REVIEW ARTICLE

Dentin Hypersensitivity after Cementation of Fixed Partial Denture: An Enigma

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Abstract

Dentin hypersensitivity is characterized by nonspontaneous short sharp pain due to the exposure of dentin in response to stimuli, which can be because of thermal, evaporative, tactile, osmotic, or chemical effect and that cannot be ascribed to any other dental defect or disease. Post-cementation, some of the patients receiving crowns or bridges on vital teeth have reported an incidence of sensitivity. Greater the diameter of exposed tubules, the greater would be the sensitivity. To reduce the incidence of postoperative sensitivity, many methods are advocated which concern with occlusion of dentinal tubules. This article reviews the mechanism and various treatment protocols followed in dentin hypersensitivity.

Keywords: Dentinal tubules, Desensitizers, Laser, Cementation.

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Introduction

Fixed prosthodontic treatment involves the replacement of teeth by an artificial substitute, which cannot be removed by the patient. Implant dentistry has revolutionized the field of fixed prosthodontic treatment. In spite of this, certain clinical situations like the decreased amount of bone, medical complications like uncontrolled diabetes, or financial constraints of the patient necessitate the use of a fixed partial denture. In most of the cases where replacement of teeth is needed, the abutments are vital. Postoperative sensitivity is a common complaint after cementation of the fixed partial denture.[1] Although tooth sensitivity after tooth preparation is a common problem, yet it is very rarely documented, and limited epidemiological data are available.

The risk of pulpal damage depends on numerous factors: heat generated by bur attrition, amount of residual dentin, dentin permeability, method of fabrication of provisional crowns, type of cement used for cementation of provisional and final crowns and marginal infiltration. Dentinal tubules are rendered patent when vital tooth is prepared for receiving a retainer for fixed partial prosthesis. This results in dentin hypersensitivity, causing discomfort to the patient. Dentin hypersensitivity is demonstrated by short sharp pain arising from the exposed dentin in response to stimuli—tactile, thermal, evaporative, osmotic, or chemical, and it cannot be attributed to any other dental defect or disease.[2] The condition is interdependent on dentin exposure and patency of dentinal tubules. Dentin hypersensitivity after cementation of interim restorations has been attributed to microleakage and formation of bacterial byproducts. However, hypersensitivity has been reported even after the cementation of definitive prostheses with the most widely used luting agents like zinc phosphate and glass ionomer cement.

Mechanism of Pain Transmission

Even though the precise mechanism of pain transmission within the dentin is a matter of speculation, there are presently three major theories—(1) the direct nerve ending theory, (2) the odontoblast receptor theory, (3) the hydrodynamic theory. Currently, the hydrodynamic theory is widely accepted. When a pain initiating stimulus is applied to dentin, there is an increase in the flow of dentinal tubular fluid. This in turn mechanically stimulates, the nerves situated at the inner ends of the tubules or in the outer layers of the pulp. Cooling, evaporation, drying, and hypertonic chemical stimuli stimulate fluid to flow away from the pulp, while stimuli such as heating or probing can cause fluid to flow towards the pulp.[3] In prosthodontics, the pressures that are generated during cementation of castings and mastication are transferred to the dentinal fluid. The cement enters the dentinal tubules before it sets, displacing an equal volume of dentinal fluid into the pulp. This could be responsible for the pain that unanesthetized patients experience during the cementation of crowns and could plausibly explain the hydrodynamic theory.[4] The most important factor which affects the fluid flow in dentinal tubule is the radius of the tubuli. If the radius is decreased by one-half, the fluid flow within the tubuli falls to one-sixteenth of its original rate. Subsequently, the creation of a smear layer or obliteration of the tubule can greatly improve the effectiveness of the treatment.[5,6]

The treatment of hypersensitive teeth should be aimed either at altering fluid flow in the dentinal tubules with tubule occlusion or modifying or blocking pulpal nerve response, chemically with agents like potassium or physically. Opium therapy was the earliest method of treatment, which dates back to 400 B.C. Various in-office use of desensitizing agents are available

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like silver nitrate, sodium fluoride, strontium chloride, calcium hydroxide, potassium nitrate, glutaraldehyde and 2-hydroxyethyl methacrylate (HEMA), potassium oxalate, casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), adhesive resins, and Nd:YAG laser. Conclusive evidence of successful treatment protocol still evades us in spite of a multitude of products being available for treatment.

