Pulp Tissue Reactions to a Dentin Bonding Agent as a Direct Capping Agent

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Abstract

The aim of this study was to investigate the response of human pulp tissue to a dentin bonding agent, Scotchbond Multi-Purpose Plus (SMPP), in exposed class V cavities. Sixteen human premolar teeth were mechanically exposed. Ten pulps were capped with SMPP and six teeth were capped with Dycal. The cavities were filled with a composite. After 40 days, the teeth were extracted and processed for histologic evaluation. Of the 10 teeth capped with SMPP, eight showed moderate chronic inflammation, one was severely inflamed, and one pulp had no to slight inflammation. None of the teeth capped with SMPP showed dentin bridge formation. Of the six teeth capped with Dycal, three exhibited incomplete dentin bridges associated with no to slight inflammation, and three showed no to slight inflammation, without formation of dentin bridges. Direct capping with Dycal with subsequent sealing with SMPP may show favorable results in pulp tissue. SMPP may cause inflammatory changes when applied directly to exposed pulp tissue.

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Like other connective tissues, pulp tissue has the potential to heal. Characteristics of exposed pulp tissue that advance healing include the reorganization of damaged soft tissue, differentiation of odontoblast-like cells from subodontoblast cells and repair of exposed dentin tissue with reparative dentin bridge formation (1). Among the factors that may compromise pulp healing are leakage of bacteria through the restoration-tooth interface (bacterial leakage), cytotoxicity of dental materials, the sensitivity of operative procedures, and aged pulps (1–5).

Calcium hydroxide cements are generally the materials of choice for successful capping procedures, because of their stimulatory effects for reparative dentin formation and their beneficial antimicrobial effect (6). Dentin bonding agents (DBA) have been examined as potential direct capping materials because of their superior ability to adhere to both demineralized enamel and dentin tissues. Hybridization of DBAs with demineralized intertubular collagen, as well as the diffusion of adhesives into the dentin tubules, may seal the vital dentin against bacterial leakage and reduce secondary pulpal inflammation. However, in vitro studies have shown that acid etchants, monomers, and other ingredients in the composition of various DBAs can be toxic to nerves, cytotoxic to cultured cell lines, and mutagenic for microorganisms and mammalian cells (7–11).

Implantation of several types of DBA in the connective tissue of rats evoked persistent foreign body responses (12, 13). Pameijer and Stanley (14) reported that direct pulp capping of primate teeth using various DBAs caused necrotic pulps in 45% of all teeth, and that microorganisms made no direct contribution to the necrosis. Kitamura et al. (15) observed that capping agents such as calcium hydroxide and zinc oxide-eugenol may have some effect on apoptosis of pulp cells, but that 4-META/MMA-TBB bonding resin may induce significantly more apoptosis during pulp healing. Kitasako et al. (16) showed that three different DBAs applied to exposed monkey tooth pulps provoked slight inflammatory reactions and the deposition of dentin bridging over time, without demonstrable bacterial leakage.

Costa et al. (17) pointed out that tests of DBA application performed on animal teeth exhibited specific pulp reactions that cannot be directly extrapolated to humans. Several studies have been published using exposed human pulps that were directly capped with various DBAs (18–22). Schuurs et al. (23) speculated that the use of DBAs and composite fillings in exposed cavities seemed promising. However, the use of DBAs in vital pulp therapies was contraindicated by Costa et al. (17). We have previously reported that one DBA, Scotchbond Multi-Purpose Plus (SMPP), did not exhibit significant deleterious effects on nonexposed human pulps (24). The aim of this study was to investigate the response of human pulp to SMPP bonding agent in exposed Class V cavities.

Materials and Methods

A total of 16 human premolar teeth from four patients undergoing orthodontic procedures were used in this study. The teeth were clinically intact and noncarious, with no more than superficial attrition and no signs of trauma.

After the patients had given their written consent to participation in the study, a local anesthetic was introduced for the operative procedures. Teeth were cleaned with 5% tincture of iodine following rubber dam isolation. Class V buccal cavities were prepared using round and cylindrical diamond burs (ISO sizes 012, 014) with an air turbine under a saline spray, extending as deep as 3 to 3.5 mm without exposing the pulp tissue. Pulps were then exposed using a round diamond bur (ISO size 010). New

burs were employed during each operation. Hemorrhage was controlled with saline irrigation and placement of sterile cotton pellets onto the pulp exposure sites.

The SMPP adhesive system (3M, USA) was used in 10 of 16 prepared cavities, in accordance with the manufacturer's instructions. The cavity and pulp tissue were etched with 37% phosphoric acid for 15 s and rinsed for 15 s. After hemorrhage was controlled, the cavity was dried for 2 s, primed for 5 s, and again dried for 5 s. Multiple consecutive coats of adhesive were applied to exposed pulps and cavity walls, and light-cured for 20 s. There was recurrent bleeding following primer and adhesive applications in all 10 cases. Sterile cotton pellets were used to stop the hemorrhage, followed by application of additional primer and adhesive. Cavities were filled with a composite (Valux, 3M) in three increments and light cured for 20 s after each increment. A light curing unit (Astralis 3, Vivadent, Liechtenstein), emitting more than 500 mW/cm^2, was used for polymerization of the adhesive systems.

