Short term human primary pulpal response after direct pulp capping with fourth-generation dentin adhesives

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The purpose of this study was to evaluate the effects of the total-etch and direct pulp capping techniques on the short-term response of mechanically exposed human primary tooth pulps using three commercially available adhesive resin systems. Class V cavities were prepared on the buccal surface of intact mandibular primary molars and exposed with a carbide bur on the cavity floor. The entire cavity except the exposure site received 36% phosphoric acid gel conditioning. Exposed pulps were capped with one of the three adhesive resins, followed by restoration of the cavities with the respective restorative materials. The teeth were extracted after 60 days and prepared according to normal histological techniques. Serial sections were stained with H/B for histological evaluations. The histopathological evaluation showed that a few of the samples in the Scotchbond Multi Purpose (SMP) and Prime & Bond 2.1 (PB) groups exhibited "attempted bridge formation", while no bridge formation was evident in the other samples. Syntac Single Component (Syntac) exhibited the most severe histological response, while the mildest reactions were observed in the SMIP group. Based on the conditions of the present study, direct pulp capping with dentin bonding agents following the total-etch technique in primary teeth can not be recommended.
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INTRODUCTION

It was traditionally believed that the primary cause of pulpal damage was the direct toxic effects from dental materials.[1,2] Subsequent studies, however, demonstrated that bacteria and their components cause pulp inflammation, even through remaining vital dentin.[3,4] Mechanically exposed dental pulp possesses an inherent healing capacity for cellular reorganization and dentin bridge formation. However, this only appears to be true when a long-term bacterial seal is effected and maintained.[8] A tight seal between the exposed pulp and the capping material is, therefore, essential to pulpal healing.

Recently developed adhesive resin systems are capable of creating hybridized zones with superficial dentin,[9-11] thus favoring the exclusion of bacteria from gaining access to the vital tissues of the dentin and pulp. Recent studies demonstrated that application of various dentin adhesive systems to vital dentin provides an effective seal against bacterial microleakage.[12,13] Several studies have evaluated adhesive resin systems as direct pulp-capping materials, in both clinical cases and with histopathological findings.[7,14,15] To date, however, there has been no published data concerning the histological evaluation of these adhesive systems on exposed human primary tooth pulp.

The reasons for limiting direct pulp capping procedures in primary teeth include: the potentials for internal resorption, calcifications, chronic pulpal inflammation, necrosis and intraradicular involvement.[16] It has also been stated that high cellular content of primary pulpal tissue may be responsible for failures of direct pulp capping in primary teeth.'[7] Undifferentiated mesenchymal cells may differentiate into odontoblastic cells in response to either the caries process or the pulp capping material, which could lead to internal resorp-

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Table 1. The adhesive resin systems and their respective restorative materials used in the study.

Bonding Agent	Cavity Code	Restoration	Manufacturer
Scotchbond Multi Purpose Plus	SMIP	Vitremer (Resin-modified Glass ionomer)	3M, St.Paul, MN, USA
Prime&Bond 2.1	PB	Dyract (Polyacid-modified resin composite)	DeTrey/Dentsply, Konstanz, Germany
Syntac Single Component	Syntac	Compoglass (Polyacid-modified resin composite)	Vivadent,Schaan, Liechtenstein

tion. Turner *et al.* [18] noted, however, that these pathological reactions were often seen when calcium hydroxide was used for direct pulp capping. It has been shown that the dentinal bridge formed under calcium hydroxide does not constitute a continuous seal and may allow bacterial leakage,[19] while calcium hydroxide loses its antimicrobial properties and disappears with time under restorations[20], leading several researchers to feel that direct pulp capping procedure in primary teeth is contraindicated.[16,17,21-23]

The purpose of this study was, therefore, to evaluate the short-term histological pulpal responses of the dentin adhesives, Prime & Bond 2.1, Syntac Single Component and Scothcbond Multipurpose Plus as direct pulp capping agents on human primary teeth.

