



Review Article

Postoperative Tooth Sensitivity in Direct Resin Composite Restorations: Etiology, Diagnosis, and Management

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CLINICAL SIGNIFICANCE

PTS could be prevented by correct diagnosis, minimal-trauma operative procedures, proper adhesive application under good moisture control, appropriate resin composite material, oblique incremental placement, and careful occlusion check.

ABSTRACT

Postoperative tooth sensitivity (PTS) is a complication associated with direct resin composite restorations, presenting as a short, sharp pain in response to stimuli. The possible mechanism is the hydrodynamic theory, which proposes that restoration defects, such as micro-gaps between the restoration and dentin, induce dentinal fluid movement that triggers pulpal nerve fibers. The incidence of PTS in direct restorations ranges from 0 to 25%. The risk of PTS has a direct relationship with cavity depth and size. Clinical evidence demonstrates no significant difference in PTS between etch-and-rinse and self-etching adhesive systems. The use of glass-ionomer cement or flowable composite liner may not reduce PTS. Errors in bonding procedures potentially lead to PTS. PTS may arise from three possible etiologies: I. transient pulpal inflammation (from preoperative insults or operative trauma); II. incomplete sealing of exposed dentine (marginal or internal gaps); and III. tooth and restoration deformation from occlusal force. Prevention strategies include minimizing operative trauma, strictly following adhesive instructions with contamination control, ensuring appropriate thickness of resin composite, incremental restoring for adaptation to cavity walls, and checking occlusion. Management strategies involve monitoring, desensitizer application, re-bonding to seal gaps or exposed dentine, repairing the defects, or replacement.

1. Introduction

Postoperative tooth sensitivity (PTS) is frequently reported as a complication following direct resin composite restorations. PTS is defined as a short, sharp pain (hypersensitivity) in response to one or more stimuli, including cold/hot water, sweet substances, occlusal force, or tooth brushing.¹ Understanding the etiology and contributing clinical factors is crucial for effective prevention and management. The objective of this review article is to describe the mechanism, clinical factors, etiology, prevention, diagnosis, and management of PTS.

2. Literature Review

2.1. Mechanism of postoperative tooth sensitivity

Dentine sensitivity is primarily explained by the hydrodynamic theory, which proposes that stimuli induce movement of the dentinal fluid, subsequently triggering pulpal nerve fibers and causing sensitivity.² In the context of PTS, defects such as micro-gaps between the restoration and the dentin allow fluid movement, leading to sensitivity after restorations.

2.2. Incidence and clinical factors

The incidence of PTS varies based on the restoration cavity, ranging from 0–25% in Class I and II restorations, typically at a low to moderate level.^{3–8} The incidence in Class II restorations is generally higher than in Class I. In some cases, PTS may decrease over time and self-relieved within 30 days.^{4,5,7} Even clinical studies report incidences of PTS in Class V restorations as usually less than 5%^{9,10}, the practical incidence is likely to be higher. The incidences in class III and IV restorations are usually rare.^{11,12} Clinical factors

that may relate to the risk of PTS are discussed as follows (Table 1):

Cavity characteristics: Cavity type, depth, and size are important factors. For cavity type, as previously mentioned, PTS is most commonly reported in Class I, II, and V restorations.¹¹ For cavity depth, the risk of PTS has a direct relationship with the depth. When the residual dentine thickness corresponds to the inner one-third of the dentine, the risk of PTS is four times greater.³ If pulp exposure, the risk of PTS is 14 times higher.³ For cavity size, the incidence of PTS increases with the size of the cavity (e.g., MOD cavity > OM/OD cavity > O cavity).⁴

Sclerotic dentine: Sclerotic dentine is characterized by lower permeability compared to normal dentine.¹³ While bonding to sclerotic dentine is less effective than to normal dentine, preserving it keeps the dentinal tubules occluded, which, according to the hydrodynamic theory, makes PTS less likely after restorations.