Pashley demonstrated that sealing dentinal tubules with polymeric resins would reduce sensitivity. However, there is a concern with the effect of pretreatment with a desensitizer on the bond strength between dentin and resin composite.[7]

Advancements in Management of Dentinal Hypersensitivity

Diode Laser

Laser therapy was introduced as a method for treating dentin hypersensitivity in the mid-1980s. Laser acts by coagulation of proteins of the dentinal fluid within the tubules, which will reduce the movement of fluid. It also causes occlusion of tubules through partial sub-melting of prepared dentin.[8] Hashim et al.[9] found that a diode laser of 810 nm with 60 seconds exposure provided a decrease in dentin hypersensitivity. According to Zach and Cohen, when the temperature rises above 5.5°C, pulpal necrosis resulted. Between 2.5°C and 5.5°C, mild and reversible pulpitis occurred. Below 2.5°C, no histological changes in pulp tissue occurred. In the laser treatment, the temperatures where below 1.5°C and hence no deleterious effects would be visible clinically.[10]

VivaSens

It is an innovative protein precipitate type of desensitizer by Ivoclar Vivadent that seals exposed dentin by the precipitation of calcium ions and proteins. According to the manufacturer, the polyethylene glycol dimethacrylate triggers the precipitation of plasma proteins in the dentinal tubules. Glutaraldehyde is a cross-linking agent, which is capable of bonding to the amine group of proteins. Potassium fluoride provides additional protection. The method of application is to rub the liquid with a brush onto the tooth surface for 10 seconds. Disperse and gently dry it with air for 10 seconds.[11]

Admira Protect

It is a light-curing ORMOCER-based protective desensitizer by VOCO GmbH. It is indicated for cervical areas and crown margins. It has a special filler technology for higher abrasion resistance and fluoride release. It consists of monomers (bisphenol A diglycidyl ether dimethacrylate, 2 hydroxyethyl methacrylate) organic acids and ormocer.

Method of application is to apply the agent on the dentin surfaces for 20 seconds. This is spread with an air jet and light-cured for 10 seconds. The oxygen inhibited layer is removed with a cotton pellet. It bonds to dentin and penetrates into the tubules creating a resinous tag.[12]

GC Tooth Mousse (GC Corporation, Japan)

It contains casein phosphopeptide amorphous calcium phosphate. This helps in the remineralization of tooth structure.

Method of application is to apply a generous layer of GC tooth mousse to the prepared tooth surface by using an applicator swab. The tooth mousse is left undisturbed for 3 minutes. Rinse the tooth with gentle water spray.

Effect of Desensitizers on Retention of Full Veneer Crowns

Yim et al.[13] stated that the bond strength of cement, which relies on the mechanical configuration of the preparation would decrease, as a result of the treatment of dentin with desensitizers. Chandrashekaran et al.[14] conducted a study to evaluate the effect of seal and protect (Dentsply) and GC tooth mousse on the retention of crowns and concluded that desensitizers when used with zinc phosphate cement reduced crown retention. Application of desensitizers provided a smooth surface, which reduced the retention. Use of desensitizers for crowns cemented with GIC and resin-modified GIC did not affect the retentive properties of the cement.

Conclusion

Even though the use of dentin desensitizers in sealing after crown preparation is scarce, their use is unlikely to cause problems. Adherence to sound principles in the various stages of crown preparation, fabrication and cementation of provisional and final restoration present a more reliable solution for preventing postoperative sensitivity.

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