

Comparing Diode Laser and Fluoride Varnish in the Treatment of Dentinal Hypersensitivity

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Abstract

Background: Dentin hypersensitivity (DH) is a “short, sharp pain arising from exposed dentin in response to stimuli, typically thermal, evaporative, tactile, osmotic, or chemical, which cannot be ascribed to any other form of dental defect or pathology.” Needless to say, it becomes a nightmare to the patient to eat or drink. **Aim:** The aim of the study was to evaluate the efficacy of fluoride varnish (sodium fluoride [NaF]), 980 nm diode laser, and the combination of both in the treatment of DH immediately post-operative, at the end of 1 week, 1 month, 3 months and 6 months. **Materials and Methods:** A total of 30 patients with minimum three hypersensitive teeth in at least one quadrant were selected and randomly divided into three groups as per their treatment modality- Group A- treated by 5% NaF varnish application alone, Group B- 980 nm gallium-aluminum-arsenide laser diode laser (0.5 W) irradiation alone, Group C- NaF varnish application, followed by diode laser irradiation. All the hypersensitive teeth were stimulated with tactile, thermal, and air stimuli and the pre-treatment and post-treatment hypersensitivity scores were evaluated with the Numeric Rating Scale (NRS) at baseline, 30 min, 1 week, 1 month, 3 months, and 6 months. **Results:** On completion of the study, there was a statistically significant reduction in NRS scores within the three groups for the tactile, thermal, and air stimuli from baseline to 6 months. Group B and Group C demonstrated a significantly higher reduction ($P < 0.05$) in DH for all the stimuli as compared to Group A at all follow-up intervals. However, a statistically significant difference ($P < 0.05$) was also present between Group B and Group C at all follow-ups. **Conclusion:** NaF varnish followed by diode laser application is significantly more effective than fluoride varnish alone and/or diode laser alone in the treatment of DH.

Keywords: Dentin hypersensitivity, Desensitizing agent, Diode laser, Numeric Rating Scale, Pain.

INTRODUCTION

Dentin hypersensitivity (DH) has been defined as the pain arising from exposed dentin, typically in response to chemical, thermal, tactile, or osmotic stimuli that cannot be explained as arising from any other form of dental defect or pathology.^[1] Several studies have reported non-carious cervical lesions and DH in adult populations, with prevalence rates ranging from 5% to 85%^[2] and 2–8% to 74%, respectively.^[3] It can lead to both physical and psychological problems for the patient. Furthermore, it can have a negative effect on the quality of a person's life, especially with regard to dietary selection, maintaining optimal dental hygiene, and beauty aspects.^[4] A number of conditions that give rise to similar symptoms of DH and so need to be differentially diagnosed include the following - dental caries, cracked teeth, defective or fractured restorations, (usually recent) tooth preparation for restorations

or restoration-induced pulp hyperemia, tooth whitening, dental trauma, occlusal trauma, cervical plaque and gingivitis, periodontal disease and its treatment and other dental pulp/endodontic problems.

Numerous in-office procedures for the relief of hypersensitivity have been tried and tested e.g., glass ionomer cement restoration, dentin bonding agents, varnish application, a commercial preparation comprising amorphous calcium phosphate and casein phosphopeptide, etc. With the dawn

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of laser technology and its growing application in dentistry, further therapeutic options such as low-level lasers like diode 660 nm and high-power lasers like diode 980 nm are accessible for the treatment of dentinal pain.^[5] The present study combines both treatment modalities, conventional treatment as well as newer advances i.e., sodium fluoride (NaF) varnish application followed by diode laser application to combine the effect of both for treating DH.

MATERIALS AND METHODS

Sixty patients (males- $n = 30$, females- $n = 30$) aged between 20 and 50 years and reporting with the chief complaint of DH to the outpatient department of Periodontia, Burdwan Dental College and Hospital, Burdwan, West Bengal during the first half of the year 2022 and willing to participate in the study were selected on a first come first serve basis. This study was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000. The intensity of pain expressed by each patient was recorded as per Numeric Rating Scale (NRS).^[6] NRS is a 1-D scale used to measure pain intensity. It is a series of numbers ranging from 0 to 10 where the two respective endpoints are “no pain” and “worst possible pain”, respectively. Patients are asked to rate their pain at the time of completing the scale by selecting the number that best represents their level of pain. The inclusion criteria in our study included those patients who had at least three teeth in one quadrant hypersensitive to tactile stimulus, cold, and air on the facial aspect and who initially responded to the stimulus with a score of ≥ 5 in the NRS. The exclusion criteria included those subjects who had used desensitizing agents or had undergone desensitizing treatment or any periodontal treatment in the past 3 months, reported of allergy or idiosyncratic responses to milk protein and/or hydroxybenzoates or had excessive dietary or environmental exposure to acids.