Adhesive mode: The comparison between etch-and-rinse (ER) and self-etching (SE) adhesives regarding PTS incidence has been thoroughly studied. Although concerns exist regarding the technique sensitivity of ER and whether SE provides a better "seal"^{14,15}, clinical evidence reveals no significant difference in the incidence or intensity of PTS between the two adhesive modes in Class I, Class II, or Class V restorations.^{5,16–19} In vitro, ER adhesives have demonstrated a comparable dentine seal to SE adhesives in fluid flow and micro-permeability tests.^{13,15} From a systematic review and meta-analysis, PTS in class I and II restorations are not different between the two adhesives, regardless of cavity types.^{20,21} For universal adhesives, the use of the SE mode resulted in lower

Table 1. Clinical factors that may associate with PTS after resin composite restoration (PTS: postoperative tooth sensitivity).

Clinical factors associated with PTS	Incidence of PTS
Cavity characteristics	Cavity type: Class II, I, and V Cavity depth: Inner 1/3 of dentine, or pulpal exposure. Cavity size: Class II MOD > Class II MO/OD. Absence > Presence
Presence of sclerotic dentine	No difference between etch-and-rinse (ER) and self-etching (SE) adhesives. Universal adhesive: SE mode < ER mode (a low incidence of PTS).
Adhesive mode and restorative technique	No difference between bulk-fill and traditional resin composite (bulk-fill vs. incremental placement).
Cavity lining	Lining with glass-ionomer cement or flowable resin composite does not decrease incidence of PTS.
Preoperative tooth sensitivity	Not increase risk of PTS.

PTS compared to the ER mode.²² However, the incidence of PTS of universal adhesive is low and not different from that of conventional ER or SE adhesive.²³ For restorative materials, no difference in PTS between bulk-fill and traditional resin composite has been reported in a systematic review and meta-analysis.²⁴ However, incremental placement of restoration to ensure adaptation to cavity and effective light-curing would be beneficial.

Presence of Lining: The use of glass-ionomer cement (GIC) or flowable composite lining has traditionally been recommended to prevent PTS.^{11,25} GIC has been theorized to offer an effective seal via chemical bond, biocompatibility, and stress absorption.²⁶ However, clinical evidence indicates that GIC lining is unlikely to reduce PTS, irrespective of whether ER or SE adhesives are used.^{5,27,28} In addition, internal gap formation tends to be higher when GIC lining is applied in a very thin layer (e.g., 0.5 mm thick) compared to a thicker layer (e.g., 1 mm thick).

Flowable composite lining may improve adaptation due to its flow capacity and act as a stress absorption layer, but laboratory studies show insignificant results between the absence or presence of a flowable composite layer.^{29,30} Clinically, PTS in class V restorations are not different between with or without a flowable composite liner.²⁵

Preoperative tooth sensitivity: The presence of preoperative sensitivity may not be related to the occurrence of PTS. Many patients who exhibited PTS after restorations do not report any

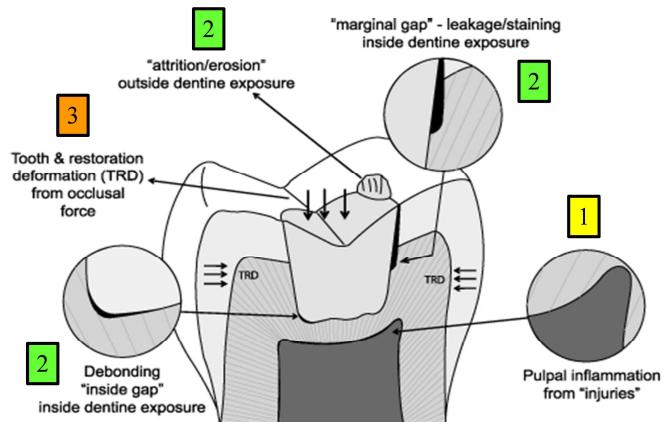


Fig. 1. Three primary types of PTS have been proposed: [1] Type I: temporary pulpal inflammation; [2] Type II: incomplete sealing of exposed dentine; and [3] Type III: tooth and restoration deformation.

preoperative symptoms.^{5,31} Conversely, the teeth with symptomatic reversible pulpitis are usually relieved after removing the causes (such as dental caries) and restoring with resin composite, which do not show any PTS.^{31,32}

2.3. Etiology and prevention strategies of PTS

From laboratory and clinical studies, three primary etiologies of PTS have been proposed³³ (Fig. 1): I. temporary pulpal inflammation, resulting from previous injuries or operative procedures; II. incomplete sealing of exposed dentine, adjacent to restorations, marginal or internal gaps; and III. tooth and restoration deformation (TRD), induced by occlusal forces.

