10.5005/jp-journals-10024-2382

ORIGINAL RESEARCH

Study to evaluate the Efficacy of Resin-modified Glass Ionomer Cement Liner as a Direct Pulp Capping Material

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ABSTRACT

Aim: The aim of the present *in vivo* study was to compare efficacy of light-cured resin-modified glass ionomer liner, Vitrebond™ (3M ESPE) with Dycal® (Dentsply) on the healing of pulpal tissue in the event of a direct iatrogenic pulpal exposure.

Materials and methods: Experimental group consisted of Vitrebond™ (3M ESPE) resin-modified glass ionomer liner, and Vitremer™ (3M ESPE) resin-modified glass ionomer cement (GIC) in comparison with the control group of Dycal® (Dentsply) as liner and Poly F® (Dentsply) dental cement. Class V cavities were prepared in 32 sound premolars that were scheduled for orthodontic extraction, and the exposures were capped according to groups. Five teeth from each group were extracted under local anesthesia after an interval of 24 hours, 35 and 60 days, and evaluated for inflammation, fibrotic changes, formation of reparative dentin and bacterial examination.

Results: The present study did not show any statistically significant difference between two groups in terms of inflammation, fibrosis, reparative dentin formation, and bacterial examination.

Conclusion: This study shows that Vitrebond™ (3M ESPE) light-cured resin-modified glass ionomer liner can be used as an alternative to calcium hydroxide as a direct pulp capping material.

Clinical significance: Light-cured resin-modified glass ionomer liner can be an alternative for the calcium hydroxide-based liner for capping iatrogenic pulp exposures.

Keywords: Direct pulp capping, Dycal, Glass ionomer cement, Iatrogenic exposure, Reparative dentin.

How to cite this article: Shameem A, Muliyar S, Thankachan RP, Kalliath JT, Mangalath U, Mangalath S. Study to evaluate the Efficacy of Resin-modified Glass Ionomer Cement Liner as a Direct Pulp Capping Material. J Contemp Dent Pract 2018;19(9):1065-1071.

Source of support: Nil

Conflict of interest: None

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INTRODUCTION

Human dental pulp has evoked much interest, as a specialized connective tissue located in an unyielding chamber of hard tissues around, with its particular arteriovenous supply and its dynamic cellular organization. Dental pulp maintains a unique environment, almost entirely encased in an unyielding chamber, often referred to as its own "coffin." In addition to this, the coronal pulp tissue lacks collateral blood supply, thereby extra nutrients and defense cells are not capable of reaching this coronal tissue as is seen in other healing tissues. Due to these reasons, the very attempt to save the pulp was viewed with suspicion and many believed that it was a fruitless effort. But now the emphasis has shifted to recovery of pulp following injury. Myriad of investigations and research has been conducted on dental pulp response and reaction to various restorative materials and procedures. Also, the ability of dental pulp to heal once exposed has been researched extensively.

Vital pulp exposure does occur in dental practice as well as in traumatic injuries. Once the exposure occurs, the clinician must make an immediate decision whether to cap the pulp or proceed with pulp removal. In case of pulp capping, there has been confusion over material to be used for the same, as in the past several years, new techniques have been promoted and older concept criticized.[1] Iatrogenic pulpal exposures capped by dental students have been found to have a success rate of 92%, wherein carious pulp exposures capped by dental students were found to be only 33%.[2] It has been observed that pulp plays a crucial role in keeping dentin moist,

supple (less brittle), and resilient, which serves to protect the teeth from forces of mastication[3] and the success of direct pulp capping is largely dependent on the clinical situation under which it is performed and age, type, site, and size of pulpal exposure.[4]

