Endodontic and esthetic management of Hypoplastic Turner’s tooth with open apex

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ABSTRACT

Turner’s hypoplasia is usually seen in premolar (as a result of infected deciduous molars) and permanent central incisors (due to trauma to the deciduous incisors). The enamel of a tooth comprises of more than 90% mineral, which breaks down in an acidic pH, such as that found at the apex of a root of an inflamed or infected deciduous tooth. As a result when the permanent tooth erupts, a roughened, irregular, or pitted area in the enamel, corresponding to the defect, may be observed. These hypoplastic teeth may become non-vital without any carious insult, cavitation or further trauma to the permanent tooth, if left untreated. We have attended a case on Turner’s hypoplastic tooth (due to trauma to the primary tooth) which had become non-vital without evidence of any carious attack or injury to the permanent tooth. Clinically tooth was mild mobile, presence of brownish discoloration and discharging sinus, tender on percussion and presence of single tooth crossbite in relation to the effected tooth. Radiographic examination showed periapical radiolucency with wide open apex. The treatment protocol was to promote a complete oral rehabilitation in both esthetics and function. Firstly endodontic treatment of affected tooth was done by using MTA apexification guided by PRF matrix and secondly esthetic rehabilitation done by light cure composite restoration with palatal contouring for correction of crossbite.

KEYWORDS: Turner’s Hypoplasia, Nonvital permanent tooth, Open–apex, Apexification, Mineral Trioxide Aggregate, Platelet Rich Fibrin

INTRODUCTION

Hypoplasia occurs as a consequence of interruption in the process of enamel matrix formation, leading to a qualitative and quantitative defect of enamel. If enamel hypoplasia affects only one tooth in the mouth, is referred to as a Turner’s tooth.⁸ If a canine or a premolar presents with Turner’s hypoplasia, the most likely cause is an infection and presence of inflamed tissues around the root of the heavily decayed primary tooth & permanent tooth bud which affects the development of the permanent tooth. If Turner’s hypoplasia is found in the anterior region of the mouth, the most likely cause is a traumatic injury to the deciduous tooth. The tooth receiving trauma (usually a deciduous maxillary central incisor) transfers this injurious force to the developing tooth bud beneath it and as a consequence affects its mineralization of enamel. Because of the normal position of the developing permanent tooth bud with respect to the primary tooth, makes the facial surface being the most affected area on the permanent tooth.⁸ & ¹⁰ The characteristics of Turner’s hypoplasia include unfavorable esthetics (White or yellow discoloration), higher dentinal sensitivity, malocclusion and dental caries susceptibility. Hypoplastic teeth, if left untreated can lead to pulpal pathosis resulting in a non-vital tooth. We have attended a case on Turner’s hypoplastic tooth (due to trauma in the primary tooth) with open-apex which had turned non-vital without any carious insult or trauma in the permanent tooth.

The absence of a natural apical constriction in a hypoplastic nonvital permanent tooth poses a challenge in endodontic therapy. There is a need arises to create an apical barrier for optimal condensation of obturating materials. In MTA apexification, the technical problem encountered is the extrusion of sealing material. A matrix material can be used to alleviate this deficiency. Platelet-rich fibrin (PRF) is an immune platelet concentrate, which can serve as a scaffold against which the obturating material can be condensed. It also helps in wound healing and repair. The article presents a case of endodontic & esthetic management of non-vital hypoplastic Turner’s teeth with open apex using MTA apexification guided by PRF as an internal matrix and esthetic restoration of the same tooth with Light Cure composite.

