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DENTINOGENESIS IMPERFECTA: A CLINICAL CASE REPORT ANTERIOR MOUTH REHABILITATION AND PUBLIC HEALTH ASPECT

Dentistry

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ABSTRACT

The size, shape, number, and tissue structure of teeth in human dentition varies greatly. Dental conditions can run in families. Tooth discoloration caused by DI conditions can range from milky blue-gray to dark tan. Due to the weakening of the dentin structure, DI patients frequently experience severe dentition wear, missing tooth structure, missing teeth, and missing vertical occlusal dimensions. A female patient, age 39, was seen with brown tooth discoloration and wear on all of her front teeth. The patient claimed that her mother was to blame for her dental issue.

KEYWORDS

Dentin; Dentinogenesis Imperfecta Type II; Brittle tooth syndrome; Zirconia crown; anterior mouth rehabilitation.

INTRODUCTION

The size, shape, number, and structure of teeth in the human dentition, as well as the composition of the tooth tissue, differ significantly. Dental disease may run in families. One such illness or disorder is dentin hypoplasia. [1]

Human dentition's structural and digital integrity is influenced by a variety of internal and external factors, which are linked to the emergence of a number of clinical diseases. Deciduous teeth and permanent dentition are both affected by the genetic disorder dentin hypoplasia (DI), which is autosomal dominant. [2]

Meyer might have made the initial discovery in 1882. The disease was initially described in a published report as a tooth defect.

Talbot published enamel, according to Witkop (1971). Finn (1933), Skillen (1937), and Hodges (1938) coined the phrase "hereditary milky white dentin" to refer to milky white teeth without a pulp cavity that is brown translucent. [3]

The pulp arc and pulp cavity vanish due to a localized mesodermal dysplasia that is characterized by milky-white teeth made of dentin that is atypically shaped and insufficiently mineralized. [4]

Hereditary milky white dentin, also known as dentin hypoplasia (DI) Type I, is an inherited condition that affects dentin development. Deciduous teeth and permanent dentition are typically impacted by DI status.

According to estimates, DI affects between 1 in 6000 and 8000 individuals. [3] Dental discoloration, ranging from milky white blue-gray to dark yellow-brown, is typically caused by DI conditions. DI patients frequently experience severe wear of the dentition, missing tooth structure, missing teeth, and missing vertical occlusal dimensions because of the weakening of the dentin structure. Additionally, dental pulp occlusion brought on by root shortening and dentin hypertrophy will occur in teeth affected by DI. Even though the thickness and radiopacity of the enamel in DI patients typically appear normal, enamel detachment is frequently brought on by potentially damaged dentin. Dentin with defects exposed is vulnerable to severe abrasion. Clinical variation between teeth is very common because of the human body's various expression capacities.

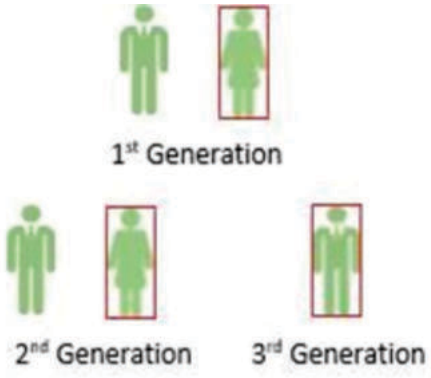
DI symptoms are divided into three categories based on the dentin and bones that are affected. Osteogenesis imperfecta (OI) and the dental phenotype known as Type I (OMIM #166200). Genetic changes in the genes for type I, COL1A1, and COL1A2.2 collagen are the most frequent cause. Mutations in the gene encoding dentin salivary phosphoprotein are linked to types II and III. [5]

Public Health Aspect:

With high penetrance and a low mutation rate, Dentinogenesis Imperfecta (DI), also known as hereditary opalescent dentin, is inherited in a simple autosomal dominant manner. Both the permanent and temporary dentition are frequently affected.

The localized type of mesoderm dysplasia seen in tissue differentiation is referred to as DI. Early diagnosis and treatment are therefore essential to achieving a good prognosis because late intervention makes treatment more difficult.