History records Phillip Pfaff performing the first pulp capping procedure using small gold pieces over exposed pulp in 1,756 to promote healing. However, calcium hydroxide which was introduced into dentistry by Hermann forms the gold standard in treating direct and indirect pulpal exposures irrespective of its poor physical and mechanical properties.[5] It has been observed that the highly alkaline pH of the calcium hydroxide causes superficial necrosis and subsequent formation of fiber-rich scar tissue. As a response to this, undifferentiated mesenchymal cells differentiate into secondary odontoblasts which stimulate the production of secondary or reparative dentin, resulting in "dentin bridge formation." Recent studies have shown this dentin bridge to have porosity and tunnel defects, leading to disintegration, paving way for microleakage.[6] This may result in infection, inflammation and, finally, death of the pulp. Presence of bacteria within the restoration gap and adjacent dentin has been considered the major factor leading to pulp inflammation and eventual necrosis.[7,8] Therefore, materials that have the advantages of calcium hydroxide and also overcome the disadvantages of the same were explored by different investigators, to varying degrees of success.

Glass ionomer cement, known for its chemical bonding and linear coefficient of thermal expansion similar to that of the tooth, has undergone a myriad of changes, since its introduction by Wilson and Kent in 1972.[9] Although literature shows conflicting views of pulpal response toward glass ionomer when used in deep cavities as liners, recent studies on Vitrebond (resin-modified glass ionomer) have shown it to have an acceptable biocompatibility when used in clinical cases having remaining dentin thickness ranging from 342.3 to 436.1 µm.[10] Therefore, the present study was undertaken to compare the efficacy of light-cured resin-modified glass ionomer liner, Vitrebond™ (3M ESPE) with Dycal® (Dentsply) on the healing of pulpal tissue in the event of a direct iatrogenic pulpal exposure.

MATERIALS AND METHODS

The present study was conducted in the Department of Conservative Dentistry and Endodontics, MES Dental College and Hospital, Perinthalmanna, Kerala, India. The study was approved by the Institutional Ethical Committee. Prior to study, treatment protocol and possible complications were explained to the patients and written informed consent was obtained.

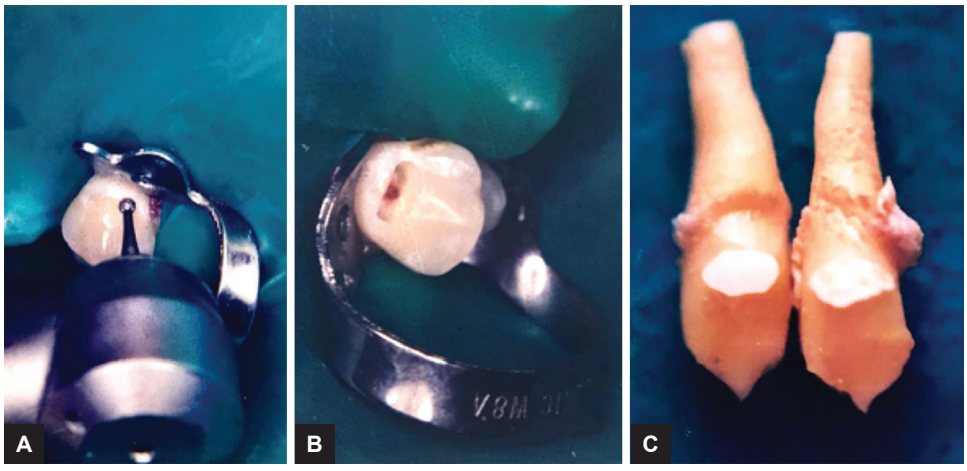
Thirty-two vital human premolar teeth free of caries or any other defects in eight patients scheduled for orthodontic extraction were included in this study. Two teeth were excluded from the study due to a processing error. Age of the patients varied from 18 to 25 years. Teeth were radiographically examined to rule out any periapical pathology, periodontal defects, and resorptions.

The sample was divided into 15 teeth each in experimental and control groups. The experimental group samples were treated with Vitrebond™ (3M ESPE) light-cured glass ionomer liner and Vitremer™ (3M ESPE) glass ionomer restorative cement. The control group was treated with Dycal® (Dentsply) calcium hydroxide liner and Poly F® (Dentsply) cement. All the selected patients underwent thorough oral prophylaxis to remove any calculus or debris that would interfere with the treatment procedure. The test was standardized according to the American National Standards Institute/American Dental Association document number 41 for *in vivo* usage test.

