Functional and esthetic rehabilitation of patients with amelogenesis imperfecta

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ABSTRACT:

Aim: Aim of the article is to do a literature review on functional and esthetic rehabilitation of a patient with amelogenesis imperfecta.

Background: Amelogenesis imperfecta (AI) is a hereditary disorder that causes developmental alterations in the structure of enamel. In addition, tooth sensitivity, missing or impacted teeth, taurodontism, altered dental esthetics and anterior open bite can also be associated with AI. AI affects the structure and clinical appearance of enamel, the enamel may be brown stained and porous. Individuals with AI may also be associated with morphologic of biochemical changes elsewhere in the body. AI is genomic in origin. The disorder may be autosomal dominant, autosomal recessive or X-linked.

Reason: It is important to know all the aspects of AI to improve their esthetics and overall life style.

Keywords: esthetic, amelogenesisimperfecta.

INTRODUCTION:

Amelogenesisimperfecta (AI) presents with a rare abnormal formation of the enamel or external layer of the crown of teeth. Enamel is composed mostly of mineral, that is formed and regulated by the proteins in it. Amelogenesis imperfecta is due to the malfunction of the proteins in the enamel: ameloblastin, enamelin, tuftelin and amelogenin.
People afflicted with amelogenesis imperfecta have teeth with abnormal colour: yellow, brown or grey; this disorder can afflict any number of teeth of both dentitions. The teeth have a higher risk for dental cavities and are hypersensitive to temperature changes as well as rapid attrition, excessive calculus deposition, and gingival hyperplasia.There are many genetic conditions that may affect an individual and result in discoloured or malformed teeth. Gene changes that affect dentin and enamel formation are being defined scientifically and we now know more about these inherited disorders. Amelogenesis Imperfecta (AI) is one group of disorders that affects the formation of enamel covering the teeth. The prevalence of AI varies from 1:700 to 1:14,000 depending on population studies. The etiology of AI involves the genetic mutation of any of the following proteins, all of which are crucial in enamel formation and maturation: enamelin, amelogenin, ameloblastin, tuftelin, amelotin, dentine sailophosphoprotein, enzymes such as kallikrein 4 and matrix metalloproteinase 20.There are four major types of Amelogenesis Imperfecta: 1. Hypoplastic; 2. Hypomaturation; 3. Hypomineralizeation (Hypocalcification); 4. Any of the above with the addition of taurodontism.1,2 The enamel deficiencies can be quantitative (hypoplastic) or qualitative (hypomaturation/hypomineralized).[1] AI may cause tooth sensitivity, loss of vertical dimension, dysfunction and poor esthetics. Amelogenesis imperfecta patients are susceptible to the restorative cycle of replacement restorations like any other patient, but start with a distinct disadvantage. [2] Management of a patient with AI is a challenge for the clinician. The treatment options vary considerably depending on several factors such as the age of the patient, socio-economic status, periodontal condition, loss of tooth structure, severity of the disorder, and, most importantly, the patient's cooperation. The clinician has to consider the long-term prognosis of the treatment outcome. This clinical report describes the fabrication of metal ceramic and all metal crowns for the restoration of severely worn teeth in a patient with AI which requires meticulous maintenance of oral hygiene and patient co-operation.[3] Amelogenesis imperfecta is a hereditary condition resulting in poor tooth development, severe anomalies, or complete absence of enamel. Enamel lesions may be the only characteristic of this condition or may be part of a generalised syndrome. Amelogenesis imperfecta can be characterised by enamel hypoplasia and/or hypomaturation or hypocalcification of the existing teeth. Restoration for patients with this condition should be oriented toward the functional and esthetic rehabilitation and the protection of the existing teeth.[4]Clinical problems of AI patients mainly include poor esthetics, sensitive teeth, loss of occlusal vertical dimension, chewing difficulties, tooth wear and open bite.8,9 Treatment plan of AI is related to many factors, including patients' age, socioeconomic status, type and severity of the disorder and intraoral condition. Treatment starts from childhood and continues throughout adolescence. An interdisciplinary approach is necessary in evaluation, diagnosis and treatment of AI including a combination of periodontal, orthodontic, prosthodontic, surgical and restorative methods.[5]Amelogenesis imperfecta (AI) is a group of genetic disorders that primarily affect the quality and quantity of amelogenesis in both primary and permanent dentitions. The main clinical characteristics are severe attrition, tooth sensitivity and unesthetic appearance.[6]It is common for AI patients to receive little or no oral healthcare during childhood. Pitted enamel surfaces may predispose AI teeth to plaque accumulation, but the spacing of the teeth may reduceinter proximal caries

susceptibility. Oral hygiene has to be maintained at a high level if a favourable long-term prognosis for restorative procedures is to be achieved.[7]The wide plethora of presentations of this disorder does not permit a single course of the treatment for every patient. The treatment protocol has to be tailored to meet the needs of individual patients and the severity of the affliction. Compromised esthetics, sensitivity to mechanical and/or chemical irritants and loss of vertical dimension leading to a breakdown of the masticatory apparatus are the most common problems associated with this disorder.[8]Clinical problems of AI patients mainly include poor esthetics, sensitivity of teeth, chewing difficulties, loss of tooth substance due to chipping and attrition, higher risk for dental caries, open occlusal relationship and decreased occlusal vertical dimension.[9,10]
Regarding etiology numerous studies have reported a variety in its inheritance pattern including either autosomal, x-linked dominant or recessive modes. More specifically, it was mentioned that enamel hypoplasia is inherited predominantly in a sex- linked, incomplete, dominant trait, whereas the enamel hypo- mineralisation in an autosomally dominant manner.[11]Rehabilitation of complicated cases poses difficulty in clinical practice, both with respect to restoring function as well as esthetics. One such condition where the clinical practitioner has to give importance to proper planning of the treatment and execution of the plan is amelogenesis imperfecta, wherein both esthetics and function are compromised.[12]