2.3.1. PTS Type I: Temporary pulpal inflammation

Temporary pulpal inflammation may arise from preoperative threads such as caries or cracks, which decrease the pain threshold of pulpal nerves.^{34,35} Moreover, operative trauma, including heat from insufficient coolant during preparation or polishing, vigorous air blasts causing odontoblast displacement, or heat generated during light curing in deep cavities, contributes to pulpal inflammation. The temporary inflammation of pulpal tissues may induce short-term PTS.¹⁸ However, when the cavity is appropriately sealed by restorations, the pulp usually exhibits self-recovery from minor operative injuries, and PTS is likely to diminish within 30-90 days.³⁻⁵

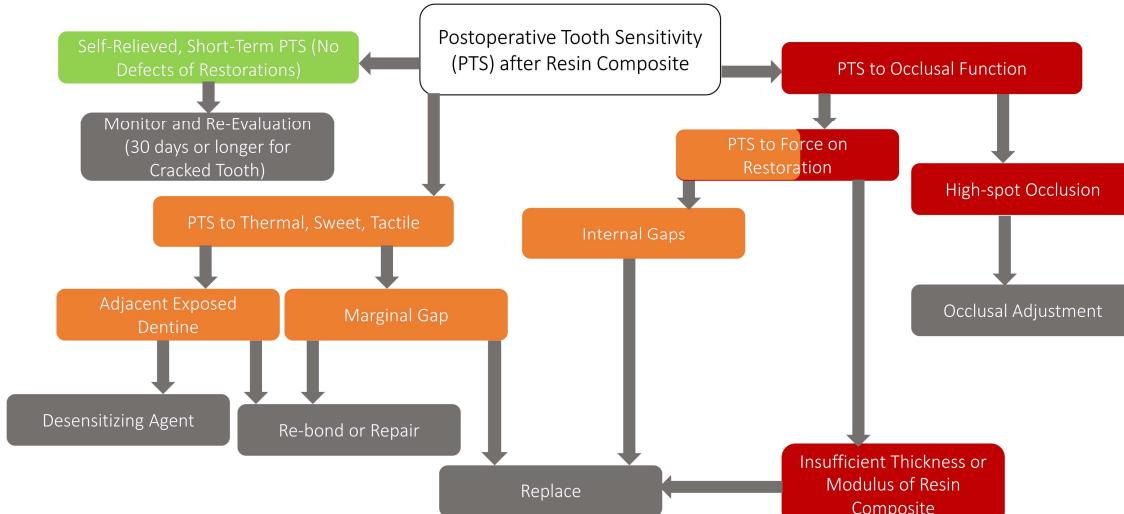


Fig. 2. Diagnosis and management of three primary types of PTS (color labeling: green- Type I, orange- Type II, and red- Type III).

Table 2: Methods used for assessment, differential diagnosis, and follow-up duration of PTS after resin composite restoration (PTS: postoperative tooth sensitivity).

Clinical symptoms of PTS	Assessment methods for PTS
<ul style="list-style-type: none"> Sensitive to occlusal force Sensitive to cold water, sweet, brushing, or others 	<ul style="list-style-type: none"> Checking high-spot occlusion using articulating paper. Testing with biting or pressing force (e.g. biting on object or tooth slope; and pressing with ball burnisher or plugger). Examining quality/any defects of restorations and adjacent exposed dentine. Radiographic examination: bitewing and/or periapical radiograph to reveal inadequate thickness of restoration or any internal gap. Try to trigger the PTS by using the stimulus reported by patients. -Cold water test under rubber dam isolation (one by one). -Sugary syrup. -Tactile force e.g. exploration. To identify the tooth and the area of PTS. Examining quality/any defects of restorations, and nearby exposed dentine. Radiographic examination: bitewing and/or periapical radiograph to reveal any marginal gap, particularly at gingival margins.
Differential diagnosis:	
<ul style="list-style-type: none"> Cracked tooth: history of crack, exploration of crack after restoration removal. Pulp pathosis: history of pulpal exposure, signs and symptoms of irreversible pulpitis, re-evaluation of pulpal and periapical status. Refer pain: other odontogenic or non-odontogenic origin. 	
Follow-up duration:	
<ul style="list-style-type: none"> PTS from operative injuries (PTS type I) without any defects of restorations should be subsided in 30 days. Teeth with PTS last longer than 30 days should be carefully re-evaluated to identify definite cause(s). If any defects are detected (PTS type II or III), the defective restorations should be sealed, repaired, or replaced immediately. 	