The teeth were polished using pumice paste with rubber cup in a slow-speed micromotor handpiece. Infiltration anesthesia was secured and rubber dam isolation obtained for the tooth to be prepared. Uniform-sized class V cavities, approximately 1mm coronal to the free gingival margin, measuring 2.5 mm cervico-occlusally and 3 mm mesiodistally with an optimal depth of 0.5 mm of remaining dentin, were prepared under efficient water spray coolant (Fig. 1A). Following this, pulpal floor was deepened using a round tungsten carbide bur in slow speed with coolant until exposure (Fig. 1B) was confirmed by the presence of hemorrhage. Bleeding from the exposure site was controlled by sterile cotton pellet compression for 1 to 2 minutes.

In the experimental group, Vitrebond was mixed as per the manufacturer's instruction. It was placed over the exposure site and the floor of the cavity, and light cured for 40 seconds. Vitremer restorative cement was mixed according to the manufacturer's instruction and placed over the Vitrebond liner/base material up to the cavosurface margin and was light cured for 40 seconds. Similarly, in the control group, standardized cavities were prepared on the opposite quadrants using the same criteria. Similarly, Dycal was placed according to the manufacturer's instruction followed by Poly-F cement restoration up to the cavosurface margin (Fig. 2).

Five teeth from each group were extracted under local anesthesia at time intervals of 24 hours, 35 and 60 days. The apical third of teeth were removed at once after extraction by means of a diamond disk using Airotor high-speed handpiece with coolant. To facilitate fixation of the pulpal tissue, the teeth were placed in 10% formalin



Figs 1A to C: (A) Class V cavity preparation under rubber dam, (B) pulp exposure evident, (C) decalcification of teeth

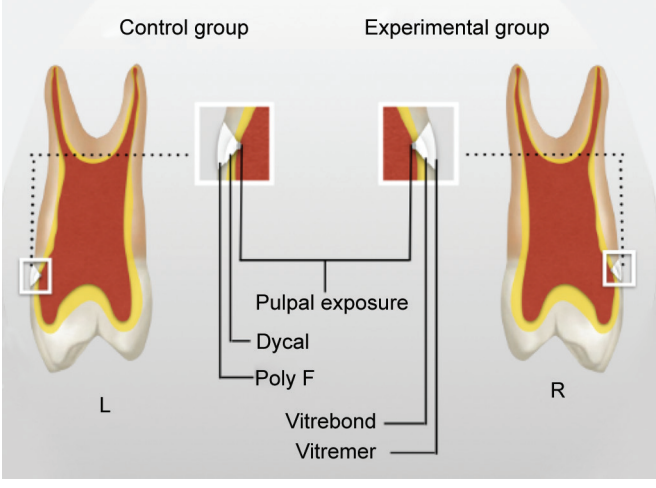


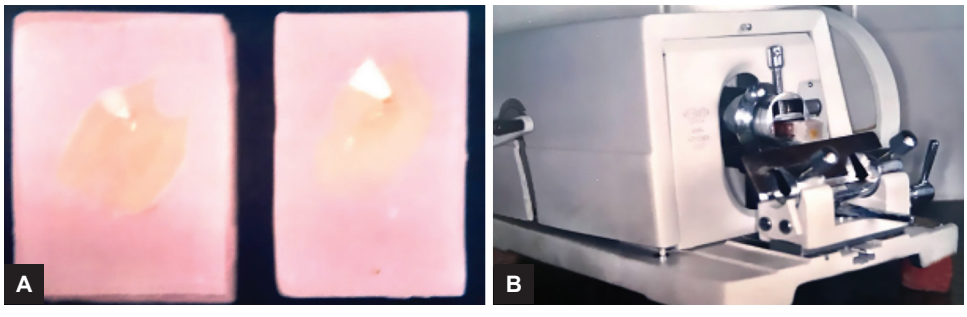
Fig. 2 Schematic representation of pulp capping