The patients were randomly divided by tossing the coin into three groups by an investigator. The treatment modalities were carried out by the second investigator who was oblivious of the patient groups. All subjects underwent a thorough clinical examination followed by oral hygiene instructions and oral prophylaxis. All the hypersensitive teeth were stimulated with 3 tests: Tactile stimuli – scratching horizontally along the CEJ with a dental explorer, thermal stimuli – using drops of melted ice and air stimuli – air blast from a three-way syringe held at a distance of 1 cm and perpendicular to the surface of the tooth for 1 s after the teeth were isolated with cotton rolls.

Group A ($n = 20$)- The hypersensitive tooth was first isolated with a cotton roll. After which, 5% NaF varnish (Varnish containing 50 mg NaF equivalent to 22, 600 ppm of fluoride) was applied on the exposed tooth surface for 2 min, with the help of a disposable applicator tip.

Group B ($n = 20$)- The hypersensitive tooth was irradiated using 980 – nm (975 nm $\pm$ 10 nm), diode laser (in accordance with the manufacturer’s (Hager and Werken GmbH and Co KG, Germany) instructions), power 2 W continuous emission form.

The tooth was irradiated for 2 min in non-contact mode using a fiber of 320-micron diameter with the laser beam directed perpendicular to the exposed root surface of the tooth as close as possible without touching the tooth surface.

Group C ($n = 20$)- The hypersensitive tooth was first treated by application of fluoride varnish for 2 min. After an interval of 5 min, irradiation treatment using 980 nm diode laser, power 2 W, and in continuous emission form, the tooth was irradiated for 2 min in non-contact mode using a fiber of 320-micron diameter with the laser beam directed perpendicular to the exposed root surface of the tooth as close as possible but without touching the tooth surface.

All the patients were instructed not to eat and drink for 1 h and to resume toothbrushing after 12 h. The NRS values were recorded at baseline, 30 min, 1 week, 1 month, 3 months, and 6 months.

Statistical analysis

Data were entered in Microsoft Excel and analyzed using the statistical package for social sciences (Version 23.0) package. The results were averaged (mean $\pm$ standard deviation) for continuous data and number and percentage for dichotomous data. The normality of data was tested using the Shapiro–Wilk test. Proportions were compared using the Chi-square test of significance. Kruskal–Wallis test and Mann–Whitney U test were used for intergroup comparison. “ P ” < 0.05 was considered as statistically significant.

RESULTS

All the sixty subjects kept their appointments as scheduled and there were no dropouts. The results of the study are presented in Tables 1 and 2. Comparison of the mean NRS scores within the three treatment groups for cold test, manual scratch test, and air blast test (Table 1) showed a statistically significant reduction in the NRS scores ($P < 0.001$) from baseline to 6 months. When the mean NRS scores between the treatment groups at different time intervals (Table 2) were compared, there was a statistically significant difference ($P < 0.001$) between Group A and Group B after 30 min and at 1 week with Group B showing a greater reduction in the mean NRS scores. However, there was no statistically significant difference between these two groups at 1 month, 3 months, and 6 months. When Group A and Group B were compared to Group C, there was a statistically significant difference ($P < 0.001$) with Group C showing a greater reduction in the mean NRS scores as compared to Groups A and B at the various study time intervals.