MATERIALS AND METHODS

Table 1 shows the adhesive resin systems and the respective restorative materials used in the study. Twenty-one healthy, non-carious mandibular primary molars (eleven, first molars and ten, second molars) from seven patients that were scheduled for orthodontic extractions were used in this study. Radiographic selection criteria included evidence of physiological root resorption, which did not exceed the apical third with no signs of internal or external resorption. The procedures, possible discomfort or risks, and possible benefits were explained fully to the human subjects involved, and the informed consent was obtained prior to the investigation. A local anesthetic (Citanest, Astra, Sweden) was given during clinical procedures.

The surface of each tooth was cleaned with a slurry of pumice in a rubber cup rotating at low speed and rinsed with water spray. A rubber dam was used to isolate each tooth, controlled with high-speed evacuation.

Buccal class V cavities were prepared with a round diamond bur (ISO size 018) at ultra high speed under water spray without perforating the pulp chamber. No beveling of the cavosurfaces were performed. A new bur was employed on every patient to ensure cutting efficiency. A round carbide bur (ISO size 010) rotating at slow speed and saline solution spray was used to expose the pulp without plunging the bur into the pulp.

The cavities were, then, irrigated with saline solution, and pulpal hemorrhage was controlled with sterile cotton pellets. Before placement of the adhesives, preparations were total-etched with 36% H_3P0_4 (DeTrey Conditioner 36, DeTrey/Dentsply, Konstanz, Germany) for 10 seconds without extending the acid gel to the exposure site, rinsed with saline solution for 20 seconds and dried with sterile cotton pellets. If secondary hemorrhage was observed, physiologic hemostasis was maintained with a sterile cotton pellet moistened with sterile saline solution.

Each patient received single cavities in each of three teeth, which were randomly treated with one of the adhesive systems listed in Table 1. Care was taken to carry the adhesives to the exposure sites without pressure. All adhesives and subsequent restorative materials were placed per instructions of the manufacturer. The restorations were finished and polished with Sof-Lex disks (3M Dental Products, St Paul, MN). Radiographs and pain histories of the teeth were taken and recorded before each extraction procedure. All teeth were extracted at 60 days after the procedures.

Each tooth was immediately fixed in 10% formaldehyde solution for one week at room temperature followed by decalcification in DeCastro's fluid (300 ml absolute ethanol, 50 gr chloral hydrate, 30 ml concentrated nitric acid and 670 ml distilled water. All chemicals were obtained from Merck, Darmstadt, Germany. After washing, the teeth were dehydrated in ascending degrees of ethanol, cleared in xylene, and embedded in paraffin. Serial sections were cut in a bucco-lingual plane and stained with hematoxylin and eosin. Sections were evaluated with a C35 AD4 light microscope (Olympus, Tokyo, Japan). Each specimen was examined independently according to the following histopathological criteria.[8,24]

Inflammatory cell response

1. **None.** Either no inflammatory cells or scattered inflammatory cells at the exposure site, beneath new hard tissue or adjacent to the exposure site.
2. **Slight**. Either (a) acute inflammatory cells (tissues are dominated by `large polymorphonuclear neutrophylic leucocytes [PMNs] and macrophages) or (b) chronic inflammatory cells (tissues are dominated by mononuclear leucocytes [MNLs]).
3. **Pronounced** (severe). Either PMNs appearing as an abscess or a dense infiltrate of MNLs that involve one third or more of the coronal pulp.
4. **Necrosis**.

Fibrosis increase in fibroblasts and collagen fibers

1. None or slight,
2. Mild,
3. Severe.

Table 2.