solution for 5 days. The teeth were then placed in 10% formol nitric acid for decalcification (Fig. 1C). The solution was replaced every 6 hours, for 7 days. An ammonium oxalate precipitate test was used to confirm decalcification. Once the decalcification was complete, the teeth were embedded in a wax block and longitudinal section was made through the exposure site (Fig. 3A), of 4 µm thickness using a soft tissue microtome (Fig. 3B). The sections were fixed on to the slide and stained by hematoxylin and eosin stains (Fig. 4). The specimens were evaluated for inflammation, fibrosis, and reparative dentin and graded accordingly (Tables 1 and 2). A separate evaluation criterion was made for evaluation at the end of 24 hours. Bacteriological examination for Gram-positive and Gram-negative organisms was carried out using modified Gram's stains[10] (Brown and Brenn stain) (Table 3). The prepared stain was standardized using positive staining of Gram-positive and -negative samples.

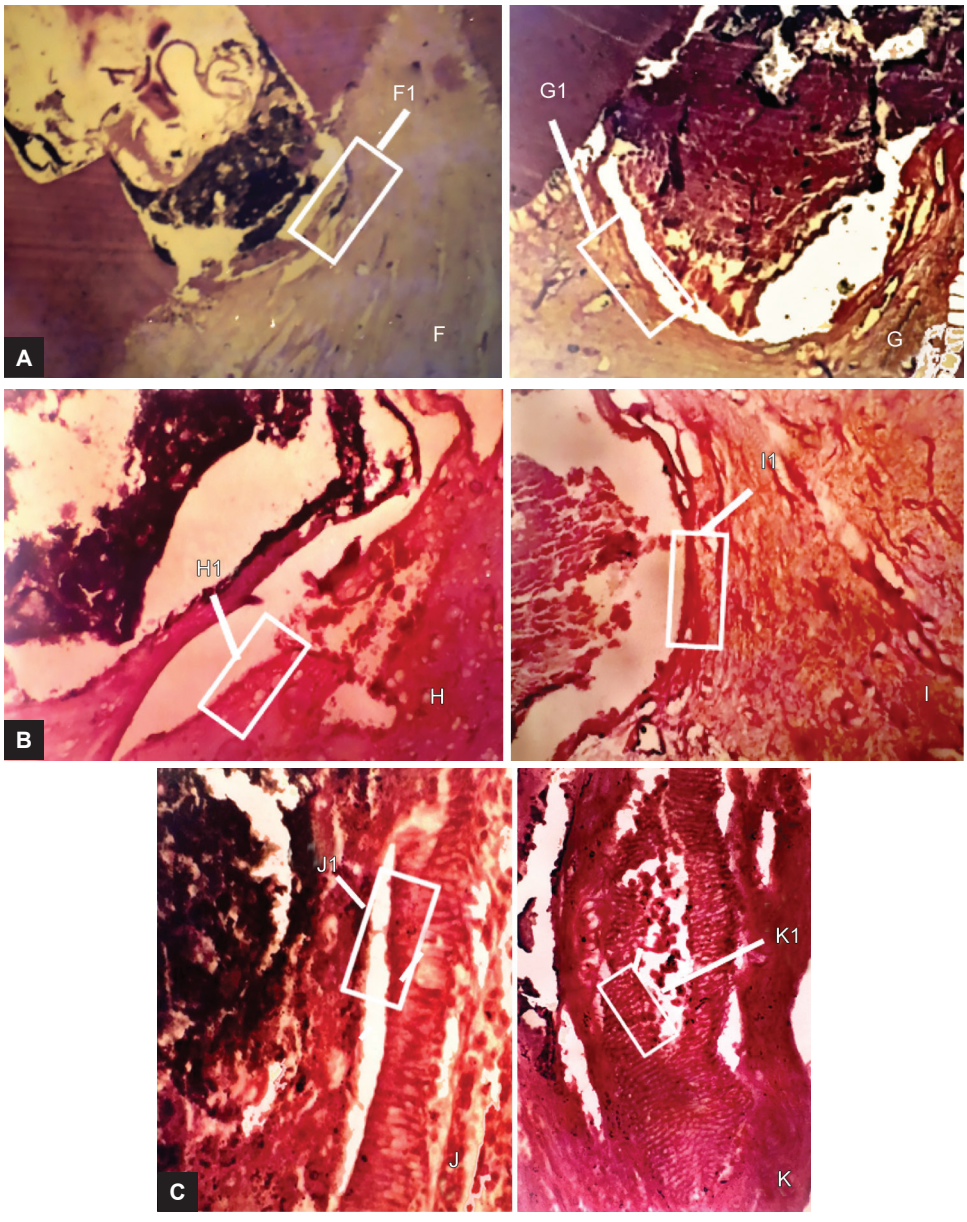
RESULTS

All the patients were free of any clinical signs and symptoms of inflammation during the entire study period. The specimens were evaluated and graded for inflammation, fibrotic changes, formation of reparative dentin at 24 hours, 35 and 60 days and also bacterial examination as per criteria. The results of study are summarized in Table 4.

In the Dycal® (Dentsply) group, it was found that at 24 hours, one specimen showed uneven odontoblastic layer with subjacent slight inflammatory changes, three specimens showed moderate acute inflammatory changes, and one showed severe acute inflammatory change. None of the five specimens showed any bacterial



Figs 3A and B: (A) Decalcified sectioned teeth embedded in wax block, (B) soft tissue microtome used to section the specimen