CASE REPORT

A ten years old male patient reported to the Department of Pedodontics and preventive dentistry of GNIDSR with the chief complain of a discolored tooth, occasional pain and swelling in upper front tooth region since three months. Clinical examination revealed brownish discoloration of the crown with grade-I mobility, the absence of caries, tender on percussion and discharging sinus in relation to 11 and presence of single tooth crossbite in relation to the affected tooth (Fig1). His past dental history revealed that at 26 months of age he had...
injured his primary maxillary central incisors during play. The injury led to an emergency treatment involving the suturing of the lip and antibiotics at a hospital, but no professional dental treatment. When the permanent successor erupted in that region, it had unsmooth, asymmetrical, pitted areas in the enamel. Other medical records of the patient were non contributory. The radiograph showed thin radicular dentinal wall with periapical radiolucency in the proximity to the open apex of 11(Fig2). Pulp vitality test showed a negative response. The treatment plan was first, MTA apexification in 11 guided by PRF matrix and secondly esthetic rehabilitation with light cure composite restoration with palatal contouring for correction of the crossbite of the same affected tooth.11

Under local anesthesia and proper isolation, access cavity was prepared on 11. Working length was determined (Fig3). The canal was then gently cleaned with minimal instrumentation followed by copious irrigation with normal saline and 2.5% NaOCl. The Tri-antibiotic paste containing metronidazole, ciprofloxacin & amoxicillin (Protocol given by Thomson and Kahler in 2010) was placed in the root canal (Fig4) and access cavity sealed with temporary restoration (CAVIT TM). After one week, the tri-antibiotic paste was removed from the canal by copious irrigation with normal saline. The canal was dried with paper points. PRF was prepared using the protocol given by Dohan et al.(Fig5) Blood (8.5 ml) was drawn by venipuncture of the antecubital vein of the patient and accumulated in a 10 ml sterile test tube without incorporating any anticoagulant. Immediate centrifugation of the tube was done at 3000 revolutions/min (rpm) for 10 min. After centrifugation, the product in the glass tube comprised of a top-most component of acellular platelet poor plasma, a middle layer of platelet-rich fibrin coagulum and red blood cells at the base.9 PRF membrane was cut into pieces & placed into the canal with hand pluggers to form an apical barrier at the level of the apex.MTA (angelus) was mixed according to the manufacturer’s instructions and condensed in the apical portion of the canal with hand pluggers against the PRF matrix till 4mm thickness achieved at the apex (Fig6). A moistened cotton pellet was placed over the MTA and access cavity sealed with CAVIT™. After 24 hrs, the Cotton pellet was discarded. For confirmation of the proper setting of the MTA barrier, a hand pluggers was tapped against it. The fiber optic post (Coltene) was inserted into the canal and cemented with resin cement (Ivoclar Vivadent) (Fig7).The discolored, pitted & chipped off crown portion was removed and restored with light cure composite (Fig8).Crossbite was managed by palatal contouring of light cure composite. After 24 hours, the surfaces were polished with flexible discs and rubber points (Fig 9).

Six months follow up of this case showed successful outcome both clinically and radiographically and the patient was instructed for further follow up at every 6 months interval (Fig10).
Disturbances in the development of permanent teeth can be the result of injury to the deciduous teeth due to the close vicinity of the root of the deciduous teeth to their permanent successors. At the age of 1-3 years, severe intrusion by primary tooth and invasion of the permanent tooth bud during the earliest phases of odontogenesis, may cause misdevelopment of the permanent tooth.\(^1\) Diana Ribeiro et al. concluded from their eight years of longitudinal study that discolorations seen in enamel or enamel hypoplasia were the most prevalent sequelae of permanent dentition due to traumatic injury.\(^8\) In addition, dislocation of the deciduous tooth root may hamper the development of the permanent tooth by causing changes in the secretory stage of the ameloblasts or, in subsequent stages, by affecting the root formation process.\(^11\) The brownish discoloration is a result of disruptions in the ameloblastic layer, leading to imperfect matrix creation generated by traumatic insults, but the dentine formation is not affected as the inner enamel epithelium maintains its inductive properties to cause differentiation of young odontoblasts.\(^8\) These structurally defective teeth are not only weak but also provide a favorable area for colonization of bacteria. The hypoplastic teeth are seven times more sensitive to carious attack compared to those without hypoplasia. Vahid Golpaygani and Mehrdad et al. concluded that the pace of caries progression in hypoplastic teeth was much higher than in normal teeth. The presence of defective enamel and open dentinal tubules predisposing to pulp necrosis as they are gateway for entry of bacteria into pulpal tissue, could have made the tooth non-vital.\(^8\) In this cases, the hypoplastic permanent tooth had turned non-vital without any history of trauma or any carious insult in the permanent tooth but the patient had a history of trauma in their deciduous dentition at 26 months of age.