Material	Code	n	Inflammatory cell response				Fibrosis			Reparative dentin formation		
			1	2	3	4	1	2	3	1	2	3
Scotchbond Multipurpose Plux	SMP	7	3	4	0	0	4	2	1	1	2	4
Prime & Bond 2.1	PB	7	2	4	1	0	2	4	1	1	2	4
Syntac Single Component	Syntac	7	0	2	4	1	0	3	4	0	1	6

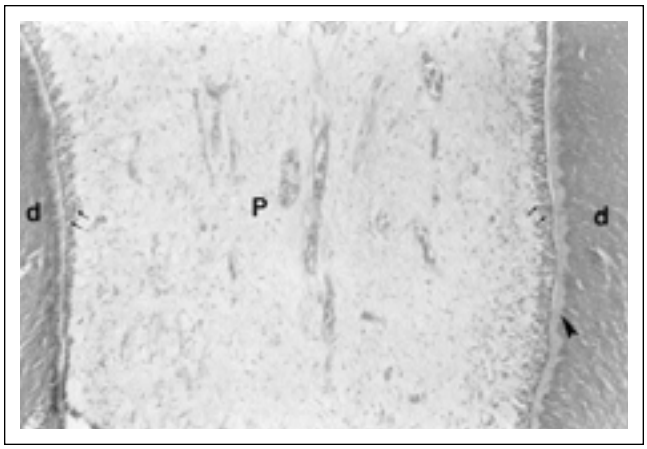

Figure Ia. A section through dentin and pulp tissue (SMP). Pulp tissue (P), odontoblastic layer (arrows), predentin (arrowhead) and dentin (d) appear to be normal. Hematoxylin-eosin, original magnification X10.

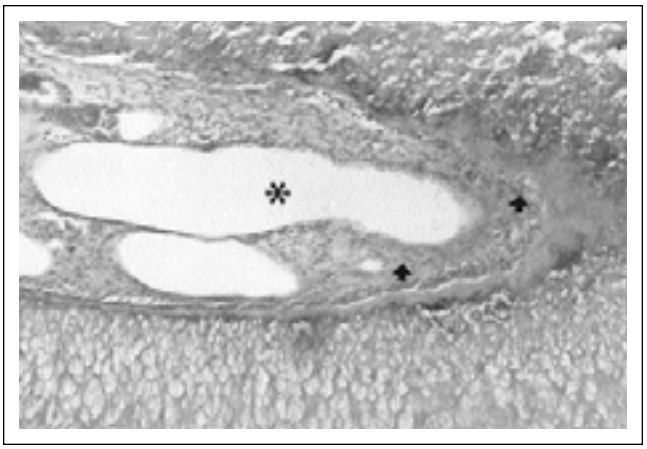

Figure Ib. Multicystic structures (*) and severe cellular infiltration (arrow) is seen. Dentin disintegration is also present. Hematoxylin-eosin, original magnification X10.

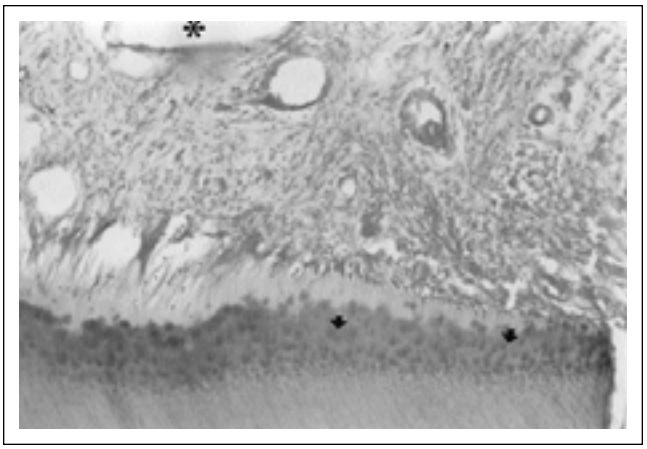

Figure 1c. Basophilic globular fragments of dentin (arrow) probably representing disintegration. Multicystic (*) and minimal fibrosis is seen.

Reparative dentin formation
1. Directly adjacent to the capping agent,
2. At some distance from the capping agent,
3. No evidence of hard tissue formation.

