Response of Pulp towards Various Dental Restorations – A Literature Review

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Abstract: There are various restorative materials have been used in dentistry. Among all these restorative materials each and every material will have their own beneficial effects and harmful effects. The primary objective of this review is to evaluate the pulpal responses and the cytotoxic effects of various restorative materials used in operative and restorative dentistry. Understanding the biocompatibility nature of the restorative material and their cytotoxicity factors helps in better decision making in day-to-day dental practice.

Keywords: Restorative, cytotoxic

1. Introduction

A caries lesion is initiated when microorganisms in dental plaque, commonly streptococcus mutans and lactobacillus to produce acids. It brings about demineralization of inorganic component of tooth and also by enzymatic reaction of organic portion. Caries is the most common reason to restore the tooth. The goals of restoration are to restore teeth to a healthy, function, and aesthetic appearance and to prevent from recurrence of caries. These restorative materials used to restore the tooth can cause mild to severe pulpal reactions. Most common permanent restorative materials used are composite, glass ionomer cement and amalgam.

2. Factors Affecting Pulpal Response

Pulp injury and regeneration appear to be influenced by the summation of the effects of the initial injury, such as the initial carious lesion, cavity preparation trauma, presence of bacteria in the cavity walls, chemical activity of restorative materials, etching treatment and patient factors, including aging and treatment history, followed by postoperative complications such as bacterial microleakage In terms of restorative materials. The most important factors appear to be the ability of the restorative materials to avoid creating pulp injuries and preventing subsequent bacterial microleakage(1,2). Various factors that influence pulpal response are as follow:

1) Remaining Dentin Thickness

Several investigators have proposed that remaining dentine thickness is an important factor in the protection of the pulp(1). With all other factors being equal, for a given chemical the diffusion rate should be inversely proportional to dentine thickness and directly proportional to the fraction of the cross-sectional area of dentine composed of dentinal tubules. Remaining dentine thickness influences the concentration and amount of chemicals diffusing through dentine to the pulp space. Thin dentine appears likely to provide markedly less protection against chemical toxins from restorative materials than does thick (2).

2) Baterial Microleakage

Bacterial microleakage complications include postoperative sensitivity, inflammation, marginal dis- coloration, recurrent caries and the eventual need for endodontic therapy. Inflammation appears to be stimulated by bacterial microleakage more than material cytotoxicity Long-term pulp inflammation in the absence of bacterial microleakage is minimal in patients with mostly all common types of restorative materials. However, unresolved severe inflammation can progress to pulp necrosis and periapical lesion development with localized bone destruction. Therefore, it is important to ensure that bacterial microleakage and unresolved pulp inflammation are always prevented(2).

Types of pulpal responses

- Pupal inflammations
- pulp injury (odontoblast survival)
- Pulpal repair (reactionary dentin secretion)

Stimulated pulp cells during the wound healing process synthesize fibro dentin, which may provide mechanical support for accumulation of specific extracellular matrix molecules and growth factors. Interactions between this dynamic substrate and competent pulp cells might further control polarization and functional differentiation of odontoblast-like cells. Native dentin contains specific information that is capable of eliciting the modulation of cell activity and/or extracellular matrix behaviour in an appropriate pulp environment. (4)

3) Materials used

Zinc phosphate

Zinc phosphate cement can cause severe pulpal damage because of its acidic nature. Toxicity is more pronounced when the cement is placed in deep cavity preparations. In deep cavities, zinc phosphate cement should not be used without intervening liner of zinc oxide eugenol or calcium hydroxide. Thick mixes should be used to minimize pulp irritation and marginal leakage. Pulp may be affected by the components of the material, exothermic heat released during setting of cement, and the marginal leakage that permits the ingress of irritants from saliva. The pulpal injury is mainly due to marginal leakage rather than its toxic chemical properties. The routes of micro-leakage may be within the smear layer, between the smear layer and the cavity varnish, between the cavity varnish and the restorative material (5).

Restorations with zinc phosphate base showed more postoperative sensitivity that may be because of its acidic nature increasing pulpal inflammation or because of lack of adequate marginal seal. (5)

Zinc carboxylate cement:

It contains modified zinc oxide powder and an aqueous solution of polyacrylic acid. It chemically bonds to enamel and dentin and has antibacterial properties. It is well tolerated by the pulp, being roughly equivalent to zinc oxide eugenol cement in this respect (5).

Amalgam:

Amalgam is considered one of the safest filling materials with least irritating properties. Even if varnish is not employed, within a period of few weeks, marginal seal develops between the tooth and the restoration due to its corrosion products. It produces discomfort to its high thermal conductivity. So, liners or bases are necessary to provide thermal insulation. It may cause mild to moderate inflammation in deep caries, inhibition of reparative dentin formation due to damage to odontoblasts, toxicity in high copper alloys, postoperative thermal sensitivity due to high thermal conductivity (5).