Figs 4A to C: (A) 24-hour evaluation (4× magnification): F—Dycal specimen; G—Vitrebond specimen; F1, G1—Dense infiltrate of PMNL cells. (B) 35-day evaluation (10× magnification): H—Dycal specimen; I—Vitrebond specimen; H1, I1—PMNL cell infiltrate. (C) 60-day evaluation (40× magnification): J—Dycal specimen; K—Vitrebond specimen; J1, K1 show reparative dentin formation at some distance from the capping material

Table 1: Inflammatory cell response

Grades	*Inflammatory cell response (24 hours)*	*Inflammatory cell response (35 and 60 days)*
I	Normal or uneven odontoblastic layer below tubules of remaining dentin with subjacent slight inflammatory changes	None, either no inflammatory cells or scattered inflammatory cells present at the exposure site, beneath new hard tissue or adjacent to the exposure site
II	Peripheral pulp disorganization beneath remaining dentin wall associated with moderate acute or chronic inflammatory changes	Slight—either acute inflammatory cells (tissues are dominated by large leukocytes and macrophages) or chronic inflammatory cells (tissues are dominated by mononuclear leukocytes [MNLs])
III	Reaction involving coronal pulp associated with severe acute or chronic inflammatory changes	Pronounced (severe)—either PMNLs appearing as an abscess or a dense infiltrate of MNLs that involve one-third or more of coronal pulp
IV	Necrotic pulp	Necrosis

contamination. At 35 days, two specimens showed scattered inflammatory cells adjacent to the exposure site, two specimens showed chronic inflammatory cells, and one specimen showed dense infiltrate of polymorphonuclear leukocytes (PMNL) cells. One specimen showed slight fibrosis, two showed mild response, whereas the rest two showed severe fibrosis. Reparative dentin was present directly adjacent to the capping in three, and in one

Table 2: Evaluation of fibrosis and reparative dentin formation at 35 and 60 days

Grades	*Fibrosis (35 and 60 days) (Increase in fibroblasts and collagen fibers)*	*Reparative dentin formation (24 hours, 35 and 60 days)*
I	None or light	Directly adjacent to the capping agent
II	Mild	At some distance from the capping agent
III	Severe	No evidence of the hard tissue formation