RESULTS

During the test period, none of the patients reported to have thermal sensitivity or pulpal pain. Neither did the periapical radiographs taken before extractions exhibit any periradicular changes. Results of the histological evaluation are summarized in Table 2.

SMP group (Figures 1a, 1b and 1c)

The major change was observed in the odontoblasts which, in general had decreased in number, while they showed an uneven distribution in 5 of 7 cases. However, there was an increase in predentin, forming a thick layer in general. Significant fibrosis was observed in one case. Chronic inflammatory changes were seen in two cases. In one tooth, cystic structures of varying size (surrounded by a capsule), were spread out in the entire pulp tissue. The presence of basophilic globular fragments of dentin was an interesting finding which could be a sign of disintegration. Yet, the findings mentioned above were more prominent around the exposure sites. Prominent dentin bridge formation was observed in only one case, while other teeth generally

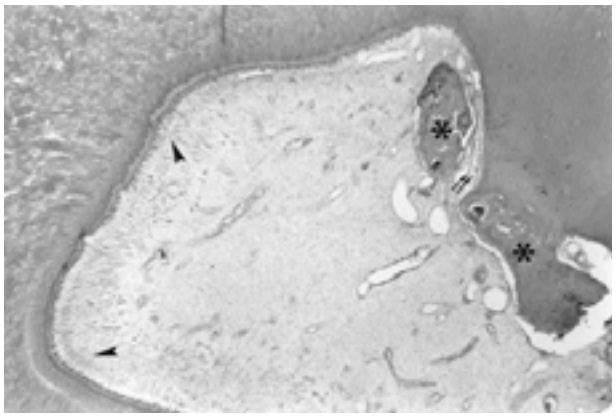

Figure 2a. A section through the pulp tissue of PB group. Relative increase in fibroblastic activity at the peripheral compartment of pulp tissue is observed (arrowhead). Odontoblastic layer was especially discontinuous in the vicinity of the cavity prepared for the filling material (arrows). Irregular dentin islands were observed in the same region (*).Hematoxylin~eosin, original magnification 10X.

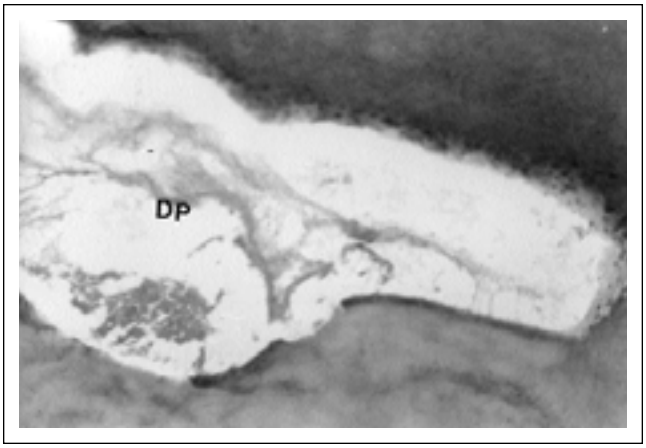

Figure 2b. Non-vital pulp (DP) consisting of necrotising tissue.

exhibited an open contact between the cavity and pulp tissue associated with some remnants of the filling material.

PB group (Figures 2a and 2b)

In this group, hemorrhage was observed in almost all of the cases and fibrotic changes in the pulp tissue was more frequently seen. Inflammatory infiltration was also a common finding, even showing an abscess formation in one case. In two cases, the pulp appeared to be normal with the exception of slight hemorrhage. Discontinuity in the odontoblastic layer together with hypocellularity was also present in most cases. Reparative dentin formation was observed in one sample.