DISCUSSION

DH is a common grievance of patients in a dental clinic. The affected teeth become sensitive to usually non-harmful environmental stimuli. Gentle touch, mild cold or hot, chemical (acidic or sweet fruits, foods, drinks) and air-flow stimuli can tempt short, sharp pain that may affect daily activities including eating, drinking, speaking, and tooth brushing.^[7]

Table 1: Comparison of mean NRS scores within the three treatment groups

Cold test						
Visit	Group	Median	Min.	Max.	Chi-square	P-value
Pre	A	7.00	6	7	1.025	0.579
	B	7.00	7	8		
	C	7.00	7	7		
After 30 min	A	6.00	4	6	29.113	<0.001
	B	4.00	3	5		
	C	3.00	3	4		
Week 1	A	5.00	5	6	28.666	<0.001
	B	5.00	4	5		
	C	4.00	3	4		
Month 1	A	5.00	5	6	21.383	<0.001
	B	5.00	5	6		
	C	5.00	4	6		
Month 3	A	6.00	5	6	23.651	<0.001
	B	5.00	5	6		
	C	5.00	3	5		
Month 6	A	6.00	5	6	22.303	<0.001
	B	5.00	5	6		
	C	4.00	3	4		
Manual scratch test						
Pre	A	8.00	6	9	0.879	0.781
	B	7.00	7	9		
	C	7.00	5	8		
After 30 min	A	6.00	3	6	28.278	<0.001
	B	3.00	2	4		
	C	4.00	3	5		
Week 1	A	5.00	4	6	27.555	<0.001
	B	4.00	3	5		
	C	4.00	3	4		
Month 1	A	5.00	4	7	23.115	<0.001
	B	5.00	4	6		
	C	5.00	3	5		
Month 3	A	6.00	5	7	24.665	<0.001
	B	5.00	4	6		
	C	5.00	4	5		
Month 6	A	6.00	5	7	22.238	<0.001
	B	6.00	5	6		
	C	4.00	3	4		
AIR blast test						
Pre	A	8.00	7	9	0.965	0.833
	B	8.00	7	9		
	C	8.00	7	8		
After 30 min	A	5.00	3	5	24.197	<0.001
	B	3.00	2	4		
	C	3.00	2	3		
Week 1	A	6.00	3	6	23.11	<0.001
	B	4.00	3	5		
	C	5.00	3	5		
Month 1	A	6.00	4	7	21.899	<0.001
	B	5.00	5	6		
	C	5.00	3	5		

(Contd...)

Table 1: (Continued)

Cold test						
Visit	Group	Median	Min.	Max.	Chi-square	P-value
Month 3	A	6.00	5	7	22.244	<0.001
	B	6.00	4	7		
	C	5.00	3	5		
Month 6	A	6.00	5	7	24.135	<0.001
	B	6.00	5	7		
	C	5.00	3	5		

NRS: Numeric Rating Scale

Even though DH is one of the most common problems met by dental professionals, universally acknowledged guidelines for differential diagnosis as well as a selection of reliable treatment modalities are deficient.^[8]

Our understanding about its etiology is based principally on data attained from *in vitro* and *in-situ* studies as well as from data obtained from epidemiological surveys.^[9] It is generally regarded that DH is linked with dentin exposure, especially exposure of open dentinal tubules and dental pulp nerve responsiveness to external environmental stimuli.^[10] Dentin exposure can be caused by physical, chemical, pathological, and biological challenges, and/or developmental abnormalities that result in dental and/or periodontal damage or defects. Various clinical situations believed to play a role in the development of DH include enamel attrition, erosion, corrosion, abrasion, and abfraction.^[11-13] Periodontal tissue loss or gingival recession is another chief predisposing factor since this leads to exposure of cervical and root dentin.^[14] Other factors, such as aging and soft tissue dehiscence, including aggressive brushing, can also cause apical shift of the gingival margins thus leading to exposure of dentin that can eventually lead to the development of DH.^[15]

A number of theories have been proposed to elucidate pulpal nociceptive transduction detected with DH. One early hypothesis held that dentin was innervated and so nociceptive nerve endings within dentinal tubules were activated directly as stimulation was applied to the exposed dentin. The theory of direct dentin stimulation was abandoned due to the lack of evidence of dentin innervation based on countless assessments including immunohistochemical and ultrastructural analyses.^[16] Since odontoblasts are positioned at the outermost layer of the dental pulp and direct processes into the dentinal tubules toward the dentinal enamel junction, it had been proposed that odontoblasts or at least their processes might themselves act as pain receptors, thereby sending pain signals to pulpal nerves that might be associated with the body of the odontoblasts within the pulp.^[17] There is, however, no evidence confirming that synaptic structures that might link odontoblasts with pulpal nerves actually exist.^[18]

