete the treatment as a result of this. Overdentures, traditional fixed or removable

prostheses, or implant-supported fixed or removable prostheses provide a more realistic face shape, arch relationship, and smile line for the patient.[32,28]. Because implant therapy is contraindicated in young individuals, removable dentures may be a viable option for replacing missing teeth. Overdentures that are tooth-supported can be used to assist preserve alveolar bone height. However, there are significant drawbacks, such as denture irritation, plaque collection, and gingivitis. In the case of teeth with short roots, set partial dentures are not indicated. Crowns are not recommended for teeth with severe DI, short roots, and severe cervical constriction. Such teeth frequently develop necrotic, and endodontic treatment is impossible in such circumstances due to the difficulty in locating the canal. Veneers are frequently utilized in individuals with dentinogenesis imperfecta, although they appear to be more beneficial in people with amelogenesis imperfecta. This is owing to the above-mentioned bonding difficulty, as well as the discoloration exhibited in certain circumstances which are difficult to conceal with veneers. This has resulted in impaired aesthetics in several circumstances. This can be avoided by incorporating an opaque porcelain layer into the restoration or cementing with opaque resin cement. Even with these, though, there is a loss of translucency, which can affect aesthetics once again.[33] Another option is to employ zirconia coping, which can then be covered with feldspathic porcelain.[34] Composite veneers, on the other hand, have the advantage of not requiring tooth structure removal and being relatively rapid. Young patients can benefit from composite veneers since they can be employed in situations with big pulp chambers.[31]

Implant treatment: Bone quality and quantity are unaffected and are comparable to people without DI. Patients with DI type I, on the other hand, have osteogenesis imperfacta, yet implants have been successful in these patients. Due to the presence of short roots, patients with DI may have a reduction in bone volume. As a result, it may be necessary to undertake guided bone regeneration prior to implant implantation. Bisphosphonate treatment is commonly used in DI type I patients. This increases the risk of jaw osteonecrosis.[35]

Endodontic therapy: Endodontic treatment is not possible since the pulp chambers have been obliterated due to deposition of reparative dentine. Enlarged pulp chambers can occur after type III DI endodontic therapy. Because morphologic changes in the dentine may occur, increasing the risk of fracture, the use of a cast post and core is not recommended. In the event of periapical infection, periapical curettage with retrograde filling may be performed.[36]

Amelogenesis imperfecta: The most common symptoms of amelogenesis imperfecta include sensitivity to hot and cold, decreased aesthetics due to discolouration of the mandibular and maxillary anterior teeth, and trouble chewing. Clinical features include enamel wear with exposed dentin, a lack of proximal connections, and a loss of vertical dimension. In some cases, an anterior and posterior open bite might be detected. In cases of amelogenesis imperfecta, a substantial proportion of teeth remain unerupted.

Preventive treatment: Temporary stainless-steel crowns can be put at a younger age as a preventive procedure. As soon as the incisors begin to erupt, they can be fitted with resin crowns.[26] They can be changed once the gingival margin is accessible. Tooth whitening products that release more than 0.1 percent hydrogen peroxide cannot be used in patients under the age of 18 according to a European Union regulatory amendment made in 2012. In pedo patients, this makes essential bleaching impossible.[37]

Restorative treatment: In individuals with amelogenesis imperfecta, the longevity of restorations is significantly

reduced, and this is related to the severity of the condition. Restorations have been observed to last roughly 50% of the time in these circumstances, and the rate of replacement is 2.5 times higher than in normal or unaffected people. When compared to other kinds of amelogenesis imperfecta, the hypoplastic type has a higher rate of restored survival. The most common reason for restorative failure is tooth fracture, which can be caused by poor enamel quality and quantity. As a result, etching is diminished, and bond strength suffers as a result. The increasing protein content of the enamel may be to blame for the decreased bond. Adhesive bonding can improve bond strength. Treatment with direct composite resin may be used to buy time until permanent restorations may be made at a later age. Bleaching with 10% carbamide peroxide can improve the appearance, but it comes with a slew of drawbacks, including sensitivity, gingival ulceration, and shade reversion. Stopping the treatment or lengthening the time between bleaching cycles, as well as employing rubber dams and correctly extended night guards, can all help to prevent these problems.