Syntac group (Figures 3a, 3b and 3c)

Most severe changes in the pulpal tissue and the odontoblastic layer were observed in this group. A generalized fibrosis was present in almost all of the cases. Even one appeared to be necrotic. Unlike the SMP and PB groups, reactive changes in the pulpal tissue was not confined to the cavity region, but had spread to the entire pulp. In addition to the loss of odontoblasts, there was also a prominent disintegration of dentin itself resulting in a regular outline facing the dental pulp. Multicystic structures seen in the PB group were also observed in three of seven cases. In one case, multiple micro-abscesses were noted. The inflammatory cell infiltration was so prominent that it exhibited the appearance of generalized pulpitis.

DISCUSSION

Recent publications have reported the potential biocompatibility of adhesive resins when applied to acid conditioned, vital dentin[25,26] or as a capping material on mechanically exposed pulps.[10,13,15,27] Cavity asepsis through sealing the exposed dentin tubules and exposed soft pulpal tissues within adhesive resin was reported to be the key of success. From a clinical standpoint, the ultimate failure of pulp capping is an inability of the medicament to provide a long-term barrier to microleakage of bacteria and other irritants. With the availability of fourth-generation dentin adhesives, however, it should be possible to provide an effective seal.[19]

It is apparent from the present study that although a few number of samples in the SMP and PB groups exhibited "attempted bridge formation,[28] the primary tooth pulp failed to form dentin bridges under the tested adhesives, in general (Table 2). Many authors maintain that regeneration of a new dentin bridge is a sign of successful tretment.[19] Others, however, have reported that dentin bridges are incomplete in the formation, leading to recurring pulpal inflammation and necrosis associated with tunnel defects in dentin bridges.[20]

Mjor,[29] Tronstad and Mjor[30] have cautioned that capping of young and healthy pulps may lead to a poor prognosis and that histological demonstration of only one section of a dentin bridge can not be considered a criterion of successful pulp capping. As discussed by others, only histological slides made in complete serial sections should be considered to provide definitive information regarding the success of complete dentin bridge formation.[19] In the present study, four samples from SMP and PB groups, which exhibited reparative dentin formation directly adjacent to the bonding agents, revealed an incomplete dentin bridge in serial sections, and were thus given the score"2".

Another fact to consider is the effects of total acid-etch technique, which has become a frequently used treatment modality for bonded restorations in restorative dentistry. Many current dentin bonding systems require application of acid conditioners to remove the smear layer from the cavity walls and to promote superficial demineralization of dentin substrate. Etch-

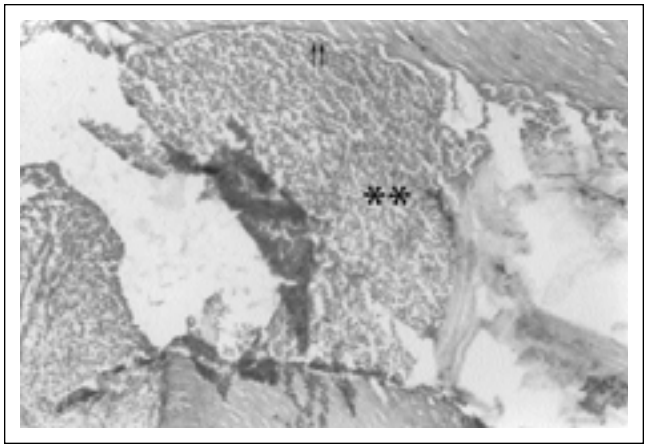

Figure 3a. Severe Inflammation (*) in the pulp compartment adjacent to the cavity with filling material (* *). Odontoblastic layer has been lost (arrows). Hematoxylin-eosin, original magnification 10X.