Prevention for Type I PTS requires minimal-trauma operative techniques, including ensuring adequate air-water coolant during cavity preparation, using a short-duration air blast, and utilizing low light-curing intensity for the adhesive layer in deep cavities.³³

2.3.2. PTS Type II: Incomplete seal of dentine (dentine exposed, marginal, or internal gaps)

Incomplete seals of restorations lead to gaps that provide a pathway for dentinal fluid movement, inducing the sensitivity.^{13,15} Gaps can be marginal (extending from enamel into dentine) or internal (at the bonded dentin interface). Marginal gap formation is more likely at sub-gingival dentine margins in Class II and V restorations due to less reliable dentine adhesion.^{36,37} Micro-gaps are formed by polymerization shrinkage, incorrect use of the

dentine bonding agent (e.g., prolonged etching, excessive drying, or inappropriate application of primer/adhesive), or contamination (saliva, blood, or astringent) during bonding procedures.³⁸⁻⁴⁰ Most errors leading to PTS are probably from improper bonding procedures. Using an intermediate layer between dentine and resin composite, such as flowable liner or glass-ionomer lining cement, does not improve gap-free restorations^{15,37,41,42} or reduce PTS.^{6,27} In contrast, placement techniques of resin composite may affect the chances of internal and marginal gap formation. The incremental layering technique of resin composite tends to decrease internal/marginal gaps compared to the bulk filling technique, which may contain interfacial voids at the cavity walls.^{43,44} However, a clinical study reported that PTS after restorations with incremental and bulk placements are not significantly different.⁴⁵

Type II PTS may occur from remaining exposed dentine adjacent to cavity margins that is not included in the restoration.³³ This exposed dentine area may not be sensitive before restorations due to dentinal sclerosis and obstruction of dentinal tubules.⁴⁶⁻⁴⁸ However, it can become sensitive after restorations, possibly from over-acid-etching or aggressive polishing that can remove the superficial sclerotic layer and then expose the normal 'sensitive' dentine.^{49,50}

Prevention for Type II PTS centers on strictly following

**Fig. 3.** Representative clinical cases of postoperative tooth sensitivity (PTS) Types I and II.

(A-B) PTS Type I associated with preoperative insult and operative trauma.

(A) Deep dental caries in the maxillary right second premolar.

(B) Following resin composite restoration, the patient reported sensitivity to thermal change (cold), which gradually decreased and completely resolved within two weeks.

(C-D) PTS Type II associated with marginal gaps.

(C) A periapical radiograph showing a clearly detectable marginal gap at the gingival margin (red arrow) of a resin composite restoration on the mandibular left first molar, associated with persistent sensitivity to cold water for several weeks.

(D) A marginal gap at the distal margin of a cervical restoration (red arrow) highlighted after staining with a caries detector dye; chronic postoperative tooth sensitivity was reported following restoration.

**Fig. 4.** A case showing PTS Type II relating to the internal gap. (A) A radiographic image of the maxillary left second molar with mesio-occlusal resin composite restoration. A radiolucent area was observed under the restoration (red arrow). The patient reported PTS to occlusal function for months, and was also sensitive to loading force from a ball burnisher or plugger when pressing on the restoration surface above the internal gap area. (B) An internal gap was clearly detected (red arrow) after removal of the restoration. This internal gap was potentially due to a poor resin-composite condensing technique.