Table 3: Examination of bacteria

Grades	*Bacteriological examination (24 hours, 35 and 60 days)*
I	Absence of bacteria
II	Present along the cavity, including the axial wall and axial floor, within the dentinal tubules and the dental material
III	Present within the cut dentinal tubules of the cavity preparation
IV	Present within necrotic pulp

Table 4: Observation results

Materials	*No. of teeth*	*Inflammatory response*				*Fibrosis*			*Bacterial staining*				*Reparative dentin*		
Dycal		1	2	3	4	1	2	3	1	2	3	4	1	2	3
	24 hours	1	3	1	0	0	0	0	5	0	0	0	0	0	0
	35 days	2	2	1	0	1	2	2	5	0	0	0	3	1	1
	60 days	2	2	1	0	1	2	2	5	0	0	0	5	0	0
Vitrebond	24 hours	0	2	3	0	0	0	0	5	0	0	0	0	0	0
	35 days	2	3	0	0	0	3	2	5	0	0	0	3	2	0
	60 days	3	0	2	0	2	2	1	5	0	0	0	0	5	0

specimen, it was at some distance from the capping material. One specimen did not exhibit any reparative dentin formation. Bacterial staining was absent in any of the specimens. At the end of 60 days, two specimens showed no inflammatory cells at all. Two showed slight chronic inflammatory cells and one showed severe chronic inflammatory cells, which occurred as dense infiltrates. Two specimens showed severe fibrosis, two showed mild fibrosis, and one showed slight fibrosis. Coagulation necrosis was seen in all the specimens adjacent to the reparative dentin. Reparative dentin was seen adjacent to the capping material in all the five specimens. Bacterial infiltration was not present in any of the specimens.

In Vitrebond™ (3M ESPE) group, it was observed that at 24 hours, three specimens showed moderate acute chronic inflammatory changes and two showed severe acute inflammatory reactions. All the specimens were negative of any bacterial contamination. At 35 days, two specimens showed a scattered inflammatory reaction and three showed slight chronic inflammatory reactions. Three specimens showed mild fibrosis and two showed severe fibrosis. Reparative dentin was evident adjacent to the capping material in three specimens and at some distance from the capping agent in two specimens. None of the specimens showed bacterial contamination. At 60 days, three specimens showed no evidence of any inflammatory cells, whereas two showed evidence of severe inflammatory reaction. Two showed slight fibrosis, two showed mild fibrosis, and in one specimen, severe fibrosis was seen. Bacterial contamination was not seen in any specimen. All the five specimens were reparative at some distance to the capping material at the end of 60 days.

Statistical analysis was carried out using Fischer's chi-square test. Analysis showed no significant difference in the results of the two materials at the end of 24 hours, 35 and 60 days.

Over the decade, different materials have been tried out to maintain the vitality of the pulp. Survival of this specialized connective tissue using various materials in the event of an exposure has been debated upon by a number of investigators. The most advocated and time-tested material for pulp capping was calcium hydroxide. The experimentation with other materials, such as mineral trioxide aggregate, tricalcium phosphate, dentin bonding agents, and cyanoacrylates gave varying degrees of success rates. Among these, resin-modified glass ionomers (RMGIs) have been reported to be cytotoxic to the pulp,[11] while in one study, it was found to have a favorable response in the deep cavity as a liner.[12] The present study evaluated the efficacy of RMGI, Vitrebond™ (3M ESPE) as direct pulp capping agent in iatrogenic pulpal exposure in comparison with Dycal® (Dentsply) as the standard.

According to Brannstrom,[7] microleakage is thought to be the culprit for pulpal death rather than material toxicity itself. A direct correlation has been observed between the pulpal inflammation and the bacterial contamination.[13] The antibacterial activity of calcium hydroxide has been attributed to release of hydroxyl ions, which causes damage to the bacterial cytoplasmic membrane, denaturation of proteins, or damage to the deoxyribonucleic acid.[14] These antibacterial effects are short-lived, as it was found by Cox et al,[8] that most of calcium hydroxide medicaments disintegrate and wash out over a period of six months, leading to a 'void' formation underneath the restoration.