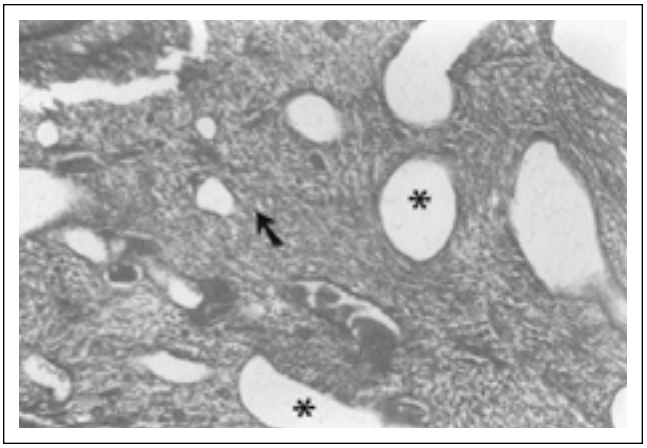

Figure 3b. An increase in fibroblastic activity (arrow), prominent inflammation and multicystic change (*) in pulp tissue is seen. Hematoxylin-eosin, original magnification 10X.

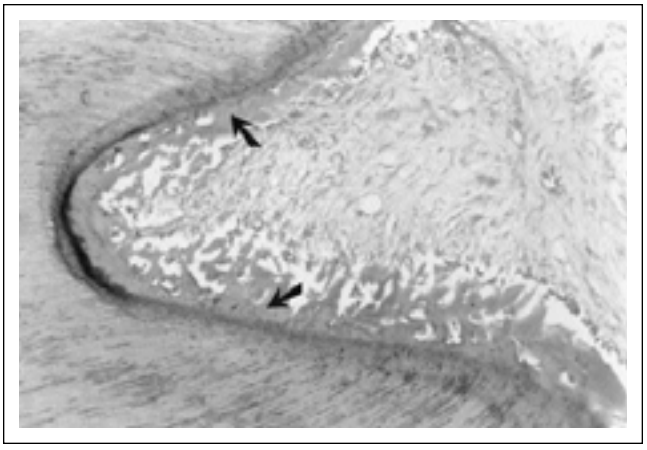

Figure 3c. Discontinuity and loss of odontoblasts and irregular outline of predentin (arrow). Hematoxylin-eosin, original magnification 10X.

ing of vital dentin was believed to increase pulp irritation by facilitating the penetration of irritants into the tubules.[31,32] During the last years, however, recommendations concerning pulp capping techniques have been made that advocate exposing pulps to acid treatment and bonding procedures.[33,34] Inokoshi *et a1*.[12] reported that total etched cavity preparations restored with Clearfil Bond system showed only slight pulpal response and no bacterial penetration.

In another study, Tarim *et al.*[35] reported that pulpal responses to total and non-etched Compoglass were statistically the same at 27 and 90 days. On the other hand, Pameijer and Stanley,[28] using the total etch technique in pulp capping procedures followed by direct pulp capping with dentin bonding agents showed that 45% of teeth became non-vital and that only 35% were capable of forming a bridge. In the same study, control groups with calcium hydroxide had 7% of teeth becoming non-vital and 82% forming dentin bridges. Within such controversy, it is not known at this time whether the unfavorable results obtained in the present study is directly related with the effects of acid gel conditioning. Provided this to be true, an explanation could be that, either the acid gel had a detrimental effect on the pulp or when the gel was washed out, the saline solution caused a diluted liquid acid which easily penetrated the connective tissue of the primary tooth pulp, or both.[28]

In other words, although the pulp hydrostatic pressure can limit the diffusion of acids through dentin, it cannot avoid its occurrence.[36] Yet, this may not sufficiently explain the divergence in severity of histopathological findings between SMP, PB and Syntac groups, since the same total-etch protocol carried out on each tooth.