The most widely accepted mechanism for DH has been the hydrodynamic theory proposed by Brannstrom.^[19] It states that environmental, mechanical, thermal and chemical changes

Table 2: Comparison of NRS scores within the treatment groups from baseline to 6 months

Visit	Group A versus Group B		Group A versus Group C		Group B versus Group C	
	Mann–Whitney U	P-value	Mann–Whitney U	P-value	Mann–Whitney U	P-value
Cold test						
Pre	56.500	0.352	76.500	0.813	71.500	0.440
After 30 min	10.000	<0.001	14.500	<0.001	00.000	<0.001
Week 1	11.000	<0.001	09.000	<0.001	00.000	<0.001
Month 1	42.500	0.201	13.500	<0.001	09.500	<0.001
Month 3	56.000	0.600	08.000	<0.001	05.000	<0.001
Month 6	48.500	0.123	13.500	<0.001	09.500	<0.001
Manual scratch test						
Pre	72.000	0.717	73.500	0.515	78.000	0.692
After 30 min	16.000	<0.001	11.000	<0.001	00.000	<0.001
Week 1	11.000	<0.001	26.000	<0.001	02.000	<0.001
Month 1	43.000	0.527	15.500	<0.001	06.000	<0.001
Month 3	46.000	0.600	21.000	<0.001	07.500	<0.001
Month 6	52.500	0.166	10.000	<0.001	00.000	<0.001
AIR blast test						
Pre	91.000	0.805	91.00	0.784	94.500	0.488
After 30 min	15.000	<0.001	12.500	<0.001	00.000	<0.001
Week 1	27.500	<0.001	17.000	<0.001	06.000	<0.001
Month 1	62.500	0.326	17.500	<0.001	12.000	<0.001
Month 3	49.500	0.158	16.500	<0.001	05.500	<0.001
Month 6	58.500	0.761	06.000	<0.001	06.000	<0.001

NRS: Numeric Rating Scale

cause the movement of fluid within dentinal tubules, which stimulate the terminals of pulpal nerve fibers positioned within the tubule inlet walls, thereby encouraging momentary acute pain. The hydrodynamic theory highlights the notion that a number of diverse stimuli can elicit similar responses. Evaporative stimuli such as air blasts as well as thermal (cold) and osmotic (sugar, acid) stimuli are believed to upsurge the outward flow of tubular fluid.^[20] Mechanical stimuli such as a dental instrument or a toothbrush drawn across an exposed dentin surface are thought to compress the surface tissue, with the expansion upon release triggering a surge in the outward flow of fluid.^[21] The intra-dental myelinated A β and some A σ fibers that send terminals into the dentin tubules are thought to respond to the fluid movements within the tubule resulting in the characteristic short, sharp pain of DH.^[20] However, how these non-noxious mechanical stimuli of dentin tubule fluid movements encourage nociceptive transduction in dental pulpal nerve fibers remains a paradox. It has been shown that DH could continue even when dentin tubules were obturated with gutta-percha and fluid movements were impossible.^[22]

The hydrodynamic theory has been challenged by emerging evidence suggesting that odontoblasts might well play an important role in the pathogenesis mechanisms of DH.^[23,24] It has been demonstrated that mechanical and/or thermal stimulation induces the release of pain mediators such as adenosine triphosphate (ATP) and glutamate from odontoblasts, providing further evidence supporting the idea that these cells express, at least in part, a neurosensory cell phenotype.^[25,26] In addition, mechanically stimulated induction of ATP release

and ATP-mediated signal transmission from odontoblasts to trigeminal neurons has been verified *in vitro* using co-culture models comprised of odontoblasts and trigeminal neurons.^[27] Recently, the existence of autocrine/paracrine mechanisms for ATP-involved purinergic signaling in cultured odontoblast-like stem cells has been noticed.^[28] If this is the case, then the need for the presence of synaptic structures associated with odontoblasts for them to participate in the transmission of pain signals is removed. Together, rising evidence indicates that external stimuli-induced mechanosensitive responses from odontoblasts and succeeding nociceptive transduction in pulpal nerves may characterize a novel clarification as to how odontoblasts contribute in a mechanosensory mechanism leading to the pain associated with DH.^[9]