Table 3: similarities and differences between ai and di.

	Amelogenesis imperfecta	Dentinogenesis imperfecta
Genes affected	Amelx, fam83h, klk4, mmp20	Dspp
Colour of tooth	Yellow brown or orange on eruption-stained brown to black with time	Dusky blue to brownish discolouration
Affects	Enamel	Dentin
Presence of osteogenesis imperfecta	Not associated with osteogenesis imperfecta	Associated with oi in some cases
Radiographic features	Enamel and dentine have similar radio opacity or enamel has reduces opacity	Bulb shaped crowns with cervical constrictions Roots spiked Obliteration of pulp chamber (shell teeth appearance)
	Pitted enamel of grooves presents on enamel	No grooves
	Autosomal dominant, recessive, X linked mode of inheritance	Autosomal dominant
	Open bite common	Not generally seen
	Dentin not affected	Opalescent dentin
	Teeth small with spacings	Open contacts
	Enamel can be removed with prophylaxis instrument	Enamel may split readily when dentin is subjected to stress

Prosthetic treatment: Due to the existence of significant pulp horns and expanded pulp chambers, the amount of preparation required when treating younger patients must be kept in mind. Otherwise, the pulp may become irritated as a result of the minimal preparation.[37] Crown lengthening can be done in cases of microdontia, however these treatments are difficult since there may be greater sensitivity after surgery, and they might be complex due to the close closeness of the roots.[39] Gold onlays or full coverage crowns can be used on the back teeth. Crowns

made of metal, PFM, and stainless steel are also available. To maintain as much dental structure as feasible, porcelain should be used exclusively in aesthetic portions and metal in unaesthetic areas.[39] All-ceramic crowns with high strength, like as zirconia, have also been employed, however the preparation for such crowns frequently results in pulpal pathology.[40] Due to the loss of vertical dimension in complex instances requiring complete mouth rehabilitation, diagnostic wax up is required. In some circumstances, crown lengthening may be necessary. Porcelain and composite veneers can also be used to improve a patient's look.[38]

Implant treatment: In situations of AI and DI in young children, particularly adolescent patients, implants are not the primary line of treatment. They should only be regarded as a therapy option for patients who have reached the end of their growth cycle. It is prescribed for people who have advanced cases of AI and whose teeth are regarded irreversible. Reduced interproximal space due to short roots and clinical crowns might be a problem when inserting implants in instances with AI.[38]

Orthodontic treatment: ZManaging the deficient enamel or dentin in such circumstances is a challenge in orthodontic treatment. The goal is to get the teeth in a better position to implant the restorations, not to get them in ideal position. First, we must establish whether the damaged enamel can survive the stress applied during appliance placement and removal. To improve application retention, plastic brackets, GIC base adhesives, and typical banded appliances can be employed. Because the enamel is not consistent, more detailed bends are required at the conclusion of the orthodontic treatment.[38]

Modes of treatment

The following is based on the clinical findings:

- In the case of an open bite, orthodontic treatment should be followed by prosthodontic rehabilitation.
- In the event that the vertical dimension is lost, a prosthodontic treatment with permanent crowns should be planned to restore it.
- Bleaching or porcelain veneers might improve your appearance.

Based on the type of amelogenesis imperfecta.

- Bleaching can be done in situations with hypoplastic amelogenesis imperfecta. Because the enamel is strong enough to hang on to bonded restorations like porcelain veneers, they can be used to protect teeth from caries.
- Bonded restorations are not recommended in situations of Hypo maturation and Hypocalcified amelogenesis imperfecta because they can cling on to the enamel. As a result, full coverage crowns are a treatment option that can be counted on.

Conclusion

The major goal in disorders like dentinogenesis imperfecta and amelogenesis imperfecta is to preserve as much dental structure as possible and retain the correct vertical dimension at an early stage so that a stable foundation for a more optimal treatment may be established later. Adhesion to AI and DI-affected teeth is unpredictably variable, but investigations have demonstrated that it is enough. The major goal of treatment should be to restore function and appearance, as well as to improve the patient's psychosocial well-being and productivity. Treatment options are determined by the severity of the disease, as well as the patient's age, socioeconomic status, and motivation. A well-thought-out interdisciplinary strategy can significantly improve a patient's quality of life.

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