A calcium hydroxide formulation, Dycal (Dentsply, USA), was placed in the exposure site and the floor of the prepared cavity in the six control teeth. After etching, the remaining cavity was restored with SMPP and composite.

After 40 days, the treated teeth were extracted. Radiographs were taken before extraction, to detect any periradicular tissue changes subsequent to the restorations. The subjects were also asked to report pain histories for the treated teeth, which may indicate pulp inflammation, before the extractions were performed.

Each extracted tooth was fixed in 10% formalin, demineralized in 0.5 M ethylenediamine tetraacetic acid, washed in distilled water, dehydrated in ascending grades of N-butyl alcohol and embedded in paraffin. Serial 6-μm sections were cut in a buccolingual plane, placed on gelatin-coated slides, and stained with hematoxylin and eosin, or Brown and Brenn's bacterial stain. Each tooth was independently examined by two investigators; in cases of disagreement, the case was re-evaluated and a consensus was reached. Inflammatory pulpal responses, hard tissue formation, and the presence or absence of bacteria were graded by light microscopy according to the criteria used by Cox et al. (2).

Results

Clinical Findings

The subjects reported no sensitivity to thermal stimulation in any of the 16 teeth during the test period. In addition, no detectable periradicular radiographic changes or tenderness to percussion were noted in any of the teeth.

TABLE 1. Distribution of teeth and materials and their pulpal responses

Material	Number of teeth	Time (Days)	Inflammatory cell response				Soft tissue organization				Reparative dentin			Dentin bridge			Bacterial staining		
			1	2	3	4	1	2	3	4	1	2	3	1	2	3	1	2	3
SMPP	10	40	1	8	1		1	9			9	1				10			10
Dycal	6	40	6				1	5				6		3		3			6

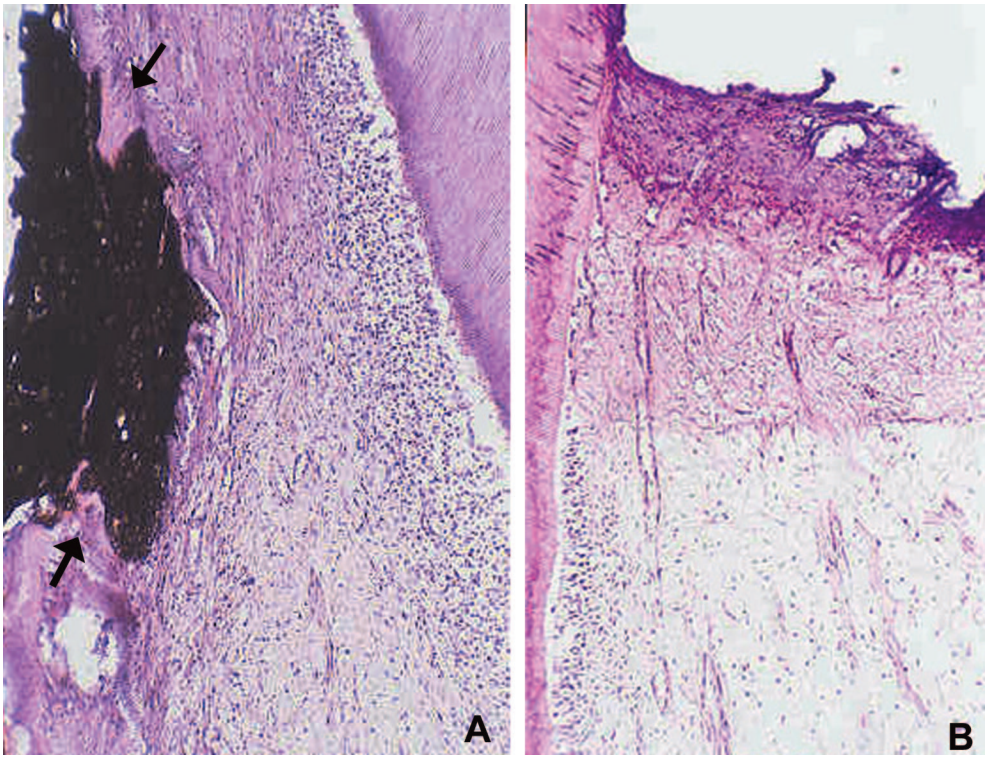

Figure 1. (A) Histology of the pulpal response of a tooth capped with Dycal. Deposition of reparative dentin directly to the Dycal material (incomplete bridge formation) is visible in some areas (arrows). Infiltration of mononuclear monocytes in the disorganized odontoblast layer on the right dentin wall can be seen (×125 original magnification). (B) Histology of another tooth capped with Dycal. A bilayered necrotic zone is present in the superficial pulp. There are no inflammatory cells present and no deposition of reparative dentin in the pulp area below the necrotic layer (×125 original magnification).