In addition to visual inspection, it is essential to evoke the characteristic transient sharp pain by applying a stimulus to the affected tooth to mimic the patient's complaint. Depending on the patient's complaint, mechanical, tactile and thermal stimuli, or air blast with an air-jet, can be applied to the location of suspected dentin exposure.^[29] We have employed all these stimuli (cold, manual scratch, and air blast) in our study. It is significant that when blowing air or delivering other stimuli to an exposed root surface with minimal gingival recession or root exposure, care should be taken to cover the soft tissues or to carefully note that the soft tissues are not being stimulated along with the exposed dentin. It is possible in some cases that blowing air or stimulating the nearby soft tissues mechanically may actually activate a neuralgic trigger point (e.g. in trigeminal neuralgia or other neuropathic conditions).^[30]

Currently, no single method of eliciting and assessing cervical dentin sensitivity may be considered ideal.^[31] In a systematic review comparing VRS, VAS, and NRS it was found that NRS reported better patient compliance than the other scales.^[32] Hence an assessment using NRS was used in the present study.

In the present study, in the 5% NaF varnish application group there was a statistically significant reduction in the sensitivity scores from baseline to 6 months. The reduced hypersensitivity in Group A can be explained by the prolonged contact time of fluoride with the tooth surface due to the sticky consistency of varnish.^[33] The varnish dries rapidly leaving behind a thin, transparent coating. There is a deposition of calcium fluoride (CaF₂) crystals on the tooth surface which blocks the patent dentinal tubules but due to the need to repeated application for effective results it is not easily accepted by the patient.^[18]

Subjects of the second group were treated using a 980 nm diode laser. The group showed a statistically significant difference in sensitivity scores from baseline to 6 months which was in accordance with a study by Mariana-Ioana *et al.*^[34] In this study, high-level diode laser was found to be effective in reducing DH as the lasers produce a melting effect with crystallization of dentin inorganic components and coagulation of the fluids contained in the dentinal tubules. In a recent *in vitro* scanning electron microscopy study,^[35] it was found that dentinal tubules can be entirely blocked after irradiation by 980-nm diode laser at 2 W power settings and with no significant morphological alterations of the pulp and odontoblasts. These findings were similar to a study by Lizarelli *et al.*^[36] where low-intensity laser therapy (LILT) at irradiation parameters of 660 nm, 40 mW was a better therapeutic method in reducing DH compared to Light Emitting Diode and the analgesic effect of LILT is related to the depolarization of C-fiber afferents which is a photo-physical change as a result of the biological light/cell interaction. In another study^[4] five treatment modalities were compared for treating DH, i.e.; Gluma Desensitizer, Seal and Protect, Oxa-gel, Fluoride, and LILT (660 nm/3.8 J/cm/cm/3mW) immediately after topical treatment, after 1 week, 1 month, 3 months and 6 months. It was found that LILT presented a gradual reduction of hypersensitivity throughout the follow-up of 6 months as observed in our study.

The third treatment group showed maximum relief from dentinal hypersensitivity and this can be explained by the combined effect of 5% NaF varnish application and 980 nm diode laser.

Strategies for management of DH

1. Oral hygiene education and brushing technique instruction for prevention of DH- Since the acids from vinegar, fruit, and fruit juices, as well as soft drinks (e.g. citric, malic, and phosphoric acid) are the major cause for dental erosion, consumption of the acidic food or beverages should be regulated in patients prone to the development of DH.^[37]
2. Behavioral control and elimination of predisposing factors- In cases with tooth wear caused by bruxism or

compromised dentition, it is recommended that the use of an occlusal guard or restoration of the worn dentition and vertical dimension be done. Overzealous brushing and other mechanical causes of gingival recession, e.g., the presence of tongue rings and studs should be eliminated.^[38]

3. Non-invasive treatments for pain relief through occluding dentin tubules and blocking nociceptive transduction/transmission- The application of desensitizing agents is the most frequently used non-invasive treatment for DH.^[39] Conceptually, desensitizing agents or analgesic treatments aim to suppress nerve impulses by either mechanical or chemical blockage of the dentin tubules or by directly stopping the nociceptive transduction/transmission occurred within the dentin-odontoblasts-nerve terminal complex of the dental pulp. Based on the mode of their administration, the desensitizing treatment can also be classified into at-home therapy or in-office therapy categories. At-home desensitizing products include toothpastes, mouthwashes, and chewing gums. In-office desensitizing products can be found in the form of gels, solutions, varnishes, resin sealers, glass ionomers, and dentin adhesives. In-office desensitizing treatments also include more sophisticated laser techniques. In general, all interventions should start with non-invasive, reversible, non-hazardous, easy to perform, and inexpensive options.^[40] It is for this reason that we have included laser and desensitizing agents in our study.