Zinc Oxide Eugenol:

EUGENOL- It is a parasubsituted phenol compound. It is acidic in nature, provides palliative and obtundent effect in low concentration (inhibits prostaglandins synthesis). In higher concentration, it chemically irritates the pulp. The pulp, reacts badly to the drilling stimulus (heat and vibration), it frequently becomes severely inflamed and precipitates a condition called acute or chronic pulpitis. This condition usually leads to severe chronic tooth sensitivity or actual toothache and can then only be treated with the removal of the nerve (pulp) called root canal therapy. The placement of a ZOE "temporary" for a few to several days prior to the placement of the final filling usually prevents the sensitivity or toothache and therefore, most times, precludes the need for the expensive and time consuming root canal procedure. Small amounts of eugenol released over time, it happens because of degradation of the surface chelates called LIXIVIATION. Heat passes through the restoration or around micro-leakage, if the released heat is around 113 F, it causes pulpal inflammation, if it is around 130 F, causes permanent pulpal damage (5).

Glass ionomer cement:

It has several important properties such as fluoride release, coefficient of thermal expansion and modulus of elasticity similar to dentin, bonding to both enamel and dentin and biocompatibility (6). Despite these advantages, conventional GICs possess limitations as restorative materials, which are related to their susceptibility to dehydration (7) and poor physical properties, such as high solubility and slow setting rate. It possesses anti cariogenic properties and is well

tolerated by the pulp. Toxicity diminishes with setting time (8).

A study by Has shown that High fluoride releasing materials (Silver Diamine Fluoride) and Type VII GIC did not induce any inflammation/necrosis in the pulp. They have good tertiary dentin inducing ability. Based on these attributes, they can be recommended as indirect pulp treatment materials for the management of deep cavities (9).

Calcium hydroxide:

A calcified barrier may be induced when calcium hydroxide is used as a pulp-capping agent or placed in the root canal in contact with healthy pulpal or periodontal tissue. Because of the high pH of the material, up to 12.5, a superficial layer of necrosis occurs in the pulp to a depth of up to 2 mm. Beyond this layer only a mild inflammatory response is seen, provided the operating field was kept free of bacteria when the material was placed. Results in formation of a hard tissue barrier may be formed. However, the calcium ions that form the barrier are derived entirely from the bloodstream and not from the calcium hydroxide. The hydroxyl group is considered to be the most important component of calcium hydroxide as it provides an alkaline environment which encourages repair and active calcification (12). The alkaline pH induced not only neutralizes lactic acid from the osteoclasts, thus preventing a dissolution of the mineral components of dentine, but could also activate alkaline phosphatase which play an important role in hard tissue formation (13,14). The calcified material which is produced appears to be the product of both odontoblasts and connective tissue cells and may be termed osteodentin. The barrier, which is composed of osteodentin, is not always complete and is porous. It has been shown that the disassociation coefficient of calcium hydroxide of 0.17 permits a slow, controlled release of both calcium and hydroxyl ions which can diffuse through dentinal tubules. Tronstad et al. demonstrated that untreated teeth with pulpal necrosis had a pH of 6.0 to 7.4 in the pulp dentine and periodontal ligament, whereas, after calcium hydroxide had been placed in the canals, the teeth showed a pH range in the peripheral dentine of 7.4 to 9.6. Calcium hydroxide helps in reparative dentin formation (10,11).

Resin modified GIC:

Developments in the field of GIC have led to the introduction of light-activated hybrid GIC versions creating the resin-modified GIC (RMGIC). The incorporation of polymerizable water-compatible monomers such as HEMA to the formulation of conventional GIC resulted in enhanced flexural strength, diametral tensile strength, elastic modulus and wear resistance, although they may not be as biocompatible as conventional GIC. The incorporation of HEMA to the formulation of conventional cements has been proven to increase their toxic effects and as a consequence, RMGIC have been shown to be more cytotoxic than conventional GIC (20). Although the degree of monomer conversion of the RMGIC has not been determined, several studies have demonstrated that measurable quantities of HEMA are released into the storage solutions used. Leached residual HEMA can easily diffuse through the dentinal tubules due to its hydrophilicity and low molecular weight, thus reaching dental pulp cells.

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Adhesive systems and resin composite:

The use of resins as restorative materials is occasionally associated with irritation and necrosis of the pulp (15,16,17), periodontal tissues. Most components of the adhesive systems and resin composites, such as bisphenol A-glycidyl methacrylate (Bis-GMA), urethane dimethacrylate (UDMA), glycol dimethacrylate triethylene (TEG-DMA), camphoroquinone, 2-hydroxyethyl methacrylate (HEMA) and others, have been shown to have definite cytotoxicity when in direct contact with fibroblasts (18). Bonding agents have been found to release camphoroquinone, photoinitiator and photosensitizer widely used to generate free radicals including reactive oxygen species (19). It has been documented that the camphorquinone acts not only as a cytotoxic agent, but also as a mutagen and its lixiviation may partly explain why these kind of resin products are considered as toxic agents.

Post-operative sensitivity is comparatively less in restorations with dentin bonding agent because of formation of hybrid layer and there will be complete sealing of dentinal tubules, reduction in fluid movement in dentinal tubules and less micro-leakage (21).