The concept of bonding, which revolutionized the field of restorative dentistry, plays an even greater role in minimizing or prevention of the microleakage. Sublining the exposure site with calcium hydroxide and then providing a base over this with GIC would, in fact, decrease the surface area for adhesion with the inherent risk of encouraging microleakage.[15] The reparative dentin formed, by pulpal tissue in contact with calcium hydroxide have been found to have 'tunnel defects', this shortcoming along with solubility of calcium hydroxide leading to microleakage, will have adverse effect on the pulpal healing.[16,17]

In the present study, mild chronic inflammatory cells were seen localized just beneath the reparative dentin formation. Accumulation of macrophages was seen more in cases of Vitrebond™ than Dycal® but not statistically significant. This may be due to the rough surface texture of the material, and pulp interface that might encourage macrophage accumulation, which describes the tendency of macrophages to prefer rough surfaces. These chronic inflammatory cells may have acted as a stimulant for the differentiation of undifferentiated mesenchymal cells into odontoblasts, which in turn formed the reparative dentin. The infiltration of small lymphocytes and plasma cells in areas of chronic inflammation and wound healing concentrate proteins for use by other cells to aid in regeneration or replacement.[18]

Necrosis was not evident in any of the examined specimens at 24 hours, 35 and 60 days, which clearly indicates an absence of any material cytotoxicity for both groups. At 24-hour interval and 35 days, both groups showed a similar inflammatory reaction. On examination after 60 days, Vitrebond™ specimen exhibited complete resolution of inflammation, wherein Dycal® specimen showed slight inflammation; however, both the groups exhibited reparative dentin formation directly adjacent to the capping agent.

As rubber dam was used and caution was taken to provide a sterile environment, none of the specimens showed bacterial contamination during the allocated time period, which supports the literature of the role of bacteria in pulpal necrosis and eventual death of pulp tissue. From the literature, it is evident that pulpal healing in case of direct or indirect exposure is dependent upon a multitude of factors, but the role of inflammation has been underestimated in the healing of pulp. Necrosis or apoptosis, resulting from localized inflammation of pulp, has been considered to be destructive[19] and the presence of inflammation cannot be just correlated to the presence of bacteria. Inflammation is body's repair function and not specifically related to the presence of microorganisms alone. As with any living tissue, when subjected to an insult either chemical or mechanical, pulp responds and reacts via inflammation. Whenever there occurs an irritation to or discontinuity in the odontoblastic lining, there occurs the accumulation of inflammatory cells as an immune response. Due to the limited collateral circulation of the pulpal tissue, it is a known fact that pulp takes time to resolve the inflammatory conditions. The inflammatory reaction has been considered as a precondition for the stimulation of progenitors associated in pulp repair.[20]

In the majority of studies related to pulpal healing or non-healing, healing following capping with medicaments direct or indirect extends to 60 days. This time frame seems to be short, for evaluation of pulp, especially when it is devoid of collateral circulation. The 90-day time frame will be a future directive to be considered to understand the pulpal healing process further. Since this study did not evaluate the 3-month time period, the persistence and effect of the inflammatory condition in long-term are not known.

CONCLUSION

The present study showed that resin-modified light-cured glass ionomer (Vitrebond™) can also be used as pulp capping agent in iatrogenic pulp exposures and the reparative dentin formed is comparable to that of reparative dentin formed with time-proven calcium hydroxide. The bacterial contamination via microleakage and not the material toxicity being the main cause for pulpal inflammation, any material that provides strong and permanent seal combined with antibacterial effect would suffice as an ideal pulp capping material. Though various other material has been proven favorable, none of the materials forms an adhesive bond with the tooth structure, thereby providing a permanent seal. Despite this, further stress is required on time period, the number of teeth and assessment of the quality of the reparative dentin formed in association with the two materials for the certainty of the outcome.

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