It has a straightforward autosomal dominant inheritance pattern, high penetrance, and a low mutation rate. Both the primary and permanent teeth are affected. Although widely accepted, Witkop and Shields' classifications are not satisfactory. Genetic research on type 1 DI established osteogenesis imperfecta's distinction from DI and demonstrated that type -2 and type-3 DI can be caused by a single mutation in the DSPP gene. Neville, therefore, provided a modified classification [6]



The Pedigree of DI type II in the patient's family

Case History Chief Complaint

The main complaint of a 39-year-old female patient was that all of her anterior teeth were wearing down and had a brown discoloration. The patient claimed that her mother gave her the dental condition. She claimed that her family had no history of bone fragility or fractures.

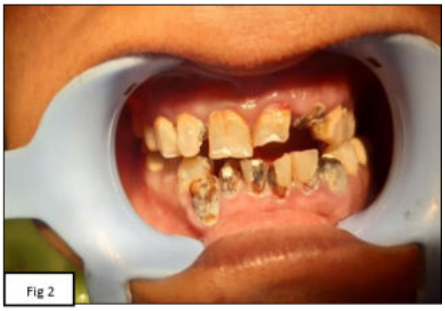
She claimed that occlusal trauma-related fractures had caused her to lose several teeth. The patient complained of having discolored teeth about three years ago, and they had received root canal treatments #35, #36, and #44. Her family history revealed that she has two other siblings, both with similar dental problems.

Clinical Examination

There were no palpable nodes or tender muscles in the patient. Lips, cheeks, tongue, oral mucosa, and pharyngeal tissues all had soft tissues that were within normal ranges. The saliva was stringy and thin. Her teeth all appear to have dark yellowish-brown opalescent discoloration, according to a hard tissue examination.

An intraoral examination revealed that the mandibular anterior and maxillary posterior teeth had the mildest generalized plaque and calculus deposits. In relation to 11, 12, 13, 14, 21, 22, 23, 24, 31, 32, 33,

34, 41, 42, 4 3, & 44, a clinical examination revealed brittle teeth and gingival recession.



Radiographic Examination

The lower lateral incisors of the patient showed root resorption on intraoral periapical radiographs. A few teeth also had radiopacities in the pulp chambers and short bulbous roots.

Diagnosis

The following diagnoses were also made in addition to DI type II: generalized mild gingivitis, fracture at teeth #21, 22, 31, 32, 33, 41, 42, & 43.

Treatment Plan

- Before beginning any treatment, the patient was given the following treatment plan and a thorough explanation was given.
- The benefits, alternatives, and risks of the course of treatment were discussed. Before beginning treatment, informed consent was obtained.
- We intended to perform scaling and root planning, give oral hygiene advice, and extract tooth #43 during the control phase. For the maxillary and mandibular arch, we intended to perform RCT AND ZIRCONIA CROWN #11, 12, 13, 14, 21, 22, 23, 24, 31, 32, 34, 41, 42, 43, & 44 during the definitive phase.
- We intended to set up a prosthodontic and periodontal recall schedule during the maintenance phase.

Treatments

Control Phase

- After scaling and root planning, tooth number 43 was removed.

Definitive Phase

- She was given the proper antibiotic prophylaxis and underwent the procedure while under local anaesthesia after a cardiologist cleared her to do so.
- RCT was used to treat teeth 11, 12, 13, 14, 21, 22, 23, 24, 31, 32, 33, 41, 42, and 44. An analysis of the histology revealed generalised chronic inflammatory changes.
- once the RCT is finished and all teeth have ZIRCONIA crowns.
- The patient experienced no pain as a result of the treatment, and she was happy with the aesthetic outcome as well as the improvement in her ability to bite and chew.