3. Conclusion

Amalgam plays a major role in longevity of the restoration, since it does not have good aesthetic properties, the usage of amalgam is getting deteriorated. RMGICs are more cytotoxic to the pulp cells than conventional GIC due to the presence of unpolymerized monomers, and should not be applied directly to the pulp tissue. Direct pulp capping with adhesive systems produces different degrees of pulp inflammation, even without bacterial presence and absence of dentin bridge formation as well as pulp repair. Monomers present in resin composites and adhesive systems (e.g.: BISGMA, UDMA, TEGDMA, HEMA) have been shown to have cytotoxic effects as a consequence of direct contact with fibroblasts. Calcium hydroxide is the best choice for conservative treatment of the pulp due to their property of stimulating the formation of sclerotic and reparative dentin as well as protecting the pulp against thermal stimuli. Recently Bio-dentin also plays a significant role in pulp protection and as a better alternate to conventional calcium hydroxide.

References

- [1] A.Hamid & W.R.Hume, The effect of dentine thickness on diffusion of resin monomers in vitro, Journal of Oral Rehabilitation 1997 24; 20-25.
- [2] Bergenholtz.G, Immuno-competent cells in normal dental pulp, J Dens Res 66(6):1149, 1987.
- [3] Sarjeev Singh Yadav, Comparison of patient sensitivity using dentin bonding agent versus zinc phosphate base beneath the amalgam restorations: An *in vivo* comparative study; Year : 2015 | Volume : 4 | Issue : 4 | Page : 236-240
- [4] Dimitrios Tziafas, Basic mechanisms of cytodifferentiation and dentinogenesis during dental pulp repair, Int. J. Dev. Biol. 39; 281-290 (1995).
- [5] Nisha Garg & Amit Garg, Textbook of endodontics: 491-95

- [6] Ogura N, Tobe M, Sakamaki H, Nagura H, Abiko Y, Kondoh T. Tumor necrosis factor-alpha increases chemokine gene expression and productionin synovial fibroblasts from human temporomandibular joint. J Oral PatholMed.2005;34(6):357-63.
- [7] Akimoto N, Momoi Y, Kohno A, Suzuki S, Otsuki M, Suzuki S, et al. Biocompatibility of Clearfil Liner Bond 2 and Clearfil AP-X system on nonexposed and exposed primate teeth. Quintessence Int. 1998;29(3):177-88.
- [8] Mount GJ. Glass-ionomer cements: past, present and future. Oper Dent. 1994;19:82-90.
- [9] Atish Korwar, Pulp response to high fluoride releasing glass ionomer, silver diamine fluoride, and calcium hydroxide used for indirect pulp treatment: An *in-vivo* comparative study; Year: 2015 | Volume: 6 | Issue: 3 | Page: 288-292
- [10] Foreman PC, Barnes IE. A review of calcium hydroxide. Int Endod J. 1990;23(6):283-97.
- [11] Stanley HR, Pameijer CH. Dentistry's friend: calcium hydroxide. Oper Dent. 1997;22:1-3.
- [12] Holland R, Souza V, Mello W, Nery MJ, Bernabé PF, Otoboni JA Filho. Permeability of the hard tissue bridge formed after pulpotomy with calcium hydroxide: a histologic study. J Am Dent Assoc. 1979;99(3):472-5.
- [13]8- Iwamoto CE, Adachi E, Pameijer CH, Barnes D, Romberg EE, Jeffries S. Clinical and histological evaluation of white ProRoot MTA in directpulp capping. Am J Dent. 2006;19:85-90.49- Keiser K, Johnson CC, Tipton DA. Cytotoxicity.
- [14] Pereira JC, Segala AD, Costa CA. Human pulpal response to direct pulp capping with an adhesive system. Am J Dent. 2000;13(3):139-47.
- [15]Baume LJ, Fiore- Donno G. Response of the human pulp to a new restorative material. J Am Dent Assoc. 1968;76:1018-22.
- [16] Stanley HR, Going RE, Chauncey HH. Human pulp response to acid pretreatment of dentin and to composite restoration. J Am Dent Assoc. 1975;91:817-25.
- [17] Stanley HR, Swerdlow H, Buonocore MG. Pulp reactions to anterior restorative materials. J Am Dent Assoc. 1967; 75:132-41.
- [18] Hanks CT, Strawn SE, Wataha JC, Craig RG. Cytotoxic effects of resin components on cultured mammalian fibroblasts. J Dent Res. 1991;70(11):1450-5.
- [19] Huang FM, Chang YC. Cytotoxicity of dentine-bonding agents on human pulp cells in vitro. Int Endod J. 2002;35:905-9.
- [20] Xie D, Brantley BM, Culbertson G, Wang G. Mechanical properties and microstructures of glassionomer cements. Dent Mater. 2000;16:129-38.
- [21] Charlton dg, moore bk, swartz ml. In vitro evaluation of the use of resin liners to reduce microleakage and improve retention of amalgam restorations. Oper dent 1992;17:112-9.