DISCUSSION:

Genetics:

Mutations in the AMELX, ENAM,MMP20,KLK-4,FAM83H,WDR72,C4orf26,SLC24A4 ,LAMB3 genes have been found to cause amelogenesis imperfecta (non-syndromic form). AMELX and ENAM encode extracellular matrix proteins of the developing tooth enamel and KLK-4 and MMP20 encode proteases that help degrade organic matter from the enamel matrix during the maturation stage of amelogenesis. SLC24A4 encodes a calcium transporter that mediates calcium transport to developing enamel during tooth development. Less is known about the function of other genes implicated in amelogenesis imperfecta.

Treatment:

The treatment of this condition depends on the type of AI that one is suffering from. This can range from providing preventive care, such as the use of Sealants and Bonding treatment.

- Orthodontic extrusion of the teeth after surgical exposure and fixed restorative approaches
- Removable acrylic over denture
- Cast removable overlay denture
- Inlay or onlay restoration
- Treatment objectives for the young adult patient also include the relief of pain and the improvement of facial esthetics and function.
- The treatment of AI must be multidisciplinary with initial extractions and periodontal therapy ranging from oral prophylaxis to surgical interventions such as crown lengthening procedures and root coverage procedures. The prosthodontic rehabilitation of these patients may require as little as few dental restorations to full mouth reconstruction to regain lost vertical dimension with occlusal and esthetic makeovers. The degree of mineralization and quantity of the enamel present is a critical deciding factor in the selection of bonded partial or full coverage restorations.[8]

Type I AI Treatment:
Based on the characteristics of an AI patient, treatment requires multiple phases to achieve function, esthetics, and maintain its form. This is often done in cases involving AI in children aged two to six years old. However, those aged 6 to 11 may have early mixed dentition, while those aged eleven and higher will be provided with permanent dentition. Restorative care has to be taken into full consideration as the social and psychological development of the child may be in trouble if AI is not treated during their early years. An abnormal set of teeth causes a lot of embarrassment and humiliation to patients, especially during their years of growing up. Permanent dentition may be a bit expensive but can ensure a normal lifestyle for suffering children.

This type of treatment is more suitable for restorative forms of therapies, such as bonding teeth to the enamel. The material used may be made of porcelain or composite resin, so that it can easily be bonded to the anterior part of the teeth. The shape, size and colour may need to be slightly altered. Orthodontic therapy may also be used so that the interdental spaces may be partially close. There are no issues in using existing amalgams and composite resins on the tooth. Nevertheless, malformed teeth may need a full dental coverage so that crowns may be placed for the assistance of dentures and the like.

Characteristics of four main amelogenesis imperfecta types :

- Hypoplastic (type I)-Multiple enamel pits, severeattrition of the enamel leads to opening of the proximal contact area, snow-capped appearance, thin enamel layer with yellow or brown colour, permanent teeth eruption problems

- Hypomaturation (type II)-The enamel is mottled in appearance, relatively normal in thickness, but it is softer than normal, the teeth appear as creamy opaque to yellow brown, often with open bite and dental sensitivity. Hypomineralization is often manifested as enamel chipping or wearing.

- Hypocalcified (type III)-The teeth is opaque white or yellowish brownthe enamel surface is rough, the enamel has a normal thickness, the enamel chips away easily, and there are dental sensitivity, open bite, heavy calculus formation.

- Hypomaturation/ hypoplasia/ taurodontism (type IV)- Similar to both the hypoplastic and hypomature types, in addition to taurodontism and anterior open bite with skeletal basis.

Amelogenesis Imperfecta Causes:

The disorder is said to result from genetic factors. The condition is linked to autosomal recessive, x-linked and autosomal dominant conditions. It is reported to be transferred from one generation to another as a dominant genetic trait. This indicates that an individual only requires a copy of the gene from either parent to suffer from the disease.

Symptoms:

The enamel of the tooth is soft and thin. The teeth appear yellow and are easily damaged. Both baby teeth and permanent teeth are affected.

Amelogenesis Imperfecta Prevention:

Calculus deposits are more prominent with AI. This makes it all the more necessary for affected children to practice strict oral hygiene because of the rough enamel surface and altered salivary flow rate (due to dental sensitivity). They may need to visit dentists more frequently for cleaning and evaluation. Deep scaling may be done to keep off deposits. Proper dental hygiene will guarantee the health of your teeth and gingiva.

CONCLUSION:

Enamel formation requires the expression of multiple genes that transcribes matrix proteins and proteinases needed to control the complex process of crystal growth and mineralization. Mutations in the amelogenin, enamelin, and kallikrein-4 genes have been demonstrated to result in different types of AI. A number of other genes critical to enamel formation have been identified and proposed as candidates for AI, including ameloblastin, tuftelin and enamelysin. AI can also be caused by an alteration in a gene that is neither known nor considered to be a major contributor to enamel formation. Continued mutational analysis of families with AI will allow a comprehensive standardised nomenclature system to be developed for this group of disorders that will include molecular delineation as well as a mode of inheritance and phenotype.

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