Hypoplastic enamel can be effectively treated by restoring the affected enamel (bonding a tooth-colored agent to the tooth in order to keep it unharmed from further wear). In most cases, interim therapeutic restorations are necessary until definitive rehabilitation is possible.\(^8\) Composite resin is a restorative material that restores esthetic with high quality, minimal wear, and durability.\(^9\) In present case crown of the tooth restores by light cure composite resin. Correction of the crossbite of the same affected tooth also done by the shaping of composite restoration.

When the hypoplastic tooth with immature root formation undergoes pulpal necrosis, the root development affected and apical closure is not achieved. Endodontic treatment during this stage is a momentous challenge, due to the canal size, the weak and brittle dentine walls and the large open root-apex.\(^1\) & \(^2\). Traditionally, treatment of non-vital immature teeth involves creating a calcified barrier to promote the formation of a hard apical barrier at the open apex, Ca(OH)\(_2\) apexitication is most commonly used. But the disadvantages of this procedure are longer treatment time, multiple visits, increased chances of re-
infection due to microleakage from failed temporary restoration. A hypoplastic tooth is already weak tooth, chanced of increased tooth fracture is more in Ca(OH)₂ apexification.

MTA is a biocompatible & bacteriostatic material, it has the good sealing ability & also helps in the formation of bone & periodontium around its interface, can be used to create a physical barrier. It promotes apical hard tissue formation with greater consistency than Ca(OH)₂ apexification. But the disadvantages of MTA is an obturating material difficulty in placement in curved canals, slow setting time and high cost. Another the technical problem encountered is controlling the overfill or underfill of MTA.

To overcome such problem Lemon in 1992 introduced the ‘internal matrix concept’. Lemon recommended the application of a matrix when the diameter of the perforation is more than 1mm to avoid extrusion of the sealing material. The usage of a matrix material aids in overwhelming this flaw. PRF was developed in France by Choukroun and Dohan. PRF is a matrix of autologous fibrin, where majority of platelets and cytokines secreted by leukocytes are trapped during centrifugation. The intrinsic incorporation of cytokines within the fibrin mesh allows for their progressive release over time (7-11 days), as the network of fibrin disintegrate. PRF membrane acts like a fibrin dressing working as a matrix to accelerate the healing of wound edges. The present case discusses a new concept of using PRF as an apical matrix membrane in hypoplastic non-vital permanent teeth with open apex.

Platelet-rich fibrin membrane possesses a soft texture and has some amount of moisture trapped in it, despite that it is a favourable matrix material for application of MTA, as MTA has a wet sand-like consistency and can be placed without applying any pressure, thus does not require a pressure resistant scaffold for the application. Besides, setting of MTA occurs in the presence of moisture and there is no need of a moisture free surrounding. Other mentionable advantage of PRF as a matrix is its reparative and wound healing promoting capability. In this case, PRF guided MTA apexification after a six month follow up showed the successful outcome of treatment both clinically and radiographically due to the absence of clinical signs, symptoms and reduction and signs of apical closure.

CONCLUSION

Traumatic injuries to primary dentition affect the permanent tooth germ. Careful follow-up of a traumatic injury is very important. Hypoplastic teeth, if left untreated can lead to pulpal pathosis resulting in a non-vital tooth. The combination of PRF as a matrix and MTA as an apical barrier can be considered as a good treatment option for periapical healing and closer to open apex in the non-vital hypoplastic immature permanent tooth. The light-cured composite resin is a good aesthetic material for restoring hypoplastic teeth.

REFERENCES


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