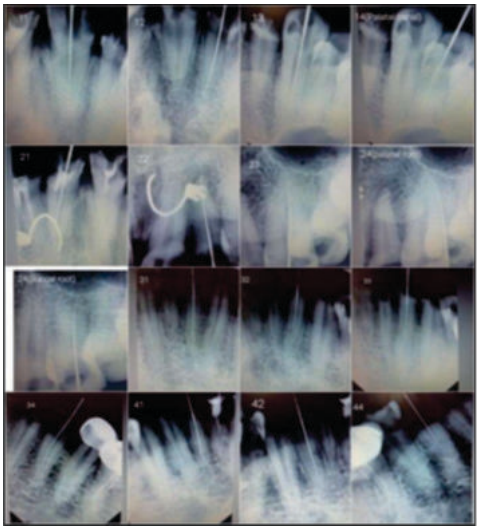


Fig. 3 Working Length IOPA

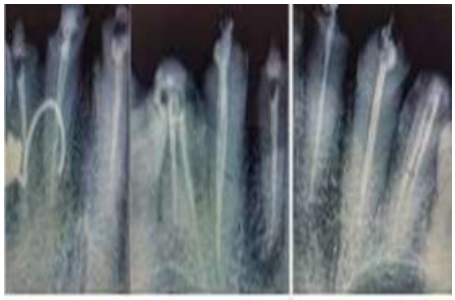


Fig. 4 Obturation IOPA

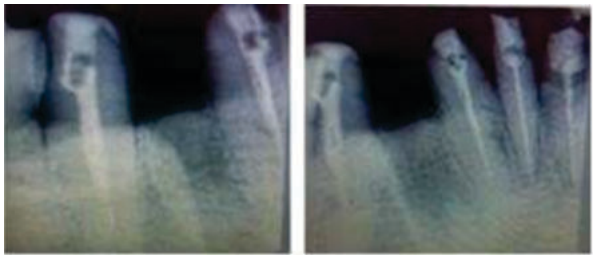


Fig. 5 Obturation IOPA

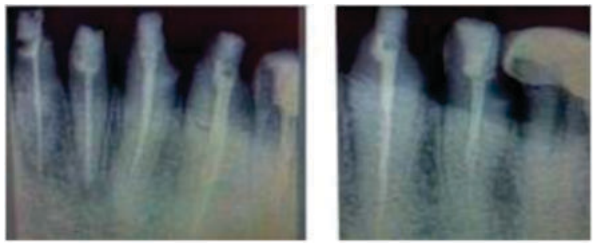


Fig. 6 Obturation IOPA

Fig. 7 Obturation IOPA

Maintenance Phase

The patient was advised to come for regular follow-up. (Fig. 8 After Treatment)

Preventive Care

Teeth that require care can be found during routine check-ups. Tartar removal, good oral care, and mouthwashes can all help with periodontal health. Desensitizers and fluoride treatments may lessen tooth sensitivity.

DISCUSSION

Dentinogenesis imperfecta (DI) is perhaps the most well-known hereditary disorder influencing the design of dentin. Robert and Schour coined the phrase "dentogenesis imperfecta" in 1939.

Dentin phosphoprotein (DSPP) and dentin sialoprotein are the two proteins involved in the synthesis of dentin (DSP). In addition to bone, kidney, salivary organs, and lungs, DSPP is communicated in a variety of tissues. However, the appearance of DSPP in dentin is significantly higher than that of DSPP in other tissues. Clinical signs of DI include irregularities in these proteins' emission, as well as their precise shape and dentinal framework's placement of hydroxyapatite gemstones. The genes at 4q12–21 are responsible for producing both DSPP and DSP. [7,8]

Clinically, the presence of teeth with DI is characterized by a high degree of clarity resembling gold and shading that shifts from yellow to pale blue dark when viewed in sent or mirrored light. Wide crowns and a tightening of the cervical region on impacted teeth give them a tulip shape. [9] The teeth appear to be strong on radiographs, devoid of mash chambers and root trenches, with only minor to moderate occlusal surface wear, short, thin roots and tightening of the cervical portion of the tooth, which gives the crown a bulbous appearance. [10] OI, Ehlers-Danlos syndrome, Goldblatt syndrome, Schimke immunoosseous dysplasia, and brachioskeletogenital disorder are disorders connected to dentinogenesis imperfecta. [11]

For people with DI, early detection and preventive interventions are essential if they are to avoid the disorder's detrimental social and functional effects.

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