However, how do these products work? The active compounds found in desensitization products could either block the openings of dentinal tubules thereby isolating the tubule contents or might directly desensitize the pulpal nerves. Potassium salts were thought to decrease the excitability of pulpal nerves and result in a reduction in dentin sensitivity,^[41] but clinical trials with sound design have failed to provide evidence that potassium is effective in desensitizing teeth.^[42] The proposed mechanism for glutaraldehyde involves the reaction with serum albumin in dentinal tubule fluid, leading to precipitate formation within tubules and subsequent narrowing or blocking of the tubules.^[43] Strontium salts, fluoride, oxalate, and arginine/calcium-containing products have been demonstrated to precipitate and occlude the tubules and form a protective layer at the dentin surface.^[44-46] Both low- and higher-output laser applications have been reported to be effective for the treatment of DH. Some experiments suggest that low output laser might operate by suppressing the excitability of pulpal nerves.^[47] Higher output laser is thought to reduce symptoms of DH by inducing the occlusion of dentin tubules.^[48]

4. Restoration or surgical treatments for dental hard and soft tissue defects- For erosion or abrasion-related DH, it is believed that direct restoration with resin-based composite or glass ionomer and indirect restoration with a crown or a veneer should provide effective long-lasting treatment for DH.^[49] Periodontal surgical procedures including

guided tissue regeneration, coronally advanced flap surgery, connective tissue grafting, and free gingival graft treatments have been proposed for the treatment of DH related to gingival recession, even though the long-term effects of these interventions are still being debated.^[50] However, if surgical correction cannot be attained, or even if there is some improvement in a recession defect and there are still symptoms of DH, then other occlusive restorative treatments as outlined above need to be considered.^[51]

CONCLUSION

The presence of dentin exposure is usually a prerequisite to the development of DH. Accurate diagnosis is the key in selecting the right treatment strategy. It can be concluded that both NaF varnish and diode laser are effective in treating DH. However, diode laser used in combination with fluoride varnish is significantly more effective than fluoride varnish alone or diode laser alone in the treatment of DH. To accept the treatment modality in general more studies with long-term follow-up are required. We anticipate that with an improved understanding of the underlying nociceptive mechanisms of DH, promising novel therapies will emerge and provide more effective relief for patients with DH.

