Oral Rehabilitation of a Patient with Amelogenesis Imperfecta - A Case Report

Mark Levin, DMD, B.Med.Sc
Managing Director, Mediclinic
Private Medical Center, Tel Aviv, Israel

Nitay Pauker, DMD, B.Med.Sc

Abstract

The following case is one of a young woman with Amelogenesis Imperfecta (AI).

A young woman of 26 years arrived in the clinic presenting severe dental and consequent psychological and behavioral problems. She was diagnosed with Amelogenesis Imperfecta Type IV. This condition resulted both in massive and extensive dental damage and many problems associated with AI - poor esthetics, loss of function, deterioration of the gingival tissues, impaired speech, self-consciousness due to difficulties to appear and speak in public and deep dental phobia. A comprehensive treatment plan was prepared, with the challenge of completing it within a single session under general anesthesia.

The treatment plan objectives were to restore esthetics, function and soft tissue health. Among the procedures performed were periodontal treatments with an Er:YAG laser, gingival recontouring, crown lengthening, root canal treatments and non-metal esthetic crowns.

This case presented many clinical challenges and requires creative solutions. In order to obtain a favorable outcome of long-term success resulting in caries free teeth and healthy soft tissues, many variables had to be taken in consideration. All of those will be elaborated in this case report.

Background

Amelogenesis Imperfecta (AI) is a complex of hereditary defects constricted to the dental enamel hard tissue.

This entity of enamel alteration targets the mineralization process and therefore affects both the deciduous and permanent dentitions. This condition was known since 1890 when Spoke described it as "Hereditary Brown Teeth". The prevalence of AI is 1:14,000.

AI is caused by mutations of several genes responsible for enamel formation. These genes are isolated and recognized as five enamel formation genes: AMELX, ENAM, KLK4, MMP20 and DLX2.

Mutation of the AMLEX gene causes x-linked AI, whereas mutation of the AMEL gene causes autosomal inherited AI.

The clinical appearance is varied and divided into four group categories:

Type I: tiny perforations scattered across the enamel surface. The distribution of those lesions can be localized as well as generalized. The damage lies within alteration of enamel matrix deposition.

Type II: characterized by hypomaturation of enamel formation. As a result the enamel appears opaque and of chalk-white coloration. The enamel layer's thickness is normal but the hardness is impaired and separates easily from underlying dentin.

Type III: the enamel is increasingly thin and has a worn brownish appearance. Its mineralization is insufficient. It separates easily from the dentin soon after the tooth erupts. The teeth are extremely sensitive to thermal stimuli.

Type IV: characterized by hypomaturation as well as hypoplastic enamel. This is the most common type of AI and it is associated with taurodontism phenomenon.

Case Report

A 26 year old female patient arrived at the clinic presenting poor oral esthetic and functional status. The patient was healthy, without any known allergies, no susceptibility to drugs.

The diagnosis showed the patient suffering from severe molar wear and failing bridgework with carious abutments due to AI type IV. The patient's chief complaint was poor esthetics and difficulty of masticatory function, considerable discomfort and sensitivity. The history did no reveal any familial AI cases.

An extra oral examination showed incompetent lips.

An intra-oral examination revealed an anterior open bite and a gummy smile. All of the permanent dentition was present. A severe wear of occlusal aspects was present causing extensive cavities in the molars.

Deep carious lesions were observed and the gingival status was found to be extremely deteriorated with severe inflammation, swelling and redness of the gingival. Pockets amounting up to 8 mm were recorded.

The oral hygiene of the patient was very poor with large plaque index and calculus.

Reference:

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