Due to the short duration of the experimental time in vivo, an attempt to correlate between pulpal inflammation and the presence of bacteria through bacteria staining was not performed. it is highly unlikely that within 60 days post-treatment, so much leakage would have occurred in the three dentin bonding groups, that this would explain the unfavorable results.[28] Although properly sealed restorations would be desirable in the viewpoint of pulpal healing, one must distinguish between pulp cap failure and failure of the restoration subsequently placed over the pulp capping agent that leads to recurrent pulpitis.[37] If a covering restoration is so degraded that a calcium hydroxide base has dissolved, even if not a capped pulp exposure, that amount of microleakage would penetrate even normal patent dentin tubules to create severe pulp pathology.[37]

In a recent study, pulpal inflammation with absence of bacteria as well as the reverse, i.e., larger number of bacteria but no pulpal inflammation was reported.[38] Logically, in a carious lesion there are billions of bacteria into the dentin tubules and many of these microorganisms must be able to find their way to the pulp tissue. Despite this, the pulp is still able to survive. By the same logic, those authors were unable to attribute cases of pulp necrosis only to the presence of bacteria at the

tooth-restoration interface.[38] Finally, it has been demonstrated that estimation of the number of bacteria and the affinity for staining are changed by the agents used during the specimen decalcification process, which make the results fairly subjective.[39]

When EDTA is used, only five bacteria are stained for each 15 bacteria present, while with formic acid, only one of 15 is stained and consequently observed under the microscope. Therefore, in categorizing the success and failure in pulp capping studies, many more factors need to be considered than just the presence or absence of microorganisms.[28,35,36]

The belief that almost any restorative material can be used for pulp capping procedures as long as the preparation has been properly disinfected is not firmly established. Although many authors pay special attention to bacterial presence at the tooth-restoration interface and a possible role in irritation of the pulpal tissue, several in vitro studies have demonstrated that resin monomers diffuses through dentin tubules[40] and cause cytotoxicity. Gwinnet and Tay[42] have shown a chronic inflammatory response when the authors applied a fourth generation dentin adhesive on to deep cavities prepared in human teeth. The authors demonstrated by TEM the movement of resin particulates through dentinal tubules. The presence of resin components in the pulp, which are not soluble and cannot be digested caused an irritating action for a long time and promoted a chronic inflammatory response, with reaction of macrophages or giant cells.

Consequently, Jontell *et al*.[43] reported that immunosupression of pulpal immune competent cells elicited by resin components may enhance the potential for bacterial injury to the pulpal tissue. Thus, increased incidence and severity of pulpal infection are justifiable concerns following exposure of the tissue to immunotoxic chemicals. Additionally, as demonstrated by several authors,[40,42] neither the hybrid layer nor a thin remaining dentin between the cavity floor and the pulpal tissue is totally capable of preventing monomer diffusion across the dentinal tubules, letting alone direct pulp capping as performed in the present case. Following dentin etching, faster outward dentinal fluid movement seems to interfere with complete polymerization of the fluid resin.

According to Hussey *et al.,*[45] the heating caused during the light curing of composite resin reverses the dentinal fluid movement and, as consequence, the inward fluid movement may carry the unpolymerized resin particulates through dentinal tubules to reach the pulp tissue.

According to the findings in the present study and those reported from relevant literature, we suggest that the monomers, which reached the pulpal tissue were one of the major factors contributing to the persistent inflammatory reaction and divergence in severity of histopathological findings. In the present study, the mildest changes were observed in the SMP group, which is in agreement with Araujo *et al*.,[46] who demonstrated that direct pulp capped human primary teeth with SMP revealed no adverse clinical or radiographic signs and/or symptoms after one year. In contrast, the results of the present study do not correlate with findings of Heitman and Unterbrink[7], who reported that Syntac could be used as a replacement for calcium hydroxide for pulpal protection. We are unable to explain whether these differences are a result of material toxicity and/or a pathologic/immunologic response of the primary tooth pulp, which remains an open area for investigation.

CONCLUSION

The preliminary data presented within the limitations of this study suggests that direct resin pulp capping could be successful in a few cases, resulting in the resolution of the inflammatory response and reparative dentin formation. However, the persistence of chronic inflammatory reaction and lack of dentin bridge formation in the majority of the samples suggests that more research is needed toward the factors contributing to the appearance of these phenomena, before direct resin pulp capping can be recommended as a routine clinical procedure in primary teeth.

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