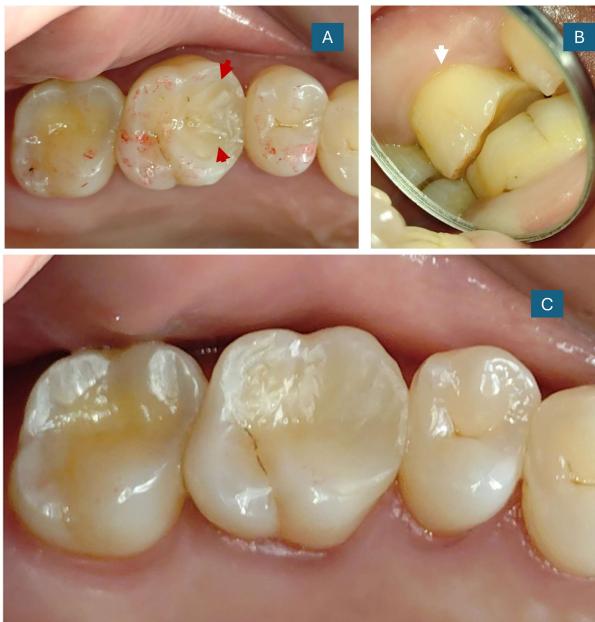


Fig. 5. A case showing PTS Type II relating to exposed dentine adjacent to the restoration. (A) On the maxillary right first molar, exposed dentine areas (red arrows) were found adjacent to the resin composite restoration. Without any preoperative symptoms, the patient reported PTS when chewing and drinking cold water. No 'red spots' from articulating paper were noticed on the restoration surface after occlusion checking. (B) Another exposed dentine was observed on the buccal side (white arrow) from a non-carious cervical lesion near the proximal margin of the restoration. Both exposed dentine areas were sensitive to cold-water testing and tactile testing. (C) At two-week recall after restoration replacement and cervical restoration, the patient reported that the restored tooth was normal and not sensitive to the stimuli.

manufacturer instructions for dental adhesives, maintaining adequate moisture control (e.g., rubber dam isolation), ensuring any contamination control, and utilizing proper placement techniques for resin composite.³³ Furthermore, exposed dentine adjacent to cavity margins should be sealed in the adhesive procedure, particularly when acid etching is used, or included in the outline of restorations. In addition, polishing of restorations should be carefully performed to prevent iatrogenic damage of adjacent dentine surfaces.³³

2.3.3. PTS Type III: Tooth/restoration deformation (TRD)

TRD might occur when occlusal forces induce mechanical



Fig. 6. A case showing PTS Type III relating to TRD due to inadequate thickness of restoration. (A) After occluso-proximal resin composite restoration on the mandibular left second molar, the patient complained about PTS when biting force close to the center of the restoration (red arrow). The restored tooth was also sensitive to the loading force when using a ball burnisher or plugger pressing on the suspected area. (B) A thin layer of resin composite at the sensitivity area was revealed in a periapical radiograph (red arrow) and after removal of restoration, which was less than 1 mm thick. After replacement, PTS disappeared by an increase in cavity depth and, subsequently, the thickness of the replaced resin composite restoration.

deformation of the enamel/dentin, leading to cuspal deflection and dentinal fluid movement.^{51,52} TRD is possible in large Class I and II cavities, particularly when the resin composite lacks sufficient modulus of elasticity or thickness.⁵³⁻⁵⁵ The larger the cavity (MOD), the higher the incidence of PTS probably due to increased TRD and cuspal tension from polymerization shrinkage stress.^{4,56,57} A minimum thickness of 1–1.5 mm of resin composite, theoretically with 60% filler loading by volume, should be used for posterior restorations to reduce deformation and resultant stress.^{53,54} Moreover, 'oblique' incremental placement of resin composite lowers shrinkage stress and cuspal deflection, by decreasing the volume of material in each layer and cavity configuration factor, compared to the bulk placement.^{58,59} However, 'horizontal' incremental placement should be avoided due to higher shrinkage stress detected.^{58,59} Traumatic occlusion or para-functional habits tends to increase the risk of TRD.^{33,60}

Prevention for Type III PTS involves proper case selection, particularly in large restorations^{61,62}, use of resin composite with appropriate elastic modulus and thickness, maintaining minimal cavity size, oblique incremental placement technique, and meticulously checking occlusion.³³

2.4. Diagnosis and management of PTS

PTS would be a symptom of reversible pulpitis to any stimuli that should be differentially diagnosed from other conditions after restorations, such as cracked teeth with persistent symptoms or restored teeth developing irreversible pulpitis.⁶³⁻⁶⁶ Diagnosis and management of three types of PTS have been proposed (Fig. 2). The diagnosis and management should rely on identifying the specific stimuli and causes inducing the sensitivity. Methods used for assessment, differential diagnosis, and follow-up duration of PTS are described in Table 2.