REFERENCES

- Mantzourani M, Sharma D. Dentine sensitivity: Past, present and future. *J Dent* 2013;41:S3-17.
- Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. *J Dent Res* 2006;85:306-12.
- Que K, Guo B, Jia Z, Chen Z, Yang J, Gao P. A cross-sectional study: Non-carious cervical lesions, cervical dentine hypersensitivity and related risk factors. *J Oral Rehabil* 2013;40:24-32.
- Aranha AC, Pimenta LA, Marchi GM. Clinical evaluation of desensitizing treatments for cervical dentin hypersensitivity. *Braz Oral Res* 2009;23:333-9.
- Nair V, Saha N. Comparing diode laser and an in-office agent in the treatment of dentinal hypersensitivity. *Int J Sci Res* 2024;13:1534-39.
- Correll DJ. The Measurement of Pain: Objectifying the Subjective. *Pain Management*. 2nd ed. Philadelphia, PA: W.B. Saunders; 2011. p. 191-201.
- Goh V, Corbet EF, Leung WK. Impact of dentine hypersensitivity on oral health-related quality of life in individuals receiving supportive periodontal care. *J Clin Periodontol* 2016;43:595-602.
- Liu XX, Tenenbaum HC, Wilder RS, Quock R, Hewlett ER, Ren YF. Pathogenesis, diagnosis and management of dentin hypersensitivity: An evidence-based overview for dental practitioners. *BMC Oral Health* 2020;20:220.
- West NX, Lussi A, Seong J, Hellwig E. Dentin hypersensitivity: Pain mechanisms and aetiology of exposed cervical dentin. *Clin Oral Investig* 2013;17:S9-19.
- Absi EG, Addy M, Adams D. Dentine hypersensitivity. A study of the patency of dentinal tubules in sensitive and non-sensitive cervical dentine. *J Clin Periodontol* 1987;14:280-4.
- Grippio JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited: A new perspective on tooth surface lesions. *J Am Dent Assoc* 2004;135:1109-18.
- Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. *Caries Res* 2000;34:182-7.
- Addy M, Absi EG, Adams D. Dentine hypersensitivity. The effects *in vitro* of acids and dietary substances on root-planed and burred dentine. *J Clin Periodontol* 1987;14:274-9.
- Smith RG. Gingival recession. Reappraisal of an enigmatic condition and a new index for monitoring. *J Clin Periodontol* 1997;24:201-5.
- Tugnait A, Clerehugh V. Gingival recession-its significance and management. *J Dent* 2001;29:381-94.
- Sigal MJ, Aubin JE, Ten Cate AR. An immunocytochemical study of the human odontoblast process using antibodies against tubulin, actin, and vimentin. *J Dent Res* 1985;64:1348-55.
- Chiego DJ Jr. The early distribution and possible role of nerves during odontogenesis. *Int J Dev Biol* 1995;39:191-4.
- Davari A, Ataei E, Assarzadeh H. Dentin hypersensitivity: Etiology, diagnosis and treatment; a literature review. *J Dent (Shiraz)* 2013;14:136-45.
- Brannstrom M. Dentin sensitivity. *Arsb Goteb Tandlak Sallsk* 1964;21:15-35.
- Matthews B, Vongsavan N. Interactions between neural and hydrodynamic mechanisms in dentine and pulp. *Arch Oral Biol* 1994;39:87S-95.
- Pashley DH. Mechanisms of dentin sensitivity. *Dent Clin N Am* 1990;34:449-73.
- Linsuwanont P, Versluis A, Palamara JE, Messer HH. Thermal stimulation causes tooth deformation: A possible alternative to the hydrodynamic theory? *Arch Oral Biol* 2008;53:261-72.
- Sole-Magdalena A, Martinez-Alonso M, Coronado CA, Junquera LM, Cobo J, Vega JA. Molecular basis of dental sensitivity: The odontoblasts are multisensory cells and express multifunctional ion channels. *Ann Anat* 2018;215:20-9.
- Liu X, Yu L, Wang Q, Pelletier J, Fausther M, Sevigny J, et al. Expression of ecto-ATPase NTPDase2 in human dental pulp. *J Dent Res* 2012;91:261-7.
- Liu X, Wang C, Fujita T, Malmstrom HS, Nedergaard M, Ren YF, et al. External dentin stimulation induces ATP release in human teeth. *J Dent Res* 2015;94:1259-66.
- El Karim IA, Linden GJ, Curtis TM, About I, McGahon MK, Irwin CR, et al. Human odontoblasts express functional thermo-sensitive TRP channels: Implications for dentin sensitivity. *Pain* 2011;152:2211-23.
- Nishiyama A, Sato M, Kimura M, Katakura A, Tazaki M, Shibukawa Y. Intercellular signal communication among odontoblasts and trigeminal ganglion neurons via glutamate. *Cell Calcium* 2016;60:341-55.
- Zhang S, Ye D, Ma L, Ren Y, Dirksen RT, Liu X. Purinergic signaling modulates survival/proliferation of human dental pulp stem cells. *J Dent Res* 2019;98:242-9.
- Migliani S, Aggarwal V, Ahuja B. Dentin hypersensitivity: Recent trends in management. *J Conserv Dent* 2010;13:218-24.
- Liu X, Malmstrom HS, Tallents R. Dental restoration induced orofacial pain and its management. *Eur J Gen Dent* 2015;4:4.
- Breivik H, Borchgrevink PC, Allen SM, Rosseland LA, Romundstad L, Breivik Hals EK, et al. Assessment of pain. *Br J Anaesth* 2008;101:17-24.
- Hjermstad MJ, Fayers PM, Haugen DF. Studies comparing numerical rating scales, verbal rating scales, and visual analogue scales for assessment of pain intensity in adults: A systematic literature review. *J Pain Symptom Manage* 2011;41:1073-93.
- Langenbach F, Naujoks C, Smeets R, Berr K, Deppe R, Kübler N, et al. Scaffold-free microtissues: Differences from monolayer cultures and their potential in bone tissue engineering. *Clin Oral Investig* 2013;17:9-17.
- Mariana-Ioana M, Dorin D, Diana L, Cosmin AB, Laura MF, Carmen T. An evaluation of the 980 nm GaAlAs high-level diode laser in the treatment of dentine hypersensitivity. *TMJ* 2007;57:280-6.
- Liu Y, Gao J, Gao Y, Xu S, Zhan X, Wu B. *In vitro* study of dentin hypersensitivity treated by 980-nm diode laser. *J Lasers Med Sci* 2013;4:111-9.
- Lizarelli R, Miguel FA, Villa GE, Carvalho-Filho E, Pelino JE, Bagnato VS. Clinical effects of low-intensity laser vs light-emitting diode therapy on dentin hypersensitivity. *J Oral Laser Appl* 2007;7:129-36.
- Rosaiah K, Aruna K. Clinical efficacy of amorphous calcium phosphate, G.C. Tooth mousse and gluma desensitizer in treating dentin hypersensitivity. *Int J Dent Clin* 2011;3:1-4.
- Bhandary S, Hegde MN. A clinical comparison of in-office management of dentin hypersensitivity in a short term treatment period. *Indian J Basic Appl Res* 2012;3:3.
- Torwane NA, Hongal S, Goel P, Chandrashekhar BR, Jain M,