2.4.1. PTS Type I

PTS resulting from reversible pulpitis (Type I) due to operative trauma is present usually when thermal stimuli induce sensitivity, but none of the exposed dentine, gaps, or traumatic occlusion is detected³³ (Fig. 3A-3B). Importantly, this PTS in non-cracked teeth tends to resolve spontaneously within 30 days after proper restorations.³

Management of PTS Type I includes informing the patient and, probably, using self-applied desensitizers that interact with neural transmission (e.g., containing potassium nitrate). However, cracked teeth with preoperative sensitivity have a high incidence of prolonged PTS after restoration with resin composite, with a few teeth later showing signs of pulpal pathology.⁶⁴ Thus, the PTS in the restored, cracked teeth should be carefully interpreted and monitored.

2.4.2. PTS Type II

PTS relating to incomplete seals of dentine (Type II) could be induced by thermal, tactile exploration, or occlusal force.³³ Marginal gaps may be visually or radiographically detected and, if extending to dentine, potentially induce sensitivity to thermal changes⁶⁷ (Fig. 3C-3D). In contrast, internal gaps are more difficult to detect (Fig. 4). However, loading tactile force or pressing on the suspected area of internal gaps may induce sensitivity due to stimulation of dentinal fluid movement inside the internal gaps.^{15,51} For the sensitivity from exposed dentine adjacent to the restorative margins, the area of sensitive dentine can be confirmed by visualization and tactile exploration.^{68,69} (Fig. 5).

Management options of PTS Type II include re-bonding to seal marginal gaps or exposed dentine, repairing the defective margins, or covering the adjacent sensitive dentine with restorations.³³ Replacement of restorations may be necessary, particularly in the internal gaps.³³ In addition, for exposed sensitive dentine adjacent to the restorative margins, professional or self-applied desensitizers (e.g., containing fluoride, glutaraldehyde,

oxalate, or bioactive glass) may be used in an attempt to occlude the dentinal tubules.⁷⁰⁻⁷²

2.4.3. PTS Type III

PTS due to TRD (Type III) is potentially sensitive to occlusal force.³³ Occlusion should first be rechecked to identify any traumatic 'high-spot' on restorations. For the non-traumatic causes, PTS to occlusal force might relate to material deficiencies (too thin or non-rigid composite), inducing cuspal deflection.^{53,54,60,73} The inadequate thickness of restorations may be detected from bitewing radiographs or after initial removal of restorations (Fig. 6). However, PTS type III should be carefully distinguished from PTS Type II, in particular the internal gaps, which both are likely to be induced by occlusal function.

For management of PTS Type III, occlusal adjustment of restorations should be firstly performed if traumatic occlusion on restorations is detected. In the non-traumatic cases, replacement of restorations should be considered, to utilize a more rigid resin composite or increase restoration thickness.³³

3. Conclusion

PTS following direct resin composite restorations is a complication defined as a short, sharp pain in response to stimuli. The risk of PTS has a direct relationship with cavity depth and size. For adhesive type, clinical evidence demonstrates no significant difference in PTS among etch-and-rinse, self-etching, and universal adhesives. The use of cavity liner/base may not reduce PTS. PTS possibly arises from three proposed etiologies: (I) pulpal inflammation due to preoperative injuries or operative trauma; (II) incomplete sealing of exposed dentinal tubules, leading to marginal or inside gaps; and (III) tooth and restoration deformation caused by occlusal forces.

Prevention of PTS is based on minimal-trauma operative techniques (Type I PTS); strict adherence to bonding protocols, contamination control, and proper restorative technique (Type II PTS); and careful occlusion checking, proper material selection, and thickness (Type III PTS). Management strategies range from monitoring by informing possibility of self-relieving in short-term PTS (especially deep cavities) to operating management, including traumatic occlusion adjustment, applying desensitizing agents, re-bonding to seal gaps or exposed dentine, repairing the marginal defects, or, ultimately, replacement of the restorations.

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