- Saxena E, *et al.* Effect of two desensitizing agents in reducing dentin hypersensitivity: An *in-vivo* comparative clinical trial. J Clin Diagn Res 2013;7:2042-6.
40. Schmidlin PR, Sahrman P. Current management of dentin hypersensitivity. Clin Oral Investig 2013;17:S55-9.
41. Orchardson R, Gillam DG. The efficacy of potassium salts as agents for treating dentin hypersensitivity. J Orofac Pain 2000;14:9-19.
42. Poulsen S, Errboe M, Lescay Mevil Y, Glenny AM. Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev 2006;2006:CD001476.
43. Ishihata H, Finger WJ, Kanehira M, Shimauchi H, Komatsu M. *In vitro* dentin permeability after application of Gluma(R) desensitizer as aqueous solution or aqueous fumed silica dispersion. J Appl Oral Sci 2011;19:147-53.
44. Olley RC, Pilecki P, Hughes N, Jeffery P, Austin RS, Moazzes R, *et al.* An *in situ* study investigating dentine tubule occlusion of dentifrices following acid challenge. J Dent 2012;40:585-93.
45. Cummins D. Recent advances in dentin hypersensitivity: Clinically proven treatments for instant and lasting sensitivity relief. Am J Dent 2010;23 Spec No A:3A-13.
46. Petrou I, Heu R, Stranick M, Lavender S, Zaidel L, Cummins D, *et al.* A breakthrough therapy for dentin hypersensitivity: How dental products containing 8% arginine and calcium carbonate work to deliver effective relief of sensitive teeth. J Clin Dent 2009;20:23-31.
47. Machado AC, Viana IE, Farias-Neto AM, Braga MM, de Paula EC, de Freitas PM, *et al.* Is photobiomodulation (PBM) effective for the treatment of dentin hypersensitivity? A systematic review. Lasers Med Sci 2018;33:745-53.
48. McCarthy D, Gillam DG, Parson DJ. *In vitro* effects of laser radiation on dentine surfaces. J Dent Res 1997;76:233.
49. Zhao X, Pan J, Zhang S, Malmstrom HS, Ren YF. Effectiveness of resin-based materials against erosive and abrasive enamel wear. Clin Oral Investig 2017;21:463-8.
50. Douglas de Oliveira DW, Oliveira-Ferreira F, Flecha OD, Goncalves PF. Is surgical root coverage effective for the treatment of cervical dentin hypersensitivity? A systematic review. J Periodontol 2013;84:295-306.
51. Chambrone L, Sukekava F, Araujo MG, Pustiglioni FE, Chambrone LA, Lima LA. Root-coverage procedures for the treatment of localized recession-type defects: A Cochrane systematic review. J Periodontol 2010;81:452-78.